Attachment 2

Public and Agency Comments on the Final EIS

Author(s)	Recipient	Date of Correspondence
Bader, Jorgen	FHWA	7/4/11
Bricklin, David	FHWA	7/18/11
Canterbury Shores Condominium Association (Mundy, Bill and Mary Ann)	WSDOT	7/11/11
Coalition for a Sustainable SR 520	FHWA	6/20/11
Coalition for a Sustainable SR 520	FHWA/WSDOT	7/15/11
Ebrahimi, Keihan	WSDOT	6/15/11
Environmental Protection Agency	FHWA/WSDOT	7/18/11
Flug-Coburn, Eliza	WSDOT	7/6/11
Fuhrman and Boyer Neighborhood Improvement Association (FABNIA)	WSDOT	7/15/11
Hansen, John	WSDOT	7/15/11
Hoffer, Louis	WSDOT	7/14/11
Kennon, Jacob	WSDOT	7/5/11
Laurelhurst Community Club	FHWA/WSDOT	7/13/11
Leeth, Shawn	WSDOT	6/10/11
National Oceanic and Atmospheric Administration (NOAA) - Stewart Tosach	WSDOT	6/22/11
Puich, Renate	WSDOT	7/8/11
Ravenna Bryant Community Association	FHWA	7/7/11
Scully, Sharon	FHWA	7/13/11
Seaton, Doug	WSDOT	6/20/11
Seattle City Light	WSDOT	6/29/11
Seattle Department of Planning and Development	WSDOT	7/1/11
Seattle Department of Transportation	WSDOT	7/30/11 *(Received 7/15/11)*
Seattle Mayor's Office	WSDOT	7/15/11
Seattle Parks and Recreation	WSDOT	6/30/11
Seattle Public Utilities	WSDOT	6/29/11
Stall, Jon	WSDOT	6/23/11
Stewart, Douglas	WSDOT	7/15/11

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RE: Final Environmental Impact Statement and
Section 4(f) and 6(f) Evaluations
SR 520, I-5 to Medina: Bridge Replacement and HOV Project

Dear Secretary Donovan, Administrator Mendez, Directors Taylor and Mainella, and Regional EPA Director and Directors McBride and Mathis:

The United States should reject the 4(f) Evaluation for the SR 520, I-5 to Medina, Bridge Replacement and HOV Project as failing to comply with **23 United States Code** ("USC") **§ 138** and its counterpart, **49 USC § 303,** and with their implementing regulations, **23 Code of Federal Regulations** ("CFR") **§ 774.** It makes these three egregious errors:

1) It excludes several acres of "4(f) property" from its 4(f) protection, namely the section labeled the "Canal Reserve," and portions of Lake Washington and it downplays other segments, namely, Lake Washington Boulevard from its intersection with the current arboretum ramps to SR 520 (just west of Foster Island Drive) westerly;

2) It greatly understates the full impact of the SR 520 project on the Arboretum by confining its discussion of mitigation measures to the core in its master plans, limiting the impacts of the project to those both caused by and occurring within that actively-planned core, and failing to discuss various elements of the project's impact; and

3) Except for park land taken under Section 106 (f) and the Bagley viewpoint, the Section 4 (f) evaluation contains no discussion or promise of replacement for park land taken and thereby diminishes the environment, the neighborhood and the patrimony of parks to be passed on to future generations. It presumes that an unspecified amount of cash will be an adequate replacement, although state law permits a municipal conversion of donated and dedicated park lands to other uses only upon replacement in kind, and Initiative 42 of The City of Seattle (Ordinance 118477) specifies replacement of any park land taken in kind and in its recitals makes particular reference to the I-5 corridor.

To remedy these failures, all regulatory agencies need to require that the Washington State Department of Transportation ("WSDOT") make an irrevocable commitment of the area, which is now occupied by the freeway ramps to be removed and the 4(f) Evaluation calls the "WSDOT Peninsula," to "Arboretum and Botanical Garden purposes" or to convey the same to the City and/or the University of Washington for such purposes; and the Federal Highway Administration ("FHWA") needs to include such a commitment as part of its Record of Decision on the Project.

Applicable Statutes and Rules

Federal laws and regulations ---

23 USC § 138 (a) and **49 USC § 303** declare a national policy to preserve "public park and recreation lands." It forbids the Secretary of Transportation from approving any program or project ... "which requires the use of any publicly owned land from a public park... unless (1) there is no feasible and planning alternative to the use of such land, and (2) such program includes **all possible planning** to minimize harm to such park, recreational area, wildlife and waterfowl planning refuge, or historic site resulting from such use." (emphasis supplied).

In *Monroe County Conservation Council v. Volpe,* 472 F. 2d 693,700 (2nd Cir. 1972), the U.S. Second Circuit Court of Appeals stated that both conditions (1) and (2) in the statute, quoted above, are independent and cumulative. It stated:

"Even if there is no feasible and prudent alternative to the taking of parkland, the Secretary [of Transportation] still may not give his approval until there has been 'all possible planning to minimize harm to such park.' This requirement also has not been met in this case."

It went on to say at 472 F.2d 700-701:

> The Secretary has nowhere made the actual implementation of these suggestions a condition of his approval, cf. *San Antonio Conservation Society, supra,* 446 F.2d at 1016-1017. Rather, several times he has refused to impose conditions because he claims that he is confident that the state officials will do all they can to minimize damage to the park; and in his statement approving the use of the park, the Secretary refers to studies underway that will determine what type of highway structure will enhance rather than detract from the park. He concluded that 'all possible planning to minimize harm has been and *will continue to* be exercised by the responsible officials.' (Emphasis added.). The statutory mandate is not fulfilled by vague generalities or pious and self-serving resolutions or by assuming that someone else will take care of it. The alternative duty to minimize the damage to parkland is a condition precedent to approval for such a taking for highway purposes where federal funds are involved; and the Secretary must withhold his approval unless and until he is satisfied that there has been in the words of the statute, 'all possible planning to minimize harm to such park..' and that full implementation of such planning to minimize is an obligated condition of the project, *see D.C., Federation, supra*, 459 F.2d at1239." *(Italics are courts)*

In the latter case, *D.C. Federation of Civic Assoc. v. Volpe*, 459 F.2d 1231 (D.C. Cir. 1971), the D.C. Circuit Court held that the secretary's approval was premature since the plans were not specific enough.

The Secretary must make an independent review of significance and adequacy of mitigation. *LaRaza Unida v. Volpe,* 337 F. Supp. 221, 488 F.2d 559 (cert den. 417 U.S. 968, 41 L.Ed 2d 1138, 93 SCt. 105, states:

> "23 USC § 138 is a solemn determination by Congress that beauty and health-giving facilities of our parks are not to be taken away for public roads without hearings, fact finding and policy determinations under the supervision of a cabinet officer --- the Secretary of Transportation."

The Secretary of Transportation can not make the federal determination and finding dependent on affirmative or negative requests of local officials. *Harrisburg Coalition Against Ruining Environmental v. Volpe*, 330 F. Supp. 918 (D.C. Pa. 1971 D.C.) The U.S. Supreme Court gave little significance to the preference of local officials to use parklands for a highway in the Overton Park case, *Citizens to Preserve Overton Park v. Volpe*, 401 U.S. 402, 411, 91 SCt. 814, 821, 28 L.Ed.2d 136 (1971). Accord: *Citizens etc. v. Volpe,* 335 F. Supp. 873, supp. Op. 357 F. Supp. 845 (D.C. Tenn.); *Pennsylvania Environmental Council v. Bartlett*, 454 F.2d 613 (3rd Cir. 1971); *San Antonio Conservation Society v. Texas Highway Department*, 446 F.2d 1013 (5th Cir. 1971) *cert den.* 406 U.S. 933, 32 L.Ed. 2d 136.

Section 4(f) applies to publicly owned land functioning as park; it need not be formally designated as park, *Stewart Park & Rescue Coalition, inc. v. Slater*, 352 F3rd 545 (2nd Cir. 3003), on remand 358 F.3d 83. *Arizona Wildlife Federation v. Volpe*, 4 ERC 1637 (D. Ariz. 1972). 23 CFR 774.11 (d).

Size is not important. The District Court of the Eastern District of Arkansas in *Arkansas Community Organization for Reform Now v. Brinegar*, 398 F. Supp. 685 affirmed 531 F.2d 864 (E.D. Ark 1975) stated:

> "The duty to make 4(f) findings does not depend on the size or nature of the park. If the park is a public park, it simply can not be used actually or constructively for federal highway purposes until a proper statutory finding has been made."

The existence of a public park presumes that it is significant unless an affirmative determination is made otherwise. *Arlington Coalition on Transportation v. Volpe*, 458 F.2d 1323 (4th Cir. 1972*) cert. den.* 409 US 1000, 34 L.Ed 2nd 261, 93 S Ct 312. The principles applicable to significance also apply to the determination of whether an area is parkland under Section (f).

23 CFR 774 repeats and implements the statutory language. 23 CFR § 774.17 Definitions, defines "*all possible planning*" as "... all reasonable measures identified in the Section 4(f) evaluation to minimize harm or mitigate for adverse impacts and effects must be included in the project." It sets a baseline. Whatever avoidance or mitigation measures are identified and reasonable must be implemented. It does not allow the highway authorities pick and choose from among the several mitigation measures identified in the Section 4(f) evaluation, nor as the foregoing cases indicate, let state and/or local officials exclude parkland or a reasonable mitigation measure from the Section 4(f) Evaluation and thereby disregard it.

23 CFR § 774.17 illustrates measures to minimize or mitigate harm as follows:

> "(1) With regard to public parks, recreation areas, and wildlife and waterfowl refuges, the measure may include (but are not limited to) design modifications or design goals; **replacement of land or facilities of comparable and function**; or monetary compensation to enhance the remaining property or to mitigate the adverse impacts of the project in other ways." (emphasis supplied)

The listing makes avoidance prime, then replacement of land, then money to enhance the remainder of the park, and finally use of funds elsewhere to offset the impact of the project. The goal appears in 23 CFR § 774.3 (c) to cause the "lease overall harm in light of the statute's preservation purposes."

Replacement of park land with other land suitable for park in the immediate neighborhood clearly best serves the statutory purpose of preserving park land. Significantly, the courts opinions in the *Overton Park* case and several of the others cited stress the importance of park as recreational open space and thereby implicitly the inadequacy of cash as a substitute. To comply with the "just compensation" clause of the 5th Amendment to the United States Constitution as applied to the states through the 14th Amendment, the highway authorities were obliged to pay the local authorities "monetary compensation."

The National Environmental Policy Act of 1969, 42 U.S.C. 4332, ("NEPA") seeks to promote beneficial effects and to prevent or eliminate damage to the environment and biosphere. It directs agencies to use all practical means and measures

... to improve and coordinate plans, functions, programs, and resources in a manner calculated to the end of fulfilling ".. the responsibilities of each generation as trustee of the environment for succeeding generations;" assure all people safe, healthful, productive, and esthetically and culturally pleasing surroundings; and attain the widest range of beneficial uses of the environment without degradation, among other goals. The Congress authorizes and directs that "to the fullest extent possible (1) The policies, regulations and laws ... shall be interpreted and administered in accordance with the policies set forth..." The State Environmental Policy Act, RCW 43.21C, replicates NEPA on a state level, and the City of Seattle has an implementing ordinance.

State laws and the common law ---

State laws and Seattle ordinances are pertinent because NEPA directs coordination with state and local governments to achieve environmental goals; federalism contemplates a comity among governments at all levels; state and local laws set policy that guides their government's actions; and in the absence of an express, direct legislative action, the statutes and ordinances often control.

In Washington, a municipality holds title to parks on behalf of the public at large. A dedication or donation of land to a municipality for only park purposes prohibits diversion of the park for other uses.[1] At least a half dozen published opinions of the Seattle City Attorney spanning several decades lay out the law against diversion of dedicated and donated park for roadway and other purposes, and are available upon request to the office. A dedicated park thereby differs from general municipal purpose property, *cf. Powell v. Walla Walla,* 64 Wash. 582, 117 Pac. 389 (1911). A municipality may change or permit the use of a dedicated or donated park only when and as explicitly authorized by statute.[2] Owners in a plat may enforce the restriction of use.[3]

RCW 35.22.280(1) allows for an exchange of a dedicated park when a reversion exists in favor of a grantor or another and the grantor can no longer be found. Accord: RCW 35.23.010 and RCW 79.125.710-.720. Then the right of the public is transferred

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[1] *Mulvey v. Wengenheim*, 23 Cal App. 268, 137 Pac. 1106 (1913); *Riverside v. Macleen,* 210 Ill. 308, 71 N.E. 408 (1904); *Alton v. Unknown Heirs,* 95 Ill App. 3rd 1072, 424 N.E. 2nd 1155 (1981). *In re Wellington,* 33 Mass 87 (1834); *Baldwin Manor, Inc. v Birmingham*, 341 Mich 423, 67 N.W. 2d 812 (1954); *Price v. Thompson*, 48 Mo. 361 (1871) and *S. Louis v. Bedal,* - Mo., 394 S.W.2d 391 (1965); *State v. Orange*, 59 NJL 331, 35 S. 799 (1896); *Porter v. International Bridge Co.*, 200 N Y 234, 93 N.E. 716 (1910); *Buffalo v.* Day, 8 Misc. 2d 14, 162 NYS 2nd 817 (1957); *Dallas v. Etheridge*, - Tex -, 253 SW 2d 640 (1952); *State v. Clark*, 161 Tex. 10, 336 S.W.2d 612; *Raynor v. Cheyenne,* 63 Wyo 72, 178 P.2d 115 (1947); *Lancaster v. Columbus*, 333 F. Supp. 1012 (D.C. Miss 1971); *Torrington v. Coles*, 155 Conn. 199, 230 A.2d 550 (1967).

[2] *Brooklyn Bridge Park Legal Defense Fund, Inc. v. New York State Urban Development Corp.*, 825 NYS 2d 347 (2006); Rhyne, *Municipal Law* (1980) 474 § 21-7.

[3] *San Antonio v. Congregation of Sisters of* Charity, 360 S.W.2d 580 (Tex, Civ. App. 1962) *cert* den 372 U.S. 967, 10 L.Ed.2d 131, 83 SCt 1093.

and preserved with like force and effect to the property received by the city in exchange. It is the only statutory authorization for converting such parks to another use. The principles that apply to a voluntary alienation apply to an involuntary transaction as well. The statutes and the common law have the effect of making replacement of land taken from a donated or dedicated park the primary remedy for the taking of park land. The statute and common law apply whether the grantor of the park land acts voluntarily or responds to an exercise of the power of eminent domain for a priority use. It pertains to the acreage taken from the Arboretum, McCurdy and East Montlake Park by SR 520.

City Ordinances ---

Initiative 42 (Ordinance 118477) of The City of Seattle—cited in the FEIS at 5-4-16 requires replacement of park land taken or use for a public project. It is attached as Enclosure "A." Its first Whereas Clauses a firm public policy against any alienation of park lands:

> ...
>
> Whereas, some of our parks are protected by bond covenants that require an equivalent replacement if those parks are taken or converted to another use; and
>
> Whereas, all of our parks need such protection in order to be preserved for public purposes and for our legacy of parks to be passed on to future generations; and
>
> Whereas, this ordinance would continue and strengthen a City policy against diversion of park lands and facilities contained in Resolution 19689, passed in 1963;"

Note: Resolution 19689 was passed in anticipation of construction of the R.H. Thomson Expressway and SR 520 through the Arboretum and called for land or funds sufficient to buy land as replacement for the property taken. The City replaced some of the land lost, which became the "Pinetum" at the westerly edge of the Arboretum. The only substantial open space land in the vicinity was the Broadmoor Golf Course, and that acreage was neither available nor acquiring it practical.

> Section 1 gives Initiative 42 its wallop. It states:
>
> "All lands and facilities held now or in the future by The City of Seattle for park and recreation purposes, whether designated as park, park boulevard, or open space, shall be preserved for such use; and no such land or facility shall be sold, transferred, or changed from park use to another usage, unless the City shall first hold a public hearing regarding the necessity of such a transaction and then enact an ordinance finding that the transaction is necessary because there is no reasonable and practical alternative and **the City shall at the same time or before receive in exchange land or a facility of equivalent or better size, value, location and usefulness in the vicinity, serving the same community and the same park purpose."** (emphasis supplied)

By Ordinance 123408 (Council Bill 116955), the City purported to "supersede" Initiative 42) within a tract described in the City's agreement with the Museum of History and

Industry ("MOHAI") as the "MOHAI Use Area" in order to divert 40% of funds received for the land to MOHAI for its new museum in South Lake Union. The validity and/or efficacy of that ordinance is questionable: the "MOHAI Use area" consists of a part of McCurdy Park, (a part of the Old Canal Reserve) conveyed by the State to the University of Washington); acreage identified in the FEIS as belonging to the Department of Natural Resources; a chunk of the southerly part of East Montlake Park (a park dedicated by the plat and a portion donated by the state under statutory stipulations restricting its use to Arboretum and Botanical Garden purposes) and street area.

Initiative 42 still applies fully to the remainder of those park properties in the Montlake neighborhood, in which the City has ownership or an ownership interest: East Montlake Park, sections of Foster and Marsh Islands in the Arboretum, portions of Montlake Playfield and Montlake Boulevard, and Lake Washington Boulevard.

OMITTED 4(f) PARK LANDS – CANAL RESERVE

The 4(f) Evaluation errs in denying 4(f) protection as park land to (a) Old Canal Reserve and (b) Lake Washington Boulevard, at least west of its intersection with the arboretum ramps. Both are part of the Arboretum and are publicly owned park that should be counted as park land taken for the project. Both that warrant replacement in kind.

4(f) Evaluation ---

The 4(f) Evaluation at page 9-30 discusses the Old Canal Reserve very curtly at page 9-30 as follows:

> "The undeveloped property north of SR 520 behind the houses facing East Hamlin Street is what remains of the Canal Reserve Land, the location of the original log canal between Lake Union and Lake Washington. This piece of land was not included in the Olmsted plans for the park, but was one of the first areas formally planted. Frederick W. Leissler, Jr., who was appointed assistant director of the Arboretum in 1936, directed WPA crews in planting Yoshino cherry trees and incense cedars on this land during the winter of 1935-1936 (BOLA and Kiest 2003). In 1963, the state Department of Highways condemned approximately 47 acres of Arboretum property for SR 520, including ost of the Canal Reserve Land. What remains of the Canal Reserve Land is located within the boundaries of the Montlake Historic District, north of SR 520, and is a contributing element to the district, but is not a part of the Arboretum."

Table 9-1, p. 9-52, captioned "Section 4(f) Uses in the Montlake Historic District under the Preferred Alternative," identifies the Canal Reserve as one acre used.

A repetitive footnote in the summaries and charts states: "The boundaries of the historic Arboretum are larger than the current park property. This use does not affect the recreational use of the Arboretum, and is only recognized as a Section 4(f) use of

Arboretum as a historic property." e.g. Table 9-2," Summary of Section 4(f)...", p. 9-58; Table 9-3. "Summary of Section 4(f) Uses under Option A". p. 9-72; Table 9-6, "Summary of Uses of Section 4(f) ....", p. 9-101 and 9-103r The text lists the 9.5 acres of the WSDOT Peninsula as affected. See also text under "Washington Park Arboretum, p. 9-68; WSDOT's analysis may apply the first sentence of the footnote to the Canal Reserve also.

Almost all the maps in the 4(f) Evaluation show the Canal Reserve as taken for the project, e.g. Exhibit 9-8, "Properties with a Section 4(f) Use..., "p. 9-44; Exhibit 9-10, "Historic Properties with a Section 4(f) Use...", p. 9-49; Exhibit 9-15, "Properties with a 4(f) Use under Option A..", p. 9-61; Exhibit 9-17, "Historic Properties with a Section 9 (f) Use under Option A, p. 9-65; Exhibit 9-20, "Section 4(f) Uses under the Preferred Alternative and Option A, p. 9-142 and 9-143, Options K and L . The Charts and text exclude its acreage from the count of park lands taken, e.g. Page 9-46 states that just 0.5 acres are taken --- a figure that necessarily excludes the acre plus taken from the Canal Reserve, Table 9-1, p. 9-52. Accord: p. 9-55. It is not listed as a park and recreation resource, p. 9-13; p. 9-21; p. 9-40 through 9-48; p. 9-159; or shown as affected park/Open space, e.g. Exhibit 9-3, Overview Map of Properties with a Section 4(f) Use ..., p. 9-17; Table 9-2, "Summary of Section 4(f) Uses...", p. 9-58; p/ 9-59 though 9-63.

FEIS References ---

The Final Environmental Impact Statement ("FEIS") takes almost the same approach as the 4(f) Evaluation. The maps and charts show the Canal Reserve as taken for the project, e.g. Exh. 5.2-4 "Right of Way Acquisitions..", p. 5-2-7; Exh. 5.4-3 "Permanent Park Acquisition..." p. 5.4-6[4]; Exh. 5.6-4 "Effects on NOAA..", p.5.6-8; Exh. 5.6-5 "Effects on Historic Properties..". p. 5.6-10; Exh. 5.6-9 "Effects on Historic Properties...", p. 5.6-17; and Exh.6.2-3 "Property Affected...". P. 6-2-4. The FEIS also omits it from the area colored green as park land under Section 4(f), e.g. Exh. 3-3 "Potential Haul Routes...", p 3-7; Exh 4.1-2 "Montlake Boulevard...", p. 4-1-2; Exh .4-1-4 "Existing Transit and HOV Facilities..", p. 4-1-7; Exh. 4-1-8 "SR 520 Montlake Interchange Area...", p. 4.1-13; Exh. 4-3-1 "Neighborhoods and Community Facilities", p. 4-3-1; Exh. 4.3-3 "Major Utilities ...", p. 4.3-8; 4.4-1 "Parks and Recreation Facilities..", p. 4.4-2; Exh. 4.6-2 "Historic Properties..", p. 4.6-4; Exh. 6.1-1 "Lake Washington Boulevard Ramp Detours", p. 6-1-4; and Exh. 6.1-2 "Road Closures..", p.6.1-5. Exhibit 4.2-1, "Existing Land Use in Seattle," p. 4.2-2 I 2 colors it grey indicating "vacant." Accord: Exhibit 5.4-4 "Permanent Park Acquisition....", p. 5-4-8. However, the University of Washington open space across the canal is colored green as park, e.g. Exh. 3-3, 4-1-7, 4.1-8; 4.2-1; 4-3-1; 4.3-8; 4.4-1, 4.4-9, 6.2-3, 5.4-5 "Permanent Park Acquisition.." p. 5.4-10; and Exh. 6.3-1 "Community Resources..", p. 6.3-2. It is omitted from acreage tabulations in Table 4.4-1 "Summary Information..." p.4.4-3, Exh. 4.6-1 "Listed and Individually Eligible Historic Properties..." , p.4.6-5,

[4] Exhibit 5.4-3 mistakenly shows the Canal Reserve as "existing right of way." It most certainly is not that. See discussion under Canal Reserve History at pages 10-13 below.

Table 5.4-1 "Permanent Park Acquisitions" p. 5-4-1 and Table 5.16-1 "Summary Comparison..", p. 5-16-4. The text, p. 4.4-1 through 4.4-10 and at 5.4-11, and 5.5-22 through 5.5-26 ignores the Canal Reserve in both its discussion of Seattle parks and University of Washington Campus Recreational Facilities, although it regards it as a contributing element in the Montlake Historic District. The FEIS Section 8.4, p. 8.4-1, even omits it and the commitment of the WSDOT Peninsula for arboretum and park use from its discussion of "unresolved controversy" although many community groups (including the Seattle Community Council Federation, the North East District Council, the University District Community Council, the Ravenna-Bryant Community Association and Friends of Olmsted Parks) have called for and still call for restoration of the WSDOT Peninsula to permanent Arboretum use.

The FEIS and the 4(f) Evaluation thereby treat the "Canal Reserve" as an orphaned bastard scarcely to be acknowledged. However, the neighborhood and parks people embrace it as park with an honorable history as part of the Arboretum and to be treated as such.

Status as parkland and part of the Arboretum ---

Black's Law Dictionary (4th Ed. 1957) p. 1271 defines "park" as "an enclosed pleasure ground in or near a city, set apart for the recreation of the public." Accord: *Batchelor v. Madison* Park, 25 Wn.2d 907, 172 P.2d 268 (1946). Webster's *Collegiate Dictionary* (1958) defines "park" as "... a piece of ground, in or near a city or town kept for ornament and recreation.." *The American College Dictionary* (1969) by Random House defines "Park" as "a tract of land set apart as by a city or a nation for the benefit of the public; a tract of land set apart for recreation, sports..."

The Canal Reserve qualifies as "park:"

+ It is open space used by the public for recreation. It is open to the public as a matter of right. The immediate neighbors use it for walking dogs and sometimes as an informal off leash area.

+ The aerial views in the FEIS and the Section 4(f) Evaluation show it as green space with trees, the traditional City park. See photographs, Enclosure "B" of ground level views. It looks like a park with neglectful maintenance.

+ It is a continuous tract. It is set apart from the homes on the north by an alley and Jersey barriers. No parking signs forbid vehicle entry beyond the alley right-of-way. Fencing, a tree-lined border, and elevation changes separate it SR 520 and the Montlake off ramp of SR 520. 26th Avenue East marks its east boundary and the Montlake off ramp and Montlake Boulevard mark its west. No fences disrupt the interior expanse.

+ Government crews mow the open space from time to time. Government signs forbid dumping and say violators will be subject to fine under RCW 70.93.060.

+ It is continuous with McCurdy Park and part of the "historic Arboretum" in the text of the 4(f) Evaluation and FEIS.

+ It has a pedigree as good as McCurdy Park and the sections of the Arboretum protected by Section 6(f). RCW 28B.20.350-.356, Chapter 45, Laws of

1947, Enclosure "C", deeded the old Canal Reserve (including this tract) to the University of Washington for "arboretum and botanical garden purposes and no other purposes." . RCW 28B.20.356 reverts the land to the State of Washington should the University divert use of the property to another usage. The University maintains an inventory of all the trees and shrubs on site. The University has never repudiated that limitation nor has the State claimed that the University is in default. The letter of the State of Washington, Recreation and Conservation Office, dated July 28, 2008, Attachment "A", Attachment "Arboretum Park Draft Boundary", contained in Attachment 2 of the SDEIS shows the former Canal Reserve easterly of 24th Avenue East as part of the Arboretum. The University in its comment on the Supplemental Draft Environmental Impact Statement (S-002) Comment #5 by Arborist Fred Hoyt noted "The plant collections at the Canal Reserve Property should be noted in the document;" and Comment # 25 states that "The Canal Reserve is unique open space property that should be called out." Each seeks 4(f) recognition.

+ The records of the King County Assessor, East ½ of Section 21-25-4 show it as owned by the University of Washington for arboretum and botanical garden purposes. Maps of Seattle, bicycle maps, trail walking maps, and park guides show a continuous green from Madison St. north to Lake Washington (save for the WSDOT wedge) and label it "Arboretum" without breaking out McCurdy Park. The grand vision of the Arboretum at the reception desk at the Graham Visitor Center includes the canal reserve as part of the Arboretum. The Don Sherwood Portfolio, Data on the History of Seattle Parks, in the Seattle Central Library, shows the Canal Reserve west of 24th Avenue East as "Arboretum – U.W." (Enclosure "D").

These factors, taken together, establish the Canal Reserve as 4(f) parkland. There's an adage that goes like this: "If it looks like a duck, it waddles like a duck, and it quacks like a duck,... it's a duck."

Canal Reserve History —-

As explained above RCW 28B.20.350 - .356 (Chapter 45, Laws of 1947) (Enclosure "C") conveyed the old Canal Reserve --- including the area renamed as McCurdy Park -- to the University of Washington for "arboretum and botanical garden purposes and for no other purposes."[5] It reflected the usage then in effect.

At that time, Washington Park extended from East Madison Street to Montlake Boulevard. In 1934, Ordinance 65130 approved an agreement with the Board of Regents of the University of Washington granting the University the right to use "all or any portion of said Washington Park" for planning, developing and maintaining an arboretum

[5] The conveyance included the portion of McCurdy Park soon to be occupied by the Museum of History and Industry. The University was authorized to reconvey possession of a portion of the tract 120' by 400' to The City of Seattle for use by the Museum of History and Industry with a reversion to the University should museum use cease, RCW 28B.20.354 (Enclosure "C") The FEIS, p. 9-20, errs in stating that this property "... is now owned by the City." The City has a right of use of a portion of the premises for the duration of the museum's occupancy.

and botanical garden with the University empowered "... to designate in writing from time to time the exact areas which it desires to devote to such use." It attached a map showing the boundaries of Washington Park. The map included the Canal Reserve area. In 1936, the Olmsted Brothers completed a plan for the University of Washington Arboretum in Washington Park. The plan, entitled "General Plan for the Seattle Arboretum," file # 2669, Plan # 73, March 1936," included the area of the Canal Reserve (Enclosure "E").[6] The University Library contains an Aerial View of Montlake, Interlaken Park, and the Arboretum taken on January 30, 1937. It shows the arboretum as extending from Madison Street to Montlake Boulevard. A 1939 aerial photograph (Enclosure "F") shows the Canal Reserve as woodsy toward the west and marshy in the east. Before SR 520 was built, Seattle residents and visitors could walk from Madison Street on the east to the Montlake Bridge on the northwest without leaving Washington Park and its plantings.

In 1961, the City and the University reached an agreement on partition of the portion of Washington Park that had been the Canal Reserve west of 24th Avenue East. The City had started condemnation proceedings for the R.H. Thomson Expressway, also known as the Empire Expressway. King County Superior Court Cause No. 566846. The area contained 141,888 square feet. The City would take 81,860 on the south for the interchange off ramp; the University would retain the remaining 60,278 square feet. The agreement was authorized by Ordinance 90723. The agreement did not remove the University portion from the restriction of use to "arboretum and botanical garden purposes." RCW 28B.20.350 still applies. For that reason, the agreement required the City to landscape areas apportioned to the University, which were disturbed by construction, up to park standards.

In 1963, the State of Washington condemned about 47 acres of Arboretum land for SR 520. The state's acquisition included the portions of the Arboretum that the City had acquired for the R.H. Thomson interchange; a portion is now called the "WSDOT Peninsula," *State vs. City of Seattle, et al.*, King County Superior Court Court No. 597685.

Resolution 24646 of the City Council of Seattle, passed August 12, 1974, adopted a "letter of clarification" of the 1934 Agreement with the University. The second paragraph of the letter "agreed that the arboretum and botanical gardens "should continue to be maintained within the confines of Washington park as provided for in the 1934 agreement and as more specifically set forth in this letter of clarification." It attached a map showing the area of Washington Park subject to the 1934 agreement. The third paragraph of the "letter of clarification" stated that "..the area 'to be designated" pursuant to the provisions of that paragraph 1 has been designated as shown on the map..." The area of the 1974 map is more limited to reflect the ballfield on the south east retracted by the City, the State's acquisitions for SR 520, and the City deed authorized by Ordinance 90723 (Enclosure "G"). After the "letter of clarification," the University could --- and

[6] The 4(f) Evaluation, page 9-30, second sentence, second full paragraph, errs. Enclosure "G" shows the Canal Reserve at the far right.

can --- develop the Canal Reserve **for arboretum purposes and no other** in any manner that the University Board of Regents decide. (emphasis supplied, citing statutory text). It no longer needs to consult with the City with respect to those 60,278 square feet of the Canal Reserve.

The 4(f) Evaluation assumes that the "letter of clarification" removed this site from the Arboretum. WSDOT staff point to City sponsored 1978 Master Plan Update and the more recent ones that omit that section from the planning area[7]. Limiting joint planning to the core area no more severs them from arboretum usage than lines on a zoning ordinance would do. All that the Master Plans really show is the scope of joint planning and management. The effect of the exclusion is to reserve the University's 60,278 square feet of the Canal Reserve segment for future "arboretum and botanical garden use" with minimum development for now.[8] My research in the records of the Board of Regents, *University of Washington, Board of Regents Records, 1861-1998,* at the University's Special Collections in the Suzallo-Allen Library of the University uncovered no document by the University to change the status of the premises from "arboretum and botanical garden" purposes or to make a formal severance of the area from the Arboretum.

For those not familiar with Seattle history, a bit of background may be helpful. Before World War I, King County had planned to build a canal through the isthmus Montlake using the route of a ditch dug earlier for floating logs from Lake Washington to Portage Bay during high water. It acquired some rights from the adjoining owners. The rights of King County were subordinate to those of the State of Washington in the marsh. Article XVII, Section 1 of the Washington Constitution asserted state ownership up to the "line of ordinary high water within the banks of all navigable rivers and lakes." When the United States announced its intention to build the Lake Washington Ship Canal, King County deeded its rights to the United States; and the State also authorized the canal. The United States, Army Corps of Engineers, chose a straight more northerly route where the canal now lies. It became known as the "Montlake Cut." The United States did not use the right of way which King County had acquired. The Ship Canal lowered the level of Lake Washington so that marsh and bog rose above Lake level and the U.S. added soil from its cut to further raise the elevation of the land.

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[7] WSDOT staff may also cite two other arguments: (a) the maintenance lags and (b) the City's 4(f) correspondence omitted it. The responses are (a) both the City and the University leave property untended, and encroachments occur into under-tended sections of parks from time to time.; and (b) The City's Parks Department letters listed only City-owned properties; its letters also omitted the University open space identified in the 4(f) Evaluation.

[8] This strategy may also reflect Ordinance # 103667 that forbids fencing, entrance fees, and construction in park lands owned by the City of Seattle. According to a UW retiree, during and after the 1990's, the UW anticipated SR 520 expansion northward and therefore left the tract alone.

In 1946, the United States deeded the property rights from King County back to King County as surplus property, (Seattle Comptroller's Files # 190837 and 194885), and the United States returned to the State of Washington all that marsh lakeside of the "line of ordinary high water" at the time of statehood. King County conveyed its property rights by quit claim deed to the City, By RCW 28B.20.350, enacted as Laws of 1947, Chapter 45, the State conveyed the old canal reserve to the University of Washington. The partition authorized by Ordinance 90723 conveyed the City's rights to the University.

Chapter 164, Laws of 1959 granted the University authority to reconvey to the state such portion of its Arboretum properties as needed for state highway purposes, the funds to be used strictly for arboretum purposes. The University did so for SR 520 construction. It is codified as the provided clause of RCW 28B.20.356.

Use of the old canal reserve for plantings dates back over a century. As part of its beautification for the Alaska –Yukon-Pacific Expedition in 1909, the Seattle Board of Park Commissioners (which managed the City's park system autonomously of the City Council) had landscaped Montlake Boulevard – then the south east entrance to the AYP Expedition grounds and extended its plantings into the Old Canal Reserve. The 4(f) Evaluation, p. 9-19, correctly states that "In the 1920's, [1925] the federal government leased a portion of the old canal right-of-way (originally reserved for the Lake Washington Ship Canal) for 99 years to the City for park use ..." It should have continued with the information contained in the Seattle Department of Transportation Records section, commonly called "the Vault." The Vault contains a copy of plats of property within the City limits; until recently, City staff would mark modifications of the original plan on the blue print to alert the researcher. A copy of an extract of Blocks 9,10, and 11 of Lake Washington Shore Lands, page 6, (Enclosure "H") shows the old Canal Reserve east of Montlake Boulevard East. It carries the annotation "Revocable license, dated June 14, 1926 revoked by license dated April 20, 1929 to use for park." If the United States had an outstanding lease or license on the premises to The City of Seattle, the 1946-47 documents would make reference to it. They don't. Neither City records at the City Clerk's office nor the Kroll maps of the 30's to the 50's at the Central Library in downtown Seattle, Seattle room make any mention of a lease or later license to the City. Rather, the area is ascribed to usage by the University. The Canal Reserve was one of the first sections replanted..[9] The public enjoyed until SR 520 construction began. The SR 520 Project guarantees that this segment will not be able to enjoy it for park or arboretum purposes again for the foreseeable future.

Law applied ---

The Canal Reserve is parkland related to, if not part of the Arboretum, and protected by Section 4(f) as such. According to the cases and regulations cited at pages

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[9] *Washington Park Arboretum History* by BOLA Archiecture and Planning and Karen Kiest (2003). Figure 21 shows the work as completed to date, April 25, 1938; and it is shown on Figure 4, p. 50 and Figure 20, p. 81.

1-5, 23 USC § 138 and 49 USC § 303 and its implementing regulation, public ownership and use as a matter of right for recreational purposes creates a presumption of parkland for Section 4(f) purposes. NEPA requires that federal laws be interpreted and administered to accomplish its policies and goals. Which best achieves the statutory purpose? That which protects the Canal Reserve as 4(f) property, honoring the statutory dedication? Or that which treats it as vacant land available for other uses irrespective of the state's donation? If the former, WSDOT's 4(f) Evaluation errs and must be corrected.

OMITTED PROEPRTY: LAKE WASHINGTON BOULEVARD

Effect of Project on westerly segment of Lake Washington Boulevard ---

The SR 520 Project will replace the arboretum ramps between SR 520 and Lake Washington Boulevard with a westbound off-ramp further west. In doing so, it converts the westerly section of Lake Washington Boulevard lying between Montake Boulevard and approximately Foster Island Road (where the arboretum ramps now are) into a freeway access roadway. The high volumes will strip away its parklike character.

When the arboretum ramps are removed, the westbound ramp from SR 520 will exit to 24th Avenue East, approximately the site of the current overpass over SR 520. At that point, 24th Avenue East will cross the east end of the lid to be constructed and connect to Lake Washington Boulevard. The design allows a left (south) turn for exiting traffic of all vehicles 24 hours per day every day, except those times that Lake Washington Boulevard is closed for a special event, such as a marathon race or a bicycle Sunday. The left turn accommodates traffic destined to the easterly neighborhoods (e.g. Madison Park and Madison Valley, Denny Blaine) and those more southerly (Madrona, Harrison and Dorfel Drive, Mount Baker and sections of the Central area). If the left turn were restricted to HOV usage, general purpose traffic would travel to Montlake Boulevard and other arterials.

The FEIS, p. 5-1-133 states: "Access to and from the south would be relocated from the Lake Washington Boulevard ramps to 24th Avenue East; this would result in an increase in trips along Lake Washington Boulevard between Montlake Boulevards and the area of the existing Lake Washington Boulevard ramps." The Transportation Discipline Report accompanying the FEIS p. 6-15 states:

> "About half of the trips that had used the Lake Washington Boulevard ramps from the south to head eastbound would move over to Montlake Boulevard. In the westbound direction, trips head south would exit at 24th Avenue East and have the option to head south along Lake Washington Boulevard or Montlake Boulevard. Similar to the shift in travel south along Montlake Boulevard and half on Lake Washington Boulevard. This pattern would be consistent in the morning and afternoon commute periods."

Exh. 5-1-23 and 5-1-24, "Traffic Volume Changes", p. 5.1-32. show an increase in the AM peak of 480 and in the PM peak of 400 in that westerly segment of Lake Washington Boulevard as compared to "No Build" In 2030. The Exhibits anticipate 850 vehicles per

hour during the AM peak and during 1010 PM peak hour under the "No Build" alternative; the increase would therefore be 56.47% and 39.6% in this segment.

Exhibits 6-1 and 6-2, "SR 520/Montlake Boulevard Interchange Area.." AM and PM peak periods respectively, show projected traffic volumes for 2030 at two locations: one east of Montlake Boulevard, the other east of 24th Avenue East. This stretch of Lake Washington Boulevard runs east-west south of and parallel with SR 520. The projections are:

		East of Montlake Blvd	East of 24th Ave. E.
AM Peak	Existing	760	840
	No Build	850	850
	Pref. Alt.	1400	1330
PM Peak	Existing	840	840
	No Build	1010	1010
	Pref. Alt	1410	1430

"Pref. Alt." abbreviates "Preferred Alternative.". The increase of traffic during the A.M. peak is 64.7% on Lake Washington Boulevard east of Montlake Boulevard and 56.47% east of 24th Avenue East. The PM figures show an increase of 39.6% and 41.58% respectively. Removal of the arboretum ramps decreases traffic volumes east and south of the WSDOT Peninsula.

The Final Environmental Impact Statement for the Washington Park Arboretum Master Plan (January 2001) in Appendix C, Figure 2, contained actual traffic counts on the vehicular volumes on Lake Washington Boulevard. In the morning, 74% of the traffic going northbound from Madison St. on Lake Washington Boulevard turned off to go to SR 520 at the arboretum ramps; the other 26% continued to Montlake Boulevard; and 35% of the traffic on Lake Washington Boulevard south of the arboretum ramps were commuters coming from SR 520. In the PM peak, 53% of the southbound traffic came from SR 520, and 45% of the traffic came from the south at Madison St. and went eastbound on SR 520. Less than one motorist in three exiting at the arboretum ramps turned west and one-seventh of the motorists using that westerly section of Lake Washington Boulevard.

4(f) Evaluation –

The 4(f) Evaluation does not consider the segment of Lake Washington Boulevard west of the WSDOT Peninsula to be a "park resource." It is omitted from the listing on p. 9-13 and the tabulations of park land taken in the same tables and exhibits as the Canal Reserve is. It lacks the green shading of parks in Exh. 9-13, "Overview Map of Properties with a 4(f) Use," p 9-17, Exh. 9-9, "Effects on the Washington Park Arboretum..", p. 9-4; Exh. 9-13, "Effects on the Washington Park Historic Park Arboretum..." p. 9-54; Exh. 9-16, "Effects on the Washington Park Arboretum under Option A," p/ 9-64; Exh. 9-18, "Effects on the Washington Park Arboretum Historic Property," p. 9-89; and Exhibit 9-29, "Section 4(f) Uses under the Preferred Alternative...", p. 9-142.

The FEIS has the same pattern of exclusion, e.g. Exh. 2-13, "Montlake Area.." p. 2-47; Exh. 3-3 "Potential Haul Routes", p. 3-7; Exh. 4.4-1 "Parks and Recreation Facilities ..", p. 4-4-2; Exh. 5-4-6 "Permanent Acquisition," p. 5-4-12. Its tables and exhibits also omit this segment from the count of park lands taken. Chapter 6, "Effects During Construction of the Project" shows increased traffic volumes for several years, Exh. 6-1-3, "Expected Traffic Volumes...", p. 6-1-3, its use as a detour, "Exh. 6.1-1 "Lake Washington Boulevard Ramp Detours," p. 6.1-4; construction truck volumes, "Estimated Daily Construction Truck Volumes...," p. 6.1-13; a construction easement, Exh. 6.2-3 "Property Affected by Construction," p. 6.2-4; and use for construction staging, Exh. 6.3-1., "Community Resources...", p. 6-3.2 and Exh. 6.4-1 "Construction Effects on Parks", p. 6.4-2. Traffic almost doubles on the westerly segment for several years of construction during the AM peak period; it increases 60% during the evening peak. Transportation Discipline, Exh 10-4, "Lake Washington Boulevard Access During Construction," p. 10-9 and Exh. 10-5, p. 10-9, and Exh. 10-10, p. 10-14. This segment is shown as eligible within the Montlake Historic District, Exh. 5.6-9, "Effects on Historic Properties," p. 5.6-17, and therefore on some exhibits shaded with a greenish tint.

The 4(f) Evaluation, "Constructive Use," p. 9-6 cites 23 CFR § 774.15 that a finding of constructive use of a 4(f) property requires a determination that the protected features are "substantially impaired." At page 9-32, it concludes that the entire length of Lake Washington Boulevard in the area of potential affects is eligible for listing on the national register of historic places. The very first sentence on page 9-33 makes this key characterization: "Lake Washington Boulevard is a transportation facility and it has served that function since its construction." WSDOT's responses to the comments of the Parks Department (L-008-008) calls it an arterial and its response to the "Coalition for Sustainable 520" (C-021-021) states that is .." not a park property because its primary use is not as a park." The 4(f) Evaluation concludes its reply by citing approval of its use as a SR 520 access roadway with modifications by the State Historic Preservation Officer. That approval may settle the matter with respect to its classification as a historic property eligible for listing. It does not resolve the issue from a park perspective. In its comment on the SDEIS, the Seattle Parks Department asked for its consideration as a 4(f) park property, (S-008-008) and so had the Arboretum Foundation, Part V, page 9 (C-037-004) and page 21 (C-037-060).

Lake Washington Boulevard as a linear park ---

Lake Washington Boulevard was designed and intended for leisurely travel in the manner of a park roadway --- not as a throughway for commuting. It is two lanes, lined with trees and ornamental old style lighting to match, and narrow as carriageways were at the turn of the century. It winds like a serpentine; there are two intersections east of the WSDOT Peninsula, and one at the 24th Avenue East overpass. Neighborhood Streets that connect to 26th Avenue East next to Lake Washington Boulevard are closed to cut through traffic. Street signs on the segment north of the intersection with East Calhoun St. are brown for park –rather than the traditional green for street. The signs say Lake

Washington Boulevard – not 26th Ave E. At East Lynn St., projected northward, the Arboretum Aqueduct, a pedestrian overpass with a drainage line, crosses over the boulevard; it is brick, arched, and so low that that truck tops sometimes bang against it.[10] The overpass is a designated City Landmark and on the National Register of Historic Places, 4(f) Evaluation 9-29.

The westerly segment of Lake Washington Boulevard lies outside the current limited access lines. It actually includes sections of roadway that are labeled on some of the FEIS exhibits as 26th Avenue East. The platted 26th Avenue East runs north and south with its most northerly end at its intersection with East Calhoun St. Washington Park Master Plan, Draft Environmental Impact Statement, Figure 10, p. 124 (May 2000). St *Montlake: An Urban Eden* by Eugene Smith in Chapter 11 describes the development of the Arboretum and Lake Washington Boulevard at pages 113-117 and 128-129. Enclosure "I" is an extract from the book. The Board of Park Commissioners laid it out within and as part of Washington Park. See the 1912 map on page 114 of the extract. It remains under the jurisdiction of the Parks Department. Various ordinances accepting deeds recite their acceptance for park purposes. The whole array may be examined in the City Clerk's office. Lake Washington Boulevard was never dedicated as street and it is subject to Initiative 42.

In some cases, Seattle created boulevards by condemning the rights from abutters, changing the right-of-way from "commercial street" to "park, drive and boulevard purposes", e.g. portions of Queen Anne Boulevard. In contrast, Lake Washington Boulevard has a park underlay throughout, and especially as part of Washington Park. As shown on Enclosures "D", "E" and "I," Washington Park extended from East Madison St. to Montlake Boulevard.

Rebuttal to WSDOT's Arguments ---

The City's street classifications as of 2002 appear on its website,www.seattle.gov/transportation/streetclassmaps. Lake Washington Boulevard is classified as a "collector arterial." That category is below "Principal Arterial" and above "access street."[11] Its function is described as ".. collects and distributes traffic from principal and minor arterials to access streets or directly to local destinations. Collector arterials are typically located within neighborhood boundaries and serve small groups of stores, schools, small apartment complexes, and residential land use." It is not a major truck street or authorized for transit. Large tracts commonly have more than one entrance and a roadway connecting them. Those large tracts include national parks,

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[10] A photograph appears in the FEIS, p.4.6-17. During public meetings for developing the Arboretum Impact Plan, citizens had asked that signage on 24th Avenue East warn motorists about the low clearance of the aqueduct over Lake Washington Boulevard. Trucks turning left on to Lake Washington Boulevard eastbound from the 24th Avenue East overpass face a dilemma: either go under the aqueduct or traverse narrow tree-lined neighborhood streets with low overhanging branches.

[11] The Recreation Discipline Report Addendum, p. 13, errs in classifying it as a "major arterial."

military posts, college campuses, and cemeteries. For example, Stevens Way makes a loop from N.E. 40th St. to N.E. 45th St. by way of Memorial Way. Those roadways were built to accommodate traffic that seeks a location in the tract a destination and in some cases for the pleasure of slow, leisurely and travel and enjoying nature. Such roads are still part of the park, post, campus or cemetery. To say otherwise, as WSDOT does with Lake Washington Boulevard, takes all together too narrow a perspective --- like having one's nose on the centerline and one's peripheral vision confined to the curbline.

The SR 520 Project escalates the westerly segment to a freeway access roadway and overshadows it park boulevard character. By analogy, on a national level the Natchez Trace fits the concept of scenic park roadways; superimposing a freeway would be a change of use. The bulge between East Louisa St. and E. Miller St. might be compared to the look-outs and picnic stops along the Natchez Trace. Making the Trace into a freeway access roadway would impose a additional servitude because the more intensive usage differs so much in degree that it amounts to a change in kind, especially at turn-out locations.

Law and common sense ---

The 4(f) Evaluation, p. 9-31, cites 23 CFR §774.11(h) that "property formally reserved for a future transportation facility" and used in the interim as park, however long the duration, does not receive 4(f) status as park. The converse should also be true. A boulevard in a park ought not to lose its 4(f) protection because commuter traffic adopts it as a short cut. Denying 4(f) status is especially inappropriate to the westerly segment where the preferred alternative makes the boulevard its prime entry and exit from SR 520. Since the 1960's, the City has prohibited left turns by eastbound traffic on Lake Washington Boulevard to the arboretum ramps and thereby kept the westerly segment from becoming a freeway access roadway.

23 USC § 138 and 49 USC § 303 require ".. all possible planning to minimize harm to such park." Supporters of the Arboretum proposed restricting the left turn at 24th Avenue East to HOV vehicles, and if that were not acceptable, to allowing usage by general purpose traffic only during peak hours. WSDOT and the City chose to allow general usage 24 hours per day seven days per week. These restrictions qualify as within a "reasonable measure" that should have been identified in the FEIS and the 4(f) Evaluation, and when so identified, adopted as part of the project. The 4(f) Evaluation at pages 9-37 and 9-38 promises "traffic calming measures and a traffic management plan..." Those elements are laid out in the Arboretum Impact Plan and in a Memorandum of Understanding with the Arboretum and Botanical Gardens Committee. These measures slow traffic speeds; they do not avoid many of the impacts from the greatly increased volume. To offset the impacts and preserve the integrity of the park, the WSDOT Peninsula needs to be committed to arboretum use.

Assuming *arguendo* that it had became a "transportation facility," the greatly increased traffic volumes to be generated by the 24th Avenue East exit amount to a

"constructive use" of the landscaped periphery to the paved lanes. That greenery should still be treated as Section 4(f) park. Between the intersection of East Louisa St. and East Miller St. projected, Lake Washington Boulevard has over one hundred feet of park width lying east of the curbline of the roadway and west of the WSDOT Peninsula right-of-way. See Exh. 9-9 "Effects on the Washington Park Arboretum...", p. 9-47. The depth equals that of an old-style Seattle City lot. Motorists can see folks relaxing or playing Frisbee or catch there. *Adler v. Lewis*, 675 F2d 1085 (9th Cir. 1982) held that a "use" occurred off-site if the project "... would substantially impair the value of the site in terms of its prior significance and enjoyment. " *Brooks v. Volpe*, 460 F.2d 1193 (9th Cir. 1972) on remand 350 F. Supp. 269 and 287, *aff'd* 487 F.2d 1344. upheld a ruling that widening I-90 west of Snoqualmie Pass had make a "use" of the Denny Creek Campground by surrounding it.

Common sense supports committing the WSDOT Peninsula to Arboretum use as compensation and mitigation for traffic rerouted by removing the arboretum ramps. After SR 520 construction is complete, the westerly segment of Lake Washington Boulevard will be serving the same function as the Arboretum ramps did. The noise and traffic that had been on the ramp closed will be moving to the boulevard that is still open. Changes will also be made at its Montlake Boulevard intersection to accommodate the traffic flow. A commitment of the WSDOT Peninsula to Arboretum use mitigates the impact on the neighborhood. The spirit of NEPA requires it.

UNDERSTATED ENVIRONMENTAL IMPACTS

The 4(f) Evaluation and the FEIS understate the full environmental impact of Seattle's park system in the "area of potential effects."

1) Each omits consideration of certain factors and impacts. For example, the detailed comments on the SDEIS of the University of Washington, the Arboretum Foundation, the Montlake Community Council, and others identify concerns that were not discussed in the supplemental draft environmental impact statement. The environmental impact statement process anticipates that drafters will lack information and knowledge known by the public and seeks to elicit it. This oversight ought to be remedied in the response to comments -- if not, it festers;

2) The responses to public comments in the FEIS and 4(f) Evaluation attempt to finesse deficiencies in the SDEIS. . The authors of the FEIS seem to feel that their initial paragraphs in the SDEIS were adequate unless there is clear error. [12] Many of WSDOT's FEIS responses cite earlier responses and/or the sections in the FEIS, which

[12] For example, the FEIS and the 4(f) Evaluation use the legislative bill numbers without identifying the session. My comment supplied the citation in the Revised Code of Washington ("RCW") and the Chapter and session used in the published session laws. These citations would assist readers and researchers, and speed finding the section on the state legislative website or code indexes. WSDOT's response replied that its bill number are accurate and made no change.

replicate the draft document. It's an implicit rejection of the comments received. A good FEIS strives to inform the decision-makers with clear, accurate, important information and where deficiencies are cited, to supply more helpful information. This FEIS aims to justify its earlier draft, making changes primarily when and where the design has been refined.

3) Both the 4(f) Evaluation and the FEIS confine discussion of mitigation to impacts of the new SR 520 bridge to those that are (a) generated by the project in the Arboretum, as constricted; (b) occur within that confined area; and (c) can be mitigated by measures there. It should, but fails to consider, impacts from outside or to the Arboretum as part of a contiguous park system. WSDOT staff set these ground rules for the Arboretum Impact Plan and its recommendations are limited accordingly.

The first failure is self-evident by reading the comments received next to the SDEIS and the FEIS and the respective 4(f) Evaluations side by each, e.g. the commentaries show how truly unique the Arboretum is and the fragility of its wetlands: 80 different species of waterfowl and 50 species of other birds use it as their habitat, and a watchful eyes can spot a bald eagle on Foster Island.[13] The prosaic text does not do justice to the sparkle of life or the beauty of the Arboretum, its serenity away from SR 520 where visitors may enjoy the sounds of nature in a crowded metropolis, and its ambience that captures and delights visitors. To do that takes a photographic essay like *The Wild Within, Wetlands of the Washington Park Arboetum* (2007), available from the Arboretum Foundation. Making the side-by-side comparison of text, comment, and FEIS response is a tedious process. It would make this letter too long to list instances. The process highlights the second error: the authors' reluctance to make corrections or to better the text in order to help decision makers and guide those who implement the plan in the course of implementing the project – the very goal of the EIS process.

The second failure alerts the reviewer to study the comments for information that ought to have been added to the text or the accompanying disciplinary studies. Take two examples from my comment letter (I-093). On page 4, I requested advance acquisition of fisheries resources for mitigation as recommended by the representative of the National Oceanic and Atmospheric Administration during the mediation process. The response I-093-063 discusses design of the Montlake lid over SR 520. Page 4 of my comment discusses the crow colony on Foster Island, one of nature's wonders:

> "Foster Island is a prime roosting area for crows, and, the place that they congregate at night. The *Street Smart Naturalist: Field Notes from Seattle*, p. 197 describes Foster Island at dusk in these vivid terms:
>
> 'I am in the center of a cosmic maelstrom. Birds arrive from the north, east, and west. Most come in groups. Many are playing, chasing each other, dive-bombing their roostmates, enjoying the last flight of the day, ... wave upon flying wave, the birds starting high above the water, then swooping low before a final climb into the leafless trees dotting the shoreline.

[13] Final Environmental Impact Statement, Washington Park Arboretum Master Plan, Jan.2001,p. 109; FEIS, Table 4.11-12, "Occurrence of Federally .. Protected Wildlife.." p 4.11-10.

'The winter dispersal and return of crows is perhaps Seattle's grandest daily natural-history display. Nowhere else in the city can one see so many wild, large, living beings at one time, except at certain sporting events.'"

The response I-093-009 refers to I-093-007, which in turn states that WSDOT formed a Park Technical Working Group relating to Bagley Viewpoint. In neither case is the FEIS response relevant to my comment. Neither the FEIS in its wildlife discussion (5.11-5.17) nor its Ecosystem Discipline Report makes any reference to the crows or bats.

The University's comment on the SDEIS (S-002) raised many issues that were not resolved. For example, Item 29 on page 3 states in part as follows:

"... The extended duration of these construction impacts, particularly those in the Arboretum strongly indicate that such impacts cannot be considered 'temporary' or 'minor.' And therefore should not be considered exceptions under 23 CFR 774.13(d) – and should be mitigated for accordingly."

It is part of a series of Items (including Items 25, 31, and 42) that explain the Arboretum wetlands are fragile and a complex ecosystem, and that construction, even with great care, by its very duration can have impacts that will take a very long time for nature to return to its earlier condition. The response S-002-079 refers to the responses S-002-025 and S-002-067 that temporary uses do not become permanent if the conditions of 23 CFR 774.13 (d) are satisfied and it is working on it. The response does not describe those impacts, their duration, or effect on the ecology, nor does it suggest mitigation by substituting other land during the period until recovery occurs. Item # 42 on indirect effects on wetland recovery receives a curt brush off. (S-002-091).

The Arboretum Foundation comment at several places expressed concern about the impact of the SR 520 structure on the ambience of the Arboretum,e.g. C-037-022. The new bridge will loom over Foster Island like a concrete overpass. The FEIS, p. 5.5-5 acknowledges that the new bigger, bulkier bridge would be "somewhat more dominant," but it opines that the new structure "would not affect overall visual quality since the bridge is already a dominant feature of the view..." The very dominance of the structure probably prompted the Arboretum Foundation's plea for extraordinary sensitivity in design and landscaping. In response the FEIS at page 5.5-26 and the 4(f) Evaluation, p. 9-7 and at p.9-125 and Arboretum Impact Plan, Attachment 9 of the FEIS, at pages 17-19, promise undisclosed "aesthetic enhancements" to address adverse effects. WSDOT has shown the Arboretum and Botanical Gardens Committee design concepts for the treatment of the bridge pylons on Foster Island and the landscaping of the trail. All the design drawings shown so far fall far short and bode ill. They're like dusting powder over a big, ugly scar: it does a little, but not much.

Blinders on the Arboretum Analysis - - -

The Arboretum is an ecological island/refuge in Seattle. The wetlands and upland are a fabric of life, rent mainly by SR 520, but still contiguous. Cutting away McCurdy Park, East Montlake Park, and the Canal Reserve affects the whole. Both the 4(f) Evaluation and the FEIS rule those impacts out of their analysis of Arboretum impacts.

limits its consideration of mitigation measures for the Arboretum to those impacts both caused by and occurring with the actively-planned areas of the Arboretum. In doing so, it excludes from consideration elements that would be considered "severance damages" under eminent domain law. 23 USC. § 138 and 49 USC § 303 were enacted to supplement eminent domain law and provide parks with added protection.

The 4(f) Evaluation takes a dual view of the Arboretum. The historic analysis discusses the Arboretum as originally laid out, FEIS p. 9-29 through 9-31 and FEIS 9-58, footnote b; the park analysis limits the Arboretum to the "main portion" of the 1977 Master Plans and its successors, excluding McCurdy Park, East Montlake Park, the Canal Reserve, and Lake Washington Boulevard. FEIS p. 9-21 and 22; pp. 9-29 through 9-33. 9-49 through 9-49. At p. 9-37, the 4(f) Evaluation refers to the Arboretum Impact Plan and a Memorandum of Understanding with the Arboretum and Botanical Gardens Committee. The 4(f) Evaluation, p 9-46 through 9.46, and at 9-53 through 9-55, acknowledges only the taking of 0.5 acres of land and 0.1 acres of submerged shoreline as do the various tabulations of acreage, FEIS p. 9-58, 9-72, 9-85 and 9-86, 9-98 and 9-99. The 4(f) Evaluation cites the measures in the Arboretum Impact Plan and the Memorandum of Understanding as providing the mitigation due. The Plan focuses on the traffic problems, which are generated by allowing all westbound traffic on SR 520 to go south on the 24th Avenue East bridge over SR 520 and then to turn east (left) and use Lake Washington Boulevard (discussed above at pages 14-19).

The FEIS Ecosystems Chapter 4, p. 4.11-3-4, describes the environment of the study area as a whole and focuses on endangered and protected species, FEIS 4-11-10, Table 4.11-2, "Occurrence of Federally Listed or Protected Wildlife ..." p. 4.11-10 through 4.11-12. It mentions only the bald eagle among avian life. The Ecosystems Discipline Report applies a very broad brush treatment. In its mitigation discussion, the 4(f) Evaluation, p. 124-5, states that WSDOT has ... identified appropriate replacement property for *part of* the land used in the Arboretum (emphasis supplied). See Chapter 10 of the Final EIS for more information." Chapter 10 contains the Section 106(f) analysis and provides for the Bryant Marina site as a substitute for the Ship Canal and Arboretum Waterfront Trail. What about the other part --- the Section 4(f) properties outside the Section 6(f) area and in the Union Bay drainage area?

This letter and its analysis does not discuss the 4(f) properties in the Portage Bay drainage basin. The Portage Bay/Roanoke Park Community Council and the Montlake Community Council, supported by the North East District Council, seek shoreline property at the south west edge of Portage Bay to replace land taken from the Montlake Playfield and its shorelands and to mitigate the adverse impacts of the SR 520 Project on the Montlake Playfield. It is called the "Frolund site." It is the remainder of several lots, which WSDOT is acquiring for the SR 520 Portage Bay Bridge; that remainder lies south of the limited access line. The request has merit. It is a separate topic from the replacement of land to the Arboretum.

Severance Damages ---

SR 520 takes 6.1 acres from the contiguous ecological Arboretum: 2.8 from East Montlake Park; 1.4 from McCurdy Park; 0.5 from Foster Island; and 1.4 acres from the Canal Reserve (60,278 square feet). This excludes Lake Washington Boulevard's conversion to freeway access, WSDOT current right-of-way, construction easements, and submerged shoreland. It does not count the 0.1 acre of the Montlake Cut that connects to East Montlake Park. These figures are taken from the 4(f) Evaluation of Table 9.2, "Summary of Section 4(f) Uses Under the Preferred Alternative, pp 9-57 and 9-58. McCurdy Park, Foster Island and the Canal Reserve were donated for "arboretum and botanical garden purposes and no other" and contain a reversion; East Montlake Park was dedicated and donated for park purposes. The shrinkage of 6.1 acres affects the remainder. Some of the area is marsh. Some waterfowl are very choosy about their habitat; reducing their specialized habitat may cause those to forego the Arboretum or reduce in their numbers. An absence of a species or drop in population may leave a niche unfulfilled or alter the ecological balance in other ways. Size, itself, is important because it provides a greater diversity and a larger population that has more in reserve for the hard times that may come. Size also matters in evaluating the development potential. Large parks commonly need space for administration, storage of equipment, or nursing plantings. The Canal Reserve acreage was available for that usage as ancillary to the Arboretum; now, making space for such ancillary activities will displace natural areas devoted to plantings.

The Section 6(f) Evaluation offers the Bryant Marina site north of the Lake Washington Ship Canal on the University campus as a replacement for the Section 6(f) properties. It is the best substitution available under Section 6(f). Nonetheless, it leaves the adverse impacts on the Arboretum cited in the previous paragraph and can not replicate an experience of wandering through a marsh on a boardwalk.

In taking McCurdy park, the SR 520 Project takes away 124 parking spaces there. FEIS Table 1-15, "Potentially Affected Parking Areas," p 5..1-64. Footnote "e" states that due to "removal of the facility that requires the parking spaces ... there would be no net loss at these locations..." Page 5.1-67 and 5.1-68 states that WSDOT is coordinating with the City for replacing parking lost at Bagley viewpoint --- no mention is made of similar replacement for the Arboretum. The parking in McCurdy Park served users of the Arboretum Waterfront Trail as well. 4(f) Evaluation, p. 9-22. Many of the strollers wandered deeper into the Arboretum. The loss of its ancillary parking affects the Arboretum.

The 4(f) Evaluation should have --- but failed --- to address the potential impact of the re-use of the WSDOT Peninsula for highway-related usage and/or commercial purposes. This is a looming threat unless WSDOT commits the site to permanent arboretum and park uses. After the arboretum ramps are removed, WSDOT obliges itself to restore the area as wetland. Federal policies forbid any loss of wetland. FEIS, p. 5.11-20 and 21. Once the wetlands are restored, WSDOT procedures require it to make use of the premises for highway purposes or surplus that area according to its procedures. In November 2010, WSDOT representatives told the Arboretum and Botanical Gardens Committee that the WSDOT Peninsula is an asset of the Transportation Fund (established

by Amendment 18 of the Washington Constitution) and that it can convey the property only in exchange for full value in money or "credits" from a land exchange. The Arboretum Mitigation Plan, p. 29, states in part, as follows: "Should all or part of the property need to be surplused, Arboretum owners would be offered an opportunity to purchase it as a contiguous landowner." The City is cutting its budgets and lacks ready capital. Within the current zoning, WSDOT could lease or sell its peninsula for concessions, such as equipment rental or refreshments or parking on days of Husky events at the nearby UW football stadium. The MOHAI lot was almost always full on days of Husky home games.

The 4(f) Evaluation should – but did not --- consider the impacts on the Arboretum of the possible use of the SR 520 right-of-way for commuter rail. SR 520 is being designed to accommodate light rail when the time comes. RCW 47.56.870 and RCW 47.01.405 (Chapter 517, Laws of 2007, §§ 2 (5) and 6. e.g. FEIS 1-12, 1-17, 2-16, 2-21 through 2-26, 2-28, and 2-32. among others. The FEIS, p. 2-60 states, in part, as follows:

> "The westbound and eastbound bridges would have a gap between the structures to be compatible with potential future light rail infrastructure, should Sound Transit determine that a light rail crossing of SR 520 is desirable at some point in the future. (No light rail crossing is currently planned or proposed as part of the SR 520, I-5 to Medina project.) ... the design would allow a potential future rail link to rise over SR 520 to connect with the University Link station at Husky Stadium."

The light rail structure would be almost entirely within the WSDOT right-of-way. The 4(f) Evaluation does not mention light rail at all. Under eminent domain law, a condemnee has one day in court and must raise all concerns at that time that would affect the property if the right-of-way were used to the fullest. The FEIS currently excludes from consideration all park land within the SR 520 right of way. 4(f) Evaluation, p 9-31. Unless the record of decision explicitly excludes impacts and damages that occur when light rail is constructed, mitigation and compensation must be provided for the Arboretum for the additional servitude of the light rail line.

Law applied ---

23 USC § 138 and 49 USC § 303 were enacted to supplement eminent domain law. These statutes increase the protection given to park land. Each recognized that cash compensation would not be sufficient alone. As supplemental, remedial legislation, these statutes contemplate that the transportation authorities will take into account at least all the elements that eminent domain law would consider. Under eminent domain law, property is considered a unitary tract when all the parcels (or portions thereof) are contiguous, used for a common purpose, and under common or affiliated ownership. 8A *Nichols on Eminent* Domain (3rd Ed. 2010) § G16.02; *State v. Windermere Co.*, 89 Wn Ap[. 369, 949 P.2d 392 (Div. 3, 1997), *rev. den.* 135 Wn.2d 1012, 960 P.2d 939; *State v. McDonald*, 98 Wn.2d 521, 656 P.2d 1043 (1983). When a portion of a unitary tract is taken, damages are due to the remainder. Here, the 6.1 acres of East Montlake Park, McCurdy Park, Foster Island, and the Canal Reserve are part of a unitary tract and the

Arboretum must be compensated, preferably in replacement property. The Section 6(f) exchange removes from this equation the acreage of Section 6(f) properties, but not the impact of their severance from the remainder of the Arboretum.

WSDOT staff may cite the Arboretum Mitigation Plan as a comprehensive resolution of remedial measures. It is not.[14] WSDOT set the ground rules for that study. By excluding severance damages and replacement land from its scope, WSDOT implicitly reserved them for further consideration through administrative processes.

WSDOT PENINSULA NEEDED AS REPLACEMENT LAND

To make the Arboretum whole, the Record of Decision must specify that the WSDOT Peninsula be committed for Arboretum use either through an easement or perpetual covenant or by conveyance of the site to the University of Washington, the City of Seattle, and/or the Washington State Department of Natural Resources for "arboretum and botanical garden purposes and no other." This restrictive use and reversion apply to almost all the properties being taken for the SR 520 project that lies north of the current SR 520 right-of-way, except East Montlake Park; and the latter was dedicated and donated for park purposes.

The WSDOT Peninsula is currently a wedge into the Arboretum that divides the area covered by the Arboretum Master Plan. If the area is returned to Arboretum use, the amount of acreage devoted to wildlife would remain or increase. Pedestrian could make a loop trip alongside the lagoon viewing the Lake Washington Ship Canal, the broad open water of Lake Washington, and a quiet cove; the vegetation, bird, and animal life differ. It would provide growing room for water loving trees and other specimens. (The lagoon could host boxes for bats; those little, flying critters gobble insect pests and spare using insecticides.) The WSDOT Peninsula would get the expert management of the Arboretum and the expertise of the University. At a meeting on June 8th 2011 at the Graham Visitor's Center in the Arboretum, the Seattle Parks Department presented three alternate concepts to the public showing how the area could be integrated into the Arboretum Master plan. About one hundred people attended and public opinion was strongly in favor of its return to Arboretum use.

The added park land would benefit not only the Arboretum. It would also help offset the adverse impact of the much heavier traffic flow on Lake Washington Boulevard to its immediate west both for the abutters on that boulevard segment and for the Montlake neighborhood. As shown on page 22 above, the SR 520 project takes at least six acres of park land from the immediate Montlake neighborhood. This total does not count the taking from the Montlake Playfield or Montlake Boulevard. The SR 520 project returns to the Montlake neighborhood use of the surface of a lid to be built

[14] RCW 47.56.870(4)(b)(v) required the Plan not only to mitigate, but to "emhance the Washington Park Arboretum." The latter assignment remains undone without returning the WSDOT Peninsula to the Arboretum.

between 24th Avenue East and 26th Avenue East. Although very beneficial, the Montlake lid does not equalize Arboretum parkland: its north and south sides will be busy freeway access roadways, and it will serve multiple purposes, such as traffic signage; a roadside bus stop, waiting areas for bus passengers and pedestrian pathways between stops and bicycle (and possibly motor scooter and motorcycle) parking for commuters and Husky stadium events. The 4(f) Evaluation offers the immediate Montlake area no park land in exchange for the acreage taken. That is a major blow to the community. Almost since its creation, Montlake has enjoyed a crescent of parks on its south; and a like crescent of lake front on its north to complete a circle. This periphery of nature and open space makes Montlake a desirable residential neighborhood despite its proximity to SR 520, the congested connecting arterials, and the nuisance of bumper-to-bumper parking on both sides of the street end-to-end on days with events at Husky Stadium and at the Hec Edmundson Pavilion. Without is parks, Montlake would not have preserved its homes and maintained its yards to merit eligibility to become a historic district.

The FEIS and 4(f) Evaluation ---

The 4(f) Evaluation identifies the Bryant Marina site as replacement for the Section 6(f) property taken for the Ship Canal and Arboretum Waterfront Trail, e.g. 4(f) Evaluation, p. 9-124 and Section 10. It serves as replacement for "part of the land used in" the Arboretum, and for a "portion of " East Montlake Park. That's good as far it goes. The 4(f) Evaluation, p. 9-124, also acknowledges that portions of East Montlake Park and the Arboretum are outside the Section 106(f) properties. The 4(f) Evaluation also identifies a replacement site for the Bagley Viewpoint,

The FEIS, p. 5.3-5, under the caption, "recreation," states that " WSDOT has made every effort to avoid permanent effects on parks..." and promises that "All loss of park acreage would be mitigated." At p 5-3-13, it boasts: "The project also would enhanse parks, particularly the Arboretum as mitigation for the increased width and bulk of the highway in this area." At p. 9-38, the 4(f) Evaluation limits mitigation to measures in the Arboretum Impact Plan: "Those mitigation measures agreed upon for the Arboretum through the consultation process with ABGC [the Arboretum and Botanical Gardens Committee] serve as Section 4(f) mitigation measures." No replacement land is offered as mitigation for the taking of the Canal Reserve or impacts on Lake Washington Boulevard although identified as contributing elements to the Montlake historic district. FEIS, pp.9-120 and 121, 9-136 through 9-139.

The 4 (f) Evaluation does not make any commitment of returning the WSDOT Peninsula for Arboretum purposes. It promises no perpetual easement nor any conveyance to the City and/or the University. The 4(f) Evaluation at p. 9-125 merely states:

> "WSDOT is evaluating the **possibility** of transferring property from the WSDOT peninsula to the Arboretum after the R.H. Thomson Expressway ramps and SR 520 ramps are removed and the area is restored to its natural condition." (emphasis supplied)

Page 9-124, under the caption, "University of Washington Open Space," states:

> "WSDOT is **proposing** to use a portion of the WSDOT Peninsula as part of a wetland mitigation project and is **exploring the feasibility** of using the remainder of the WSDOT-owned land in the peninsula area for mitigation for effects on parks in the project area." (emphasis supplied)

There is a telling contrast between "**proposing**" and "**exploring the feasibility**" of using the area as mitigation.. The UW Open Space on campus *north* of the Lake Washington Ship Canal. The return of the WSDOT Peninsula to arboretum or park use is not among the proposals "... to avoid or minimize harm" or mitigate for impacts and effects listed for Historic properties or park properties *south* of the Ship Canal. 4(f) Evaluation pp 9-126 through 9-140.

The FEIS, p. 5-11-20, and both the Arboretum Impact Plan and the Memorandum of Understanding call for restoration of the wetland after the arboretum ramps are removed. Pages 5.11-20 and -21 identify the WSDOT Peninsula as compensatory wetland under the federal policies of "no net loss" of wetland. The Arboretum Impact Plan (December 22, 2010) at p. 28 offers the City a preferential right to purchase as an abutter before placing the site on the real estate market should the area be surplussed. The Memorandum of Understanding was silent upon its ultimate disposition. Neither makes any permanent commitment of it for park or arboretum purposes. The responses to the comments of the University, Item # 28, and the Arboretum Foundation cite the 6(f) Replacement and are silent as to the WSDOT Peninsula, e.g. S-002-059 and -088; C-037-037; and I-093-063 and -155 and -156 respectively.

The University in its comment on the SDEIS, Items 28 and 37 through 39, called for replacement land for property taken. The Arboretum Foundation comment, p. 6 (C-037-015 and -016) and p. 18 (C-037-037), asked that the WSDOT Peninsula continue to be used as park land and properly protected. WSDOT's response referred to the sections on measures to protect historic properties and the Section 106(f) substitution. It elicited no commitment to using the WSDOT Peninsula as replacement land. My comment called for return of the WSDOT Peninsula to arboretum use in multiple places. The responses (I-093-009, -155, and -156) are essentially same: each cites the Section 6(f) exchange and claims a net gain of park land. More than a dozen citizens and organizations asked for permanent use of the WSDOT Peninsula for arboretum and botanical garden purposes and got similar responses. It shows public support for re-integration of the WSDOT Peninsula into the Arboretum inasmuch as the focus of WSDOT's public presentations and the SDEIS was at selecting among alternative designs and on traffic planning. Section 8.4 of the FEIS, p. 8.4 et seq. should have listed it as a controversy yet to be resolved, but failed to do so. This failure is likely to arise in litigation contesting the project.

Need for Commitment ---

The laws to protect the environment and parks aim to make the area of potential effects whole from the project. Replacement land can do so. Here, the replacement is at the doorstep and available. Nothing else is of "comparable value and function.." to the

4(f) properties being taken in the Montlake area. This is true from the perspective of wildlife habitat, from the perspective of neighborhood impact, and from the perspective of recreational experience --- each as described earlier in my letter. A cash payment won't do so for several reasons:

a) Cash is not land. It erodes with time just as the Arboretum Capital Improvement Fund established by Ordinance 92511 for proceeds from the building of SR 520 did.

b) The responsibility rests with WSDOT. Paying money shifts implementation to other governments. To achieve compliance, WSDOT would need to impose strict and enforceable conditions, such as a precise exchange as done with the Section 106(f) properties. Otherwise, parks and the environment would have little less protection than that prevailing under eminent domain and the common law without 23 USC § 128 and USC § 303. Nothing in the 4(f) Evaluation suggests that WSDOT plans such stipulations.

c) If cash were paid, the City and the University would then have to buy the WSDOT Peninsula. Unfortunately, by Ordinance 123408 and 123132 the City has already committed 40% of the cash received from the SR 520 proceeds of the "MOHAI use area" to the Museum of History and Industry for use in South Lake Union. The "MOHAI Use Area" lies largely within the 6(f) properties of McCurdy Park, but extends into East Montlake Park. This commitment was made despite the fact that WSDOT paid MOHAI for its museum and relocating its exhibits and that MOHAI has no ownership in any of the land in the so-called MOHAI use area. That's a bad precedent for relying on the City, especially since City officials say it lacks money to maintain its park buildings.

d) The allocation process of cash proceeds subjects the Montlake community and friends of the Arboretum to the vagueries of Citywide budgeting. The 4(f) properties are coming from Montlake and/or for Arboretum purposes The funds would replace parks dedicated or donated for the use and enjoyment of the public *in Montlake and/or arboretum uses.* The funds are imbued with a trust for the public with a particular emphasis on the Montlake region and/or arboretum purposes. Experience with the METRO's payment and Ordinances 123408 and 123132 casts doubt on whether the City would implement the trust by acquiring the whole WSDOT Peninsula. When METRO paid Seattle for a large section of the Elliott Bay beach in order to expand its sewerage treatment plant at Discovery Park, the City dispersed the funds for waterfront capital "improvements" scattered throughout the parks system. Many of them have passed their functional life. Land is permanent and would be part of the patronage passed on to future generations; and

e) Initiative 42 (Ordinance 118477) cited at page 6, contemplates replacement land for park land taken.

A permanent commitment of the WSDOT Peninsula to arboretum uses serves ancillary advantages:

+ It fulfills the promises at 5.3-5 that " WSDOT has made every effort to avoid permanent effects on parks..." and promises that "All loss of park acreage would be mitigated." It carries out the implication of this quotation in the SDEIS, Draft Parks Mitigation Technical Memorandum (December 2009) WSDOT Response to the Seattle Board of Park Commissioners, Answer to Question 1: "WSDOT proposes to exchange

this property with the Seattle Department of Parks and Recreation as part of the mitigation for both alternatives.;"

+ It was an assumed premise during the mediation process from September 2007-December 2008. Advocates for Alternative A cited that return as a major of advantage for that design, e.g. Westside Project Impact Plan (December 2008), p. 5-3, 6-3, 6-9, 6-11; Statement of the University District Community Council, p. 1. It was probably a factor in evaluation of the alternatives in the local decision making process, e.g. Seattle City Councilmember Richard Conlin in his comment on the 2006 DEIS called for its return;

+ It removes a source of controversy. As knowledge of WSDOT's retention and potential use or disposition for other uses spreads, the public will demand action for its commitment. It's usually better to anticipate and then react and repair afterward.

+ Litigation on the project is likely. Removing this issue helps WSDOT's case., e.g. *Brooks v. Volpe*, 460 F2d 1193 (9th Cir. 1972) stated that substituting 180 acres of greenbelt was important as a measure to minimize harm. A settlement now moots the failure of the 4(f) Evaluation on the issues raised in this letter and averts the possibility that the citizen organizations in the lawsuit may prevail. If the plaintiffs were to prevail, their attorneys would likely seek an award of attorneys' and expert witness' fees. That could be costly.

The Law applied ---

As stated at pages 2 and 3, 23 USC § 138 and 49 USC § 303 condition federal approval on a program that "...includes **all possible planning** to minimize harm to such park, recreational area, wildlife and waterfowl planning refuge, or historic site resulting from such use." "Possible" means "feasible to minimize and mitigate harm to the park, and that "full implementation of such planning is an obligated condition of the project. Vague generalities and reliance on the good faith of state and local officials will not suffice. *Monroe County Conservation Council v. Volpe,* 472 F. 2d 693,700 (2nd Cir. 1972). Except for the Bagley Viewpoint and the Section 106(f) properties, the 4 (f) Evaluation offers little more. The 4(f) Evaluation , p. 9-117, purports to discuss "reasonable measures carried forward for consideration to minimize harm or mitigate for adverse impacts." The "consideration" will extend to "evaluating the possibility" and "exploring the feasibility." That is not a plan; it's preliminary thinking. There's no committing to take action on the ground nor any serious discussion of the benefits of committing the site to arboretum use or what might happen without such a commitment. Moreover WSDOT's ground rules for the Arboretum Impact Plan put transfer of the WSDOT Peninsula as replacement land outside the scope of analysis, although it did mention allowing the City/University/Department of Natural Resources a prior right to purchase as an abutter.

23 CFR § 774.17, quoted in the 4(f) Evaluation at pp. 9-116 and 9-117, states that "... all reasonable measures identified in the Section 4(f) evaluation to minimize or mitigate for adverse impacts and effects must be included in the project." This regulation implicitly assumes --- and thereby requires --- that "all reasonable measures" must be

described in full. If a measure is reasonable, an agency may not exclude it's consideration and its implementation by taking it off the table when preparing its 4(f) Evaluation and in its procedures leading up to it. The return of the WSDOT Peninsula to arboretum use is the best mitigation measure available. It can't be put off as the 4(f) Evaluation tries to do. It is too obvious a remedy, too important to the Arboretum, too much sought after by the neighborhood and friends of the Arboretum, and too necessary in order to comply with federal, state, and local policies and public. The 4(f) Evaluation simply can not be approved on the current record.

Perhaps, WSDOT has concerns about making a commitment for arboretum use, e.g. determining when restoration is complete, getting recognition for off-setting value in eminent domain proceedings and negotiations, defining the exact boundary of the area to be conveyed, possible hazardous wastes on site, and so on. The Record of Decision may recognize and identify those concerns, e.g with language such as "... on the understanding that...." or "it is assumed that..." Such a list of qualifications or reserved items may modify the commitment, but are not an excuse for refusing to make it all. The stipulation may request the affected parties to work the implementation out in good faith negotiations. "Good faith" would apply to all parties: WSDOT, the City, the University, and the State Department of Natural Resources. So far, the interagency discussions have been primarily in private and the actions of those other agencies (e.g. Ordinance 123408 and 123132) may have influenced WSDOT's stance. If the Record of Decision follows my recommendation, both the Arboretum and Botanical Gardens Committee and the Arboretum Foundation should be invited to participate in the resolution of the matter.

CONCLUSION

The Record of Decision must stipulate that WSDOT must either (a) commit the WSDOT Peninsula to Arboretum use through an easement or perpetual covenant or (b) convey the premises to the City, the University of Washington and/or the Washington State Department of Natural Resources for arboretum and botanical garden purposes. The conveyance must describe the entire excess area; it may name the grantees as tenants in common as their respective interests may appear in the property taken for the project. The record of decision my qualify this stipulation by reserving to WSDOT the ability to set the conveyance off as special benefits from the project under RCW 8.04.080 or in a settlement, its functional equivalent, and allowing other administrative details to be worked out in good faith negotiations.

This letter asks for a final result: the return of the Arboretum *south of the SR 520 right-of-way* insofar as practical to its condition before SR 520 was first built. The responsibility rests on government officials for finding the manner of achieving that goal. The mediation process persuaded most of its participants that the current SR 520 is a festering sore; the preferred alternative with the WSDOT Peninsula would still be a scar in Montlake as freeways are, but it would be built and mitigated to the state of the art.

If you need more references or documents, please write me at the addesss on my letterhead or call me at (206) 525-9070.

Yours truly

Jorgen Bader

List of Enclosures

A Initiative 42 (Ordinance 118477)
B Photographs of Canal Reserve
C RCW 28B.20.350-.356 (Chapter45, Laws of 1947)
D Don Sherwood's Portfolio, Extract, History of Seattle Parks
E Olmsted Brother's sketch of Arboretum (1936)
F 1939 Aerial Photo
G Ordinance 90723. Partition of westerly portion of Canal Reserve
H Extract of Plat, Blocks 9, 10,and 11, Lake Washington Shorelands
I Extract, *Montlake:An Urban Eden*

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For more information, call
323-1562 or 324-5218, or write:

P.O.P.
2102–24th Ave. S.
Seattle, WA 98144

Please mail by Nov. 18, 1996

INITIATIVE MEASURE NO. 42

AN ORDINANCE REQUIRING PRESERVATION OF ALL LANDS AND FACILITIES HELD NOW OR IN THE FUTURE BY THE CITY OF SEATTLE FOR PARK AND RECREATION PURPOSES; STIPULATING THAT SUCH LANDS AND FACILITIES MAY ONLY BE CHANGED FROM PARK USE AFTER A PUBLIC HEARING AND THE ENACTMENT OF AN ORDINANCE FINDING THAT SUCH ACTION IS NECESSARY, AND PROVIDING FOR A SIMULTANEOUS EXCHANGE OF LAND OR FACILITIES OF EQUAL OR BETTER VALUE.

WHEREAS, developers and others are making demands on City officials to take over our parks for other uses; and
WHEREAS, some of our parks are protected by bond covenants that require an equivalent replacement if those parks are taken or converted to another use; and
WHEREAS, all of our parks need such protection in order to be preserved for public purposes and for our legacy of parks to be passed on to future generations; and
WHEREAS, this ordinance would continue and strengthen a City policy against diversion of park lands and facilities contained in Resolution 19689, passed in 1963;
NOW THEREFORE,

BE IT ORDAINED BY THE THE CITY OF SEATTLE, AS FOLLOWS:

SECTION 1.
All lands and facilities held now or in the future by The City of Seattle for park and recreation purposes, whether designated as park, park boulevard, or open space, shall be preserved for such use; and no such land or facility shall be sold, transferred, or changed from park use to another usage, unless the City shall first hold a public hearing regarding the necessity of such a transaction and then enact an ordinance finding that the transaction is necessary because there is no reasonable and practical alternative and the City shall at the same time or before receive in exchange land or a facility of equivalent or better size, value, location and usefulness in the vicinity, serving the same community and the same park purposes.

SECTION 2.
Within thirty days of the effective date of such an ordinance, any person may seek review in the Superior Court. The Superior Court shall set aside the proposed transaction if it is not necessary or the proposed substitution is not equivalent or better than the park exchanged. The Superior Court shall make its decision on the evidence as an issue of fact.

SECTION 3.
Section 1 permits by duly enacted ordinance after a public hearing: a boundary adjustment of equivalents with an adjoining owner; or the transfer of a joint use agreement with Seattle School District No. 1 to another school site. Section 1 also permits by duly enacted ordinance after a public hearing and without providing replacement property: a transfer to the federal, state, or county governments for park and recreation uses; the reversion of right–of–way continuously owned by a City utility; the opening of an unimproved street for street use; a sub–surface or utility easement compatible with park use; and franchises or concessions that further the public use and enjoyment of a park.

SECTION 4.
This ordinance shall take effect as provided by Article IV, Section 1 of the City Charter. However, if the City should sell, transfer, or change the use to a non–park use of any park property held on or after May 17, 1996 (including Bradner Playfield), the City shall replace it in kind with equivalent or better property or facilities in the same vicinity, serving the same community, unless the City has already received as good or better land and facilities for park use in the same vicinity, serving the same community, in exchange for that transaction.

ENCLOSURE A

City of Seattle Legislative Information Service

Information retrieved on October 3, 2010 6:45 PM

Council Bill Number: 111606
Ordinance Number: 118477

AN ORDINANCE adopting Initiative 42, enacting it as an ordinance of the City of Seattle.

Date introduced/referred: Jan 21, 1997
Date passed: Jan 27, 1997
Status: PASSED
Vote: 9-0
Date filed with the City Clerk: Feb 5, 1997
Date of Mayor's signature: Feb 4, 1997
(about the signature date)

Committee: Full Council
Sponsor: DONALDSON

Index Terms: PARKS, LAND-ACQUISITION, SALES, INITIATIVES-AND-REFERENDA

Fiscal Note: *(No fiscal note available at this time)*

Text

Note to users: {- indicates start of text that has been amended out
-} indicates end of text that has been amended out
{+ indicates start of text that has been amended in
+} indicates end of text that has been amended in

AN ORDINANCE adopting Initiative 42, enacting it as an ordinance of the City of Seattle.

WHEREAS, citizens of the City of Seattle circulated petitions seeking the enactment of Initiative 42 into law; and

WHEREAS, King County certified to the City of Seattle that Initiative 42 bore a sufficient number of validated signatures to qualify for transmittal to the City Council; and

WHEREAS, the City Council received Initiative 42 on December 16, 1996; and

WHEREAS, City Charter Article IV provides that the City Council may enact or reject such an initiative; and

PM

TWO-SIDED

transfer, or change the use to a non-park use of any park property held on or after May 17, 1996 (including Bradner Playfield), the City shall replace it in kind with equivalent or better property or facilities in the same vicinity, serving the same community, unless the City has already received as good or better land and facilities for park use in the same vicinity, serving the same community, in exchange for that transaction.

Section 5. This ordinance shall take effect and be in force thirty (30) days from and after its approval by the Mayor, but if not approved and returned by the Mayor within ten (10) days after presentation, it shall take effect as provided by Municipal Code Section 1.04.020.

Passed by the City Council the _____ day of _____, 1997, and signed by me in open session in authentication of its passage this _____ day of _____, 1997.

_____

President _____ of the City Council

Approved by me this _____ day of _____, 1997.

_____

Mayor

Filed by me this _____ day of _____, 1997.

_____

City Clerk

(Seal)

January 22, 1997

GEKgh
111606.DOC
(Ver. 1)

ENCLOSURE B

twenty-nine degrees six minutes fifty-three seconds (29°06'53") east, a distance of nine hundred twenty-four and twenty-four one-hundredths (924.24) feet to the beginning of a curve to the left having a uniform radius of one hundred fifteen (115) feet; thence southeasterly along the arc of said curve, a distance of one hundred twenty and fifty-one one-hundredths (120.51) feet to the point of beginning. [1969 ex.s. c 223 § 28B.20.340. Prior: 1913 c 24 § 1. Formerly RCW 28.77.280.]

28B.20.342 University site dedicated for street and boulevard purposes—Local assessments barred against site. No assessments for the opening, improvement or maintenance of any public street upon the tracts of land described in RCW 28B.20.340 shall ever be levied, assessed or collected upon any portion of section 16, township 25 north, range 4 east, W.M., or upon any portion of blocks 7 and 8 Lake Washington shorelands. [1969 ex.s. c 223 § 28B.20.342. Prior: 1913 c 24 § 2. Formerly RCW 28.77.290.]

28B.20.344 University site dedicated for street and boulevard purposes—Eminent domain may not be exercised against site. The power of eminent domain of any municipal or other corporation whatever is hereby declared not to extend to any portion of said section 16, township 25 north, range 4 east, W.M., and blocks 7 and 8 of Lake Washington shorelands. [1969 ex.s. c 223 § 28B.20.344. Prior: 1913 c 24 § 3. Formerly RCW 28.77.300.]

28B.20.350 1947 conveyance for arboretum and botanical garden purposes—Description. There is hereby granted to the University of Washington the following described land, to wit:

Lots two (2) and three (3), Block eleven-A (11-A) of the supplemental map of Lake Washington shorelands, filed September 5, 1916 in the office of the commissioner of public lands, to be used for arboretum and botanical garden purposes and for no other purposes, except as provided in RCW 28B.20.354. [1969 ex.s. c 223 § 28B.20.350. Prior: 1947 c 45 § 1. Formerly RCW 28.77.310.]

28B.20.352 1947 conveyance for arboretum and botanical garden purposes—Deed of conveyance. The commissioner of public lands is hereby authorized and directed to certify the lands described in RCW 28B.20.350 to the governor, and the governor is hereby authorized and directed to execute, and the secretary of state to attest, a deed of said shorelands to the university. [1969 ex.s. c 223 § 28B.20.352. Prior: 1947 c 45 § 2. Formerly RCW 28.77.315.]

28B.20.354 1947 conveyance for arboretum and botanical garden purposes—Part may be conveyed by regents to city of Seattle. (1) The board of regents of the University of Washington is hereby authorized to convey to the city of Seattle that portion of said lot three (3) of the shorelands described in RCW 28B.20.350 which is within the following described tract, to wit:

A rectangular tract of land one hundred twenty (120) feet in north-south width, and four hundred (400) feet in east-west length, with the north boundary coincident with the north boundary of the old canal right-of-way, and the west boundary on the southerly extension of the west line of Lot eleven (11), Block four (4), Montlake Park, according to the recorded plat thereof, approximately five hundred sixty (560) feet east of the east line of Montlake Boulevard.

(2) The board of regents is authorized to convey to the city of Seattle free of all restrictions or limitations, or to incorporate in the conveyance to the city of Seattle such provisions for reverter of said land to the university as the board deems appropriate. Should any portion of the land so conveyed to the city of Seattle again vest in the university by reason of the operation of any provisions incorporated by the board in the conveyance to the city of Seattle, the University of Washington shall hold such reverted portion subject to the reverter provisions of RCW 28B.20.356. [1969 ex.s. c 223 § 28B.20.354. Prior: 1947 c 45 § 3. Formerly RCW 28.77.320.]

28B.20.356 1947 conveyance for arboretum and botanical garden purposes—Reversion for unauthorized use—Reconveyance for highway purposes. In case the University of Washington should attempt to use or permit the use of such shorelands or any portion thereof for any other purpose than for arboretum and botanical garden purposes, except as provided in RCW 28B.20.354, the same shall forthwith revert to the state of Washington without suit, action or any proceedings whatsoever or the judgment of any court forfeiting the same: PROVIDED, That the board of regents of the University of Washington is hereby authorized and directed to reconvey to the state of Washington block eleven-A (11-A) of the supplemental map of Lake Washington shorelands, filed September 5, 1916 in the office of the commissioner of public lands, or such portion thereof as may be required by the state of Washington or any agency thereof for state highway purposes. The state of Washington or any agency thereof requiring said land shall pay to the University of Washington the fair market value thereof and such moneys paid shall be used solely for arboretum purposes. Such reconveyance shall be made at such time as the state or such agency has agreed to pay the same. [1969 ex.s. c 223 § 28B.20.356. Prior: 1959 c 164 § 2; 1947 c 45 § 4; No RRS. Formerly RCW 28.77.330.]

28B.20.360 1939 conveyance of shorelands to university—Description. The commissioner of public lands of the state of Washington is hereby authorized and directed to certify in the manner now provided by law to the governor for deeding to the University of Washington all of the following described Lake Washington shorelands, to wit: Blocks sixteen (16) and seventeen (17), Lake Washington Shorelands, as shown on the map of said shorelands on file in the office of the commissioner of public lands. [1969 ex.s. c 223 § 28B.20.360. Prior: 1939 c 60 § 1; No RRS. Formerly RCW 28.77.333.]

28B.20.362 1939 conveyance of shorelands to university—Deed of conveyance. The governor is hereby autho-

ENCLOSURE C

rized and directed to execute, and the secretary of state to attest, a deed conveying to the University of Washington all of said shorelands. [1969 ex.s. c 223 § 28B.20.362. Prior: 1939 c 60 § 2; No RRS. Formerly RCW 28.77.335.]

28B.20.364 1939 conveyance of shorelands to university—Grant for arboretum and botanical garden purposes—Reversion for unauthorized use—Reconveyance for highway purposes. All of the shorelands described in RCW 28B.20.360 are hereby granted to the University of Washington to be used for arboretum and botanical garden purposes and for no other purposes. In case the said University of Washington should attempt to use or permit the use of said shorelands or any portion thereof for any other purpose, the same shall forthwith revert to the state of Washington without suit, action or any proceedings whatsoever or the judgment of any court forfeiting the same: PROVIDED, That the board of regents of the University of Washington is hereby authorized and directed to reconvey to the state of Washington blocks 16 and 17 of Lake Washington shorelands, or such portions thereof as may be required by the state of Washington or any agency thereof for state highway purposes. The state of Washington or any agency thereof requiring said land shall pay to the University of Washington the fair market value thereof and such moneys paid shall be used solely for arboretum purposes. Such reconveyance shall be made at such time as the state or such agency has agreed to pay the same. [1969 ex.s. c 223 § 28B.20.364. Prior: 1959 c 164 § 1; 1939 c 60 § 3; No RRS. Formerly RCW 28.77.337.]

28B.20.370 Transfer of certain Lake Union shorelands to university. Block 18-A, Second Supplemental Maps of Lake Union Shore Lands, as shown on the official maps thereof on file in the office of the commissioner of public lands, is hereby transferred to the University of Washington and shall be held and used for university purposes only. [1969 ex.s. c 223 § 28B.20.370. Prior: 1963 c 71 § 1. Formerly RCW 28.77.339.]

28B.20.381 "University tract" defined. For the purposes of this chapter, "university tract" means the tract of land in the city of Seattle, consisting of approximately ten acres, originally known as the "old university grounds," and more recently referred to as the "metropolitan tract," together with all buildings, improvements, facilities, and appurtenances thereon. [1999 c 346 § 2.]

Purpose—Construction—1999 c 346: "The purpose of this act is to consolidate the statutes authorizing the board of regents of the University of Washington to control the property of the university. Nothing in this act may be construed to diminish in any way the powers of the board of regents to control its property including, but not limited to, the powers now or previously set forth in RCW *28B.20.392 through 28B.20.398." [1999 c 346 § 1.]

*Reviser's note: RCW 28B.20.392 was repealed by 1999 c 346 § 8.

Effective date—1999 c 346: "This act is necessary for the immediate preservation of the public peace, health, or safety, or support of the state government and its existing public institutions, and takes effect immediately [May 17, 1999]." [1999 c 346 § 9.]

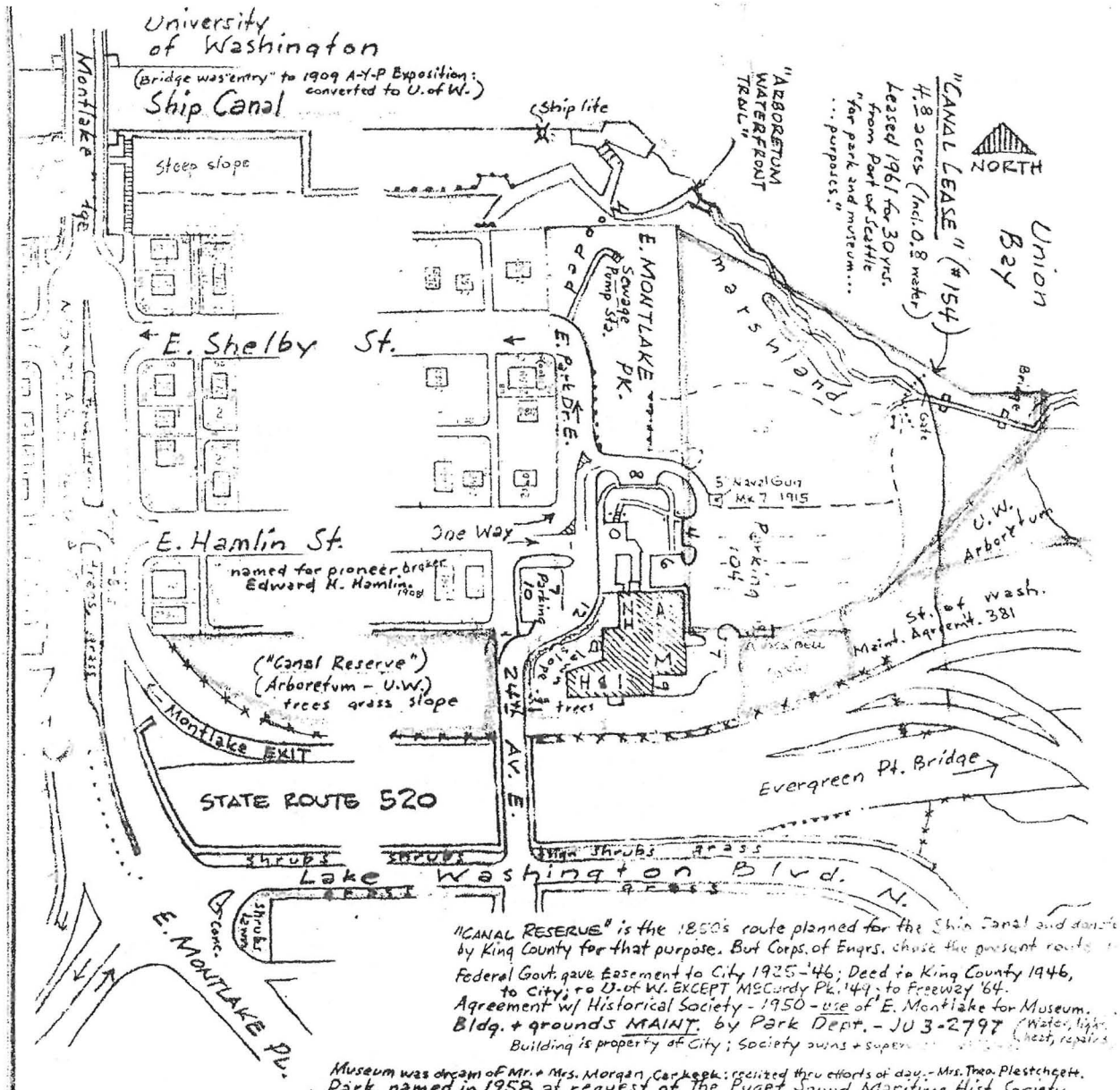
28B.20.382 University tract—Conditions for sale, lease, or lease renewal—Inspection of records—Deposit of proceeds—University of Washington facilities bond retirement account. (1) Until authorized by statute of the legislature, the board of regents of the university, with respect to the university tract, shall not sell the land or any part thereof or any improvement thereon, or lease the land or any part thereof or any improvement thereon or renew or extend any lease thereof for a term of more than eighty years. Any sale of the land or any part thereof or any improvement thereon, or any lease or renewal or extension of any lease of the land or any part thereof or any improvement thereon for a term of more than eighty years made or attempted to be made by the board of regents shall be null and void until the same has been approved or ratified and confirmed by legislative act.

(2) The board of regents shall have power from time to time to lease the land, or any part thereof or any improvement thereon for a term of not more than eighty years. Any and all records, books, accounts, and agreements of any lessee or sublessee under this section, pertaining to compliance with the terms and conditions of such lease or sublease, shall be open to inspection by the board of regents, the ways and means committee of the senate, the appropriations committee of the house of representatives, and the joint legislative audit and review committee or any successor committees. It is not intended that unrelated records, books, accounts, and agreements of lessees, sublessees, or related companies be open to such inspection. The board of regents shall make a full, detailed report of all leases and transactions pertaining to the land or any part thereof or any improvement thereon to the joint legislative audit and review committee, including one copy to the staff of the committee, during odd-numbered years.

(3) The net proceeds from the sale or lease of land in the university tract, or any part thereof or any improvement thereon, shall be deposited into the University of Washington facilities bond retirement account hereby established outside the state treasury as a nonappropriated local fund to be used exclusively for the purpose of erecting, altering, maintaining, equipping, or furnishing buildings at the University of Washington. The board of regents shall transfer from the University of Washington facilities bond retirement account to the University of Washington building account under RCW 43.79.080 any funds in excess of amounts reasonably necessary for payment of debt service in combination with other nonappropriated local funds related to capital projects for which debt service is required under section 4, chapter 380, Laws of 1999. [1999 c 346 § 3; 1998 c 245 § 17; 1996 c 288 § 27; 1987 c 505 § 13; 1980 c 87 § 10; 1977 ex.s. c 365 § 1; 1974 ex.s. c 174 § 1.]

Purpose—Construction—Effective date—1999 c 346: See notes following RCW 28B.20.381.

28B.20.394 University tract—Powers of regents—Agreements to pay for governmental services. In addition to the powers conferred upon the board of regents of the University of Washington by RCW 28B.20.395, the board of regents is authorized and shall have the power to enter into an agreement or agreements with the city of Seattle and the county of King, Washington, to pay to the city and the county such sums as shall be mutually agreed upon for governmental services rendered to the university tract, which sums shall not exceed the amounts that would be received pursuant to limitations imposed by RCW 84.52.043 by the city of Seattle and



"CANAL RESERVE" is the 1800's route planned for the Ship Canal and donate by King County for that purpose. But Corps. of Engrs. chose the present route.
Federal Govt. gave Easement to City 1925-'46; Deed to King County 1946, to City; to U. of W. EXCEPT McCurdy Pk. 1.44; to Freeway '64.
Agreement w/ Historical Society - 1950 - use of E. Montlake for Museum.
Bldg. + grounds MAINT. by Park Dept. - JU 3-2797 (Water, light, heat, repairs
Building is property of City; Society owns + supe

Museum was dream of Mr. + Mrs. Morgan Carkeek; realized thru efforts of day. - Mrs. Theo. Plestcheeff.
Park named in 1958 at request of The Puget Sound Maritime Hist. Society C.F. Clise, v.p., to honor Horace W. McCurdy, prominent civic + business leader; Puget Sound Bridge + Drydock, Lockheed Aircraft, P.S. Dredging + Pacific Nat. Bank; const. projects incl. first Lk. Wash. Floating Bridge; his artifacts in Maritime Wing.
Holder of a B.S. degree in Mech. Engineering from M.I.T., Cambridge, Mass., he was named "Construction Man of the Year" (1969), "Maritime Man of the Year" (1951), "Civil Engineer of the Year" (1969) and honored with "First Citizen Award" of Seattle-King Co. Board of Realtors.

= Seattle Fire Bell

HISTORICAL MUSEUM
2161 E. HAMLIN ST.
EA 4-1125
98102

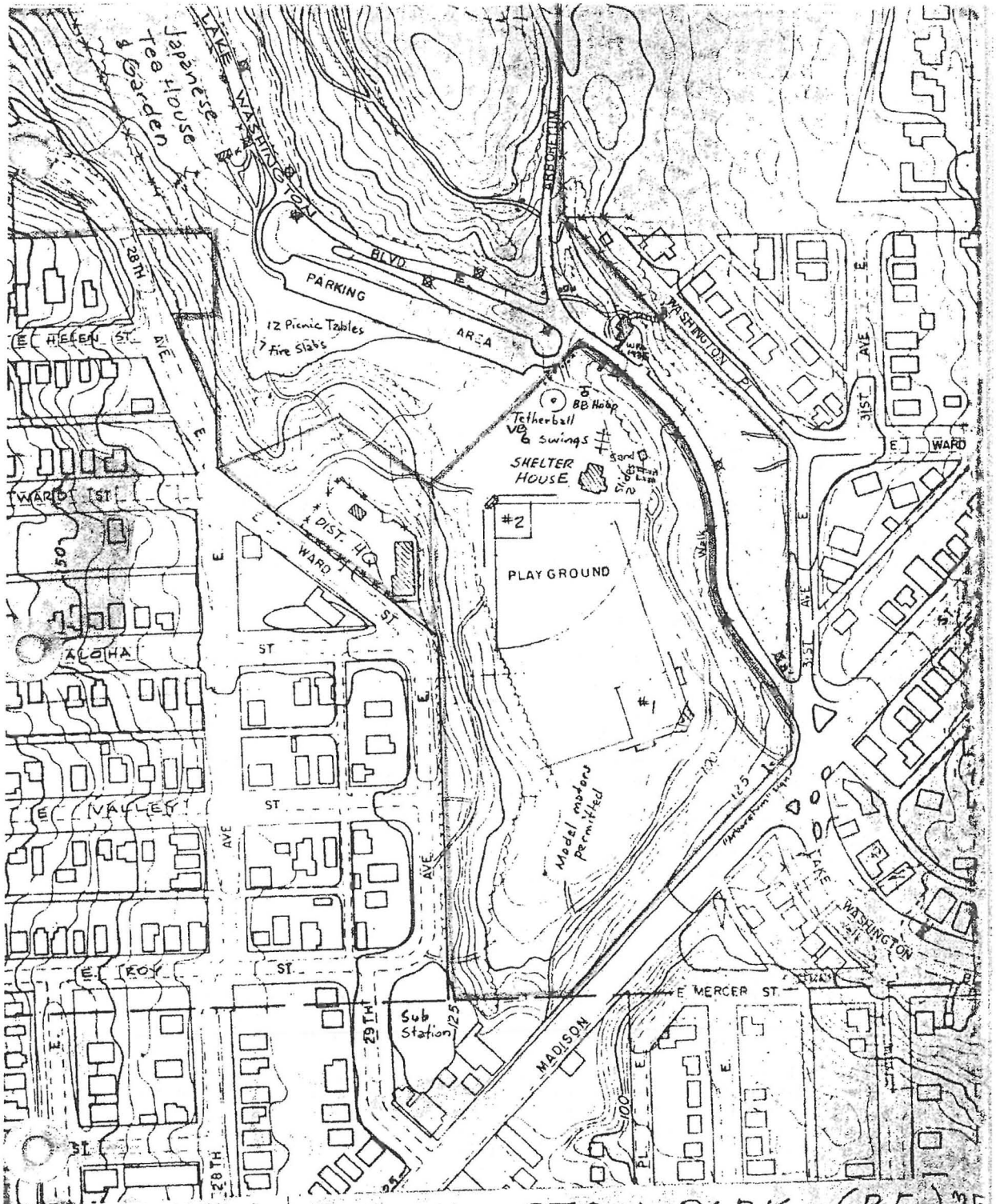
History + Industry (1952)
Maritime (1958)
Natural History (1961)
Auditorium (500 seat) (1971)

1.1 Acres
Deed: 1946 "to the City"
Use since 1925

H. W. McCURDY PARK

ENCLOSURE D

Two-sided

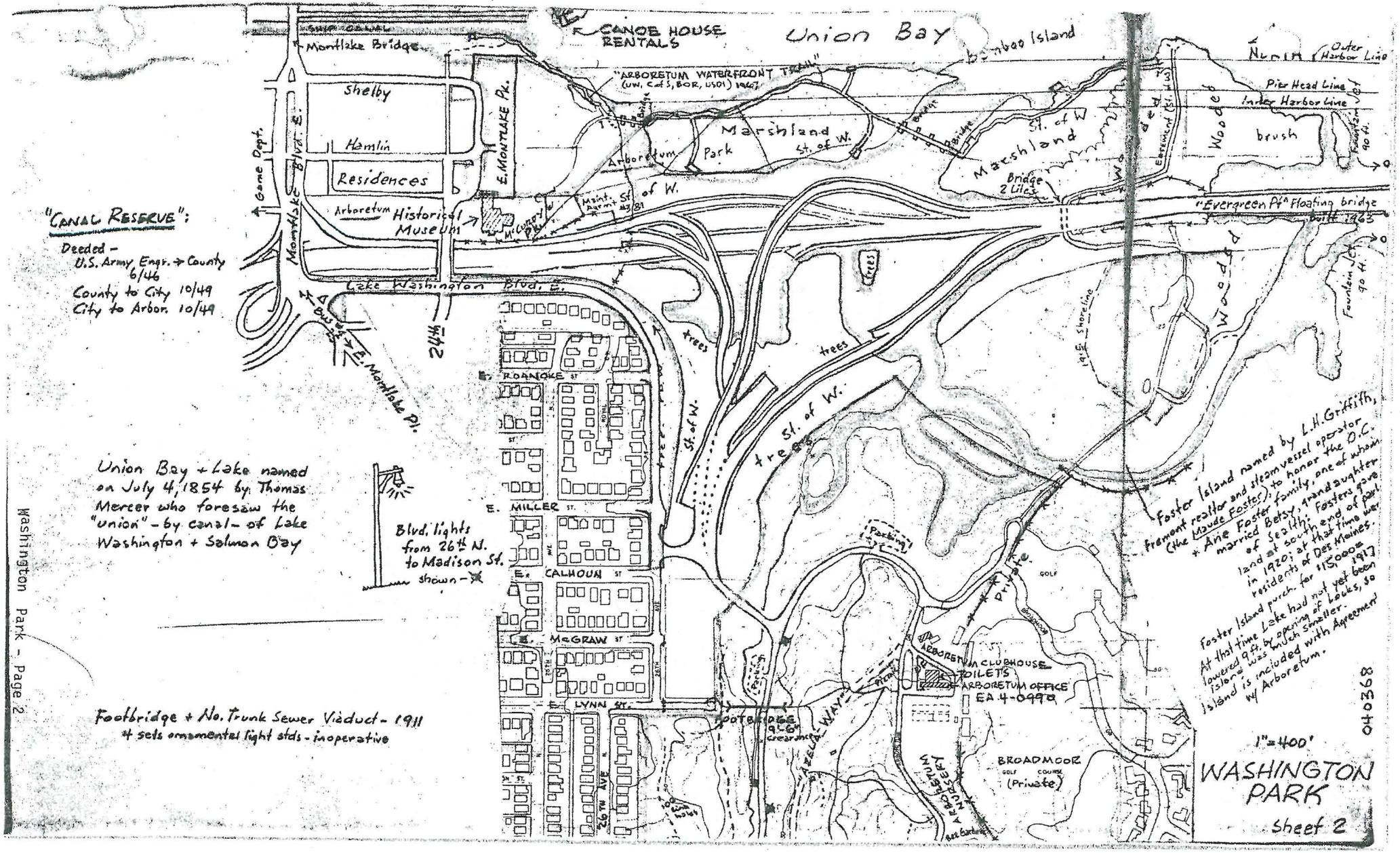


WASHINGTON PARK (P.F.)

Dist. H.Q. 1.5 Ac. ← (Modification Agreement 12¼ Ac) → 10.6 A.

Sheet 3

0714-67



"CANAL RESERVE":

Deeded -
U.S. Army Engr. → County 6/46
County to City 10/49
City to Arbor. 10/49

Union Bay + Lake named on July 4, 1854 by Thomas Mercer who foresaw the "union" - by canal - of Lake Washington + Salmon Bay

Blvd. lights from 26th N. to Madison St. shown - ⊗

Footbridge + No. Trunk Sewer Viaduct - 1911
4 sets ornamental light stds - inoperative

FOSTER ISLAND: Purchased in 1917 for $15,000; it was considerably smaller for the lake was 9' higher prior to the opening of the Ship Canal and locks. During dredging of the canal, nsiderable filling was done (with approval) "in the marshy areas, amongst the reeds d cattails." The Island was named by a Fremont realtor who also operated a steam vessel, The Maude Foster, which he named to honor O. C. and Ane Foster, one of whose family married Betsy, the grand-daughter of Chief Sealth. In 1920 the Fosters were residents of Des Moines and gave some property at the south end of the park. (Off Vancouver Island is a Foster Island which was named in 1865 by Captain Pender to honor British Major George Foster.) In 1963 the Evergreen Point floating bridge bisected the Island, and intended to slice off the park's west side with Thomson Expressway (first proposed as "Empire Way" in 1930) but vigorous continuous lawsuits resulted in voter rejection of the project in 1972. The freeway to the north under Union Bay - - -

In 1967 "the marshy areas, reeds and cattails" were recognized as a valuable resource and the "Arboretum Waterfront Trail" was established by the U.W., B.O.R., Department of Interior, and City of Seattle.

BOULEVARD LIGHTING: Rustic cedar poles and fixtures were made as a WPA project in 1936. By 1944 operation and maintenance were a problem that worsened until a new system was installed in 1970; a compromise in design and illumination level by the University of Washington, Traffic Department, City Light, Parks Department and Design Commission.

HISTORY

Travel northward by land from the pioneer town of Seattle was squeezed between Union Bay, Lake Union and Salmon Bay, and the wagon road along the "Montlake Ridge" became the latter-day Montlake Boulevard. Just south of Union Bay on the shore of Lake Washington, a pioneer judge, John J. McGilvra, staked his land claim in the 1880s and cut a road on n almost straight line "through the wilderness" to the town on Elliott Bay, the road hat became Madison Street. At best the roads were rough, dusty or muddy, and the journey long beset with the danger of bears or other wild animals. To promote the sale of real estate "so far from town" the judge gave 21 acres for a park at the foot of Madison Street and formed a company to build one of the "new toy" cable cars from town to Madison Park. It became a very popular Sunday summer outing. The Puget Mill Co. owned property in this area and, having logged the best timber, wished to sell their real estate. Improvements like the cable car were a big inducement to sales, so they made a deal with the City wherein they would give 62 acres of ravine for (Washington) park in exchange for $35,000 worth of watermain work in an adjacent subdivision they were developing.

This was 1900. The new park land was a rough ravine sloping abruptly to the "living stream of water running the entire length", from about 33rd Avenue into Union Bay. It was necessary for McGilvra's road to ford the creek, but upon construction of the cable car, a trestle bridge crossed the ravine. (The trestle was replaced with a fill about 1915.) The ravine was covered with a dense growth typical of Northwest forests with trees that had survived the loggers or second growth. About 1896 a system of bicycle paths was developed around the town, one route from Lake Union following the contours along the bluff that became Interlaken Park and boulevard, but it did not enter this ravine, staying at a higher contour so as to intercept Madison Street at the west end of the bridge over the Washington Park Creek (29th Avenue). At Madison Street it was a 50' deep ravine! (City Engineer contour map 7003/1903.)

At the time of acquisition there was a private park named "Washington" which was bought by J. M. Frink and later given to the City and, about 1910, renamed Frink Park. Bu 1902 this park was identified as WASHINGTON PARK. (In 1889 the Congress chose to honor GEORGE WASHINGTON as the name for the new (42nd) state on the 150th anniversary of his birth. The lake whose Union Bay forms the north end of the park was named for Washington

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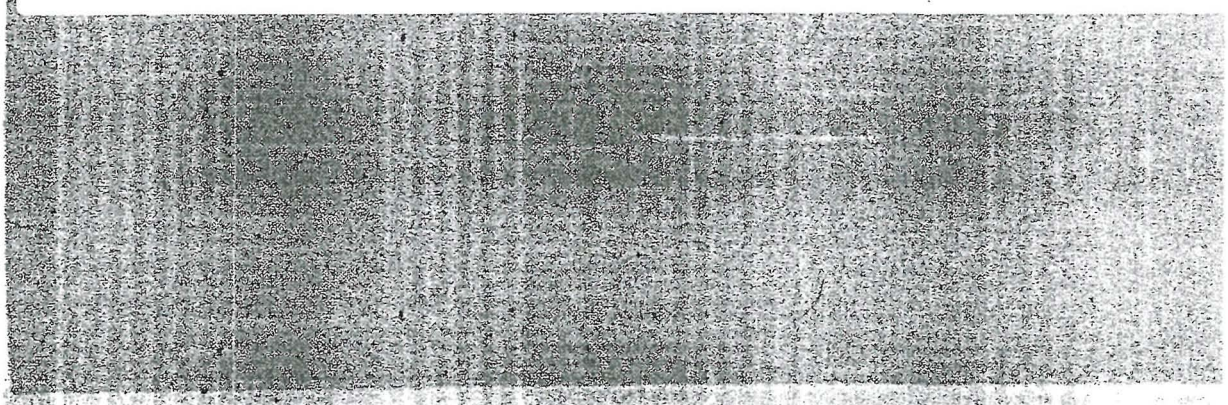

(boulevard) system will be." It was so popular for automobiles, carriages, horsemen and pedestrians that a mounted patrolman was necessary - the Park Department furnishing the horse and the Police Department the officer: the next year the horse had to be replaced with a motorcycle!

The automobile was still a novelty for the rich and the "sports", so the still numerous horse owners formed a Speedway Organization which raised $9,520 toward development of a public course for the "speeding" of harness horses (Azelia Way) together with sheds for cooling the horses and a barn. Horsepower was still the backbone of the Department's work force and since Washington Park was then "the center of the Boulevard System", a stable for 8 horses plus accommodations for steam rollers and other tools and a headquarters "barn" was built in 1909.

By this time a huge fill had been placed across the ravine, north from Madison Street, and an "athletic field" (baseball) had been established, the sloping sides of which made an ideal natural grandstand. (The pro-ballfield was nearby, at Madison Park.) "Games of the Bank League and numerous commercial teams are pulled off on (Washington Park) grounds." (It was a sanitary fill by the City Garbage Department.)

The 1913 Report notes a decline in the demand for the Speedway - "due to the advance of the automobile." Meanwhile, the sanitary fill continued, being done now in the marsh area near Union Bay; when dredging operations began for the new Ship Canal more fill was placed in the marsh, reeds and cattails around Foster Island, originally a small island until the dredging and lowering of the lake level by 9' upon the opening of the Ship Canal in 1917. (The island was owned by a Fremont Realtor who operated the steamer The Maude Foster, named to honor the daughter of O. C. and Ane Foster; one of the family married Betsy, the granddaughter of Chief Sealth. (Near Vancouver Island is a Foster Island named in 1865 by Captain Pender to honor British Major Foster.) In 1920 residents of DesMoines named Foster gave property to the south end of the park.) Foster Island was purchased in 1917 ($15,000).

Excepting for the foregoing improvements noted, this "huge ravine" had been left in a natural state. "Considerable work had been done adjacent to the driveway in the way of walks, lawn areas, flowers and shrubs, etc." So in 1915 came a surge of interest in the game of golf. The first municipal course in Seattle had just opened on Beacon Hill: Jefferson Park. Now came the proposal for a course in the north end at this "undeveloped" park. In 1919 "certain gentlemen of this city" offered to form a corporation to lease and develop a course in Washington Park. The Board questioned the legality of such use and held the park development should be for "the general public." Soon after this property along the east boundary of the park was resubdivided as the exclusive Broadmoor development with a private golf course around three sides. An easement for a roadway across shorelands to permit the development and public use of Foster Island had been granted by the State in 1917; that road was along the northeast edge of the park, so it was quite convenient to locate a north entry to Broadmoor onto this "public road". There were proposals to develop the "Lakeside Boulevard" along the shoreline from 43rd to the University, but much filling was required to accomplish it. So the roadway across the north end of Washington Park became the north access route for Broadmoor.

Horseriding facilities continued in the park but with decreasing popularity until 1935, the surrendering of the concession contract of the riding academy. The old Speedway had been abandoned by 1919, "closed on account of the rotting away of a bridge." Before long it was replaced with grass and became "Azelia Way" (W. C. Hall, Park Engineer.) The barn (minus cooling sheds?) was leased by concession for riding clubs and academies. The park barn and service yard, located in the meadow below Helen Street, was relocated in 1950 upon the request of the Arboretum Board which planned to build an exhibition hall there. The new site was up the hill from there, fronting on Ward Street. But the new site was found to be composed of 325,000 cobblestones from Madison Street

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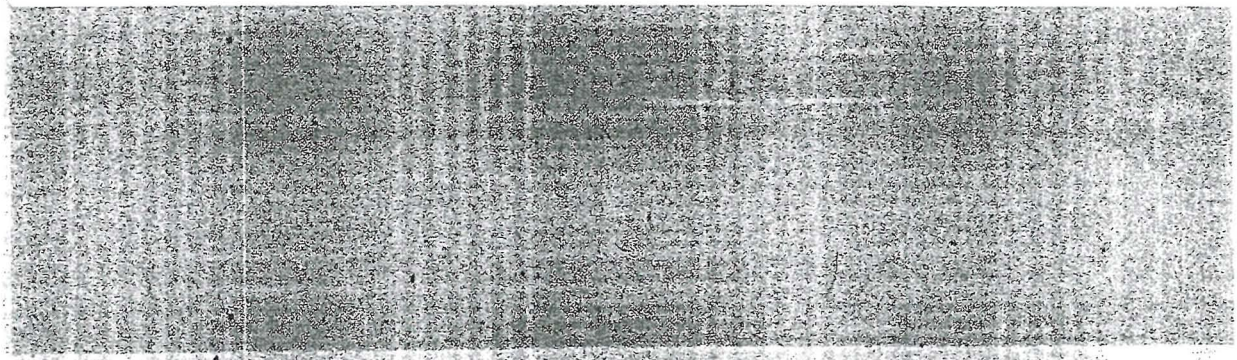


arterial traffic off the Park boulevard.); the fence to prevent theft of rare plants and to protect wildlife from dogs. A great controversy arose (1936): Park Board Chairman H. M. Westfall declared "it was all a hoax to crystallize public opinion." A "temporary" fence was built along the east boundary (and golf course) to remain until the thickly planted hedge grew as high as the fence. Fencing for the Arboretum became another hotly contested controversy after the State Legislature in 1972, faced with the necessity to cut the University budget, recommended relief from the management of the Arboretum. The U.W. declared that the use of the Arboretum was that of a public park rather than a scientific classroom. As such, the area received abuse not related to an arboretum. But the City declared it did not have funds to maintain the park as an arboretum. The U.W. proposed fending the arboretum - east of the boulevard. The opposition was heated. The U.W. objected to placing a unit of the reactivated mounted police (in an effort to cope with increased muggins, rapes, without assurance that the frail plant and soil conditions would be protected from the horses). (Pat Hemenway had been shot by a robber, causing a spinal injury that made her totally disabled, unable to find any financial support; the State Legislature listened to her plea for recompense for all victims of such attacks and authorized such legislation. Despite her great courage, she lost her fight for life.)

Meanwhile, the "tug of war" continued to rage until a settlement was reached in 1974 with the Letter of Clarification wherein total maintenance of the 1974 level "or better" would be financially shared equally by the U.W. and the City. The Seattle Times editorialized that the U.W. position had softened with a change in policy under the new U.W. president, Dr. John Hogness.

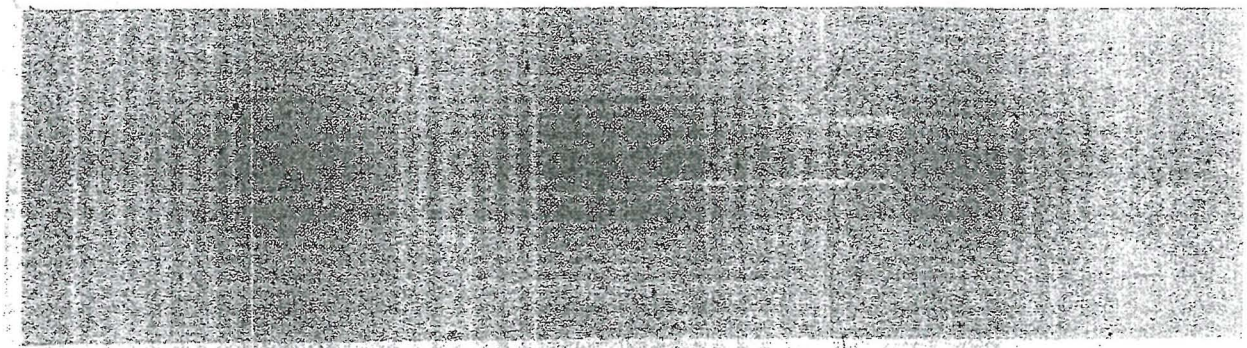
Among the first visitors to the Arboretum (in 1938) were two distinguished ones and 600 unique ones: the mother and the wife of President Roosevelt who also visited another WPA project - West Seattle Golf and Recreation Area - and of course visiting the wife of the P.I. editor, Anna Roosevelt Boettiger, daughter of the "First Family"; the unique visitors were 2000 larvae from which 600 fireflies matured - these immigrants from the east coast were an attempt to transplant the fascinating insects into the Arboretum and the northwest - the suggestion of an invalid daughter of a Department of Agriculture official who was honored at the ceremony releasing the fireflies.

The entire Washington Park (including Foster Island) was included in the origianl Agreement with the Arboretum (U.W.) in 1934. When it became known to the ballplayers that the athletic field was about to be replaced with a rose garden, another storm of disapproval arose. The result was modification of the agreement, in which the playfield as well as the proposed new service use were excluded from Arboretum use (1948).

Efforts to establish a Japanese Teahouse and Garden began as early as the 1909 AYP Exposition. It was a logical part of the Pacific Rim celebration and its contribution to northwest culture and trade. After the Expo the Teahouse was purchased by Emma Watts and "placed in Madison Park." 10 years later a $5,000 teahouse existed at the southwest corner of 5th and University. A request was made to the Department to permits its relocation in Volunteer Park or elsewhere as a concession sponsored by the Japan Central Tea Association. In 1937 the Arboretum Society renewed the dream, but it did not take form until 20 years later when Mrs. Neil Haig went to the Japanese Consul, Yoshiharu Takeno, who sought aid from cities in Japan. The first response came from Kobe, Seattle's sister city. Tokyo gave enormous gifts - the work of the eminent designers, Mr. K. Inoshita and his associate Mr. Juki Iida, and a magnificent teahouse. Funds for the work came mainly from a generous Arboretum member. The value of all gifts and work was $200,000. Seattle craftsmen performing the work, supervised by Mr. Iida and Mr. Kitamura, were the Yorozu Co., Ishimitsu Co. and Yamasaki and Kubota. The garden was dedicated in 1960.

Arsonists completely destroyed the teahouse in 1973.

continued

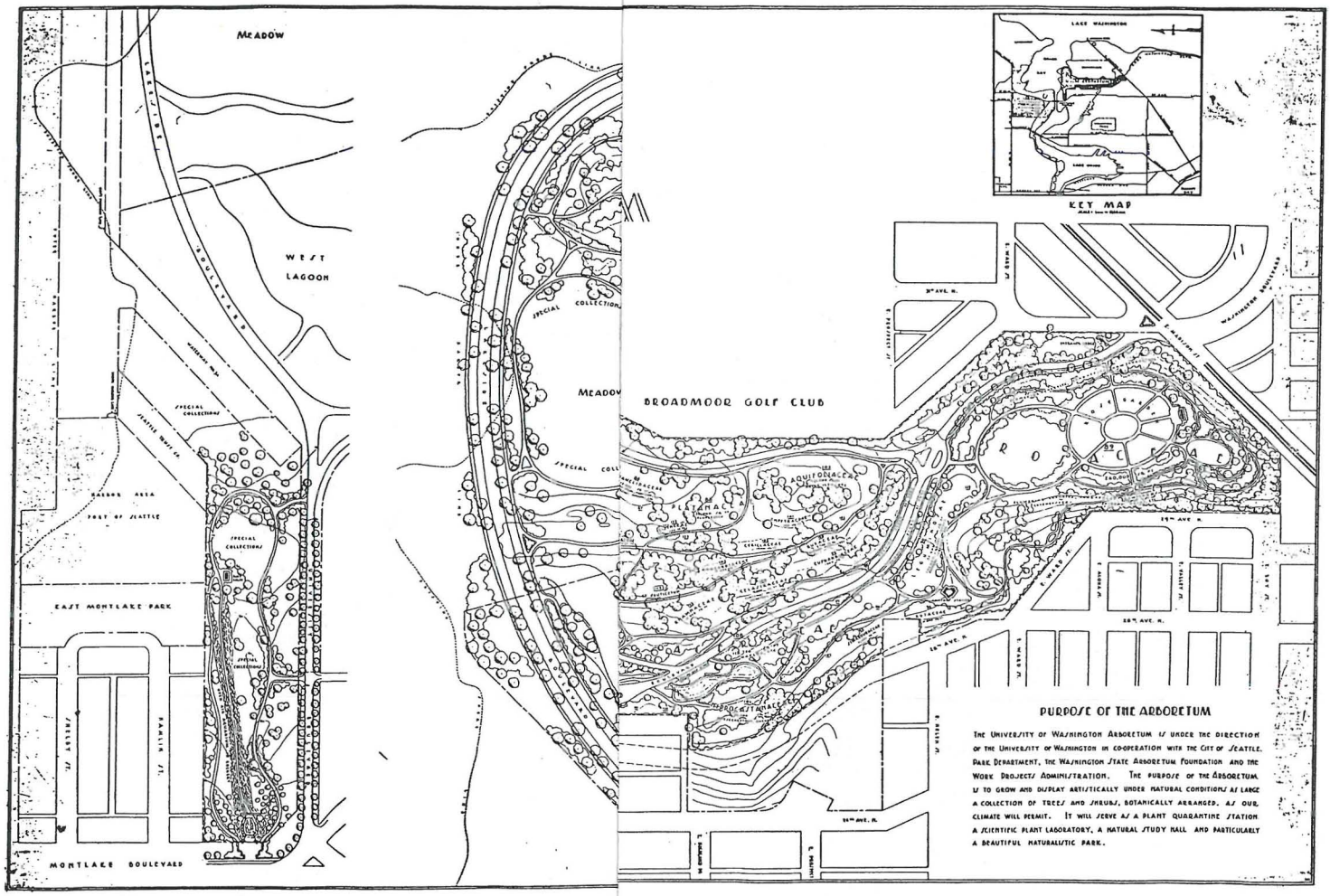


WASHINGTON PARK - History (continued) AND ARBORETUM

The growth of suburbia east of Lake Washington demanded relief from the crowded Mercer Island Floating Bridge, so another one was constructed in 1963 from the Evergreen Point across the north end of the park - mainly Foster Island - creating a new "Bamboo Island" and a wide interchange of ramps intended to connect with the north/south Expressway known in 1928 as Empire Way and later proposed as the Thomson Expressway. Empire Way had been proposed along the west side of Washington Park, taking the whole side from Ward to Lynn Streets, and/or the strip of residences along 26th, at least. But the community and residents had long ago stopped Empire Way at Madison Street with a series of vigorous and continuous lawsuits. So the 1963 interchange ramps deadended abruptly at the north end of the park onto Lake Washington Boulevard. Further construction into the park waited . . . until 1972 when the voters rejected the Expressway. But the expectant bulldozers had excavated for the Expressway to Lynn Street, and it remained as a blighted scar where little would grow except parking for cars, especially during football games. Some homes had been bought and boarded up until . . . . .

The marshy areas, reeds and cattails on the north side of the Freeway were recognized (finally) as a valuable wildlife resource and the Arboretum Waterfront Trail was built on pontoons by the U.W., Bureau of Outdoor Recreation and City Arboretum Trust Fund. Completion of the Waterside Trail along the Canal in 1971 caused it to become a National Recreation Trail.

DS:d
9/20/74


MEADOW
WEST LAGOON
BOULEVARD
SPECIAL COLLECTIONS
MEADOW
BROADMOOR GOLF CLUB
KEY MAP
LAKE WASHINGTON
AQUIFOLIACEAE
ROSACEAE
EAST MONTLAKE PARK
MONTLAKE BOULEVARD
PURPOSE OF THE ARBORETUM
THE UNIVERSITY OF WASHINGTON ARBORETUM IS UNDER THE DIRECTION OF THE UNIVERSITY OF WASHINGTON IN CO-OPERATION WITH THE CITY OF SEATTLE, PARK DEPARTMENT, THE WASHINGTON STATE ARBORETUM FOUNDATION AND THE WORK PROJECTS ADMINISTRATION. THE PURPOSE OF THE ARBORETUM IS TO GROW AND DISPLAY ARTISTICALLY UNDER NATURAL CONDITIONS AS LARGE A COLLECTION OF TREES AND SHRUBS, BOTANICALLY ARRANGED, AS OUR CLIMATE WILL PERMIT. IT WILL SERVE AS A PLANT QUARANTINE STATION A SCIENTIFIC PLANT LABORATORY, A NATURAL STUDY HALL AND PARTICULARLY A BEAUTIFUL NATURALISTIC PARK.

ENCLOSURE E

THE

UNIVERSITY NATIONAL BANK

OF SEATTLE

Member Federal Deposit Insurance Corporation

it has been (privilege to
and have the confidence c
University of Washington a
residents of the great dist
which surrounds it.

U N I V E R S I T Y O F W A S H I N G T O N C A M P U

ENCLOSURE #2

Ordinance No. 90723

AN ORDINANCE providing for the acquisition of certain real property from the University of Washington for the Montlake Interchange by the exchange of property with, and payment of consideration to, said University, and making an appropriation from the Seattle General Arterial Improvement Bonds 1954 Fund in connection therewith.

FILE NO. 241473

Council Bill No. 82196

INTRODUCED: NOV 13 1961	BY: Parks and Public Grounds, Streets & Sewers, Finance
REFERRED: NOV 13 1961	TO: Finance, Parks & Public Grounds, ~~Streets & Sewers~~
REFERRED:	
REPORTED: NOV 20 1961	SECOND READING: NOV 20 1961
THIRD READING: NOV 20 1961	SIGNED: NOV 20 1961
PRESENTED TO MAYOR: NOV 20 1961	APPROVED: NOV 20 1961
RETD. TO CITY CLERK: NOV 20 1961	PUBLISHED:
VETOED BY MAYOR:	VETO PUBLISHED:
PASSED OVER VETO:	VETO SUSTAINED:
ENGROSSED: VOL..... PAGE.....	BY:

SM 10-59 HALL

MCG

GGW:EM
11-13-61

ORDINANCE 90723

AN ORDINANCE providing for the acquisition of certain real property from the University of Washington for the Montlake Interchange by the exchange of property with, and payment of consideration to, said University, and making an appropriation from the Seattle General Arterial Improvement Bonds 1954 Fund in connection therewith.

WHEREAS, the City requires certain property hereinafter described for construction of the Montlake Interchange contemplated by Ordinance 90098 and the University of Washington has offered to convey its interest in such property to the City for a consideration including the payment of Thirteen Thousand Four Hundred Seventy Three Dollars ($13,473), the conveyance to said University of the city's interest in certain property hereinafter described and certain other conditions and the City Engineer in C. F. 241873 has recommended acceptance of such offer; Now, Therefore,

BE IT ORDAINED BY THE CITY OF SEATTLE AS FOLLOWS:

Section 1. That upon receipt of a quitclaim deed from the University of Washington to The City of Seattle, conveying to the city the University's interest in the following described real property to wit:

That portion of Old Canal right of way (Canal Reserve) in Section 21, Township 25 North, Range 4 East, W.M., described as follows: Beginning at a point on the east margin of Montlake Boulevard distant 155.93 feet south of its intersection with the center line of Hamlin Street; thence south 1° 25'23" west along said east margin 213.94 feet; thence south 42°28'57" east 150.11 feet; thence south 88°33'57" east along the north margin of Lake Washington Boulevard a distance of 590.23 feet; thence north 0°43'27.5" east 202.22 feet; thence north 88°34'37" west 131.88 feet to the production south of the east line of the north and south alley as platted in Block 4, Montlake Park Addition, according to plat thereof recorded in Volume 18 of Plats, page 20, Records of King County, Washington; thence north 1°25'23" east along said produced line 120.00 feet to the south line of the east and west alley in said block; thence north 88°34'37" west along said south line 15.00 feet; thence south 17°48'44" west 88.60 feet; thence south 1°25'23" west 76.50'; thence north 86°21'37" west 34.18 feet to a point of curvature; thence westerly along the arc of a curve to the right, having a radius of 400 feet, an arc distance of 49.68 feet to a point of tangency; thence north 79°14'37" west 255.24 feet to a point of curvature; thence westerly and northwesterly along the arc of a curve to the right, having a radius of 220 feet, an arc distance of 199.22 feet to a point of tangency; thence north 27°21'37" west 3.00 feet; thence north 88°34'37" west 26 feet to the point of beginning.

Containing an area of 141,404 square feet more or less.

together with a conveyance of a temporary easement over the following described real property for use during construction of the Montlake Interchange under Ordinance 90098:

Ordinance No. 90723

AN ORDINANCE providing for the acquisition of certain real property from the University of Washington for the Montlake Interchange by the exchange of property with, and payment of consideration to, said University, and making an appropriation from the Seattle General Arterial Improvement Bonds 1954 Fund in connection therewith.

11/14/61 - Pass

FILE NO. 241973

Council Bill No. 82196

INTRODUCED: NOV 13 1961	BY: Parks and Public Grounds, Streets & Sewers, Finance
REFERRED: NOV 13 1961	TO: Finance, Parks & Public Grounds, ~~Streets & Sewers~~
REFERRED:	
REPORTED: NOV 20 1961	SECOND READING: NOV 20 1961
THIRD READING: NOV 20 1961	SIGNED: NOV 20 1961
PRESENTED TO MAYOR: NOV 20 1961	APPROVED: NOV 20 1961
RETD TO CITY CLERK: NOV 20 1961	PUBLISHED:
VETOED BY MAYOR:	VETO PUBLISHED:
PASSED OVER VETO:	VETO SUSTAINED:
ENGROSSED: VOL..... PAGE.....	BY:

BM 10-59 HALL

MCL

PUB
A. A. (BS
BLDG
ENG.
R. C.
L. & E.
C. O.
LIGHT

BALANCES AS NOTED ORD. 93220

Deck #15863

ENCLOSURE G

That portion of Old Canal right of way (Canal Reserve) in Section 21, Township 25 North, Range 4 East, W.M., described as follows: Beginning on the east margin of Montlake Boulevard distant 155.93 feet south of its intersection with the center line of Hamlin Street; thence south 88°34'37" east 26 feet to the true point of beginning; thence south 27°21'37" east 3.00 feet to a point of curvature; thence southeasterly and easterly along the arc of a curve to the left having a radius of 220 feet, an arc distance of 199.22 feet to a point of tangency; thence south 79°14'37" east 255.24 feet to a point of curvature; thence easterly along the arc of a curve to the left having a radius of 400 feet, an arc distance of 49.68 feet to a point of tangency; thence south 86°21'37" east 34.18 feet; thence north 1°25'23" east 61.50 feet; thence south 89°54'26" west 334.72 feet; thence westerly and northwesterly along the arc of a curve to the right, having a radius of 215 feet and the center of said curve bearing north 10°45'23" east, an arc distance of 194.94 feet; thence north 88°34'37" west 5.71 feet to the true point of beginning.

Containing an area of 12,380 square feet more or less.

the Mayor and City Comptroller are authorized and directed to execute and deliver to the University of Washington a quitclaim deed, subject to easements for such sewer and water pipelines as now exist, of the city's interest in the following described real property to wit:

That portion of the Old Canal right of way (Canal Reserve) in Section 21, Township 25 North, Range 4 East, W.M., described as follows:
Beginning at a point on the east margin of Montlake Boulevard East 155.93 feet south of its intersection with the center line of Hamlin Street; thence south 88°34'37" east 26 feet to the true point of beginning; thence south 27°21'37" east 3.00 feet to a point of curvature; thence southeasterly along the arc of a curve to the left, having a radius of 220 feet, an arc distance of 199.22 feet to a point of tangency; thence south 79°14'37" east 255.24 feet to a point of curvature; thence easterly along the arc of a curve to the left, having a radius of 400 feet, an arc distance of 49.68 feet to a point of tangency; thence south 86°21'37" east 34.18 feet; thence north 1°25'23" east 76.50 feet; thence north 17°48'44" east 88.60 feet to the south line of the east and west alley in Block 4, Montlake Park Addition as recorded in Volume 18 of Plats, at Page 20, Records of King County, Washington; thence north 88°34'37" west along said south line and same produced to the true point of beginning.

said property being a portion of that heretofore authorized to be conveyed to the University of Washington by Ordinance 78354.

Section 2. As further compensation to the University of Washington for the conveyance to the City contemplated in Section 1 hereof, there is hereby appropriated from the Seattle General Arterial Improvement Bonds 1954 Fund the sum of Thirteen Thousand Five Hundred Dollars

($13,500) or so much thereof as may be necessary for the removal of shrubbery and other botanical specimens from the property to be used by the City for highway purposes, and the City Comptroller is authorized to draw and the City Treasurer to pay the necessary warrants, as recommended by the City Engineer in C. F. 241873.

Section 3. And as a part of the consideration to said University for the conveyance to the City contemplated in Section 1 hereof the City Engineer and Board of Public Works are authorized and directed to provide for the relandscaping of the University property to be used by the City as a temporary construction easement for the preservation or relocation of sewer and water lines serving the property herein authorized to be conveyed to the University of Washington, and for the disposal of excavation waste material, all as contemplated by C. F. 241873.

(To be used for all Ordinances except Emergency.)

Section 4. This ordinance shall take effect and be in force thirty days from and after its passage and approval, if approved by the Mayor; otherwise it shall take effect at the time it shall become a law under the provisions of the city charter.

Passed by the City Council the 20 day of November, 19 61

and signed by me in open session in authentication of its passage this 20 day of November, 19 61.

Elmira Larsen
President of the City Council.

Approved by me this 20 day of November, 19 61.

Gordon A Clinton
Mayor.

Filed by me this 20 day of November, 19 61.

Attest: O. H. Richardson
City Comptroller and City Clerk.

(SEAL)

Published

By W. A. Perino
Deputy Clerk.

The City of Seattle--Legislative Department

MR. PRESIDENT:

Date Reported and Adopted

Your Committee on Finance, Parks & Public Grounds, and Streets & Sewers

to which was referred C.B. 82196, NOV 20 1961

providing for the acquisition of certain real property from the University of Washington for the Montlake Interchange by the exchange of property with, and payment of consideration to, said University, and making an appropriation from the Seattle General Arterial Improvement Bonds 1954 Fund in connection therewith,

RECOMMEND THAT THE SAME DO PASS.

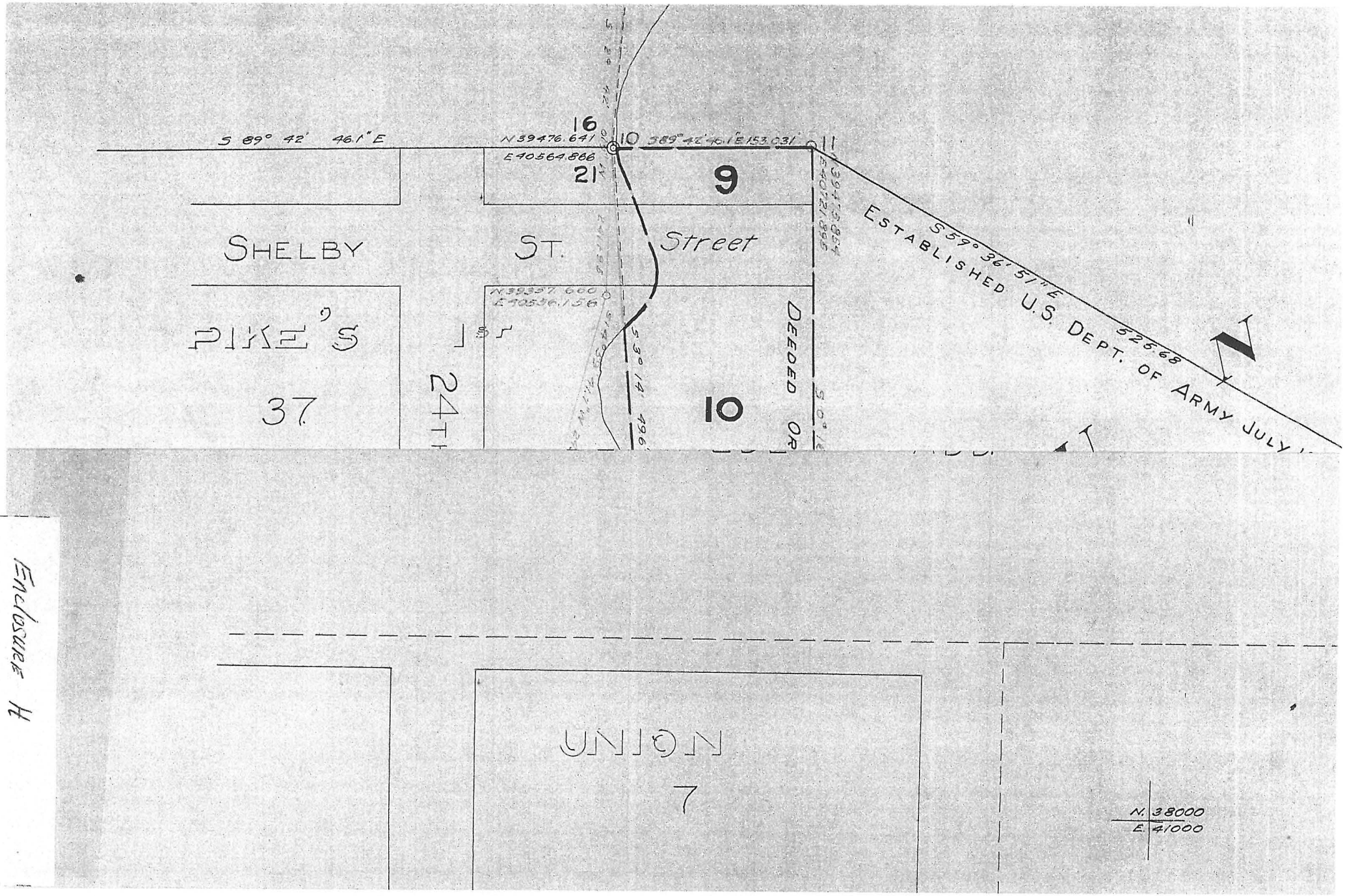
Fin. Chairman

Committee

P&PG Chairman

S&S

Committee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SHELBY
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24TH
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S 59° 36' 51" E
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ESTABLISHED U.S. DEPT. OF ARMY JULY
UNION
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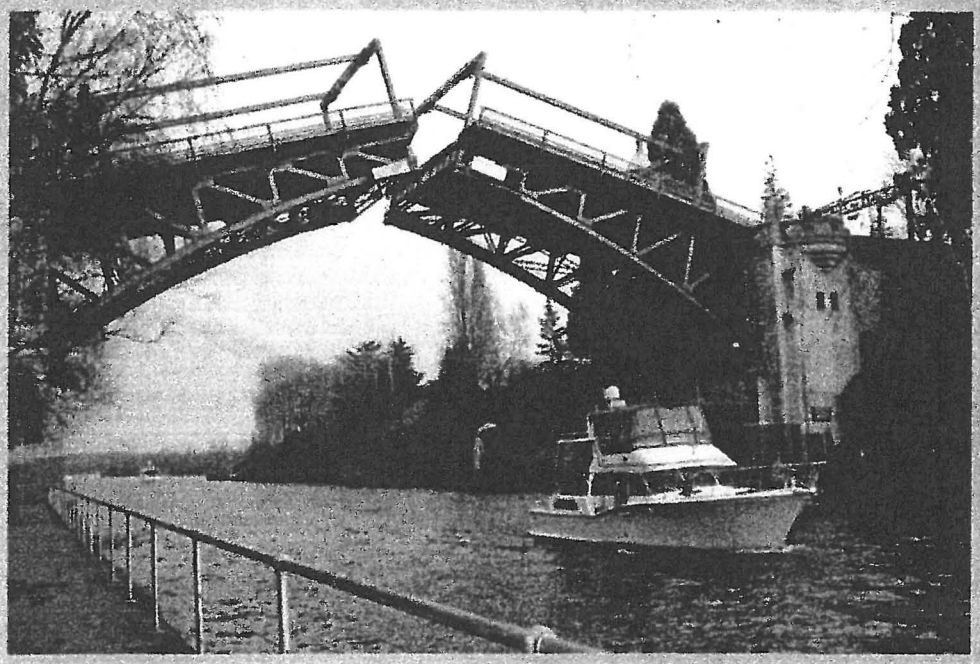
N. 38000
E. 41000

Enclosure H

MONTLAKE: AN URBAN EDEN



A History of the Montlake Community of Seattle

EUGENE SMITH

ENCLOSURE I

CHAPTER 11

Parks and Garbage--Within and Nearby

As real estate agents are fond of pointing out to prospective buyers--who may not already have discovered it for themselves--Montlake abounds with parks. The two largest, Interlaken and Washington, are not actually within the loose Montlake boundaries, though they seem so near physically and psychologically that most Montlake residents have historically treated them as integrally connected. The smaller areas designated as parks or parkway function as pleasant open spaces--dividers between lake and residence and between busy lanes of car and truck traffic. The creators of these spaces seem to have had exactly these purposes in mind.

East and West Montlake Parks and Montlake Boulevard Centerstrip

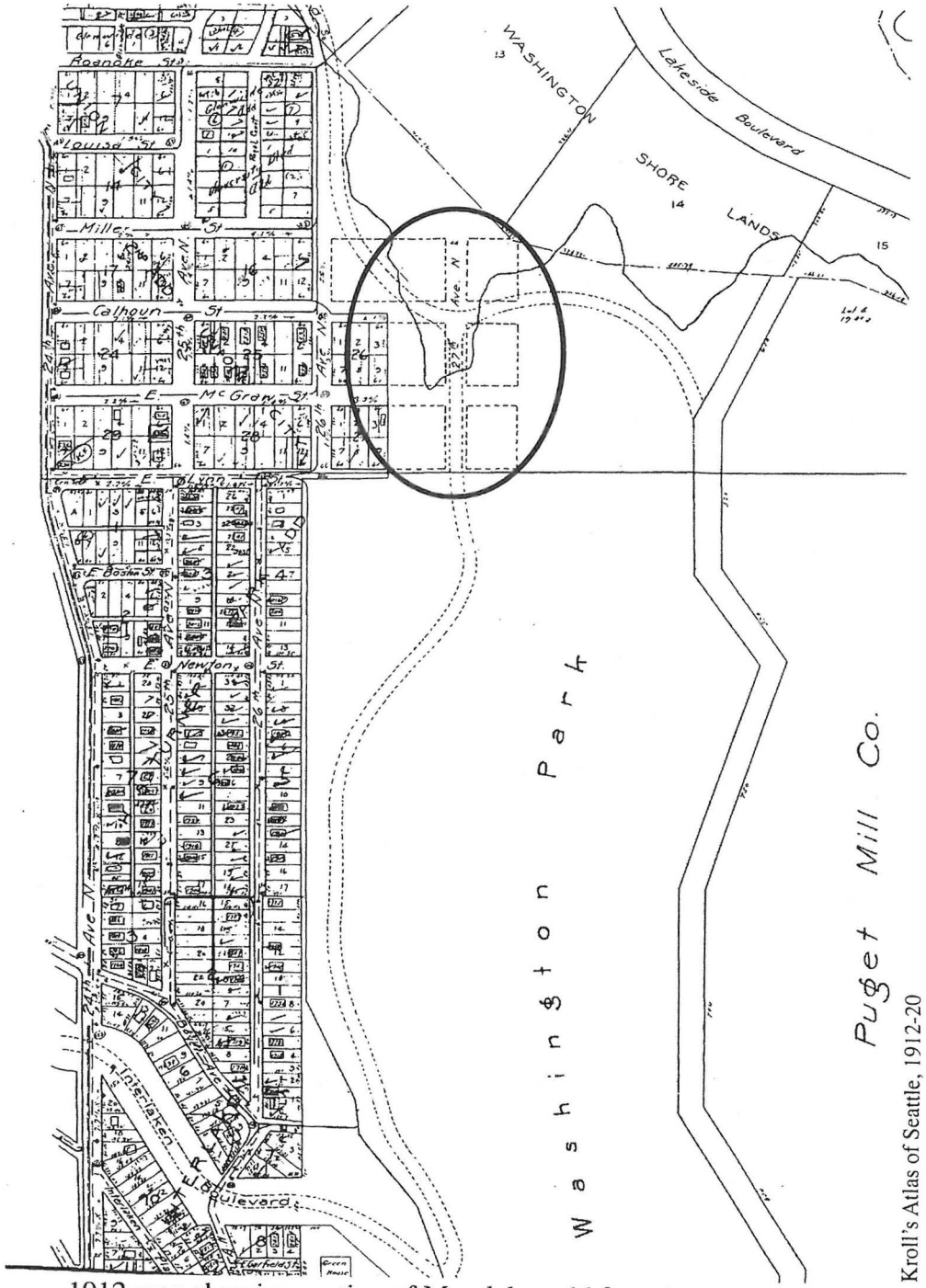
Even before Hagan and Hagan submitted their Montlake Park Addition plat in 1909, negotiations had begun with the Seattle Park Board to connect the early version of Lake Washington Boulevard--from Washington Park at the south--to the university grounds. John C. Olmsted--who, in 1903, had laid out a comprehensive park and boulevard system for Seattle and had strongly recommended a parkway connection between Washington Park and university grounds--reiterated that recommendation with greater force in 1906: ". . . this project will be of greater value to the Park system than any other which has been contemplated."[1] His proposal assumed greater urgency as planning for the Alaska Yukon Pacific Exposition accelerated because several city officials were looking for a graceful and efficient south entrance. The first plan was to extend the boulevard northward, along the Union Bay shore (on the east side of the isthmus). To that end, Ramsey and Baillargeon, owners of three acres of land in the Montlake Park Addition, proposed to sell at $4,000 per acre.[2] The Park Board, meeting in 1907, offered $10,000. (The Panic of 1907 had hit.) A month later, one of the Hagan brothers met with the board, doubtless with their projected replatting in mind. Though the minutes of the meeting did not quote him directly, they stated that he "protested the taking of land for the carrying out of the route chosen by the Board" and "offered a plan whereby another route than that which had been selected might be secured."[3] Furthermore, he said, he represented "the holdings of Messers. Baillargeon and Ramsay." Hagan evidently stated his case vigorously, causing the board members to go into executive session. When they emerged, they passed a motion reaffirming their decision and directing continuation of condemnation proceedings.

At the same time, additional land south of the Montlake Park Addition (formerly, Pike's First Addition to Union City) was needed for the projected boulevard extension. This major purchase, at $500 per lot,[4] took out two and a half blocks of what could have been Montlake, projected farther into Washington Park Arboretum than it now is. Perhaps subsequent Montlake residents have a better historic claim on Washington Park Arboretum than they had suspected! As the map on page 116 shows--with dotted lines indicating blocks in original plat of Pike's Second Addition to Union City--the gently curving Lake Washington Boulevard that came to occupy land that had been owned by the Union Trust Company; 12 lots in Block 15, 6 lots in Block 26 and 27, all east of 26th Avenue N., became park property.

Hagan had reason to mistrust the board's decision when he learned of ship-canal-superintendent Major Chittenden's edict about location of a bridge across the government canal: "a bridge across the canal at 22nd Avenue will be the only one allowed by the government."[5] That meant that the Union Bay-hugging route was out of the question. Hagan, seeing an advantage for his proposed Montlake Park Addition, came up with an offer impossible to resist: he would donate enough land on the isthmus ridge to accommodate a 150 foot-wide boulevard--no mere street. Not only that, he offered to donate land for parks fronting both

lakes at each end of the new addition. Faced with the Chittenden decision and this magnanimous offer, the board accepted the Hagans' proposition, which would include 75 feet in the center of the boulevard for park land. Furthermore, the two parks--later named West and East Montlake Parks--"[give] the Park Board the entire water front of Lake Washington and Lake Union without any expense to the Park Board . . ."[6] The board also agreed to dismiss condemnation proceedings for the previous route.

When the Hagans submitted their plat plan a few months later, it showed the two parks--still mostly un-

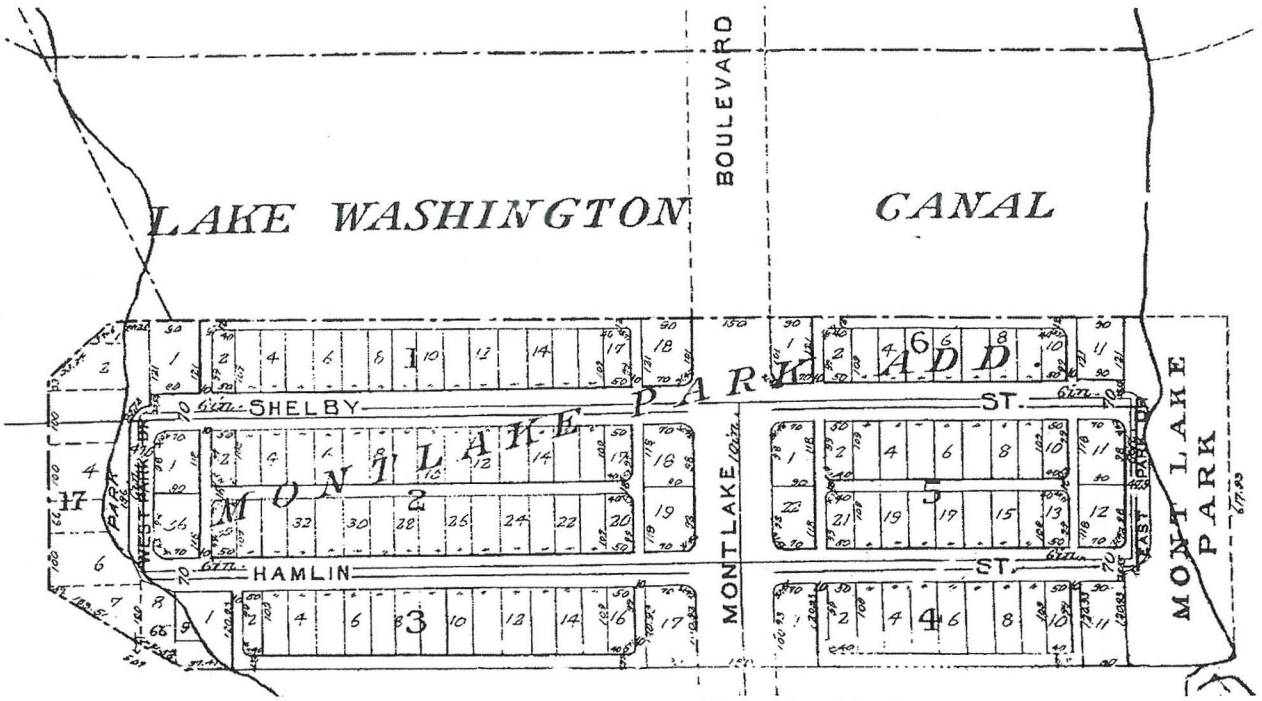


Kroll's Atlas of Seattle, 1912-20

1912 map showing portion of Montlake sold for arboretum use (blocks with dotted lines in circle)

der water--approached by East and West Park Drives, both extensions in the form of parentheses of Shelby and Hamlin Streets. On city maps, the centerstrip of Montlake Boulevard was clearly marked as parkway, though that term did not necessarily imply protection from other uses. The City Engineer, indeed, thought it would be an excellent spot for street cars that would transport A-Y-P visitors; he presented that proposal to the park commissioners, who approved it.[7] However, two months later, they reconsidered that action, deciding that parkway was parkway and that a commercial enterprise--the street railway was under private ownership at that time--should be "outside of this strip."[8] John C. Olmsted, active in planning Washington Park and the A-Y-P grounds, caused them to reverse their position once again: he recommended that "the street railway tracks [two tracks with poles for trolleys] occupy the central parkway strip, with the commercial roadway on the west side and boulevard on the east side."[9] His report, with obvious sensitivity to aesthetics, also proposed grass to be planted on top of the sleepers (or, railroad ties), four rows of tulip trees and shrubbery on the central and side strips, as well as vines up the trolley and lighting poles. To assure that overzealous owners of property adjoining the boulevard did not spoil his vision, he further proposed that nothing should be built less than 40 feet from the boulevard, that no commercial uses should be permitted, and that any house should be no more than two stories--these restrictions to apply for 99 years. As a final touch and an eye to the future, he urged that the Parks Department should care for the plantings in the centerstrip and the trees on the east side. The board adopted that report.

As grading, laying of tracks, and paving proceeded in 1909, the Parks Department somewhat ungenerously presented the Hagans with a bill for $2,280 for boulevard grading--probably determining that this work was a necessary part of the Hagans' preparing the area for sales of real estate. They couldn't pay, however, because incomplete utilities had retarded or prevented sales. When the property owner, James M. Corner, and the Hagans countered with a request to use the Park Department dock on Union Bay for unloading sidewalk-construction materials[10]--part of utilities construction--the park superintendent refused permission because of the unpaid claim. Legal complications lasted into the 1920s, when Hagan and Corner lost title to some of the property in order to satisfy the debt.[11]



1912 map of Montlake Park Addition showing area of East Montlake Park that was then submerged

Another facet of readying Montlake Boulevard for use was construction in 1916 of a timber bridge across the old canal site[12]--later replaced with fill and even later breached for a freeway. The contract for another timber bridge--the first Montlake Bridge--was let in April, 1909 (just two months before A-Y-P opened[13]) over the on-going dig, then called the Erickson Cut (later Montlake Cut). The cost, $7004.58, was borne by the Park Board, Seattle Electric Co. (i.e., the street railway system), and the city's general fund. That bridge must have accommodated only street cars, however, because in March, 1910, Hagan & Hagan requested the Park Board to appropriate $238.50 for building a suspension bridge over the canal-to-be.[14] The total cost of that bridge was to be $1090, with remaining funds to come from the city's general fund and from owners of adjoining property. The combination of streetcar and pedestrian bridges was evidently unsatisfactory since just a few years later a clamor arose for replacing both with the present Montlake Bridge.

Don Sherwood, the most prolific historian of Seattle's parks, has written that development of West Montlake Park was closely tied to the desires of the Seattle Yacht Club, which a few years later had bought the former "Casino Grounds" (probably not intended for gambling) of A-Y-P for their building. He stated that, "[s]ince most of the Park area was in the water, the State deeded that portion to the city in 1909. . . . A wooden bulkhead was built out in the water on the Pierhead Line and filling began behind it, using cinders [probably residue from coal and wood fuel] from various public buildings."[15] Over the next 10 or 15 years, "the fill material had changed to refuse [Montlake's first city dump?] and there were objections to 'the stench' created." Furthermore, in 1918 construction of a series of moorage piers abutting the park bulkhead, including a boathouse, caused neighbors to object to blockage of their view. The boathouse was moved to the south pier, and 13 poplar trees were planted along the shoreline, giving the area a more parklike feel. In 1932, the Parks Department constructed a concrete seawall and completed the fill,[16] presumably with something other than garbage.

In contrast with the park on the west, East Montlake Park simply emerged--its existence dependent on the lowering of the water level of Lake Washington. Baist's map of 1912 (five years before the lowering; see p. 115) shows its supposed rectangular shape. When the canal finally opened in 1917, the park land became an object of contention between the city and the federal government because of its location on or near the government canal reserve. The city wanted to use the newly dried-out land as an extension of Washington Park[17]; the government asserted that it must be a separate park with no permanent structures. But the area in the 1920s must have looked somewhat unpromising as a park because of its boggy, cattail-ridden quality, not to mention debris from the time when logs had been assembled there for floating through the old canal. That very quality of wildness was to become, in later years, its most desirable aspect as a takeoff point for a waterfront trail, with rustic log walkways covering the bogginess and making it possible to walk comfortably through the cattails and other plants.

By 1931, though, its future was problematic. A Park Department plan for the next ten years, a depression period, assessed East Montlake Park as unimproved. It had been recommended by anonymous sources as "a neighborhood bathing place,"[18] but the board concluded that "[i]ts improvement should await the development of . . . [the] adjoining lands." In contrast, the same report noted that West Montlake Park "is fully improved with lawn and flowers" and that, because railway tracks still ran through the centerstrip of Montlake Boulevard, that area "will not permit of any further improvement beyond the lawn and trees which are now maintained there."

Though the 1.1-acre East Montlake Park had been deeded to the city in 1946,[19] it became a focal point for visitors in the early 1950s, when an immediately adjacent part of the old canal right-of-way became the home of the Museum of History and Industry, which includes a maritime collection. From its earliest days, the museum included a home for the Puget Sound Maritime Historical Society, one of whose heroes was Horace W. McCurdy.[20] A past commodore of the nearby Seattle Yacht Club, he had been head of Puget Sound Bridge and Dredging Company since 1922 and had made himself beloved of people who were

Seattle Municipal Archives, Don Sherwood History Collection, photo by Olmsted Brothers, 1903

1903 photo of area that was to become East Montlake Park at east end of Montlake Park Addition

thus allowing for the later creation of the Broadmoor golf course, which was not only a nice amenity for residents but also protected them from the intrusion of whatever activities--several of a decidedly lower class type--might occur in Washington Park.

With this entirely untamed chunk of prime property in hand, the Park Commissioners felt pressure to start planning exactly what to do with it--hence their decision to hire the Olmsted Brothers to create a comprehensive park and parkways plan that would capitalize on the "existing long, narrow Washington Park."[62] Olmsted's 1903 report, adopted by the Park Board, said, tactfully but forthrightly, that the board hadn't acquired enough land; the potential park needed to be enlarged by widening on the west side and by acquiring frontage on Union Bay west of the park, all of Foster Island, and a narrow piece through Union City that would connect Washington Park with the university grounds. All of these acquisitions, he pointed out, would bring plentiful returns to the city because of "the increased valuation and taxes . . . from the adjoining private lands," on both east and west sides. He also wanted the borders of Washington Park to be "curvilinear" so that "a graceful border street or a parkway" could more or less follow the western boundary--what later became Lake Washington Boulevard. Slashing and clearing for this parkway began right after Olmsted submitted his report, with an amazing $200,000 appropriation. A revised plan for this road by Olmsted became the basis for further work "in conformity with the contour of the ground," and by June, 1905, the mile-long, metalled roadway was "thrown open to the public."

Olmsted was struck by what remained of native forest ("only in places are there groups of very large firs and cedars") and the brook, "derived mainly from springs," that he hoped could be kept in very good condition by augmenting its sources. Most of the land needed to be partially or wholly cleared, he suggested, "and the surface covered with grass . . . so as to adapt it for use by large crowds." Though not much money was available to develop the park and no thought had apparently been given to developing it as an arboretum,* by 1907 the road designated by Olmsted had been macadamized from Madison Street to the fork with Interlaken Boulevard and graveled to Union Bay in a "very satisfactory" way,[63] according to the Park Commissioners. Through private contributions from horse owners, a 3/4-mile speedway was also under construction at the north end; other parts of the driveway through the park had become "more and more popular for automobiles, carriages, horse men, and pedestrians," so popular that the Park Board supplied a horse and the police department a patrolman. Clearing continued, much of it done by the otherwise unemployed of Seattle[64] (a practice that continued during the 1920s and peaked in the 1930s with the federal Civil Works Administration and the later Works Progress Administration[65]), and the park became headquarters for maintenance of the boulevard system, requiring a barn for steamrollers and other tools and a stable for eight horses.

More ominously, the board's 1907 report notes that a "garbage crematory" would most likely be constructed within Washington Park "on account of the scarcity of sites available."[66] Later records of the use of two areas of Washington Park for garbage disposal refer to them as "sanitary fills,"[67] with no reference to burning that the word *crematory* suggests. At the north end, the fill extended from 26th Avenue and East Miller, immediately adjacent to Montlake, to the marshes of Union Bay. (In the 1970s, this dump was discovered by neighborhood kids and adults and for a few months was both an exciting place to explore and the source of saleable old bottles and other memorabilia.[68]) At the south end, just north of Madison Street, "a huge fill had been placed across the ravine" by 1909; that fill presently serves as a soccer field.

While garbage was going in at the south end, the other end of Washington Park was being prepared as a

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*The idea for an arboretum in Washington Park began to develop in the early 1920s, when the University of Washington proposed that all of the park should be given to the university for use as an arboretum. The Board of Park Commissioners accepted this proposal in 1924, but lack of funding prevented the transfer from happening. [Guide to papers on University of Washington Arboretum, "History," <www.lib.washington.edu/specialcoll/manuscripts. arbor. html>]

suitable entrance to the Alaska-Yukon-Pacific Exposition, though it had to pass over declivities that would make travel awkward. The commissioners, working hard to do their part in assuring a successful exposition, reported in 1908, about a year before the June, 1909 opening, that the road was being hurried:

> It has been necessary to cross some of this low ground on bridges supported by piling and these are in place, and the grading of the balance is well under way. The road branches off Washington Park Roadway about one-eighth of a mile from the end at Union through the center of the recently platted Montlake Addition to the Plaza, the south entrance of the A.Y.P. Exposition.[69*]

Whatever increase in development momentum occurred because of A-Y-P had slowed by 1931, though land acquisitions had increased the size of the park to 197.05 acres.[70] A large part remained undeveloped--"in much the same condition as when acquired, a tract of logged-off land covered with second growth timber and brush." Furthermore, the equipment barn was "an eyesore" and must be replaced. Montlake residents were less concerned about that than about the need for a children's play area near the intersection of 26th Avenue N. and E. Lynn--the site of the present Tot Lot. By 1931, Park Commissioners had confirmed that "the area is now used rather extensively by the children of the neighborhood. It is recommended [to Mayor Frank Edwards] that a children's playground be developed there."

Even more noteworthy is the recommendation in this report that at least 85% of the park "be set aside for use as an arboretum." Ideas for development had been actively circulating in the city since at least 1924. In that year the Park Commissioners established "a botanical garden of trees, shrubs and flowers . . . in the north end of Washington Park"[71] in cooperation with the Chamber of Commerce and U.W. faculty in botany and forestry. The Chamber saw this project as a way to attract worldwide attention to Seattle, perhaps rivaling botanical gardens in St. Louis, Boston, and London.[72] Additionally, U.W. faculty needed a place for scientific plant study, as Edmond Meany had suggested when he promoted the university's relocation from downtown; their first garden, near the present Drumheller fountain on the campus, had been destroyed in 1909 to make way for A-Y-P.[73**]

Seattle's mayor in 1927, Bertha K. Landes, lent her support to the arboretum idea when she declared Washington Park "an ideal site for an arboretum, otherwise known as a botanical garden."[74] She also referred to "some talk" of a university-city joint effort to combine park and arboretum. This talk had a solid basis of planning at high levels within the university, city, and state, extending over about a decade. Tracing their inspiration to Professor Edmond Meany, they incorporated the Arboretum and Botanical Society of Washington with the aim of creating "gardens which will be second to none in the world."[75] With much of the enthusiasm coming from a sub-committee of the Seattle Chamber of Commerce--who knew a potential revenue-enhancing tourist attraction when they saw it--these gardens were to be kept open to the public, subject only to conditions necessary to caring for and preserving the plant collections.

According to *The Seattle Times*, "the first public meeting [April, 1930] at which the plans of the recently organized Arboretum and Botanical Society of Washington"[76] were announced occurred before the Montlake-Interlaken Community Club. Speakers included Hugo Winkenwerder, Dean of the U.W. College of Forestry, and R.J. Fisher of the Seattle

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*The building of an aqueduct bridge across Lake Washington Boulevard at E. Lynn Street probably occurred in this same decade. Called the Arboretum Aqueduct, it was constructed to support the North Trunk Sewer [City Engineer's Plan 782-5]; it is now a pedestrian overpass. Because of its architectural/engineering distinction and the influence of its designer, in 1976 it was officially designated as a Seattle Landmark. [Seattle City Ordinance 106070, December 13, 1976]

**Meany is reported to have said in 1895, "I want this land [immediately north of Union City and its first addition] for the University because we want an Arboretum." No one disagreed because few people knew what an arboretum is. To Meany's disappointment, the arboretum idea became "lost in the shuffle" as the university developed in its first few years. ["Hearing All About," *The Seattle Times*, June 13, 1935]

Bill Mundy, Chairman
Canterbury Shores SR520 Committee
And
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July 11, 2011

SR520
Final Environmental Impact Statement Administrator
Washington State Department of Transportation
Olympia, WA.

Dear Administrator:

The following are several comments regarding the adequacy of the SR520 FEIS. These are matters that were either not dealt with, such as navigation, or were indirectly and/or inadequately addressed.

- South Lake Union Navigation. Because of the planned construction zone south the of the existing bridge it will not be possible to get sailboats and large power boats to and from the North Madison Park (NMP) area year-around. Also, it will not be possible to get power boats or shallow draft vessels in or out during the summer due to the milfoil and lilies that grow along the shoreline.
- Dust and particulate matter and air pollution during construction and permanent. This was inadequately addressed. Your data uses averages. During the summer dust/particulate matter is a severe problem in northeast Madison Park with the wind blows out of the north.
- Vibration. Inadequately addressed. Best management practices (BMP) will not suffice. During the driving and extracting of piles damage occurs immediately, not incrementally over time. When the threshold is reached that causes mortar and wallboard to crack there is not time to deal with BMP. Either pile driving/extraction should not be allowed or a specific written agreement between WSDOT and NMP property owners prior to construction specifying how damage

will be quantified and mitigated. This deals with BOTH vibration and noise.

- Transition from old to new bridge. This deals with the fact that the entire project will not be built as one, but will be segmented in two, the second or final segment being the Seattle Segment, from the West Highrise to I-5. There are several problems with this segmentation approach that were not addressed in the SDEIS or FEIS.
    - First, the stated purpose for rebuilding SR520 was for safety reasons, the possibility a floating segment might sink during a storm and the deteriorated poor quality columns the west segment (west highrise to Foster Island and Montlake to I-5) that are not seismically sound and are likely to collapse if there is an earthquake. The earthquake prone segment of 520, that is from the West Highrise to I-5, will not be repaired. Segments that are perfectly sound are being rebuilt instead.
    - Second, the "transition bridge" that will funnel west-bound traffic from the new lanes to the old lanes just east of the West Highrise. We don't know what this is or where it will be. We don't know what kind of traffic effects this will have as three westbound lanes are transitioned (via some kind of an "S" curve) and squeezed into two lanes. Traffic effects include:
        - Congestion;
        - Air pollution from idling vehicles;
        - Increased particulate matter, especially from truck exhaust;
        - Accidents due to slowing and/or stopped traffic and the "S" curve.
    - Construction of the "transition" bridge. Nothing was discussed about how long this will take, what the cost will be, what the impact on the environment will be during construction, how it will affect navigation or how it will affect traffic on the existing bridge.

Respectfully submitted,

Bill Mundy, Ph.D.

Coalition for a Sustainable SR 520

Madison Park.. North Capitol Hill.. Montlake.. Laurelhurst..Roanoke Park/Portage Bay. .Boating community
2636 10th Ave East, Seattle WA 98102, fran@roanokecap.com

Ms. Gloria Shepherd
FHWA Associate Administrator
Office of Planning, Environment & Realty
1200 New Jersey Ave., SE
Washington, DC 20590

June 20, 2011

Re: Washington State Department of Transportation State Route 520 Project

Dear Ms. Shepherd,

We represent the Coalition for a Sustainable SR 520, the voice for thousands of Seattle residents and property owners who will be impacted by the above referenced project. We write at this time to correct some important errors and mis-leading Statements in the recently published FEIS. Please investigate these issues yourself so that your ultimate decision on this project is based on an adequate and accurate record.

WSDOT is expecting you to issue a Record of Decision for this project on or soon after July 11, 2011. Part of the *Final Environmental Impact Statement* was published on June 10, 2011, with the rest published on Friday the 17th. The three prequalified contractors submitted bids on June 8, 2011.

Due to the incomplete, misleading and erroneous information in the FEIS, the time is far from ripe for a ROD on this project. We ask you, as the federal agency with primary oversight, to take a hard look at the facts and defer issuance of a ROD until several serious problems are remedied. Chief among them:

1. The *FEIS* glosses over the reality of the plan WSDOT seems most likely to implement (because of financial constraints): to build a bridge part way across Lake Washington and then stop, out of funds for many years, maybe forever. This partial bridge would not improve mobility and would leave severe safety issues on the west (Seattle) side of Lake Washington unaddressed. It makes no sense to spend billions on the partial bridge unless there are assurances that funding can be obtained for the rest of the bridge—but those assurances are lacking.

The state plans a "partial bridge" (in blue): the red section may never be built.

I____I-5 to Medina project_____ I_____east side project, already underway_____I
I_partial bridgeI
I__west side _____I

The FEIS does discuss this "partial bridge" as a "Construction Phase One" phasing *(FEIS 5.15*), but gives only cursory mention of the very real probability that it will be a very long term or permanent condition. The FEIS does only a perfunctory analysis of impacts, but even this would convince any disinterested party that it makes no sense to expand the bridge to 6 lanes part way across the lake, then have traffic merge into the existing 4 lanes, while leaving the safety problems on the west side unaddressed. In fact, the analysis indicates that spending $1.7 billion for a partial bridge does not improve round trip travel times over the "no build" option, either for HOV or for general commuters (*FEIS, pages 5.15-5 and 5.15-3)*.

The State admits to being short the $2 billion necessary to build the western part of the I-5 to Medina project, with no foreseeable way of covering the enormous shortfall. $2 billion is a huge sum relative to the State's highway improvement program, which for the 2011-2013 biennium has $1.5 billion for <u>all</u> projects other than the two megaprojects, SR 520 and SR 99. The $ 2 billion shortfall also represents 50% of the total $ 4.0 budget for the 520 project from I-5 to Medina.

A long-term or permanent partial bridge is very different from the alternatives studied in the 2010 SDEIS. Consequently, the public has had no opportunity to consider and comment on this partial bridge plan, and no opportunity to compare it with reasonable alternatives … particularly to compare it to an alternative of fixing all the safety problems rather than expanding the number of lanes on part of 520.

A ROD should not be issued for the partial bridge until its impacts as a stand-alone project have been adequately and fairly analyzed and until the public has had an opportunity to comment.

2. The applicable Metropolitan Transportation Planning Organization, the Puget Sound Regional Council (PSRC , has approved the "Partial Bridge" for construction (subject only to release of the FEIS). However, the PSRC has not approved the full I-5 to Medina project and cannot do so, since the funding is not available. Because the PSRC has not approved the whole I-5 to Medina project, a ROD for this project cannot be issued lawfully.

3. The PSRC approved the partial bridge based on the State's assertion that it has funding for this part of the project. In reality, funding has been authorized for the partial bridge, but it is unlikely to materialize. Funding for even a partial bridge is dependent on toll revenue bonds and on bonds backed by anticipated future federal highway funds (see Exhibit A), but both of those funding sources are highly problematic.

    i. Bonds on 520 tolls: A Statewide voter initiative (No. 1125) is headed toward the ballot in November. This initiative, which would restrict the type and use of tolls, has recently received funding of $500,000, and many of its sponsor's previous initiatives have passed. It is highly unlikely that the State can float bonds based on tolls until this issue is settled. This source for acquiring $800 million is not realistic in the short term, and may not be in the long term.

ii. Bonds on future grants of federal highway funds: Because of the uncertainty of the amounts and timing on grants of future federal highway funds, it is highly unlikely that the State can float large amounts of bonds backed by anticipated future grants. This source for acquiring another $800 million is not realistic in the short term, and may not be in the long term.

We would be pleased to supply you with much more detail on the financial situation.

Because even the partial bridge project does not have funding, a ROD should not be issued for even that limited section.

4. You should be skeptical about WSDOT's preference for its preferred alternative. WSDOT committed itself to this alternative long before it completed the FEIS. As you know, agencies are required to keep their minds open until they get the environmental analysis in the FEIS. But WSDOT has demonstrated over and over again that it made its mind up long before the FEIS was done. Now, WSDOT is in the uncomfortable position of having devoted tens of millions of dollars to its preferred alternative. Do you think that agency, with that much on the line, is able to make a reasoned decision among alternatives? Do you think it is even capable of even preparing an objective FEIS? Of course not. The FEIS is terribly biased in key sections as its authors strive to justify a decision already made. Your agency, free of these constraints, should insist on a new, objective evaluation and a new decision after that truly objective analysis is completed.[1]

We are aware the State will say this project has been studied for 14 years, with much community input. The 14 years is true, and many of us have participated for that long in good faith efforts to get a good solution. However, we have observed that the community input is taken only with respect to small tweaks of the State's plan. Questions on major issues like those described above are routinely ignored.

We believe that the Washington State officials are actively denying and ignoring the effects of their preferred alternative, in order to ram it through, irrespective of its impacts. They chose their plan long before environmental review was complete; they refuse to look at options which might well have better results and cause less damage; they avoid acknowledging, and thus having to mitigate, damage that will clearly result; and they generally elevate form over substance in complying with environmental laws. They are rushing to implement their plan before objections gain traction.

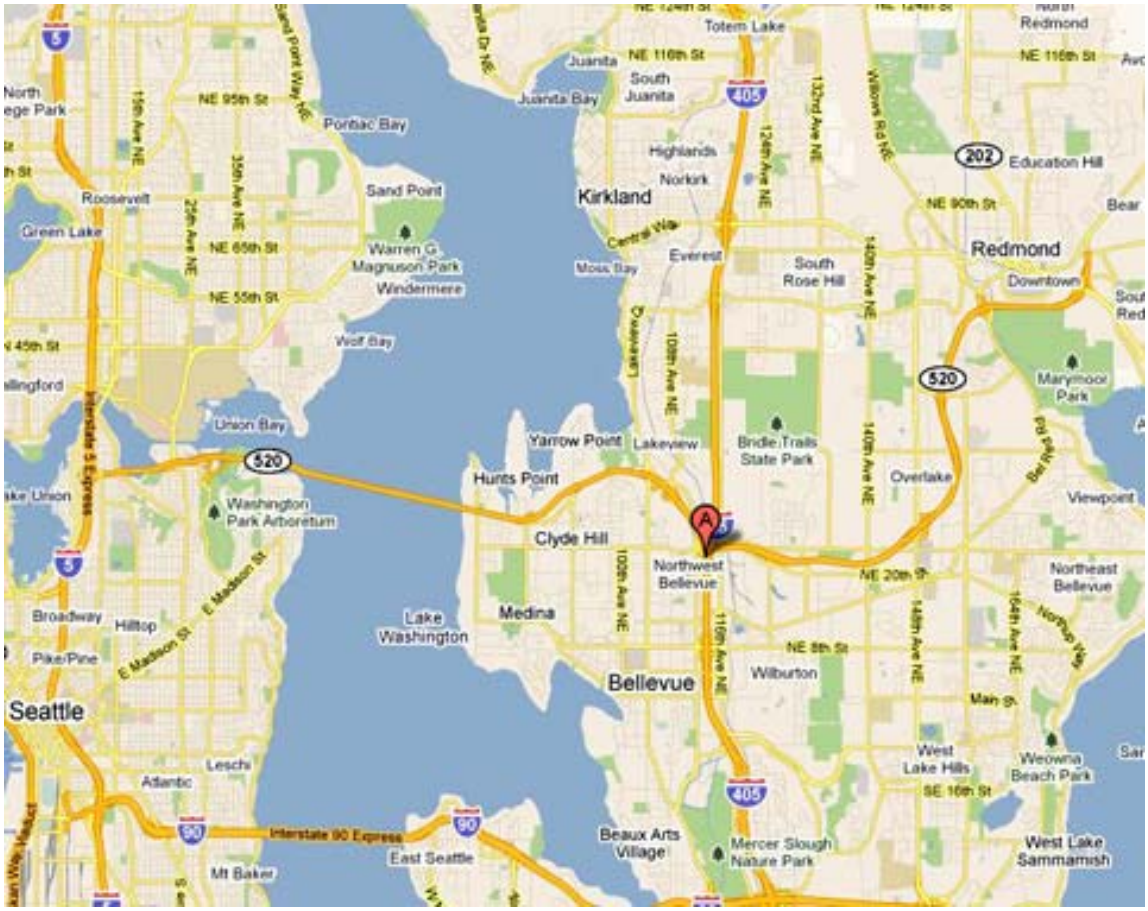
What has happened is bullying and whitewashing, not analysis. We ask you not to issue a ROD until these issues are resolved.

[1] WSDOT's SR 520 project is three miles from downtown, the site of WSDOT's State Route 99 tunnel project. It probably is not a coincidence that WSDOT is using the same approach on both projects: making its mind up first; committing huge sums to its pre-ordained choice; and then struggling to create an EIS that justifies the decision already made, instead of creating an objective EIS to be used in making a valid decision in the first instance.

BACKGROUND

The Coalition for a Sustainable SR 520 represents the communities adjacent to the highway. We do not object to expanding SR 520, if it is done in a way that will enhance long-term mobility and minimize damage to the natural and human environments. The Coalition has strong objections to the State's current preferred alternative plan.

The highway SR 520 is primarily a commuter highway across Lake Washington, connecting with the major north-south interstates: I-5 in Seattle and with I-405 on the east side of the lake.



The 520 expansion project was initially a single project, but WSDOT broke it into 3 segments. Two of these are already underway, in spite of their interdependence with the third segment. This remaining segment, the western portion of the project, the "I-5 to Medina" segment, stretches from SR 520's termination at I-5 to the eastern shore of Lake Washington. An *SDEIS* for this segment was published in January 2010.

OTHER ISSUES

While presenting thousands of details, the FEIS manages to ignore the big realities.

A) On the densely-settled Seattle side, the Preferred Alternative is much wider than 6 lanes.

The State's currently preferred alternative talks of a seemingly modest expansion of SR 520 in Seattle from four lanes to six (if funding were available). But the planned expansion is really much more than that. Most of the highway is now about 60 feet wide, 4 lanes plus on and off ramps. The Preferred Alternative expands the highway on the Seattle side to as much as 262 feet wide: 6 lanes, but also wider shoulders plus two sets of on and off ramps (HOV and general purpose).

The proposal would force this massively enlarged freeway through densely settled neighborhoods, wetlands, bays, and recreational open space. The western end of the SR 520 project runs directly through the heart of Seattle's Montlake, Portage Bay, Roanoke Park and Capitol Hill communities, and passes between the communities of Laurelhurst and Madison Park. Thrusting a major highway near surface level through an area like this is a 1950's solution, not a 21st century solution.

Roanoke area: FEIS 2-40

Montlake area: FEIS 2-47

East of the Montlake interchange, the highway will be 160 to 262 feet wide! Few citizens seem aware of this vast expansion of the footprint. The FEIS certainly does a poor job of communicating this information. Most people still think the planned expansion is simply adding a single travel lane in each direction.

B) On the west side, most of the area to be used for expanding 520 will be taken from wetlands, open space, parks, and properties which should be classified 4(f).

WSDOT's routing was chosen in part because it was cheaper and easier than others; that is, because it is mostly open space rather than housing. Congress passed 4 (f) precisely to halt agencies' proclivity to make the "easy" choice of converting park land to highway use. But that is exactly what WSDOT has done. Having chosen this route, the FEIS concludes that there is no way to avoid taking 4(f) and other protected properties! The preferred alternative would forever change the whole area, which is oriented toward the water and open spaces. Contrary to Statements in the FEIS, the bays and wetlands are heavily used for swimming, canoeing, kayaking, other small boats, bird watching, wildlife viewing, etc.

The planned expansion is more than twice as big as today's 520, and almost all of the land to accomodate it will come from parks, wetlands, open space, and open water used by wildlife and for recreation.

We will write more on this subject when we have had a chance to fully digest the FEIS, but we want to bring it to your attention now, since the FEIS does not make it clear. We strongly believe that alternatives are available which would do less damage to the environment.

C) The preferred alternative does not address the earthquake safety problems.

The project was sold to the public primarily as a safety fix. However, the State's current plans make it clear that highway expansion, not safety, is the goal:

- WSDOT worked on the non-safety parts of the project first. WSDOT has already hired contractors for the project east of Lake Washington, which has no safety issues, and work there is underway. Meanwhile, the safety issues in Seattle take a back seat.

- While WSDOT devotes money to non-safety portions east of Lake Washington, it says it has no money to fix the worst safety problems in Seattle. The bridge is subject to two threats: a windstorm could damage the floating portion and earthquakes could damage the western portion (which rises above Lake Washington and Portage Bay on piers). Construction of the

"partial bridge" would fix safety problems of potential windstorms on the floating portion of the bridge. But it will leave the hollow piers that support the west side of 520 today vulnerable to earthquake. It essential to fix the safety problems on the whole length of I-5 to Medina now, instead of expanding one portion of the project to 6 lanes and leaving the other portion with its safety problems untouched.

- The current plan for the I-5 to Medina segment is to expand four lanes to six on the floating (east) part of the bridge and then await further funding for an indefinite period before re-building and expanding the pier-supported western section. Thus, for many years, the worst safety problems in Seattle will remain unabated—while WSDOT completes non-safety improvements on the eastside of the lake and expands to 6 lanes over part of the lake.

D) The preferred alternative reduces capacity on I-5, the north-south interstate through Seattle

The preferred alternative has only one additional connector to I-5, connecting to the reversible express lanes which travel south in the morning and north in the afternoon. There are no additional ramps or lanes connecting with I-5 in three of the 4 directions;[2] consequently, additional vehicles from the expanded 520 will have to squeeze onto existing ramps going in these three directions.

The one new connecting ramp will actually land on an existing I-5 express lane, thus changing its use from north-south commuters and freight to east-west commuters. Taking a lane for 520 commuters means that only 3 lanes, instead of today's 4 lanes, are available for north-south traffic. (Exhibit B) This configuration also causes several new weaves, which by themselves will slow I-5.

Reducing capacity on I-5 will certainly cause increased congestion and delay on the express lanes, spreading to the main lanes. Although the FEIS claims that I-5 has enough capacity for this because other I-5 chokepoints will not be remedied, (*FEIS 5.1-23),* common sense tells us that an interstate highway which is already congested during much of the day, in a region which is projected to grow rapidly, should not have capacity reduced. At the very least, it seems imprudent to remove capacity from the main interstate in an area where geography prevents future expansion:

please see image on next page.

[2] There are no new ramps connecting I-5 southbound with 520; connecting 520 with I-5 northbound; or connecting I-5 and 520 into\out of downtown Seattle opposite the reversible lane connection.

Image from Wikipedia, with our annotations. Bottom of page is north; downtown is to the south.

Until we discovered this issue a few months ago, in its numerous public meetings and publications WSDOT did not mention reducing I-5 express lanes from 4 to 3 near SR 520. A diagram like the one on Exhibit B attached is essential to understand what is going on, yet we have not found one in the FEIS. The Executive Summary of the FEIS says only "Add an HOV connection to the I-5 express lanes that would operate westbound to southbound in the morning and northbound to eastbound in the afternoon." (p.30. There are two similar references, on pages 26 and 28.)

In the FEIS itself, one has to go page 108 of the Transportation Discipline Report in Attachment 7, before finding a clear articulation of the plan: "The Preferred Alternative would reduce the number of lanes from four to three in the Express Lanes across the Ship Canal Bridge to provide space for a single new HOV/transit ramp to and from SR 520…."

Consequently, there has been no public discussion of the important tradeoffs involved, or about the wisdom of taking a lane away from I-5, or of creating new merges and weaves. Instead, there is a lively civic discussion on the need to relieve I-5 congestion by removing bottlenecks to increase its throughput. This is yet another example of WSDOT trying to hide the ball and use the EIS, not to inform a decision to be made, but to justify a decision already made.

Better alternatives are available.

The State has failed to use the environmental process to evaluate realistic alternatives which might produce better mobility with less environmental damage.

- The *EIS* for this project considered alternatives only in conjunction with a set of assumptions which doomed them: *e.g.*, comparing the six lanes with tolls to a "no-build" base case without tolls. The process also eliminated some promising alternatives on the basis of preliminary and biased cost estimates. Again, because WSDOT had its mind made up and was spending many

millions on its pre-determined choice, it had no interest in presenting a fair analysis of reasonable alternatives in the EIS.

- In the EIS, the "no build" base case was invalid—to do nothing to 520. We all agree that safety improvements and small fixes are necessary, so "do nothing" is not a valid base case. In addition, tolls are now to be put on SR 520 within a few months. To date, WSDOT has compared a "no build" scenario without tolls to its preferred alternative with tolls. The base case—or another alternative--should include safety fixes, minor improvements, and the same assumption on tolls that is used for all alternatives. (The FEIS does include a 'No Build Toll Sensitivity Analysis" in Attachment 19 to the FEIS, but the base case is still invalid.)

- There has been a failure to review other alternatives once the environmental impacts of WSDOT's preferred alternative were understood. The process eliminated some alternatives, like an improved four lanes, before the huge environmental impacts of the State's preferred alternative were known. Now that those impacts are more apparent, tradeoffs should be considered.

In the light of the financial shortfall, the *FEIS* should consider:

- Make fixing the safety problems the first priority, or simply reconstruct a four lane highway from I-5 to Medina. Then when funding is available in the future, implement a better plan for expansion.

- Use congestion pricing tolls plus improvements in the four lanes to increase throughput.

If and when it is appropriate to evaluate alternatives assuming that financing will be available, the following should be included:

- Using lanes 5 and 6 for transit only, which could eliminate much of the environmental damage (by reducing the need for extra on and off ramps) and improve mobility by eliminating the congestion which will be created on the Seattle side;

- Using immersed tube tunnels which have been successfully used in several parts of the world at reasonable costs to reduce environmental consequences while improving mobility ;

- Keeping the segment from the Montlake interchange to I-5 at its current four lanes, which the independent consultant Nelson Nygaard has already indicated is feasible . This would avoid much of the environmental damage.[3]

[3] This is different from WSDOT's "partial bridge" alternative in that it extends the six lanes all the way across the lake to the Montlake interchange (where many vehicles will exit). WSDOT's partial bridge plan, in contrast, terminates the six lane segment before reaching the interchange, forcing a huge backup as six lanes narrow to four.

ABUSE OF PROCESS

WSDOT, the governor, and certain legislators chose an alternative long before the environmental review was done and have not been open to other alternatives which might provide better mobility with less damage to the environment.

NEPA creates a process intended to ensure that environmental information is first obtained and then used to make informed decisions. But time and again, WSDOT has demonstrated that it has made its mind up in advance and is going through the NEPA process as a bureaucratic formality, creating analyses to justify decisions already made.

Our SDEIS comments in April 2010 lay out indicators of WSDOT's bad faith in the process to that date. Since then, the same patterns of behavior have become more intense:

1) In February, 2010, shortly after publication of the SEIS, the State speaker of the house, the mayor of Seattle, other politicians and various groups joined us in saying that alternatives like using lanes 5 and 6 for transit only must be considered. (They are not considered in the *SDEIS*.)

   In respon*se,* the governor said looking at changes in configuration would set back the project, and "our commitment to ensuring public safety does not allow that kind of delay". (Never mind that the governor's proposal would leave in place for a decade or longer the hollow, earthquake-prone pillars supporting 520 west of the floating bridge.) Likewise, a leading State representative said, "We have an agreement, let's move forward." Proponents of the State's plan held a press conference at which the House Transportation Chairwoman, Judy Clibborn, D-Mercer Island, reiterated the argument that a redesign of the car-pool lanes would delay the project up to two years. All this before comments on the *SDEIS* were even submitted and before a preferred alternative was officially chosen.

2) The preferred alternative was officially announced at the end of April 2010, less than two weeks after the *SDEIS* comment period and long before anyone could have read and absorbed the hundreds of submitted comments. The choice had obviously been made long before.

3) Long before the Final SEIS was released, the governor, WSDOT, and State legislators presented the preferred alternative as a final decision, awaiting only paperwork details to be implemented. "We have a new 520 and are ready to move forward to open the bridge in 2014," Gov. Chris Gregoire said.

4) In op-ed pieces, media interviews, and ads, the governor, WSDOT and State legislators, together with some business interests, pushed hard for people to stop questioning the plans., "It's time for action on the 520 bridge!" This pressure strengthened the perception that the decision had been made and that opposing it was dangerous. It trivialized the environmental process mandated by NEPA (and its State counterpart, SEPA).

5) The governor set a tight timetable for construction that would not permit any further discussion or consideration of alternatives that were, or should be, analyzed in the EIS.

a. The Section 106 process for analyzing and mitigating impacts on historic areas was driven by the deadlines. Legitimate requests for additional assessments were brushed aside in the name of meeting deadlines.

b. The Metroplitan Transportation Planning Organization (the Puget Sound Regional Council) was asked last month to approve and did approve going ahead with construction of the "partial bridge" *before* the FEIS was published, because the State said it needed to move fast with construction of its preferred alternative.

c. WSDOT seeks to have a ROD in July, just a month after the *FEIS* is released and long before you can reasonably be expected to consider all the information contained in the EIS and listen to those who can show that its analysis is biased, incomplete and inaccurate.

IN SUMMARY

As the State does not have and cannot get funds *t*o actually build what it describes in the *FEIS* anytime soon, we believe WSDOT's only real purpose in getting a ROD issued so soon is to get its preferred alternative set and started before its problems become widely known—to build bureaucratic momentum for an alternative in which it has prematurely invested.

To champion this design, chosen long before its impacts were analyzed, the State has refused to acknowledge those impacts or its lack of funding. It has refused to give serious consideration to alternatives or provide good mitigation. This is a travesty of the environmental process. We ask you to step in and ensure that the process meets the intent of the law. A far better project is still possible and will result if you provide the hard look required by law.

We would like to talk with you, and will be happy to provide more backup of the statements here, or additional information.

Signatures on next page …

Coordinator for the coalition

Fran Conley

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Roanoke Park/ Portage Bay Community Council

Ted Lane

Roanoke Park/ Portage Bay Community Council

Anne Preston

Montlake Community Council

Jonathan Dubman

Montlake Community Council

Anita Bowers

Laurelhurst Community Club

Colleen McAleer

Laurelhurst Community Club

Jean Amick

North Capitol Hill Neighborhood Association

Nancy Brainard

Boating Community

Gary Stone

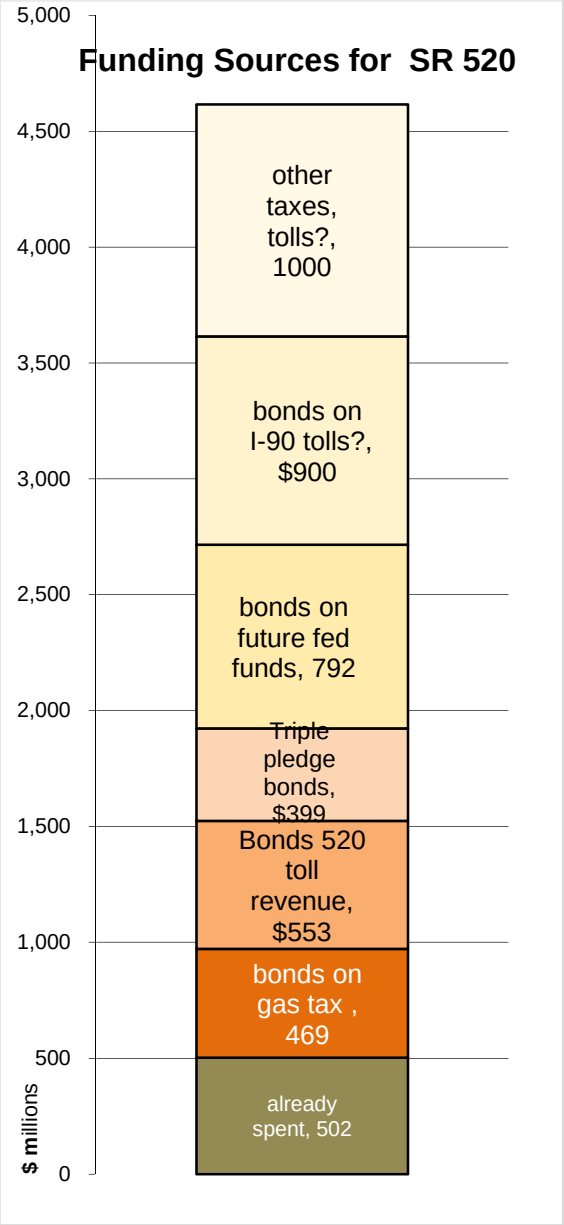
Madison Park Community Council

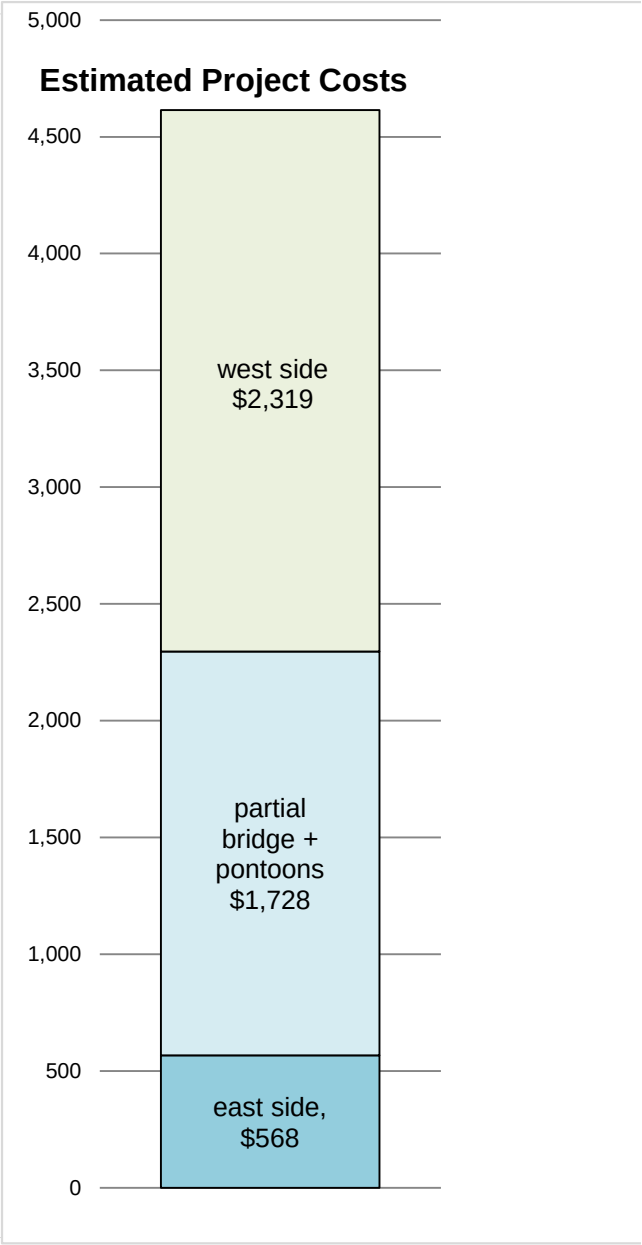
Maurice Cooper

Madison Park Community Council

Gail Irving

Exhibit A





SOURCES OF FUNDS:	SR 520 million $
already spent	$ 502
bonds on gas tax	$ 469
Bonds on 520 toll revenue	$ 553
Triple pledge bonds	$ 399
bonds on future fed funds	$ 792
bonds on I-90 tolls?	$ 900
other taxes, tolls?	$ 1,000
Total construction cost	**$ 4,615**

PROJECT COSTS	SR 520 million $
east side	$ 568
partial bridge,pontoons	$ 1,728
west side	$ 2,319
Total	$ 4,615

draft 4/4/11

source: WSDOT 11/10 Program Comparison Chart

Souce LEAP draft 3/11 and 520 financial plan 3/11

I-90 tolls estimate from legislative workgroup materials

"Triple pledge" bonds: toll revenue, gas tax, and full faith and credit of state

Exhibit B : Impacts on I-5

Left: new ramp from 520 lands on an existing express lane.

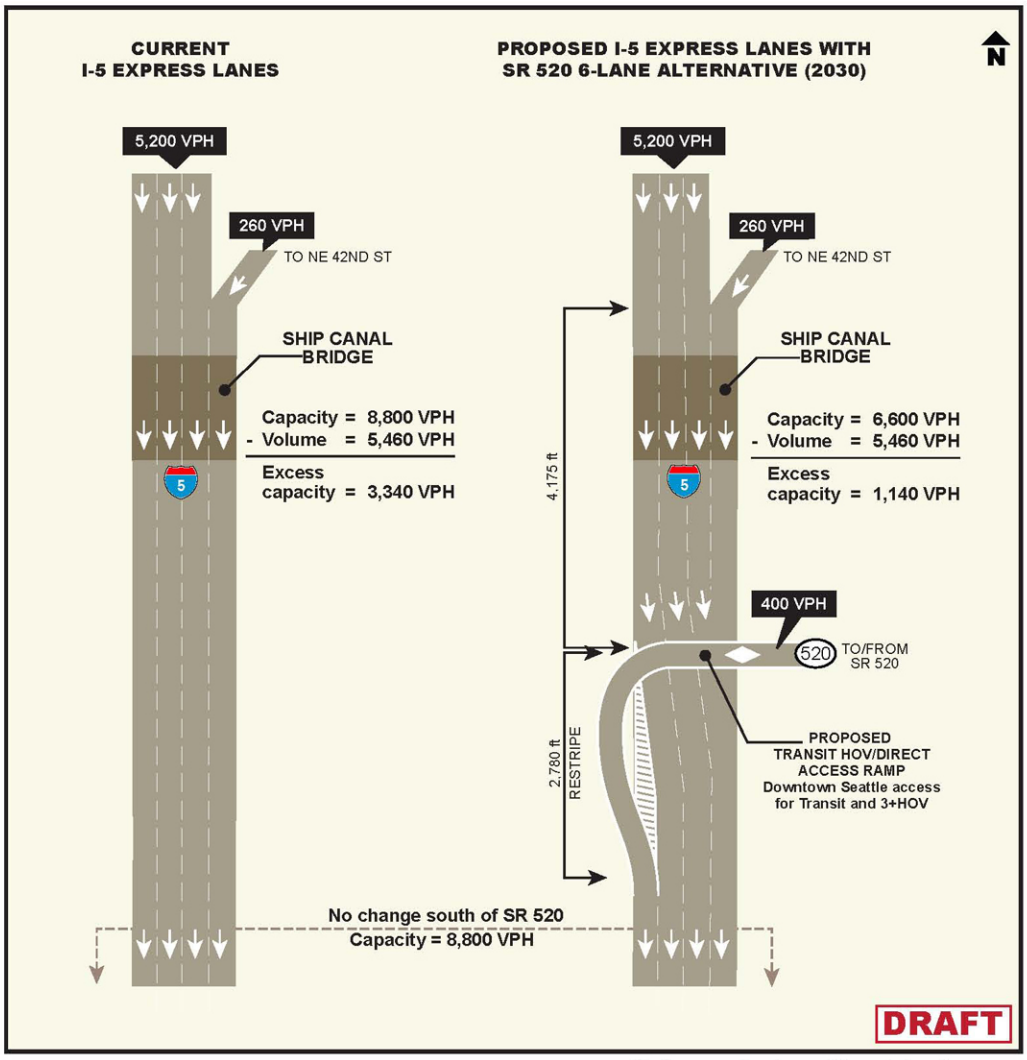
Below: near 520 interchange, today's 4 lanes for north-south travellers would become 3 lanes near the 520 intersection.

Washington State Department of Transportation

SR 520 Bridge Replacement and HOV Program

STATE ROUTE 520

Existing and future I-5 express lane operations - AM traffic



NOTE:
- For illustration purpose only.
- VPH = Vehicle per hour
- I-5 mainline configuration not affected by this change.
- Similar operation for northbound direction
- The I-5 express lanes extend from downtown Seattle to Northgate Mall at North 103rd Street.

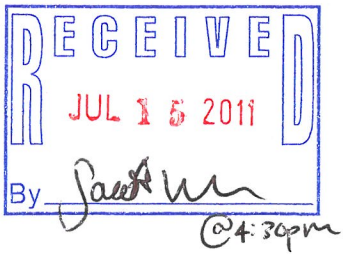
Coalition for a Sustainable SR 520

Madison Park.. North Capitol Hill.. Montlake.. Laurelhurst..Roanoke Park/Portage Bay. .Boating community
117 East Louisa St. #205 ... Seattle, WA 98102-3203

July 15, 2011

Julie Meredith
Director 520 Program
WSDOT
Seattle, WA

Gloria Shepherd
FHWA
Washington, DC



Dear decision makers at WSDOT and FHWA,

Here are the comments of our coalition on the FEIS.

1) Comment period.

The FEIS was issued on June 17, with comments due today, July 15 in order to precede the ROD, which may be issued in a few days. The FEIS has 11,000 pages, including an appendix which has 572 pages, 18 documents and no index. It is not possible for anyone to become familiar with the FEIS in one month. The ROD should be deferred to allow adequate time for the public (not to mention FHWA and WSDOT decision makers) to review the FEIS. Environmental impact statements are created not to satisfy legal obligations, but to be *used* by the agencies in the decision making process. It would be impossible for the agency decision makers to truly use this document in their decision making process if they have only 30 days to analyze and digest it.

Because of the short time period, we may not have found all the data we need from this massive document. We may supplement this letter as we review more of the FEIS.

2) New material

There are a number of surprising new plans in the document, and some of them have important impacts on the environment. The public has had no opportunity to comment. These include:

- The way the Montlake interchange works is very different from the SDEIS. The new low ramps which direct traffic on to the top of the "lid" and then across the lid to go south are an example of differences.

- FEIS 9-9: "The Portage Bay Bridge would operate 110 feet north of the current bridge". This is news, and would have considerably more severe impacts such as loss of usability of Portage Bay for recreation, and increased noise and health effects on homes nearby. In addition, it is not presented in the main document, but only in an appendix which is inconsistent with the main document.

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Ted Lane, Roanoke Park Community Council ;Anita Bowers, Jonathan Dubman, and Robert Rosencrantz, Montlake Community Council; Maurice Cooper, Madison Park Community Council; Colleen McAleer and Jean Amick, Laurelhurst Community Club; Nancy Brainard, North Capitol Hill Neighborhood Association; Gary Stone boating community.
www.sustainable520.org

- The FEIS removes the lid over I-5, which was a significant feature in all the alternatives of the SDEIS. The reason given is possible future expansion of I-5. But no such expansion is planned, and if it were, it would be material to the disclosures in the FEIS.

- This is the first time we have seen the 4(f) mitigation plan and it is completely and woefully inadequate. We have had no chance to analyze it or comment about it.

- There is new traffic analysis in a number of places in the FEIS, and we have not had a chance to put the various analyses together and analyze them.

- The idea of constructing a bridge part way across the lake, and then running out of money for an extended period, is new. Please see 12) below.

3) Assertion of funding adequacy is not factual.

The FEIS states at page 1-2 that "Full funding is reasonably anticipated to be available for completion of all phases of the project within the time period anticipated for completion of the project. " This is not factual. The state's highway improvement plan for the next 10 years includes only Construction Phase One. Likewise, the MTPO (the Puget Sound Regional Council) has only included the Construction Phase Onepiece in the MTP. To finish this project would require massive new taxes or tolls in an era when people are quite resistant to them. In addition, there are serious funding shortfalls with even Construction Phase One, discussed below, and serious funding shortfalls with another highway megaproject. One could hope that funding might be available, but one could not reasonably anticipate $2 billion more in taxes or taxes and tolls.

Please see attachments 3A, the state highway project funding plan; 3B, the 520 financial plan; and 3C; our summary of sources and uses of funds.

4) Lack of disclosure on funding uncertainties for Phase 1

Although funding for Construction Phase 1 has been authorized by the legislature, it is not in hand and may be unobtainable. State Initiative 1125, sponsored by Tim Eyman and funded with more than $1 million, will go to voters in November. It would prohibit the variable tolls which are planned for SR 520, and also prohibit use of tolls from I-90 to fund SR 520. In addition, the plan to raise money on anticipated future grants of federal highway and bridge funds is extremely shaky in the current national political environment.

5) Potential Exposure of the general fund

The FEIS neglects to disclose that if a contract is signed and the state does not have funding to complete the contract, the general fund could be exposed to large outflows. The same losses could occur if the state puts its full faith and credit behind bonds which tolls are inadequate to cover. This information would influence a reasonable decision-maker.

6) Risks to tolling revenues

The tolling revenues depicted in the FEIS are dependent on achieving the projected traffic volumes. All of WSDOT's previous projections of traffic volumes have been much higher than the subsequent reality. If tolls do not come in as planned, the general fund is at risk, see below.
Please see attachment 6A and its links, incorporated here by reference.

7) Inconsistent and unreasonable assumptions on behavior and tolls

For the "No Build" alternative, the FEIS assumes that tolls would be removed from the current bridge before 2030, even though those tolls are meant to control congestion. This is not a reasonable assumption. In addition, it makes it impossible to compare the un-tolled "No-build"alternative with all the other alternatives, which are tolled.

> In the No Build Analysis in Attachment 19, the FEIS has a few traffic numbers on a tolled no build option, but the mobility parameters addressed are different from the mobility parameters addressed for other options, making meaningful comparisons among the alternatives impossible.

The FEIS then assumes that a very high percent of drivers would turn to carpools with 3+ people. This is also an unreasonable assumption, because drivers crossing 520 are mainly commuters going to many different destinations, and because history shows in Seattle and nationwide that use of carpools has been steadily decreasing. Even programs to encourage carpooling are unlikely to achieve that much result.
Please see Attachments 7 A and 7 B.

Then the FEIS assumes that the public will allow the HOV lanes to be quick and almost empty, rather than allowing single person vehicles (SOVs) to drive in them for a fee. This is both an unreasonable assumption, and contrary to recent history in the state, where carpool lanes have been and are about to be opened to SOVs who pay a fee.
The implications of these assumptions are material. If fewer people become carpoolers than expected, or if carpool lanes become available to SOV's who pay, then the number of vehicles on SR 520 will be much higher than the FEIS shows, and the impacts on traffic, environments, and neighborhoods will be more severe.

This assumption on carpooling also drives the conclusion that fewer vehicles will use the expanded highway than would use the "No Build"... a conclusion which is farcical on its face. If WSDOT did not think that more vehicles would use the highway, they would not be expanding it.

The impact of congestion on I-5 and on I- 405 is portrayed inconsistently in different analysis on the EIS. We have not had time to develop details, but different assumptions are used in different places.

8) Mis-characterization of Open Space

Although the FEIS has many details about the open space, it neglects to portray the reality... that west of the SR 520 highrise bridge, most of the area to be taken consists of open space: bays, wetlands, and surrounding open space rich with birds, beaver, and

other wildlife, and a destination for used for canoeing, swimming, hiking, and other recreation.
This is some of the last remaining space of its type in the heart of Seattle, and is literally irreplaceable. The expansion of the highway would destroy much of its usability.

The open space here is important enough that tour boats include it in their trips, and numerous newspaper articles describe it as an attractive destination. For a sampler of these articles, see Attachments 8A, 8B, 8C, and 8D.

9) Conclusion on tolled 4 lanes alternative is inconsistent with FEIS analysis.
The FEIS concludes that the alternative of a tolled 4-lane highway does not meet the project mobility goals. However, the FEIS indicates that with a $4 toll, the results would be close to those of the PA.

Furthermore, no tradeoff analysis is done. If the tolled 4 lanes are close to the PA in terms of mobility, but do much less damage to the environment and the neighborhoods, (and cost much less) is that not a better alternative? That critical analysis is missing.

If a good bus rapid transit system were funded in conjunction with an improved 4 lanes, the state could get most of the mobility advantages along with an affordable price tag. That analysis is missing, too.

10) FEIS gives undue priority to mobility, and inadequate weight to other objectives.
Four objectives are stated in the "purpose" for the project (FEIS 1-5):

1) to improve mobility,
2) to [create] safety and reliability,
3) to be cost-effective, and
4) to avoid impacts on neighborhoods and the environment.

The logic of the FEIS is to discard alternatives based on their lack of achieving objective 1, mobility. The results would be very different if the FEIS looked first at the other objectives, or at least gave them heavy weight. For instance,

- The PA does not meet Objective 2, Safety. If safety were a dominant concern, then the scarce financial resources would be used first to fix all the safety problems, including those on the earthquake-vulnerable piers on the west side, which otherwise will be left for many years until funding is found. This obviously endangers reliability, because if the west side collapses, an expanded east side will do no good. Instead, WSDOT is spending scarce resources to improve the corridor east of Lake Washington where the safety issues are much less by WSDOT's own account.

- As shown in 9) above, the tolled 4 lanes would not have been discarded if WSDOT had based its decisions on its own data and had given appropriate weight to objectives 2,3, and 4.

- If objective 4 (environment) were given appropriate weight, once the damage to this irreplaceable open space was known, the FEIS would have analyzed in detail other alternatives and/or design changes including use of tunnels, narrower footprint, and fewer off ramps in the ecologically-sensitive areas.

- The FEIS does not consider an alternative which we and many others requested in comments on the SDEIS; lanes 5 and 6 for transit only, with fewer on and off ramps and a smaller footprint. The FEIS does not even consider the tradeoffs between funding a good bus rapid transit system and the preferred alternative. The FEIS gives detailed history of years of process on transit decisions, but the fact that public bodies have made some decisions does not excuse the FEIS from developing and presenting data which might change these decisions. If the best way to achieve the project objectives is with more or different transit, the FEIS should present this and the state should advocate for it.

11) The FEIS has inadequate analysis and unreasonable assumptions on local traffic impacts

The people most familiar with Seattle street traffic.... including both the Seattle Department of Transportation and the members of this coalition... have concluded that the PA will have a significant impact on traffic on a large segment of Seattle: from Madison Street to NE 75th Street, and from I-5 to Lake Washington Boulevard. Both SDOT and the coalition have asked for analysis of the SR 520 impact on traffic in this area. However, the FEIS does not look at the whole area, but only a much smaller area right next to SR 520's Montlake interchange.

WSDOT staff members tell us orally that the study isn't needed because there will be little impact. So because there is no study, no one can prove that there is indeed a significant impact!

Furthermore, even if WSDOT models suggest there will be no impact, but all the knowledgeable people say there will be, the odds are high that the models are wrong . There is considerable evidence that traffic models are very fallible in this kind of analysis. At the least, they are dependent on the assumptions they are given, which in this case might be highly optimistic.

The FEIS says that intersections near the Montlake interchange are given an "F" (fail) now and will be given an "F" in the future. However, even a failing intersection can become worse. This one will, and the traffic will spill over into the local area ins ways that are not considered in the FEIS, so no mitigation is planned.

12) The impacts on I-5 are not adequately disclosed or analysed.

The preferred alternative will cause reduced capacity on I-5. This is not disclosed in the main body of the FEIS, and is not made clear until FEIS itself, page 108 of the Transportation Discipline Report in Attachment 7, "The Preferred Alternative would

reduce the number of lanes from four to three in the Express Lanes across the Ship Canal Bridge to provide space for a single new HOV/transit ramp to and from SR 520...."

Even this disclosure is not accompanied by the diagram, like Attachment 12, which is necessary to understand the impact on I-5, and which is not anywhere in the FEIS.

Consequently, there has been no public discussion of the important tradeoffs involved, or about the wisdom of taking a lane away from I-5, or of creating new merges and weaves. Instead, there is a lively civic discussion on the need to relieve I-5 congestion by removing bottlenecks to increase its throughput.

This is yet another example of WSDOT trying to hide the ball and use the EIS, not to inform a decision to be made, but to justify a decision already made.

Please see Attachment 7.

13) The "Partial Bridge" is new, is inadequately analyzed, and will have material impacts.

There is a new Partial Bridge in the FEIS called the "Construction Phase 1" bridge. The FEIS discloses a plan to build from the east side to the western highrise bridge, with a merge from 6 lanes back to 4 lanes just west of the highrise (where there is no interchange). This is a partial bridge, a bridge to nowhere, because the two added lanes don't go to any destination, but simply merge back into the existing 4 lanes.

Contrary to assertions in the FEIS, this plan is very different from the "phased implementation" discussed in the SDEIS and it is significantly different from the alternatives analyzed in that document. There has been no opportunity for public comment.

The differences include:

- The SDEIS phased implementation was to be a short time. All parties agree that this Partial Bridge might be in place for a long time.

- The SDEIS phased implementation connected to land, at an offramp where at least 30% of the traffic leaves 520. The Partial Bridge does not connect to land or to any exits.

The FEIS does not adequately analyze the Partial Bridge. The merge of 6 lanes to 4 will create heavy congestion for some hours each day, with noise and emissions directly over fragile wetlands and the bay with its fish and wildlife. The noise and emissions will affect the nearby neighborhoods of Madison Park and Laurelhurst. The visual impacts of the merge are significant. And on the west side, traffic will continue to back up on I-5 because there is no relief of the chokepoint getting on to SR 520.

Furthermore, will the bike/pedestrian path simply stop at the end of the Partial Bridge? We have not found anything in the FEIS about this.

The long-term partial bridge will also mean that the highway damage will continue to be done to the wetlands and bays of the west side. The stormwater problems, the continued dropping of matter into the bays, will not be fixed. The pavement of the west side, which is old and very noisy, will not be improved.
Please see Attachment 13.

The FEIS statement that impacts will be similar to the phased implementation of the SDEIS is not borne out with analysis, and is inherently unbelievable.

14) Inadequate disclosure of design

After numerous requests, we have still never seen depictions of the Montlake Interchange from ground level, or of the Portage Bay bridge in comparison to the current bridge, or of other areas. Such depictions are necessary for us and the decision makers to understand the plans and what might be approved for construction.

15) Changes after the FEIS.

In discussions, WSDOT has already indicated that it may change the FEIS plans for the placement of the Portage Bay bridge. Any change here would have profound impacts on the many homes which see and hear traffic on the bridge, and on the families whose lives would be affected. We cannot know the impacts of such significant change until we see documents. Given the post-FEIS timing of these revelations, this critical information obviously is not in the FEIS.

16) The PA was chosen before, and outside of, the EIS process.

WSDOT, the governor, and certain legislators chose an alternative long before the environmental review was done and have not been open to other alternatives which might provide better mobility with less damage to the environment.

NEPA creates a process intended to ensure that environmental information is first obtained and then used to make informed decisions. But time and again, WSDOT has demonstrated that it has made its mind up in advance and is going through the NEPA process as a bureaucratic formality, creating analyses to justify decisions already made.

Our SDEIS comments in April 2010 lay out indicators of WSDOT's bad faith in the process to that date. Since then, the same patterns of behavior have become more intense:

1) In February, 2010, shortly after publication of the SEIS, the State speaker of the house, the mayor of Seattle, other politicians and various groups joined us in saying that alternatives like using lanes 5 and 6 for transit only must be considered. (They are not considered in the SDEIS.)

   In response, the governor said looking at changes in configuration would set back the project, and "our commitment to ensuring public safety does not allow that kind of delay". (Never mind that the governor's proposal would leave in place for a decade or longer the hollow, earthquake-prone pillars supporting 520 west of the floating bridge.) Likewise, a leading State representative said, "We have an agreement, let's move forward." Proponents of the State's plan held a press conference at which the

House Transportation Chairwoman, Judy Clibborn, D-Mercer Island, reiterated the argument that a redesign of the car-pool lanes would delay the project up to two years. All this before comments on the SDEIS were even submitted and before a preferred alternative was officially chosen.

2) The preferred alternative was officially announced at the end of April 2010, less than two weeks after the SDEIS comment period and long before anyone could have read and absorbed the hundreds of submitted comments. The choice had obviously been made long before.

3) Long before the Final SEIS was released, the governor, WSDOT, and State legislators presented the preferred alternative as a final decision, awaiting only paperwork details to be implemented. "We have a new 520 and are ready to move forward to open the bridge in 2014," Gov. Chris Gregoire said.

4) In op-ed pieces, media interviews, and ads, the governor, WSDOT and State legislators, together with some business interests, pushed hard for people to stop questioning the plans., "It's time for action on the 520 bridge!" This pressure strengthened the perception that the decision had been made and that opposing it was dangerous. It trivialized the environmental process mandated by NEPA (and its State counterpart, SEPA).

5) The governor set a tight timetable for construction that would not permit any further discussion or consideration of alternatives that were, or should be, analyzed in the EIS.

a. The Section 106 process for analyzing and mitigating impacts on historic areas was driven by the deadlines. Legitimate requests for additional assessments were brushed aside in the name of meeting deadlines.

b. The Metropolitan Transportation Planning Organization (the Puget Sound Regional Council) was asked in April to approve and did approve going ahead with construction of the "partial bridge" before the FEIS was published, because the State said it needed to move fast with construction of its preferred alternative.

c. WSDOT seeks to have a ROD in July, just a month after the FEIS is released and long before you can reasonably be expected to consider all the information contained in the EIS and listen to those who can show that its analysis is biased, incomplete and inaccurate.

Submitted on behalf of the Coalition for a Sustainable SR 520

Fran Conley

Attachment 3A

2011-13 Biennium Senate Proposed Transportation Budget Project List
LEAP Transportation Document 2011-2 ALL PROJECTS as developed April 19, 2011
Highway Improvements Program (I)
(Dollars in Thousands)

Route	Bin	Project	Leg Dist	-- Fund Source -- TPA	Nckl	Othr	2009-11	2011-13	2013-15	2015-17	2017-19	2019-21	2021 +	Total (incl. Prior)
SR 500, Vancouver to Orchards - Corridor Improvements							**7,674**	**40,044**	**636**	**0**	**0**	**0**	**0**	**56,961**
500	450000A	SR 500/St Johns Blvd - Build Interchange	49	☑	☐	☑	7,674	40,044	636	0	0	0	0	56,961
SR 502, I-5 to Battle Ground - Corridor Improvements							**13,741**	**47,377**	**18,000**	**233**	**0**	**0**	**0**	**140,307**
005	400599R	I-5/SR 502 Interchange - Build Interchange	17, 18	☐	☑	☑	826	0	0	0	0	0	0	52,521
502	450208W	SR 502/I-5 to Battle Ground - Add Lanes	18	☑	☑	☑	12,915	47,377	18,000	233	0	0	0	87,786
SR 503, Battle Ground to Vancouver - Improvements							**5,342**	**423**	**0**	**0**	**0**	**0**	**0**	**7,377**
503	450305B	SR 503/4th Plain/SR 500 Intersection - Add Turn Lane	17	☑	☐	☑	587	214	0	0	0	0	0	811
503	450393A	SR 503/Lewisville Park Vicinity - Add Climbing Lane	18	☑	☐	☑	4,755	209	0	0	0	0	0	6,566
SR 509, SeaTac to I-5 - Corridor Completion							**3,825**	**20**	**0**	**0**	**0**	**0**	**0**	**61,537**
509	850901F	SR 509/I-5 to Sea-Tac Freight & Congestion Relief	30, 33	☑	☐	☐	3,824	20	0	0	0	0	0	26,541
509	850902A	SR 509/I-5/SeaTac to I-5 - Design and Critical R/W	33	☐	☑	☐	1	0	0	0	0	0	0	34,996
SR 510, Yelm - New Freeway							**8,734**	**687**	**0**	**0**	**0**	**0**	**4,860**	**36,006**
510	351025A	SR 510/Yelm Loop - New Alignment	02	☑	☐	☑	8,734	687	0	0	0	0	4,860	36,006
SR 518, Burien to Tukwila - Corridor Improvements							**3,170**	**733**	**0**	**0**	**0**	**0**	**0**	**42,127**
509	850919F	SR 509/SR 518 Interchange - Signalization and Channelization	33	☑	☐	☑	304	75	0	0	0	0	0	5,831
509	850919G	SR 509/SR 518 Interchange - Interchange Improvements	33	☐	☐	☑	94	0	0	0	0	0	0	465
518	851808A	SR 518/SeaTac Airport to I-5 - Eastbound Widening	11, 33	☑	☐	☑	2,772	658	0	0	0	0	0	35,831
SR 519, Seattle - Intermodal Improvements							**42,705**	**0**	**0**	**0**	**0**	**0**	**0**	**84,015**
519	851902A	SR 519/ I-90 to SR 99 Intermodal Access Project - I/C Improvements	37	☐	☑	☑	42,705	0	0	0	0	0	0	84,015
SR 520, Seattle to Redmond - Corridor Improvements							**500,907**	**1,263,266**	**629,813**	**24,005**	**25,000**	**0**	**1**	**2,645,415**
520	152040A	SR 520/W Lake Sammamish Parkway to SR 202, Stage 3 - Widening	48	☐	☑	☑	45,930	1,408	86	0	0	0	0	79,397
520	8BI1003	SR 520/ Bridge Replacement and HOV (Nickel/TPA)	43, 48	☑	☑	☑	388,087	1,247,769	629,727	24,005	25,000	0	1	2,478,632
520	L1000033	Lake Washington Congestion Management	43, 48	☑	☐	☑	66,890	14,089	0	0	0	0	0	87,386

Alt 3A, p2

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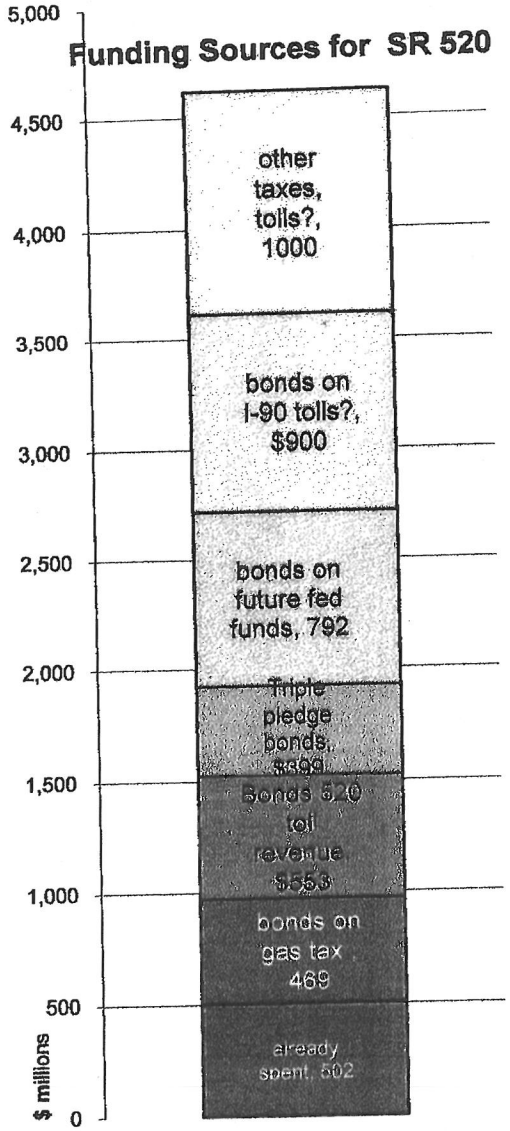
Highway Improvements Program (I)							**2,896,968**	**4,103,038**	**2,210,747**	**797,823**	**288,830**	**261,823**	**1,127,925**	**17,834,773**
SR 3, Mason/Kitsap County - Improvements							**1,925**	**4,480**	**11,338**	**0**	**0**	**0**	**24,823**	**47,019**
003	300344C	SR 3/Belfair Bypass - New Alignment	35	☑	☐	☑	316	435	0	0	0	0	11,188	14,533
003	300344D	SR 3/Belfair Area - Widening and Safety Improvements	35	☑	☐	☐	1,585	4,045	11,338	0	0	0	0	18,154
003	300348A	SR 3/Fairmont Ave to Goldsborough Creek Br - Replace Bridge	35	☑	☐	☐	0	0	0	0	0	0	13,635	13,865
003	300348B	SR 3/Jct US 101 to Mill Creek - Safety	35	☑	☐	☐	24	0	0	0	0	0	0	467
I-5 / SR 16, Tacoma Area - HOV & Corridor Improvements							**220,337**	**320,835**	**288,269**	**153,979**	**36,395**	**79,667**	**121,261**	**1,612,262**
005	300504A	I-5/Tacoma HOV Improvements (Nickel/TPA)	25, 27, 29	☑	☑	☑	209,690	319,562	288,269	153,979	36,395	79,667	121,261	1,477,351
016	301636A	SR 16/I-5 to Tacoma Narrows Bridge - Add HOV Lanes	27, 28, 29	☐	☑	☑	10,511	1,273	0	0	0	0	0	127,451
016	301638B	SR 16/36th St to Olympic Dr NW - Add HOV Lanes	26	☐	☑	☑	136	0	0	0	0	0	0	7,460
I-5, Everett Area - HOV & Corridor Improvements							**645**	**1**	**0**	**0**	**0**	**0**	**0**	**261,990**
005	100540F	I-5/164th St SW to SR 526 - HOV and Interchange Modifications	01, 21, 38, 44	☐	☐	☑	4	1	0	0	0	0	0	41,872
005	100543M	I-5/SR 526 to Marine View Drive - Add HOV Lanes	38, 44	☐	☑	☑	641	0	0	0	0	0	0	220,118
I-5, Lewis County Area - Corridor Improvements							**86,951**	**98,388**	**71,224**	**740**	**0**	**0**	**0**	**368,364**
005	300581A	I-5/Grand Mound to Maytown - Add Lanes and Replace Intersection	20	☐	☑	☑	53,603	16,403	40	0	0	0	0	115,335
005	400507R	I-5/Rush Rd to 13th St - Add Lanes	20	☐	☑	☑	980	0	0	0	0	0	0	53,660
005	400508W	I-5/Mellen Street I/C to Grand Mound I/C - Add Lanes	20	☑	☐	☑	32,368	81,985	71,184	740	0	0	0	199,369
I-5, Puget Sound Area - Improvements							**68,850**	**61,581**	**2,416**	**26**	**17,431**	**21**	**19,362**	**356,273**
005	100505A	I-5/Pierce Co Line to Tukwila Interchange - Add HOV Lanes	11, 30, 33	☐	☑	☑	103	0	0	0	0	0	0	138,946
005	100522B	I-5/Express Lane Automation	43	☐	☐	☑	2,832	2,993	0	0	0	0	0	5,825
005	100529C	I-5/NE 175th St to NE 205th St - Add NB Lane	32	☐	☑	☑	32	0	0	0	0	0	0	8,735
005	100536D	I-5/SR 525 Interchange Phase	01, 21	☑	☐	☐	0	0	0	0	0	0	19,357	20,001
005	100537B	I-5/196th St (SR 524) Interchange - Build Ramps	01, 21	☑	☐	☐	18,879	11,174	203	0	0	0	0	33,775
005	100541M	I-5/128th St SW (SR 96) - Interchange Improvements	21, 44	☐	☐	☑	364	600	0	0	0	0	0	1,872

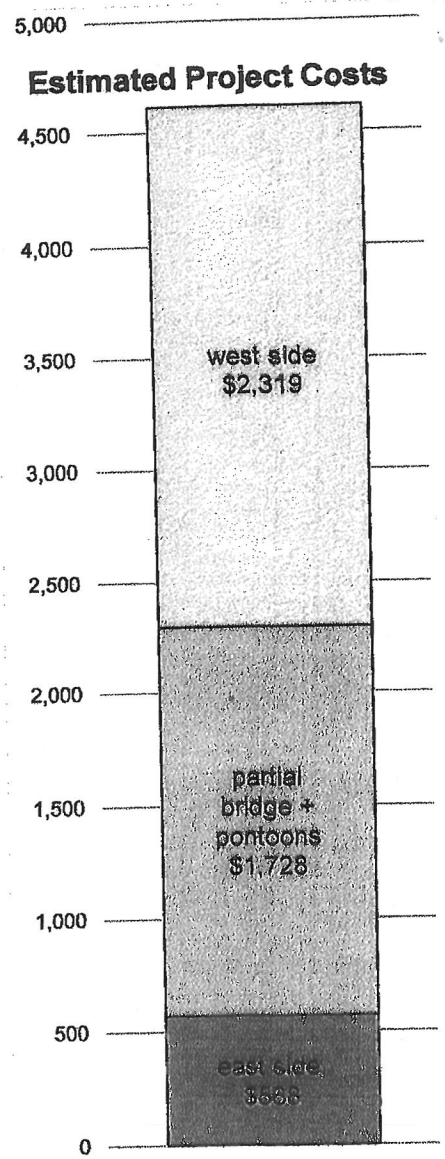
Enacted 2009-11 Budget as base			Enacted 09-11	Supp	09-11	11-13	13-15	15-17	17-19	19-21	21-23	23-25	25-27
Beginning Fund Balance			0	0	0	1,932	797	584	221	15,497	44,703	47,377	49,739
Source of Funds													
Treasury Deposit Earnings		S	0	(13)	(13)	79	319	(150)	(20)	200	200	200	250
Treasury Deposit Earnings from acct 495		S	0	26	26	180	220	250	260	270	280	290	300
Toll Revenue transfer from account 495		S	12,305			111,128	143,142	167,235	181,904	190,912	200,222	209,916	220,009
Deductions for Free Trip Incentives		S	0										
Customer-Initated Payment Discount		S	0			(450)	(471)	(449)	(444)	(437)	(431)	(424)	(417)
Reserves		S	0	0	0	(18,280)	(2,269)	(4,067)	(13,451)	(1,179)	(463)	(1,719)	0
Transponder Revenue		S	8,005			7,969	3,366	3,536	3,715	3,904	4,101	4,309	4,527
Fees		S	1,373			2,828	3,223	3,194	3,227	3,194	3,132	3,039	2,908
Administrative Transfer from Civil Penalties Account		S	190	(190)	0	754	946	957	993	989	947	926	907
Bond Proceeds Triple Pledge		B	231,754	0	231,754	[illegible]	[illegible]	0	0	0	0	0	0
Bond Proceeds - Senior Lien Revenue Bonds		B			0	[illegible]	[illegible]	0	0	0	0	0	0
Bond Proceeds - Subordinate Revenue Bonds		B			0	[illegible]	[illegible]	0	0	0	0	0	0
Bond Proceeds GARVEE		B			0	[illegible]	[illegible]	0	0	0	0	0	0
Federal Surface & Bridge Funds	*for debt service*	S			0	14,649	74,656	256,294	188,093	302,996	264,697	0	0
Withholding for Existing Debt Service		S			0								
Withholding for Estimated Debt Service		S			0	(33,792)	(91,874)	(105,956)	(113,927)	(119,608)	(125,621)	(129,386)	(138,095)
Debt Service pd by Federal Funds		S			0	(14,649)	(74,656)	(256,294)	(188,093)	(302,996)	(264,697)	0	0
Total Sources			255,628	(18,432)	236,596	1,052,841	584,342	64,550	62,257	78,225	82,367	87,141	90,389
Other Agency													
Sales Exp/Cost of Issuance		B	83		83	68	185	0	0	0	0	0	0
Underwriters Discount Expense		B	1,308		1,308	1,075	2,949	0	0	0	0	0	0
Total Other Agency			1,391	0	1,391	1,143	3,134	0	0	0	0	0	0
Operations													
B00 Toll Maint/Opns		S	24,000			0	0	(1,068)	3,571	4,798	5,907	10,143	8,548
B00 Toll Maint/Opns unalloted		S				(933)	(577)	(577)					
B00 Toll Maint/Opns - DP	*Service Center reduction*	S	0		0	34,306	34,539	42,557	43,410	44,221	45,026	45,876	46,765
S00		S	264		264	0	0	0	0	0	0	0	0
Total Operation			24,264	(20,829)	3,435	33,373	33,962	40,912	46,981	49,019	50,933	56,019	55,313
Capital													
I0C Improvements - Deferred Sales Tax		S									28,760	28,760	28,760
I0C Improvements		S					22,853	23,206	0	0			
	Moved under-run of bond expenditures in Feb update from 09-11 to 11-13. This must be appropriated as Bond Expend. in the 11-13 Budget Bill, but must be coded in TEIS Fund Balance as state cash expenditures. Includes 10-11 Feb Update as Cash Expenditures.	S											
		S											
Bond Proceeds Triple Pledge		B	231,754		231,754	[illegible]	[illegible]	0	0	0	0	0	0
		B											
		B											
Bond Proceeds - Senior Lien Revenue Bonds		B				[illegible]	[illegible]	0	0	0	0	0	0
Bond Proceeds - Subordinate Revenue Bonds		B				0	[illegible]	0	0	0	0	0	0
Bond Proceeds GARVEE		B	0		0	[illegible]	0	0	0	0	0	0	0
Total Capital			231,754	(1,925)	229,829	1,019,460	547,459	24,001	0	0	28,760	28,760	28,760
Total Uses of Funds			257,418	(22,754)	234,664	1,053,976	584,555	64,913	46,981	49,019	79,693	84,779	84,073
Ending Fund Balance (SR 520)			(2,390)	4,322	1,932	797	584	221	15,497	44,703	47,377	49,739	56,055

Attachment 3B

Attachment 3C

~~Exhibit A~~





SOURCES OF FUNDS:	SR 520 million $
already spent	$ 502
bonds on gas tax	$ 469
Bonds on 520 toll revenue	$ 553
Triple pledge bonds	$ 399
bonds on future fed funds	$ 792
bonds on I-90 tolls?	$ 900
other taxes, tolls?	$ 1,000
Total construction cost	**$ 4,615**

PROJECT COSTS	SR 520 million $
east side	$ 568
partial bridge,pontoons	$ 1,728
west side	$ 2,319
Total	$ 4,615

draft 4/4/11

source: WSDOT 11/10 Program Comparison Chart

Souce LEAP draft 3/11 and 520 financial plan 3/11

I-90 tolls estimate from legislative workgroup materials

"Triple pledge" bonds: toll revenue, gas tax, and full faith and credit of state

Attachment 6A

Sightline Daily
News & Views for a Sustainable Northwest

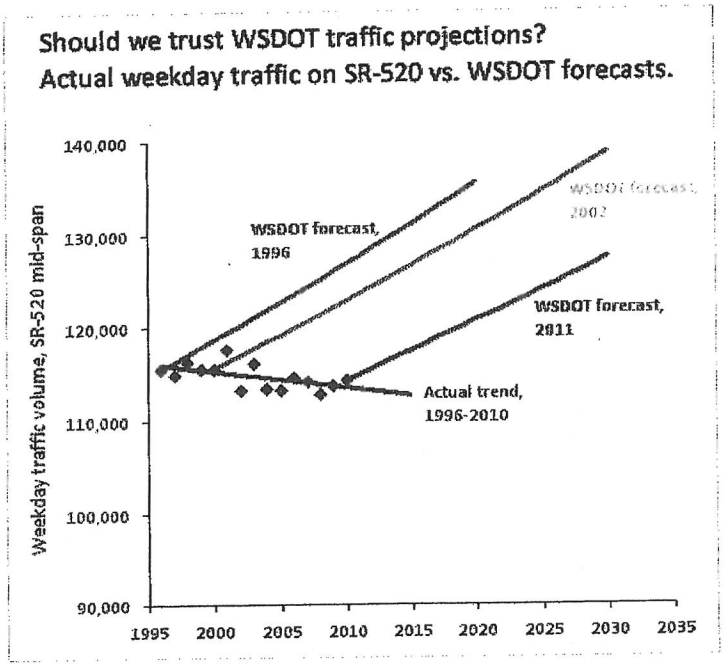
WSDOT vs. Reality

Puget Sound traffic forecasts don't even pass the laugh test.

Clark Williams-Derry on July 13, 2011 at 1:35 am

This post is 12 in the series: Dude, Where Are My Cars?

I wish I were making this up. The Washington State Department of Transportation continues to insist that traffic volumes on the SR-520 bridge across Lake Washington are going up up up—even though actual traffic volumes have been flat or declining for more than a decade! Here's a chart that makes the point.



In a charitable mood, you could forgive the 1996 projections. Back then, rapid traffic growth on SR-520 was a recent memory: up through about 1988, traffic growth was both steady and rapid.

By 2011, however, it should have been perfectly obvious that the old predictions were proving inaccurate. Yet WSDOT just kept *doubling down on their mistakes*—insisting that their vision of the future remained clear, even as their track record was looking worse and worse. So now they've wound up with an official traffic forecast, in the final Environmental Impact Statement no less, that doesn't even pass the laugh test.

It would be funny—if the state weren't planning billions in new highway investments in greater Seattle, based largely on the perceived "need" to accommodate all the new traffic that the models are predicting will show up, any day now.

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In case you don't believe me about the numbers, feel free to check out the sources directly. I'd be happy to be corrected.

The data on recent traffic volumes—the blue dots—come from three sources. I start with

AH 78B, p3

lines with those that have existing lines or have invested in new ones, a correlation between rail and transit use is apparent. Cities with no rail saw far smaller declines in automobile mode shares than their rail counterparts; they also saw declining non-automobile mode shares, compared to increases in the rail cities. These differences were especially considerable when considering rail cities outside of Texas; excluding them, transit saw no mode share change, whereas single-person commuting by car decreased (albeit by a minuscule amount).

This may indicate that rail lines can play an important role in encouraging the population to try modes other than the automobile. The non-automobile mode share, which includes transit, biking, and walking, is particularly interesting from this perspective because it may reflect the number of people choosing to live in areas where it is acceptable to use transportation other than the private car. Is this conclusive evidence that rail works better than bus service to encourage people out of their cars? Not necessarily, but it's certainly a part of the overall equation.

Looking city-by-city, modal share changes reflect some overall trends. Automobile usage continues to decrease in the nation's older, densely developed cities: The places recording the largest declines in overall car share were, in order, Washington, New York, Boston, San Francisco, Seattle, Portland, and Chicago. Those with the largest declines in non-automobile share were largely sprawling cities, including, in order, Columbus, Houston, Dallas, Fort Worth, Las Vegas, and Nashville.

% Change in Mode Share, 2000-2009 in America's Biggest Cities

	Total Auto	Total Non-Auto	Driving alone	Carpooling	Transit	Biking	Walking
Austin	-5.1	4.5	-1.2	-25.2	12.0	11.9	-11.4
Baltimore	0.5	-6.6	11.0	-37.1	-12.7	209.6	0.7
Boston	-11.9	9.7	-10.9	-16.4	6.9	117.7	8.4
Charlotte	-3.7	24.3	-1.6	-16.2	8.5	3.6	59.4
Chicago	-6.0	4.1	1.4	-31.5	1.6	129.2	4.7
Columbus	0.3	-24.0	4.3	-29.1	-39.7	107.3	-18.6
Dallas	0.6	-20.8	10.8	-40.0	-28.1	9.3	-2.3
Denver	-2.4	-3.3	1.7	-23.8	-7.5	89.8	-15.5
Detroit	-3.3	7.8	4.1	-33.1	-12.0	192.4	58.4
El Paso	-2.4	14.4	4.3	-35.0	2.5	47.8	26.5
Fort Worth	-1.5	-16.4	4.7	-29.9	1.5	-18.2	-31.5
Houston	0.7	-23.5	5.3	-19.8	-33.0	-17.9	-0.4
Indianapolis	-0.3	-2.6	3.0	-21.8	-17.1	129.1	1.1
Jacksonville	-1.1	-11.3	0.4	-10.4	-18.5	-4.1	-4.7
Las Vegas	-0.1	-13.7	5.5	-27.5	-28.5	-10.7	18.7
Los Angeles	-3.6	9.2	2.0	-28.7	10.7	63.8	-4.2
Memphis	-1.5	-7.9	2.7	-22.2	-7.8	-78.7	-4.0

AH TB, PZ

crawls to life every weekday morning before dawn, when a stretch of Interstate 95 turns into a glittering river of headlights moving so slowly that drivers need to leave up to two hours to cover a 30-mile trip.

"Painful," said a 55-year-old accounting firm employee who tries to pick up other riders at designated places in Woodbridge so she can use the restricted, faster lanes.

"Books on tape, music, it doesn't help," she said about the daily trip (most of the commuters interviewed here asked that their names not be used). "All I'm thinking is, 'Oh, God, this is going to hurt.' "

The grind of the drive provokes such frustration that commuters do odd things to stay calm. One commuter waiting for a ride at a meeting point here said that one driver had become notorious among the regulars — "the puppet guy," who apparently used hand puppets to act out arguments to manage his anger over being stuck in traffic.

The population of the Washington suburbs has exploded in recent years, up by more than 60 percent since 1980. Still, the congestion has not served as an impetus for car-poolers, whose numbers, as a portion of all drivers, have fallen.

In fast-growing Prince William County, where Woodbridge is located, the number of car-poolers has actually grown, but not nearly as much as number of people driving alone, which has tripled since 1980.

The census data also show that different races car-pool at different rates. According to the census, black, Hispanic and Asian commuters car-pool far more than white workers.

In 2000, the car-pool rate for Hispanic workers was 28 percent, double the rate for whites, partly because of new immigrants sharing rides to jobs at construction sites or factories. But even Hispanics are relying less on group rides: by 2009, the rate for Hispanics had fallen to 19 percent.

"As cars became more affordable and life became easier, the big car pools broke up," said Alan Pisarski, a consultant who studies transportation trends.

Car-pooling first cropped up as a policy idea in the United States in the 1940s, when oil and rubber shortages limited the use of personal cars, according to Erik Ferguson, a professor of urban planning and the author of a 1997 article called "The Rise and Fall of the American Carpool."

Car-pooling was first seriously studied by academics and urban planners in the 1970s, the decade of the oil embargo, "a time of great hope for car-pool enthusiasts," Mr. Ferguson wrote.

A+ 7B, P3

Lew Pratsch, who organized shared rides for federal workers while working for the Energy Department in the 1970s, remembers that decade as a golden era for car-pooling, when big companies like Xerox and Chevron organized car pools for their employees. He picked up his future wife on their first date with a car-pool van.

But since then, profound demographic and economic shifts occurred. Companies spread out more, and the workday became less predictable. Women went to work in large numbers, raising the incomes of households as well as their ability to own a car.

"It's economic," said Roger F. Teal, a former professor of civil engineering whose Illinois software company, DemandTrans Solutions, helps municipalities with transportation issues. "If people have a car available, they will use it."

With today's high levels of car ownership, "the strongest motivation for people to car-pool disappeared," said Mr. Teal, who conducted one of the early comprehensive studies of car-pooling. Car ownership has outstripped even population growth, as the number of cars parked in American driveways has risen by nearly 60 percent since 1980, while the number of Americans has grown by a third.

What remains, of course, is traffic, and in places like Washington, where it adds hours to commutes, people car-pool to take advantage of the fast-track car-pooling lanes.

People car-pool here with strangers in a practice called "slugging" — the term comes from fake bus tokens, because bus drivers sometimes mistake car-poolers, who often wait near bus stops, for bus riders. Each waiting spot has its own destination, like the Pentagon or L'Enfant Plaza, and drivers call them out as they drive up.

The practice can bring surprises, some more welcome than others. One commuter said she picked up some great financial advice from her carmates.

But another said she once had to defend a fellow passenger after the driver started lecturing her about Christianity. "It's O.K. to spread the word of God, but technically he was holding her hostage," she said.

As car-pooling has continued to decline, mass transit use has increased in the past decade. In the Washington area, it represents about 14 percent of commuters, compared with 11 percent in 2000, according to the data.

Another big change has been the number of people working from home at least one day a week, which has tripled since 1998, to about 600,000, according to Nicholas Ramfos, director of Commuter Connections, a network of agencies and local governments that coordinates ride-sharing programs.

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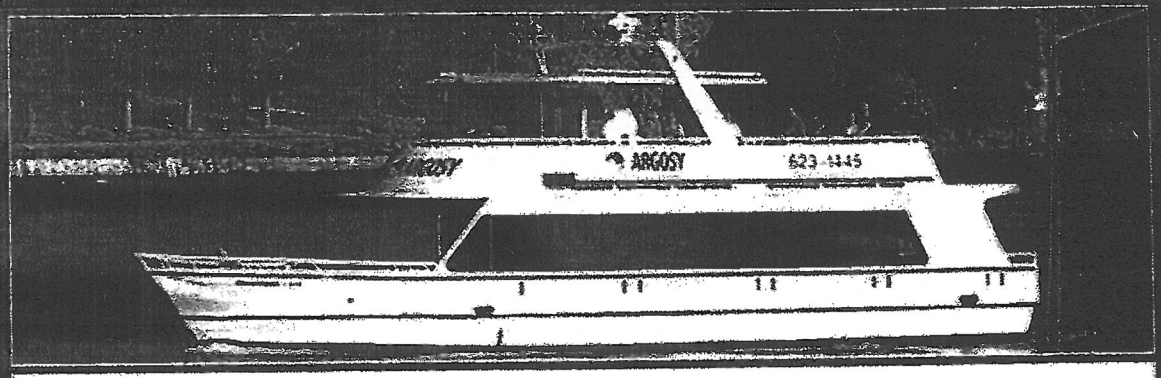

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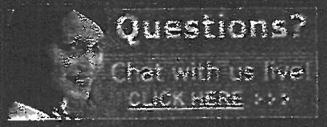
- Locks Cruise
- Seattle Lakes Cruise
- Kirkland Lake Washington Cruise
- Tillicum Village Blake Island Cruise



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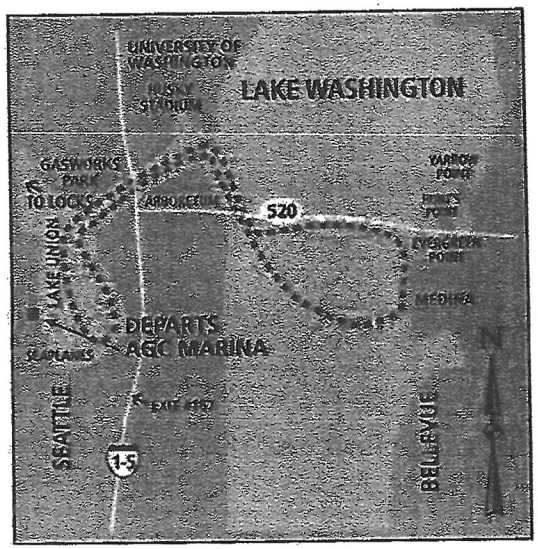

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DEPARTURES

PRICES

Attachment 8A

Attachment 8B

Originally published October 5, 2006 at 12:00 AM | Page modified June 18, 2007 at 5:05 PM

A Few of Our Favorite Things

Where to see fall foliage

Turning the tables this week, we offer favorites from a few writers on the Seattle Times travel staff: For a lavish dose of fall...

Turning the tables this week, we offer favorites from a few writers on the Seattle Times travel staff:

• "For a lavish dose of fall color, drive through **Tumwater Canyon** on Highway 2, just west of Leavenworth. Vine maples, cottonwoods and aspens put on a red-gold show alongside the highway and the churning Wenatchee River." — *Kristin Jackson*

• "Walk along the south side of Seattle's **Montlake Cut**, starting on Portage Bay. A narrow gravel trail starts near the west end of East Hamlin Street in a leafy park adjacent to Seattle Yacht Club, with views of the hilly Roanoke neighborhood, and passes under the turreted Montlake drawbridge before ending at the edge of Lake Washington. Across the water are the pretty slopes of Laurelhurst and panoramic views of the Eastside's tree-covered shores and the Cascades beyond." — *Tyrone Beason*

• "Cool nights at higher elevations produce early, eye-popping colors on scenic **Blewett Pass** on Highway 97. Take a short detour to the old gold-mining 'ghost town' of Liberty. Or head over the **North Cascades Highway**, where golden larches at the pass complement crimson vine maples below."

— *Brian J. Cantwell*

enlarge BRIAN J. CANTWELL / THE SEATTLE TIMES
Vine maples gleam in the rain along Highway 20, the North Cascades Highway, east of Marblemount.

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Video

Practice for Bon Odori, dancing in honor of loved ones and community
Members of the local community practice for the Bon Odori celebration on Saturday and Sunday, July 17 and 18. The public dances honor recently lost loved ones in Japanese buddhist tradition, but welcome all nationalities and faiths to participate.

Raw video: Colorful underwater life off San Juan Island

Running of the Bulls on Queen Anne

Seafair Pirates land ashore with Army assistance

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9. Behind the increase: Why Netflix is raising prices

Attachment 8C

Saturday, April 25, 1998 - Page updated at 12:00 AM

A Good Paddling -- Glide Through Seattle's Waterways In A Canoe Or Kayak

By Cathy Reiner
Seattle Times Staff Reporter

The rhythmic dip of paddles in the water. The flutter of a seagull overhead. The glitter of the waves. A fast escape from the landlocked world.

Paddling a canoe or kayak in the many waterways of greater Seattle can be a delightful adventure for families with children of about age 6 and up.

It's a safe, fun sport, as long as parents make sure that they and their children know what they're doing, before they get far from a beach or dock.

"Most important: Make sure everyone wears a Coast Guard-approved life vest," says Dolph Diemont, a Coast Guard boating safety specialist. "Next, parents should check the weather ahead to make sure they're not heading into a storm or high waves, and they should know their strengths and limitations.

"A paddling lesson is a good way to get started. You'll learn about safety that way."

Though paddlers soon learn to maneuver a canoe or kayak, preventing a capsize or righting a boat requires more practice, as does rescuing a "man overboard."

In a lake, a river, or particularly in chilly Puget Sound, a capsize can quickly become a life-or-death situation.

Proper instruction and practice also is important to make sure that everyone likes paddling and knows to behave himself, says Dan Hendrickson of Cascade Canoe and Kayak Center at Bellevue's Enatai Beach Park. "The dangerous kid is one who doesn't know what he's doing, or who gets bored and can't sit still in the boat for long."

Whether your family is thinking of long paddling trips or just getting out on the water, your first outings can be in rental boats, and enhanced by lessons and guided tours.

You can rent canoes, kayaks and other small rowing craft at a number of area lakes and saltwater beaches for $6 to $18 an hour, depending on the size and type of craft. Many outlets offer discounted half-day or longer rental rates.

Most outlets offer one-time or series group or private lessons, and guided tours for kids and adults. These are usually by reservation, but sometimes can be signed up for at the last minute. Introductory group lessons or tours cost $20 to $35 for about 90 minutes, including boat rental. Many places also offer weekend and summer kids programs.

Rental shops provide paddles and life vests (called personal safety devices, or PFDs) and require the vests be worn. Paddlers should bring spare dry clothing, sunglasses, sunscreen, water and snacks, all in a waterproof or tightly sealed plastic bag. Drinking water is particularly important because paddlers get thirsty and it's not safe to drink lake or sea water.

Families usually rent double kayaks or canoes so a parent or older sibling can be teamed with a younger child. The stronger or more experienced paddler takes command from the back seat.

Canoes are usually paddled by two people, sometimes with a child or extra passenger seated in the open middle. Paddlers kneel inside and use a single-bladed paddle on one side of the boat.

Kayaks come in single or double-seater models - one or two "holes" in the covered deck of most models. Paddlers are seated inside and dip a double-bladed paddle alternately, side-to-side. Kids often look forward to "graduating" to a single they can maneuver by themselves.

"I can't wait to take my own boat out," said Billie Andrews, 8, who recently was paddling a canoe with his parents in Mercer Slough, near Bellevue's Enatai Beach Park.

Last summer Billie and his father, Phil Andrews, always rented a double kayak, or sometimes a canoe so Billie's mom could go along, too. This summer Billie and Phil are planning some longer kayak expeditions, first in Lake Washington, then on some rivers. At times, Billie will be in his own kayak.

"He's strong and he's careful," says his father. "He can handle it. We have a great time on the water."

---------- THIS WEEKEND

Paddlefest: Test-paddle new boats, listen to sea-savvy experts, watch or paddle recreational races, meet manufacturers, retailers and club representatives, and see their products and programs, 9 a.m. to 4 p.m. today and tomorrow at Stan Sayres Memorial Park, 3800 Lake Washington Blvd. S., Seattle.

Admission $5, kids under 9 free. Introductory lessons (50 minutes) at 9:30 and 10:30 a.m. each day to first 18 people to sign up (age 10 and up), $5. Kids under 18 must have parental-release form to test-ride boats

Attachment 13

Pete and Wendy DeLaunay

2524 Boyer Ave. E. #212/210 Seattle, Washington 98102

Pete@Delaunay.com (206) 323-9128

July 13, 2011

TO: Department of Ecology – SEA Program

Federal Project Coordinator

ecyrefedpermits@ecy.wa.gov

FR: Pete DeLaunay, 25+ Year Portage Bay Area Resident, Seattle, Washington

Project Name: SR 520 I-5 to Medina Bridge Replacement and HOV Project

Application for State of Washington 401 Water Quality Certification & Coastal Zone Management Act Consistency

We have lived on the shoreline of Portage Bay which lies between Lake Union and Lake Washington in Seattle, Washington for the past 25+ years -- and just south of the existing Portage Bay viaduct -- on Boyer Ave. E. Before the Washington Department of Transportation built the existing SR 520 bridge, specifically over the Portage Bay area, our bay had a pristine shoreline with sufficient depth for abundant sea life and recreation – and a unique urban environment.

While Portage Bay remains somewhat fishable and a wonderful place for smaller human power boating, **WSDOT's negligence over the past 40+ years has had incredible impacts on water quality, toxic silt build up, invasive plants and significant reduction in fish and water fowl. No one can swim there any longer.**

To demonstrate how serious WSDOT's negligence has been, my wife created a website and video web cast of the filthy water from the existing structure that pours directly into Portage Bay during Seattle's somewhat frequent rain storms. These drains have been in place and pouring untreated filthy water from the existing bridge deck into the bay for the past 40+ years!

It is disgusting to view, but visit www.build520right.net to see it for yourself. The dirt from this runoff has resulted in silt build up of up to 90 feet in some places during some 40 years time.

Although WSDOT has said in the DEIS that Portage Bay is not considered a recreation area, pictures and people paint a different story in spite of the conditions WSDOT has wrought on the waterway and water quality of this unique urban area.

Att 13, p2

WSDOT said in a recent meeting that they are not accountable for past water runoff from the bridge and its serious impacts on water quality in Portage Bay. They have told us they will not mitigate the damage untreated runoff has had on the shoreline, water quality and water depth.

The new bridge, they say, will treat water runoff responsibly...and we are certain environmental officials such as DOE and others will require that. We ask that you insist, however, that WSDOT make up for past sins...not unlike the way in which government has held industry responsible and accountable for water quality from unbridled chemical deposits in waterways here (the Dumamish) and elsewhere.

State Environmental Policy Act intentions:

We request the reclamation of the South Portage Bay waterway and shoreline by WSDOT before any permits are granted by DOE or federal agencies.

Original SR 520 construction affected the bay in many ways: slit build up, water quality, shoreline, native species, native plants, and salmon habitat. Reclaiming South Portage Bay with removal of silt, invasive plant life, restoration of shoreline (see www.build520right.net) and better recreational access will provide an important dimension to this unique urban environment. And make up for WSDOT's past negligence, holding them accountable as government holds industry accountable for past mistakes.

Thank you for your attention and response to the issues we have raised on behalf of many neighbors and enthusiasts for responsible environmental mitigation of past sins. We request your vigilance to mitigate impacts of the SR 520 project fairly.

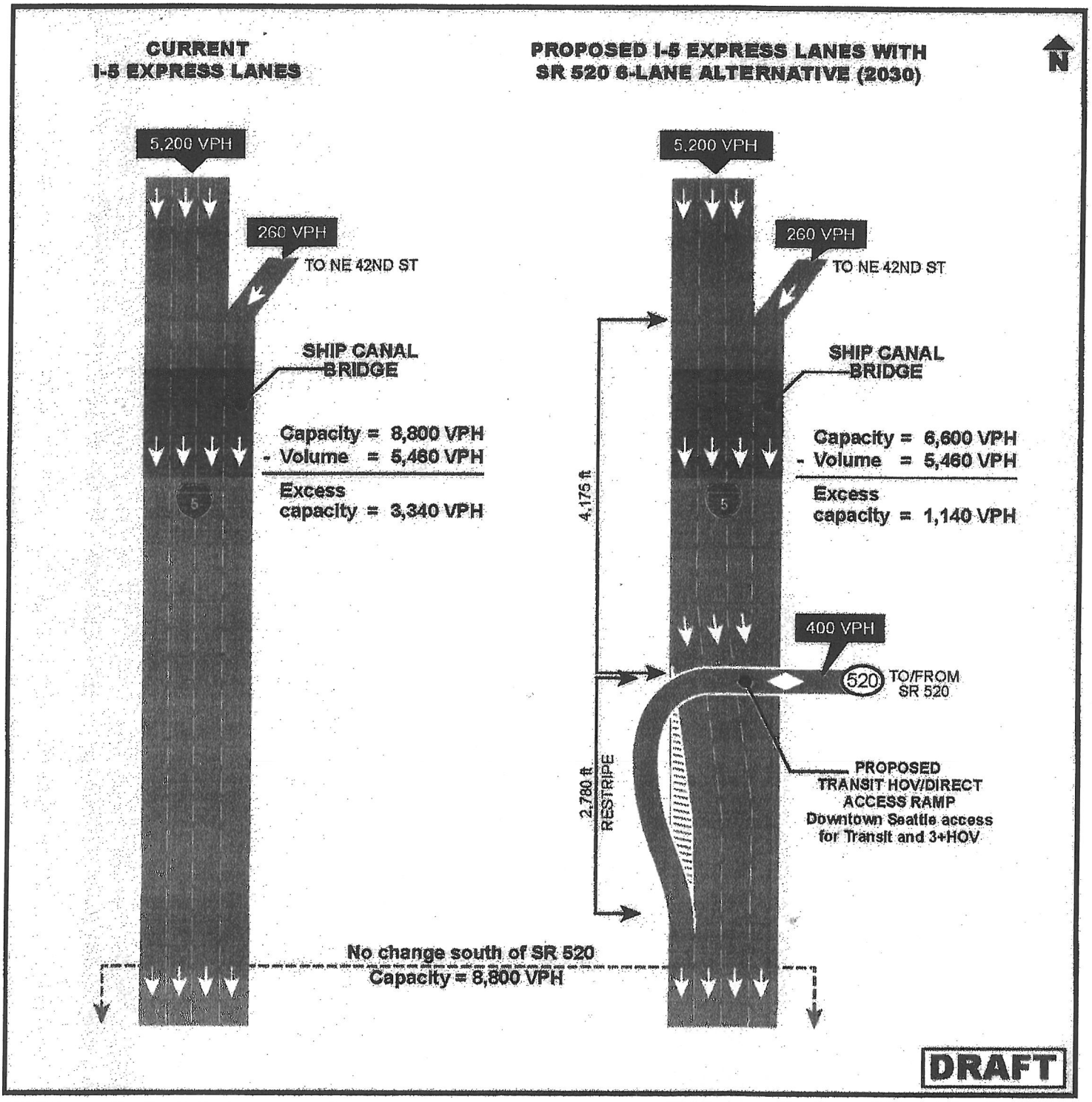
Attachment 12

Washington State Department of Transportation

SR 520 Bridge Replacement and HOV Program

STATE ROUTE 520

Existing and future I-5 express lane operations - AM traffic



NOTE:
- For illustration purpose only.
- VPH = Vehicle per hour
- I-5 mainline configuration not affected by this change.
- Similar operation for northbound direction
- The I-5 express lanes extend from downtown Seattle to Northgate Mall at North 103rd Street.

Communication #15191

Received: 06-15-2011

Communication: Hi,

I scanned through the EIS briefing chart, Final EIS presentation, June 13, 2011, slide 9, and noticed there are no plans presented about the terrible bottle neck issue at the I-5 south bound to 520 eastbound interchange, where cars getting on I-5 from the N.E. 45th street south bound on-ramp have to negotiate 3 lanes over to connect with the east bound 520 exit.

Seems that is a just as important of a traffic flow improvement as the traffic flow improvements planned for the 520 west bound to I-5 southbound interchange. Is this a separate project or is it going to be included in the SR 520 Bridge Replacement program?

Thanks for your consideration.
Cheers,
Keihan Ebrahimi
Systems Engineering
Integrated Simulation Management
66-6B-Q320

425-342-0759
Fax 425-342-9641

Note: Refers to the 6/13 City of Seattle FEIS presentation,
http://www.wsdot.wa.gov/Projects/SR520Bridge/Library/meeting.htm#SeaCouncil

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION 10
1200 Sixth Avenue, Suite 900
Seattle, WA 98101-3140

OFFICE OF
ECOSYSTEMS, TRIBAL AND
PUBLIC AFFAIRS

July 18, 2011

Ms. Jenifer Young, Environmental Manager
SR 520 Project Office
600 Stewart Street, Suite 520
Seattle, Washington 98101

Mr. Randolph L. Everett
Federal Highway Administration
711 S. Capitol Way, Suite 501
Olympia, Washington 98501

Re: SR 520, I-5 to Medina Bridge Replacement and HOV Project Final Environmental Impact Statement. Region 10 Project Number: 00-013-FHW.

Dear Ms. Young and Mr. Everett:

The U.S. Environmental Protection Agency (EPA) has reviewed the SR 520, I-5 to Medina Bridge Replacement and HOV Project Final Environmental Impact Statement (FEIS). We are submitting comments in accordance with our responsibilities under the National Environmental Policy act (NEPA) and Section 309 of the Clean Air Act.

The project team is to be commended for their dedication and hard work in bringing this project forward. The extensive involvement of and collaboration with the public, agencies, and tribes has led to beneficial outcomes and a Preferred Alternative that represents and reflects all those involved. Thank you for your responsiveness to our comments and those of other project participants. We appreciate the new design features incorporated into the Preferred Alternative that would reduce environmental impacts, such as, innovative noise reduction measures, bridge height and width adjustments that reduce shading, visual, and cultural resources impacts, and that improve future light rail compatibility. We particularly note and appreciate several new additions to the EIS: a quantitative analysis of mobile source air toxics, an analysis of vehicular emissions from the Montlake lid, inclusion of the SR 520 Health Impact Assessment (HIA) as Attachment 14 to the FEIS, and incorporation of numerous recommendations from the HIA.

Thank you for the opportunity to review and comment on the SR 520 Bridge Replacement Project. If you have questions or would like to discuss these comments, please contact me at (206) 553-1601 or email at Reichgott.christine@epa.gov or Elaine Somers of my staff at (206) 553-2966 or email at somers.elaine@epa.gov .

Sincerely,

Christine B. Reichgott

Christine B. Reichgott, Manager
Environmental Review and Sediment Management Unit

Communication #15251

Received: 07-06-2011

Communication:

-----Original Message-----
From: Eliza Coburn [mailto:elizaflug@mac.com]
Sent: Wednesday, July 06, 2011 5:12 PM
To: SR 520 Bridge Replacement & HOV Project
Cc: Eliza Coburn
Subject: Bridge plans and the Arb

To Whom it may concern;

It is my understanding that with the 520 floating bridge plan, as it stands, that no impact statements have been taken by the local residents and store owners of Madison Park, Broadmoor, and Washington Park. This makes this project highly suspect to these residents who pay taxes and care for their neighborhood very deeply.

Growing animosity towards this project because of the demolition of the Hunts Point Exit and destruction of the forestation of that area has created deep seeded animosity amongst all of the residents. This project destroys the quiet nature of these neighborhoods-who pay the highest taxes and use the bridge to get to and from their homes and to work. It is as if their feelings and investments to make these neighborhoods what they are is being overlooked by selfish, greedy and poorly informed individuals attempt to take over these places of sanctity and make them public domain-exploiting the resources, making accessibility number one over privacy and devaluing property and family history.

If you were to poll and discuss this issue with the majority of actual home owners and business owners, we are all horrified and saddened that people who have no business or ownership of the area have so much pull and so little foresight into this terrible plan that has been poorly conceived. I-90 was made larger and destroyed the quiet sanctity of the surrounding neighborhoods-creating highly urban crime infested problems. The idea of making the lake more for the whole city-as if it were not already, will further devalue property. People will leave it and it will fall in to ruin.

Madison Park residents chose their neighborhood so they could be quiet residents, safe, clean, and not encumbered by the urban feel of other places in the area. This idealism is shared with all faiths, cultures, and ethnicities, who live in this area. Everyone takes great pride in their lives and has worked so hard-and now, it will be destroyed by a minority of people who have been given too much power and not enough foresight or education on this predicament.

This sentiment is felt amongst all residents and the feeling that the lake will also be damaged by the tools and toxins released during construction-have not been thought out thoroughly. Building a bridge so Microsoft employees can get to work may be imperative, but there are other people and businesses here too, and no one seems to care.

The Arboretum businesses need the flow of traffic to get down to Madison

Park and the residents of this area will be adversely affected-tripling times to get places, and ruining the commutes for many children who go to school in a variety of different parts of the city.

This 14 year plan may be concocted by the same geniuses who destroyed a little town called Bellevue-and those who only look to create an Communist approach to its resoruces-which is good and bad, as history has proven, but the people of these neighborhoods will suffer-and then others will as well-it is basic trickle down theory.

It would be our hope to have the exit kept leading on to 520 through the Arboretum-so that the residents of the area to not clog and bottleneck 23rd and Montlake-which will see a huge spike in crime due to this moronic escapade in poor city planning.

If you want to take a look at good preservationist projects, take a look at the West Side highway in NYC, or the projects in place with the Bay Bridge-it took forever to complete but they did not devalue property or neighborhoods in the process.

Sincerely,

Eliza Flug-Coburn

July 15, 2011

Ms. Julie Meredith,
Program Director.
SR520 Program Office
600 Stewart Street. Suite 520
Seattle, WA 98102
SR520bridge@wsdot.wa.gov

Ms. Meredith:

FABNIA has helped develop 4 letters submitted during the SR 520 EIS development process. They were on the draft EIS, the supplemental EIS, Section 6(f) final report and the purchase of the 3 Frolund properties on Boyer Avenue. Countless hours were spent by neighborhood residents reviewing the published material that was available for public comment. Though no response is required for a comment letter submitted after publishing the final EIS, we believe it is useful to have our reaction to WSDOT's responses to our comment letters be part of the project record.

Unfortunately, our members have concluded that WSDOT did not adequately consider our specific comments in three of the four letters. On the other hand, WSDOT's fourth letter does provide at least a minimally adequate level of response.

Draft EIS Comment Letter

We provided joint comments with the Portage Bay-Roanoke Park Community on the 2006 Draft EIS. WSDOT responses to these comments were not provided until this June's final EIS documentation. Actual responses are only included in a Comment Summary Report. This report does not provide specific responses to any of our letter's comments. Two examples of our reaction to this inadequate level of response are hereby provided. These examples cover WSDOT's response to our comments on neighborhood traffic impacts.

Our letter comment C-028-007 includes concerns on the project's permanent neighborhood traffic impacts. WSDOT response comment C-028-007 refers to section 5.3 of the Comment Summary Report. Part 5.3 notes that future project traffic impacts were only modeled for Seattle arterials with 5% percentage change. Our Fuhrman-Boyer arterial apparently was not even included in that analysis.

Letter comment C-028-001 discussed construction period noise and traffic impacts. WSDOT's response comment C-028-002 refers to part 7.1 of the Comment Summary Report that covers neighborhood issues. Information provided in part 7.1 refers to the revised Social Elements Discipline Report pages 44-71. Only page 57 of that report discusses construction period and noise impacts in the Portage Bay/Roanoke neighborhood. This page does not discuss construction traffic impacts! This is not an adequate response as Fuhrman-Boyer must be a traffic detour route as well as being a designated traffic haul route. Entry to the street from a staging area will likely require temporary lane closures by a flagger. Additionally, Fuhrman-Boyer Avenue has traffic calming circles, medians and bulb-outs not designed for the passage of large construction project haul trucks.

Supplemental EIS Comment Letter

The FABNIA comment letter on the Supplemental EIS discussed permanent and construction impacts to our neighborhood's traffic/transit, land-use, park use and shoreline habitat. The following discussion again provides examples of how our comments were not adequately addressed.

Letter comments marked C034-003, 004, 005 and 006 discussed the traffic impacts that will affect Fuhrman –Boyer and other neighborhood arterials. Corresponding WSDOT comment responses failed to note that no neighborhood arterial traffic analysis was included in the revised Transportation Discipline Report. Letter comment C034-004 noted that there is no additional ramp to/from southbound I-5 to SR 520. This will increase cut through traffic our Fuhrman-Boyer Avenue and other neighborhood arterials. Our neighborhood streets will be used to provide a by-pass to the I-5/SR 520 junction and SR 520/Montlake Boulevard intersection congestion. The additional backed up traffic on both SR 520 and I-5 will add to noise and air pollution problems. WSDOT response comments C034-004 to C0034-07 do not cover these concerns.

Letter comment C-034-008 discusses construction detour and hauling traffic impacts and Metro Route 25 usage of Fuhrman-Boyer Avenue. WSDOT response comment C-034-008 states that construction "is not expected to affect Metro Route 25." How is this possible with 5 years of congestion on Fuhrman-Boyer Avenue from detour traffic, haul traffic and Boyer Avenue bridge structure placement?

Our letter comment C-034-011 discusses that a 4-lane alternative will intrude less into the wetland habitat of south Portage Bay. WSDOT response comment C-034-012 states "There would be no adverse long term effects on recreational boating in Portage Bay." This response comment notes that there will be less bridge support columns. "With fewer columns the boating

experience will be enhanced." These comments make the dubious assumption that a water area to be covered by the preferred option bridge structure up 2.5 times wider (at each end of Portage Bay) will provide a desirable boating area!

The natural shoreline habitat uniquely remaining in this area is also part of the park's attraction. Our organization working with Seattle Parks has just completed a shoreline trail and kayak boat launch. We also completed shoreline and upland native plant restoration for the western end of the park. A wider, higher and closer bridge structure will adversely impact both adjacent wetland habitat and kayak/canoe recreational use.

Our letter comment marked C-034-009 notes that construction noise levels along Boyer Avenue will exceed 90 dBA. Our letter noted that no statement was made on the need to provide residential sound proofing or vibration mitigation.

WSDOT response comment C-034-009 states that "WSDOT will develop a construction vibration monitoring plan to avoid damage to sensitive properties and structures during construction in the Montlake and Portage Bay area. Monitoring would take place if vibration from impact construction levels, such as pile driving and vibratory sheet pile installation is expected to exceed a certain threshold." These comments makes no commitment to provide any mitigation for shoreline sensitive areas or our residential structures that must endure 5-6 years of project construction.

Related letter comment marked C-034-007 also noted that construction dust and noise will affect kayak recreation use. It will also impact beavers, herons, eagles and other species that use the south Portage Bay shoreline area. WSDOT response comment C-034-013 states " many of the animals that occur adjacent to the SR 520 corridor are accustomed to living in urban areas and may not be disturbed by construction-related activities and habitat alteration. Wildlife that is more sensitive to disturbance would be displaced to other areas of suitable habitat."

Impacts to adjacent historic residence or park areas for a long period of construction may require mitigation as discussed in the CFR 771.135 p 5 "constructive use" regulation. This responsibility was not even discussed in any of the WSDOT responses to our two EIS comment letters.

Our two EIS draft letters supported a 4 lane rebuild alternative. The 4(f) report on historic and park properties does recognize that the No-build 4 lane alternative will avoid use of all Section 4(f) properties (page 9-105). The no-build alternative however is dismissed as not preventing bridge structural failure. A 4 lane rebuild alternative would repair all bridge structural deficiencies. It is also noted that a 4 lane No-build alternative is not acceptable as it will not meet "mobility" project objectives of increasing highway traffic flow. We believe that the same

conclusion could be made about the Preferred Alternative. That is if the EIS project area is realistically widened to include the I-5 traffic flow north of SR 520 or Seattle arterial traffic flows.

Section 6(f) Final Report Letter

Members of the general public were not given an opportunity to provide input or comment on the 6(f) mitigation site selection process. A replacement mitigation site is needed for project construction impacts to both the ship canal and Marsh Island - Foster Island trails. General public comment letters could be submitted only on the final 6(f) report that was released after the mitigation site selection. We have received an acknowledgement from WSDOT on the receipt of our comment letter. Our letter did not support the selection of the Bryant site on the north site of the ship canal to mitigate for impacts to the two trails. The Bryant site likely is contaminated. It is located in a commercial and industrial area that does not provide equivalent recreation use or habitat value.

Frolund Property Acquisition

FABNIA developed a letter signed by 31 residents and endorsed by the Northeast District Council on Fuhrman-Boyer Avenue haul routes and the purchase of the three adjacent Frolund properties. Two of the three properties were purchased for an expanded staging area without public notice. The purchase of only one of the properties was identified in the Supplemental EIS. The letter made 4 requests. They were (1) that the Frolund properties and adjacent undeveloped WSDOT property after construction become a public park (2) that Fuhrman-Boyer Avenues and adjacent sidewalks be reconstructed as necessary after construction, (3) that WSDOT use all appropriate construction BMPs and mitigate any related property damage and (4) that the south Portage Bay water and its adjacent shorelines be restored.

We found that WSDOT's May 11 response to this letter was not as unresponsive to our specific concerns as was the case in the other letters. It stated that WSDOT will receive community input on the use of the Frolund property after construction, restore streets including traffic calming improvements damaged by construction, and develop a shoreline habitat mitigation plan that will meet permit mitigation requirements. The letter also noted that WSDOT will meet with residents and the contractor to provide necessary BMPs. These BMPS will be in a community construction management plan (CMMP). Though the letter's response provides for a minimal commitment, as we may well question the adequacy of the shoreline habitat management plan and CMMP, this letter at least specifically addressed all comments.

We hope that the May 11 letter provides a responsible level of response that can followed in future communications.

Sincerely,

Anne Preston
President
FABNIA

Communication #15265

Received: 07-15-2011

Communication:

From: Johnski2sea7047@aol.com [mailto:Johnski2sea7047@aol.com]
Sent: Friday, July 15, 2011 11:41 AM
To: SR 520 Bridge Replacement & HOV Project
Cc: fran@roanokecap.com; bill@mundyfarms.com; mamundy@comcast.net
Subject: SR520 bridge replacement

Dear WSDOT - SR520 Bridge Officials

I request that the following information be incorporated in the Final EIS for the subject project:

I am writing for myself and the owners of boats moored at the dock of the Canterbury Shores Condominiums, 2500 Canterbury Lane E., Seattle, WA, on the south shore of Union Bay. The WSDOT plan for the work area designated for the SR520 bridge replacement project is very close to the south shoreline from the NE point of Madison Park west to several hundred yards beyond the Canterbury Shores dock . The water passage left between the work area and the shore to accommodate boat traffic from the Canterbury Shores dock to the waters of Lake Washington is unvavigable due to being too shallow for sailboats with keels and too choked with lily pads and milfoil for motor boats.

How will this situation be mitigated to provide for navigable operation of vessels from the Canterbury Shores area to Lake Washington during the construction period for the project? Also, upon completion of the project, will there be at least the depth of water that now exists for safe navigation through this area? It must be understood that the water level of these waters lowers as the summer progresses due to water being used the Hiram Chittenden Locks.

This information was conveyed in writing by me to your representatives at a meeting held by
WSDOT at the Montlake Field House several years ago and at that time, I was told I would receive a response to my communication. This has not occurred.

Receipt of this communication is respectfully requested.

Thank you.

John Martin Hansen
2502 Canterbury Lane E., #309
Seattle, WA 98112-2562
206-726-9674

July 14, 2011

Paula Hammond
Secretary of Transportation
Washington Department of Transportation (Hand Delivered)

RE: SR 520 Bridge Replacement – Final Environmental Impact Statement – Response

I believe Olympia is making a grave error on insisting to go with the current preferred conceptual design for the SR 520 bridge replacement.

I am well versed in most of the conversations, debates and studies dating back to the original Parametrix Study on Alternative Designs and 520. I was on Mayor Nickels 520 Advisory Board and worked with Senator Ed Murray to secure the funds for the 520 Mediation Group.

Olympia and Seattle are under the impression that an Immersed Tunnel Tube (ITT) on Lake Washington would cost $8 billion dollars and that particular design (done by COWI) didn't even go all the way across Lake Washington. Olympia, Seattle and the general public couldn't be more misinformed on this subject.

Based on current and finished ITT projects in Northern Europe where the cost of skilled labor is greater than ours here in the Northwest, Immersed Tunnel Tubes cost in Denmark, Sweden and Northern Germany approximately ($US) $500 million per mile. This is the cost for a four-chambered submerged tube underwater. Cost based on the completed Oresund Tunnel, Sweden to Denmark four-chambered tube completed in 2000 (project sheet attached) and the just announced Fehmarn Belt Tunnel connecting Denmark to Germany again four chambers for vehicle and trains, 11 miles long in 131 feet of water making it the longest ITT in the world. There are dozen more completed ITT projects that support this cost figure per mile enclosed in this packet.

SR520 is only 3.7 miles from I-5 to the Medina shore that would be only $1.8 billion dollars for a four-chambered tube from Medina to I-5.

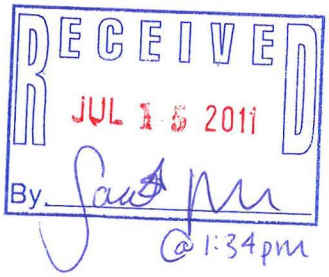
Governor Gregoire is telling us that the 520 design above ground and floating high above the water on stilts like a viaduct will cost $3.8 billion. That is $2 billion dollars more than a tube.

Even with additional ramps south of the ship canal dispersing traffic to the north and south before I-5 the total cost for an ITT still would be less than Governor Gregoire's $3.8 billion monstrosity.

Governor Gregoire has already spent $500 million on pontoon construction...perhaps they can be recycled into rain barrels?

Regards,

Louis Hoffer
1523 Parkside Drive East
Seattle, WA 98112
(206) 919-1664 - p



July 14, 2011

Seattle Mayor Mike McGinn
Seattle City Hall
600 Fourth Avenue, Seattle, WA.

RE: SR520 – Response to Final Environmental Impact Statement FEIS

Dear Mayor McGinn,

Today is the final day for responses to the FEIS for SR520 and the Bridge Replacement. I want to thank you in particular and all of our leaders listed below on this letter for the work that you have done along with community groups, community councils and individuals to bring a better solution to the Seattle 520 Corridor.

In the coming months as you all can imagine a chain of events (both legal proceedings and at the voting polls) will occur that will bring Olympia's decisions regarding the 520 Bridge to a forefront in King County and Seattle.

I wanted the four of you to have the information in this packet for you and your staff to be aware of actual costs per mile for an Immersed Tunnel Tube (ITT) projects planned and completed in other parts of the world to have as a comparison and hopefully as a tool for you to work from.

Over the past several decades China specifically but also most of Asia have been constructing ITT projects at a rapid pace. These projects are difficult to attach a quantitative figure as their labor costs are greatly reduced and their site priorities and challenges (if any) are different. However, these projects have proven to be an effective testing ground and have propelled projects with many environmental, topography, air quality, seismic and construction challenges forward in Northern Europe where cost for skilled labor is higher than in the Northwest here in the United States.

This attached packet shows from a large number of planned and completed ITT projects with high vehicle and in most examples light rail capacity a cost per mile (in US$) to be far less than what Olympia and Governor Gregoire have led the general public to believe.

The general cost per mile currently in Northern Europe is $500 million (US$) per mile. SR520 is 3.7 miles from I-5 to the Medina Shore. The base cost for a four-chamber tube underground and underwater from I-5 to Medina would cost $1.8 billion. Governor Gregoire currently is planning to construct a monstrosity for $3.8 billion.

Of course, on/off ramps south of the Montlake Ship Canal would increase the cost and improvement options such as a connection to Sound Transit and an additional 520-Downtown Seattle connection to the Mercer Street exit bypassing I-5 for Seattle/Eastside traffic could all be added at additional costs.

Japan has also benefited greatly from (ITT) developments in China and for the past decade leading up to the devastation that country has recently endured constructed all of it's main transit lines in Immersed Tunnel Tubes. As a result Japan had zero destruction in it's immersed tunnel tubes allowing all main transit lines to remain at full operation during and after the earthquake much the same way as BART did in San Francisco in 1989.

I propose and I have a team prepared to do a three-month study that would produce recommended designs and options and more importantly actual costs associated with each design option.

Capita Symonds is among the top three largest tunnel engineering firms in the world. I have attached some information on their company and I am in constant contact with them as well as the other top producers in the engineering world for this kind of work.

I have been a resident of Seattle for a long time, my wife's family was one of the pioneers of our city's commerce, transportation, banking and insurance industry. I and as I know the rest of you want a transportation solution for this corridor that not only is cost effective, safe for our environment but also something we can be proud of. A legacy to our families and to all Western Washington residents and visitors, something as monumental as our iconic Space Needle could be created in this corridor if we all just pause, think and look around and try to do the right thing.

I can be connected at anytime and would take great pleasure in assisting in any way I can with this endeavor. It is not too late.

Best Regards,

Louis Hoffer
1523 Parkside Drive East
Seattle, WA 98112
(206) 919-1664 p

cc. Senator Edward Murray
Representative Frank Chopp
Representative Jamie Pedersen

CAPITA SYMONDS

14th July 2011 **Our ref** . RCL/CS/900017-02

Mr M McGinn
Seattle City Mayor
Seattle City Hall
600 4th Avenue 7th Floor
Seattle, WA 98104
United States

Dear Mr McGinn

Lake Washington Immersed Tunnel proposal

I have been in discussion with Louis Hoffer about the possibility of constructing an immersed tube tunnel across Lake Washington as replacement for the current SR 520 bridge. From our discussions I believe that such a tunnel is possible and within the current state of knowledge for the design and construction of immersed tunnels.

The maximum depth of the lake appears to be around 190ft. This is comparable with the depth of the immersed tunnel that has recently been completed across the Bosphoros in Istanbul, so although the tunnel is deep it is not beyond the current state of knowledge. Costs depend on the specifics of the country and the site but to take the Øresund Tunnel between Denmark and Sweden as an example this was 2.55 miles long and accommodated a dual carriageway road and twin track high speed railway. The tunnel was completed in 2000 at a cost of US$960m or US$376m per mile. Allowing for around 60% inflation to 2011 prices the cost today would be around US$600m per mile. This is only an approximate calculation but indicates that a cost of US$550 – US$650m per mile should be achievable depending on differences between the US and European construction industries. This is inclusive of all the electrical and mechanical installations in the tunnel.

Immersed tunnels have been constructed across waterways where a bridge would be environmentally unacceptable. For example the first such tunnel in the UK at Conwy was built across the Conwy Estuary, an important salmon river, to protect the World Heritage site of Conwy as a bridge would have dominated the landscape. There would inevitably be some temporary environmental impacts during construction but these can be mitigated and generally a solution that is acceptable to al interested parties can be found. Once the tunnel has been completed the environment is returned to its existing condition.

Seattle is in a seismically active area and immersed tunnels are able to accommodate the movements associated with this. Many immersed tunnels have been built and performed successfully in such countries as Japan and Greece and the recently completed Bosphoros tunnel in Turkey is only 15 miles from the very active North Anatolian Fault.

An immersed tunnel is a particularly adaptable form of construction in that it can accommodate combinations of road and rail. It is relatively easy and cost effective to provide for future expansion in such a tunnel, for example by constructing bores to accommodate future light rail plans. Ground level intersections at each end of the tunnel allow for effective flow and dispersion of traffic into the adjacent road network.

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CAPITA SYMONDS

In summary, an immersed tunnel could provide an acceptable solution to the replacement for the floating bridge and approaches on the Seattle side that would provide a safe efficient transport corridor without long term detriment to the environment.

Yours sincerely

R C Lunniss
Consultant
For Capita Symonds Ltd

Tel +44(0)1342 327161
Fax +44(0)1342 315927
Email richard.lunniss@capita.co.uk

Sent from:	Jake Kennon
Address:	
City:	
State:	WA
County:	
Zip:	
Email:	jacob.kennon@gmail.com
Phone:	

Comments:
Hello, My name is Jake Kennon and I am a researcher for the Sightline Institute, a Seattle-based research group. We are currently doing analysis on the proposed SR 520 bridge replacement project and hope you can clarify a technical detail for us. In the recently-released Final Transportation Discipline Report for the project pages 92-93 cite data for existing traffic conditions (Daily Vehicle Demand Volume) on the bridge midspan. The map on page 92 puts the number at 115,000 vehicles, while the table on page 93 indicates only 101,700. First, does "Daily Vehicle Demand Volume" refer to the average across the whole week or just weekdays? Second, can you explain the discrepancy between the two numbers or point us to the proper person who can? Thank you in advance for any help you can provide. ---- Jake Kennon / Research Team Sightline Institute / 1402 Third Avenue, Suite 500 / Seattle, WA 98101 www.sightline.org / T.206.447.1880 / F. 206.447.2270

Laurelhurst Community Club

Serving Seattle's Laurelhurst community since 1920

July 13, 2011

To: Daniel Mathis, Washington Division Administrator Federal Highway Administration
Megan White, Director, Environmental Services Office, Washington Department of Transportation
Jenifer Young, SR520 Bridge Replacement and HOV Project Environmental Manager

Dear Mr. Mathis, Ms. White and Ms. Young:

The Laurelhurst Community Club (LCC) submitted a lengthy analysis of the SDEIS for the SR520 Bridge Replacement in April, 2011.

The final EIS for such project was published with over 1100 pages on June 20th, 2011.
While LCC has not scrutinized the entire report, we have selected findings that undermine the integrity of the final EIS and outline them below:

1. Introduction to the Project

a. Section 1 on Project Overview on Native American Tribes (page 6 of the Executive Summary, and 1-25 and 1-26 of the full EIS) reveals that the Preferred Alternative is not actually approved by the Muckleshoot Indian Tribe. The EIS is not tenable without their approval. The more than doubling of the original footprint may be completely unacceptable to their ancestral rights and covenants.
If this is the case, the western structure from the floating bridge to I-5 may have to be re-designed which would trigger another SDEIS to analyze such new impacts.

b. Section 1, page 3 (Executive Summary) notes the safety purpose of the project:
"The ever -present possibility of an earthquake in the Seattle area poses additional risk sin the SR520 corridor. The columns of the Portage Bay Bridge and both west and east approaches to the Evergreen Point Bridge are hollow and do not meet seismic standards."
However, on page 7, "When would the project be built?" T he EIS again states:
"The most vulnerable structures (the Evergreen Point Bridge and east approach)would be built in the first stage of construction."
Page 8 further states "However, the funding for the full corridor program falls approximately $203 billion short of the $4.65 billion total".
Last paragraph states," Should full funding not be available, the project would be phased, with the floating bridge and the landings comprising the initial construction phase.

Since the project was first proposed, the economic conditions, shortfall of funds will impact the ability of WSDOT to secure financing for re-building the most vulnerable parts of the corridor. The concerns by LCC for "phasing" of the project is discussed in the "alternatives" section #2.

2. Alternatives

Phased bridge building, Section 2.8, pages 2-74 and 2-75
"Due to the funding shortfalls, the FHWA and WSDOT still believe it is prudent to evaluate the possibility of phased construction should full project funding not be available by 2012. Accordingly, this final EIS discusses the potential for the floating bridge and landings to be built as the first phase of the SR520 , I-5 to Medina project. This differs from the SDEIS Phased Implementation scenario which included the west approach and the Portage Bay bridge in the first construction phase."

Exhibit 2-29 illustrates the plan for the "partial bridge" construction. It shows that the HOV lanes and shoulders built to the north section will end before the western high rise, and, page 2-76 states,
" To connect the western end of the floating span to the existing western approach, WSDOT would construct a new interim connection, four lanes wide and 1500 feet long, between the new west transition span and the existing west approach bridge "(exhibit 2-28)

"To address the potential for phased construction, the final EIS evaluates construction of the floating bridge and landings separately as a subset of a "full build analysis".

The Laurelhurst Community Club opposes any "partial bridge " that leaves a super structure of 116 feet width suspended mid air for an unpredictable term (best case is 2018 assuming collection of gas taxes, tolling of I-90 and non-existent federal dollars) while full funding becomes available to complete the Medina-I-5 project.

This "phasing" is likely to become a semi-permanent design for the SR520 re-build, which **was not considered** as "an alternative" in the State Mediation process, nor fully vetted for its impacts in the SDEIS, 2.4. Only general schemes were identified, but their environmental impacts were not analyzed in the SDEIS.

Specifically, LCC has these concerns about a "phased" construction plan:
.
a. Environmental damage caused to the habitat of Lake Washington and Union Bay with excess footprint structures (twice), noise, pollution and emissions from construction.

b. Traffic bottlenecks created on Lake Washington caused by the merge of 6 lanes from the Eastside to the narrower 4 lane existing structure, causing air/water pollution for fish, wildlife and adjacent neighbors. The two key benefits of widening the existing bridge profile, specifically, giving transit/HOV priority, and the ability to cross in a non-motorized mode, will be lost for the foreseeable future with this "phased", partial bridge, as these lanes will not be built.

c. Building the structure "twice" with interim exit ramps, construction bridges, pile driving, hauling excess waste and double the noise for local residents, especially in Laurelhurst and in Madison Park

d. Local traffic impacts from a "partial bridge" that will result from reduced capacity from the increased volume of vehicles, including HOV, plus general purpose merging into 33% fewer lanes. Back ups on Montlake Boulevard from 520 to University Village and NE 45th Street would deteriorate to over 35-45 minutes in the pm peak commute.

e. Funding for the fully built Preferred alternate is predicated on many unpredictable sources. Since the EIS and SDEIS were published, ballot measures limiting tolling funds from I-90 will be put to voters in November, in addition to the reduction in revenues from the long delay in tolling the existing SR520 itself.
These funding problems will no doubt exacerbate the limited construction funds for SR520 and result in partial bridge building, much farther out on the original timeline.
Thus, adjacent neighborhoods will endure the effects mentioned above for a longer duration than predicted in the EIS which does not include these deficient funding factors.

f. The other alternatives developed include an Alternative L which was only supported by one stakeholder, Mark Weed, a developer who represented the transportation committee for the Seattle Chamber of Commerce. Virtually, no other Seattle Mediation group voted to even include this option, and several, times requested that WSDOT drop it, and include other acceptable alternatives. This was vetoed in the process, and the L alternative continued in the process. It is **not** listed on pages 2-10 or 2-11 in the full EIS, only mentioned that it was ruled out in advance of the EIS on page 2-26.

g. One option, M, was offered as a more viable, economical, and less environmentally adverse alternative, but was never fully included. It was considered "too damaging and expensive" even thought the cost for the tunnel under the Montlake Cut was $49.5 million, not an extra $1.5 billion that was given to the Legislative Workgroup the night before the vote on the alternatives. The tunnel M was most acceptable to adjacent Seattle neighborhoods to reduce the impacts from a higher and wider super sized concrete viaduct design.
Its omission leaves out a key alternative that offered greater mobility and lesser neighborhood impacts.
Plan M offered the only alternative to have local traffic flow without building a second bascule bridge, providing a separated egress to and from SR520 from local traffic.

h. Location of the centerline of the Preferred Alternative new bridge total structure. Chapter 2, page 2-38 describes the location of the Preferred Alternative as location 190 feet north of the existing bridge. Through the mediation process with WSDOT, the adjacent communities agreed on a settlement that the western structure centerline would be no greater than 100 feet moved northward. Clarification of this statement is requested by LCC of WSDOT to ensure that this alignment corresponds with their overall agreed upon scheme since it is not fully depicted in the shortened exhibits showing the old vs. new structure's overall footprint.

3. Traffic analysis and tolling

There is a fundamental assumption that is illogical in the traffic analysis with regard to tolling. Page 18 of the Executive Summary notes that the No Build Alternative used for traffic comparison would not be tolled, yet the comparison is to a 6 lanes that would be tolled. The comparison should logically compare a tolled 6 lane to a tolled 4 lane to measure any difference in travel times. This is a simple math issue, that is a fallacy in the SDEIS traffic assumptions and subsequent analysis, which is carried into the EIS.

Page 32 of the Executive Summary states:
The project would improve mobility on SR520 and would meet local traffic concurrency standards. WSDOT have identified several potential intersection improvements that may benefit local traffic operations and will work with SDOT to determine their effectiveness. (section 5.1 of the EIS). Most of the EIS analyses center exclusively around the Arboretum traffic, and not a fully scoped to include local street impacts in adjacent Seattle neighborhoods and intersections . In fact, Peter Hahn of SDOT has repeatedly requested a full analysis in regard to the local traffic impacts for the Preferred Alternative from Madison St to NE 75th St, and this has not been accomplished before the EIS was issued.
Thus, the EIS is insufficient, and the Preferred Alternative local traffic impact data must be included to identify its effectiveness as a design for a transportation corridor for westside users, and local streets and neighborhoods.

4. Visual Quality

The Executive Summary has a section on visual quality on page 42, and a section 5.5 , pages 5.5. 1- through 5.5-25, in the complete EIS. No viewpoints/photos from the Laurelhurst neighborhood (any views looking south onto the new structure) are included in the EIS. This omission is completely unacceptable, and we consider this a serious omission from the EIS. The new section bridge will be built entirely to the north, directly impacting the visual quality of homes in the Laurelhurst neighborhood. Photos were submitted by the legal graphic photographic (Legal Media, Inc) in our LCC comments of April, 2011. The exclusion of any such photos from their firm, and WSDOT identifying the visual impacts on affected Laurelhurst residents renders the EIS incomplete.
The visual impacts of the new Preferred Alternative are significant and will affect the value of the homes in the view corridors in a negative way, and mitigation should be required.

5. Air Quality

The Executive Summary (page 50) states that " all SDEIS alternatives reduced congestion and improved traffic speeds" resulting in a "slight improvement in air pollutant emissions compared to no build" See point #3 that simply tolling the "no build" might also reduce congestion and emissions, although this data is not included in the EIS.

With any lengthy "partial bridge" or segmented bridge, the results are actually an increase in congestion from merging 6 into 4 lanes. In addition any advantage gained from the HOV lanes and non-motorized crossing of bikers and pedestrians are lost in the "phased bridge". There will logically be **more** congestion sitting in a traffic bottleneck on top of Lake Washington as HOV's, and SOV lanes merge and the result is an **increase** in emissions, and **worsening** of air quality for the habitat of Lake Washington and the people who inhabit the adjacent neighborhoods of Laurelhurst and Madison Park. Thus, any partial bridge is not acceptable as it creates more pollutants for an indefinite period.

6. Water Resources

The Executive Summary (page 52) states that the Preferred Alternative will increase the amount of land covered by pollutant-generating impervious surfaces within these areas by 35-45% to complete this project. The construction effects will result in direct disturbance of water bodies, including turbidity and spill of pollutants. Dewatering will take place and severely impact the wetlands (page 53).
In addition, of the 3 alternatives (page 54, Ecosystems)," the Preferred Alternative would result in the most wetland shading in the western approach" "The Preferred Alternative and all of the SDEIS options would affect wildlife by permanently removing vegetation and wildlife habitat, and by increasing shading".
Further, on page 56, the Executive Summary notes, "Construction would also result in areas with reduced fish habitat functions, primarily due to increased shading by work bridges and barges. " The Preferred Alternative and SDEIS options would require substantial in-water pile driving to build construction work bridges in shallow water areas that cannot be accessed by barge. ".....the underwater sound levels generated.. can disturb or alter the natural behavior of fish and aquatic species and cause mortality....

The mitigation is not clear in the EIS, and as such, is unacceptable. On page 56 in the Executive Summary, it states, "WSDOT will continue to work with the City of Seattle and the UWA to develop mitigation planting strategies to offset construction effects on shoreline habitat in Portage Bay and Union Bay."

This statement is woefully inadequate for the final EIS. WSDOT should have a specific analysis and plan to save the irreplaceable habit of the wetlands and its ecosystems in Portage and Union Bays **before** issuing this EIS.

In summary, the EIS issued in June 2011 for the SR520 re-build is inadequate in terms of its omissions of the impacts for the affected water and air quality environments, local traffic impacts, lack of analyses of any "partial bridge" phasing, the incomplete predictability of funding for the entire corridor, omission of Plan M tunnel alternative, the lack of final approval from the affected tribes, and the omission the visual impacts of the Preferred Alternative on the view sheds from northern residents where the new bridge will be built . It also is not clear where the northern footprint is compared to agreements with adjacent communities on the western approach.

Building a new transportation corridor through these environmentally sensitive ecosystems and densely populated neighborhoods, requires a better design solution, and the EIS does not provide such an optimal solution.

The Laurelhurst Community Club requests that the Federal Highway Department and WSDOT complete the above analysis before issuing any Record of Decision for the final design of the replacement of the SR520 bridge corridor.

Sincerely,

Colleen McAleer, Mediation Representative
Transportation Chair
3137 West Laurelhurst Drive NE
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cc. Governor Christine Gregoire
Paula Hammond, Secretary of Transportation
Randolph Everett, Federal Department of Highways
Senator Ed Murray
Speaker Frank Chopp
Senator Scott White
Representative David Frockt
Seattle City Council President Richard Conlin
Mayor Mike McGinn
Seattle Councilmember, Transportation Chair, Tom Rasmussen
Seattle City Council members

FEIS

Howery, Stacey (Consultant)

From: Shawn Leeth [shawnleeth@gmail.com]
Sent: Friday, June 10, 2011 8:48 AM
To: SR 520 Bridge Replacement & HOV Project
Subject: West side of Lake Washington expansion

Categories: FEIS

I am curious as to whether or not the 520 bridge replacement program scope includes lane expansion on the west side of Lake Washington? On your web site there is talk of expansion on the east side (though it is 3 lanes already right up until the bridge) but no talk about the west side. Is the plan for now to increase the bridge to 3 lanes but bottleneck the west side down to 2 lanes?

I am sure this isn't the plan because you wouldn't spend $5 billion to create a bottleneck but I can't seem to find information on west side expansion. Thank you.

-Shawn Leeth

From: Stewart, Holly (Consultant)
Sent: Monday, June 27, 2011 12:13 PM
To: Gardner-Brown, Tessa (Consultant); Young, Jenifer (Consultant)
Cc: Howery, Stacey (Consultant)
Subject: FW: SR 520 technical report drafts: air quality, noise, and vibration

Hi Tessa and Jenifer -

Here's the agency comment that I mentioned in our meeting this morning. It is actually from Stewart Toshach at NOAA. This is being uploaded to the comment database today.

--Holly

-----Original Message-----
From: Brandt, Sarah (Consultant)
Sent: Monday, June 27, 2011 10:57 AM
To: SR 520 Bridge Replacement & HOV Project
Cc: Haner, Sara (Consultant); Stewart, Holly (Consultant); Gitlin, David (Consultant)
Subject: FW: SR 520 technical report drafts: air quality, noise, and vibration

Hi team,

This question came to me last week from our NOAA NWFSC folks, and Jenifer asked that we include it (and the response that I wrote with Jenifer's review) in the official NEPA comment record for the FEIS. I assume we could take doc control credit for it, too...

Thanks!
Sarah

-----Original Message-----
From: Brandt, Sarah (Consultant)
Sent: Wednesday, June 22, 2011 2:50 PM
To: 'Stewart Toshach'
Cc: Walton W Dickhoff; jim herkelrath; Bob Lohn; Matthew Erwin; Ruth, Kerry
Subject: RE: SR 520 technical report drafts: air quality, noise, and vibration

Hi Stewart,

Nice to hear from you, and thanks for your question. The answer is that the technical reports are not included in the analysis presented in the Final EIS, except in the comment response section addressing NOAA's letter (we reference them there when we note that additional technical analysis is underway to best address your concerns).

I spoke with Jenifer Young, our NEPA lead, and she explained that often this kind of technical analysis, which is focused on one particular property and beyond the level of detail required by NEPA, is more appropriately considered part of detailed mitigation and/or real estate discussions. Even though the technical reports are not included in the NEPA documents, they will certainly be relevant to our decision-making about appropriate mitigation for the NWFSC. As you know, the final mitigation package will also ultimately be committed to in the Section 106 Programmatic Agreement.

Thanks for the confirmation about your external review of the reports. We'll look forward to hearing the results.

Hope you're enjoying the start of summer!
Sarah

-----Original Message-----
From: Stewart Toshach [mailto:Stewart.Toshach@noaa.gov]
Sent: Wednesday, June 22, 2011 1:52 PM
To: Brandt, Sarah (Consultant)
Cc: Walton W Dickhoff; jim herkelrath; Bob Lohn; Matthew Erwin
Subject: Re: SR 520 technical report drafts: air quality, noise, and vibration

Sarah,

I hope you can help with a quick question on the draft technical reports.

I was looking through the Final EIS and attachments to see how this material has been included and so far have not seen it. Since the EIS and the Noise Discipline report are long and my search was not exhaustive I might have missed it, or maybe it is not included? Please let me know if the techical reports are a part of the Final EIS and if so where is the material located?

Also, to confirm what we have mentioned in meetings with WSDOT, we are arranging an external technical review to assist in our review of the technical draft reports.

Thanks,
Stewart

Brandt, Sarah (Consultant) wrote:
>
> Good afternoon,
>
> As promised, here are our draft technical reports on air quality,
> noise, and vibration related to the Northwest Fisheries Science
> Center. We would like your review of these documents so that we can
> respond to your comments/questions. We’re doing our best to schedule
> our next workshop, which looks like it may occur during the week of
> April 18. Ideally, we’d be able to discuss these reports and your
> input at that workshop. We’ll be in touch again soon to get this
> meeting confirmed and scheduled.
>
> Please be in touch with any questions. Thanks!
>
> * *
>
> Sarah Brandt
>
> Project Communications
>
> SR 520 Bridge Replacement and HOV Program
>
> Washington State Department of Transportation
>
> 206-770-3637 direct | 206-770-3500 main
>
> 600 Stewart Street, Suite 520 |* *Seattle, WA 98101
>
> Visit us at our Web site:*
> http://www.wsdot.wa.gov/projects/sr520bridge/*

>

Washington State Department of Transportation

OFFICE OF THE SECRETARY
MS: 47316

Referral Slip

Letter Ref No.
2703182

DATE: July 20, 2011

TO: Julie Meredith - SR 520 Bridge Replacement

FROM: Paula Hammond **E-MAIL:**

NAME: Renate Puich **PHONE:** 206-323-1199

SUBJECT: SR 520 bridge replacement project and impacts to Montlake area

ACTION NEEDED: Please prepare a DRAFT response for David Dye's signature. Email the draft response to HQ Customer Service; include this referral email with the draft response.

PLEASE CC:

REFERRAL FROM:

PLEASE NOTE:

DUE DATE: **Wed, Jul 27, 2011**

(July 8TH - 2011) RE: 520 PROJECT

Dear Paula Hammond, P.E.
Sec. of Transportation - WSDOT:

I am a native of Seattle and our parks are world class. Very few cities in the world host as beautifully natural parks as Montlake or capital Hill, even the U.W. district bridges and University are very old and beautiful. Accommidateing Bellevues burgeoning traffic issues with the few options that seem quite unimaginitive in their designs, is an assault on the beauty of the regions I have mentioned. People in Montlake, like myself, who are homeowners appear to be the big losers in this massive and invasive plan. What happened to engineering ideas from the fine designers of the U.W. educated individuals?

Why are the only engineers from WSDOT? Bellevue benefits and is enabled to continue as business as usual while our antiquated roads, bridges and parks are about to be disrupted terribly. There is little or no mention of the real impact to Montlake. Shunting cars with one driver through our neighborhoods at our expense seems to be WSDOT's prefered plan: (a title most inappropriate). I think Montlake has been left out of your Final Impact nonsence and that better engineers should be consulted to bring less invasive plans to this project.

Thank you - Renate Finch

224 E. McGraw St.
Seattle, WA. 98112

(206) 323 1199

Ravenna-Bryant Community Association
6535 Ravenna Ave. N.E.
Seattle, WA 98115

July 7, 2011

U.S. Secretary of Transportation Ray LaHood
Federal Highway Administrator Victor Mendez
1200 New Jersey Avenue S.E.,
Washington D.C 20590

Director Daniel Mathis
Washington Division, FHWA
711 Capitol Way, # 501
Olympia, WA 98501

RE: Omissions in the Washington State Department of Transportation's SR 520 FEIS

Dear Secretary LaHood, FHWA Administrator Mendez, and FHWA Division Director Mathis:

The Washington State Department of Transportation's (WSDOT) Westside SR 520 FEIS, released in June 2011 was sent to the USDOT/FHWA for review/approval. Under current NEPA/EIS law, there is no opportunity for public comment on 3 serious omissions. Our Ravenna-Bryant Community Association (RBCA) has been advised that public comments should be made directly to the relevant federal agencies. We look forward to your response and positive solutions to our three concerns with the SR 520 FEIS, and in the SR 520 Federal Record of Decision. This letter was reviewed and authorized by the RBCA Board of Directors on July 5, 2011 at our regular Monthly Meeting.

RBCA's Board represents a community of about 23,000 residents in the north-end of Seattle, Washington. Our Board is writing to request resolution to 3 FEIS issues. Two are major omissions in the SR 520 FEIS and relate to 4(f) Washington Arboretum Park issues. The other is our request in previous SR 520 EIS and SDEIS comments to WSDOT, to include a SR 520 Corridor Management Agreement. They are discussed below. (Our RBCA Board and community have been represented for the past 5 years on the SR 520 citizen's review process by Virginia Gunby, a former WSDOT Transportation Commissioner, 1973-79.)

The RBCA is opposed to the negative impacts of a recent revised design for the SR 520 Montlake exit. It is not discussed in the Sec.9 4(f) of the SR 520 FEIS. RBCA is concerned over WSDOT's plans to use the Olmsted-designed historic Lake Washington Boulevard as a Westside SR 520 freeway area exit ramp. This proposed design would allow exiting westbound SR 520 vehicles to travel south through the length of the Washington Park Arboretum. If built, it would permit Westbound exiting SR 520 vehicles to turn left from the SR 520 Montlake exit ramp and cross a new North/South lid connector (one block long) road on the proposed new landscaped lid near Montlake Boulevard. The park-like lid also has Pedestrian/Bicycle Paths and nearby Transit Stops. We find this new feature could be a safety hazard for the users of all of these modes, and a major cause for backups on the SR 520 Westbound exit ramp (See Attachment *SR 520 FEIS 5.1-45*). This feature was added by WSDOT after the SDEIS, from recommendations made by a transportation consultant to the City of Seattle during their review of the project.

Lake Washington Boulevard is an Olmsted-designed, historic two-lane road through our unique Washington Park Arboretum, with a south exit at a major city arterial, at East Madison Street. This recent design change was made after the SR 520 SDEIS process, and had little analysis of future traffic, or the short and long term environmental impacts. There was no opportunity for written public comments. RBCA's concern is that this change continues to cause further environmental degradation of the Arboretum. The increase of traffic, noises,

and fumes creates hazards for park visitors trying to cross the Washington Park Arboretum Boulevard all day long. It reduces the value for the Arboretum visitor's enjoyment of what should be a unique and valuable personal experience in a unique, urban park-setting.

Another 4(f) issue -- after tolling begins and the Westside construction is completed -- is the WSDOT 's operations on the new Montlake exit route that could be amended to allow changes and a new category of SR 520 HOV lane users to enter and exit SR 520 using the new lid road to reach the adjacent Montlake SR 520 HOV/Transit ramps. A new WSDOT post-FEIS HOV operation's policy could even allow single occupant vehicles, to "buy into" the HOV lanes. WSDOT has discussed how the HOV lanes could become "HOT" lanes in the future. The change in SR 520 operations would increase revenue from SR 520 tolls for WSDOT and is being considered for an easy post-construction operational change although not discussed in the FEIS. FHWA should discuss the potential of this operational revision and its long term impacts, and reserve the right to review this potential decision, in a future Environmental Review of this decision. The current plan for boulevard speed-reducing cushions will not reduce the number of vehicle trips on Lake Washington Boulevard, given the "short-cut" the Boulevard provides, to and from SR 520, to Capitol Hill, First Hill and to South Seattle.

The good 520 news for RBCA and our regional public is that WSDOT selected the preferred design option for SR 520 which includes finally removing the two 1963 built SR 520 on- and off-ramps that are currently operating over our park's freshwater wetlands, within the 230 acre historic Arboretum Park. These on- and off-ramps to and from the SR 520 limited-access freeway have, for 48+ years, caused significantly increased auto use on the two-lane Lake Washington Boulevard. The flow of vehicles to and from the two SR 520 ramps has seriously impacted the adjacent park environment and the park users on both sides of the Boulevard. The Lake Washington Boulevard was originally designed for 4000 cars per day, primarily Washington Arboretum Park users. We support the removal of the ramps which should be approved as part of the SR 520 FEIS and the Federal Record of Decision.

Lake Washington Boulevard is a 4(f) Historic and Park Resource. On March 22, 2010, the City of Seattle Parks and Recreation Department submitted a SR 520 SEIS review with 4 pages of comments on the SR 520 Supplemental EIS which stated on page 2 "*that Lake Washington Blvd is referred to in the SR 520 SDEIS as a city street throughout the Supplemental Draft Environmental Impact Statement. The SDEIS "4f" evaluation fails to identify Lake Washington Boulevard as either a historic resource or a park and recreation resource. The officially designated park boulevard is a 204-acre, 9.2 mile-long linear park wholly owned by the City and under the jurisdiction of Seattle Parks and Recreation. It is a crucial element of the 1903 Olmsted Plan for the Seattle boulevard system, sometimes referred to as the 'Emerald Necklace'". (NOTE: The SR 520 FEIS, repeats the same omission.)*

The RBCA urges USDOT and FHWA to treat Lake Washington Boulevard as a protected 4(f) Park property. Why hasn't the Boulevard been listed in either the 520 SDEIS or the FEIS 4(f) evaluation, as a valued, historic, Olmsted-designed public park and recreational resource? WSDOT should be asked for an evaluation of the impact of this post-SDEIS lid load design change, and to evaluate the new lid road's future and potential impacts on Lake Washington Blvd. This crucial change, made late in the SR 520 design, must be evaluated in the SR 520 4(f) section, and not be overlooked again in the SR 520FEIS and the Federal Record of Decision. In addition, under US Executive Order EO 1318, the lead agency should report on its future progress in monitoring and assessing the effectiveness of project mitigation.

Weak traffic calming measures are contained for Lake Washington Boulevard in the "Mitigation Measures" found in an agreement between the city of Seattle, SDOT, WSDOT and the Arboretum Botanical Gardens Committee (ABGC). The FEIS states that the "reconstructed SR 520, with the ramps removed, will have "less traffic than "No Build," but is not quantified or considered when the new post-SDEIS landscaped lid road was added. In the future, the RBCA's SR 520's citizen oversight of SR 520 will continue to seek our major objective to support the Preferred Alternative Westside 520 Design and the long-term protection of our Olmsted-planned and designed publicly owned historic Washington Park Arboretum, with the reduction of traffic on Lake Washington Boulevard. This issue must not be overlooked as a Federal "4(f)" issue.

The 4(f) Federal law can only be applied if the project sponsor has listed the 4(f) issue in its mitigation information in the project's FEIS. The public has never had a chance to offer comments to WSDOT after 2010 SDEIS on the impacts of the new road on the lid, or on the design or the exit ramp's left turn road. We are opposed to revisions in the SR 520 HOV lane operations in the future to move more and more SR 520 vehicles entering and exiting onto Lake Washington Boulevard surpassing the vehicle traffic that currently exists from the soon to be removed existing ramps.

Our final issue is the lack of WSDOT response to our many DEIS/SDEIS requests for a new WSDOT policy to adopt an SR 520 Corridor Performance Management Agreement. Over 10 years ago, during WSDOT's "Translake Study," WSDOT received a USDOT Grant of about $850,000 for an SR 520 Corridor Management Agreement Study, to study the benefits of such a new program. The study was completed, and is at the Puget Sound Regional Council (RTPO's) Library gathering dust. There has been no application of the final study's information by WSDOT, or the SR 520 Staff. If the findings were implemented, it would improve the overall, long-term sustainability and multimodal performance of WSDOT's rebuilt SR 520. It could transform WSDOT long-term management of the new reconstructed SR 520 corridor. Our suggestions for long-term SR 520 multimodal performance monitoring and reporting this information regularly to the public could help to meet the objective of constantly seeking ways to improve the new SR 520 corridor's overall performance. Our objective has been to achieve a new thoughtfully managed, integrated, multimodal, environmentally friendly, limited access state corridor. There would be an agreement between the relevant communities, transportation, business, and other public Interests. The overall agreement's goal would be to constantly improve the rebuilt SR 520 corridor's long-term operations and the multimodal performance in serving all of the users, and being a good neighbor to the nearby communities. WSDOT's 2011 FEIS does not include our 2008 and 2010 EIS recommendation for new Corridor Performance Monitoring. If used in the future it would result in significant reductions in the long-term impacts, improve SR 520's performance and decreased public costs while adding community support and involvement.

Thanks for your consideration of our letter's issues, and for any assistance and wisdom you and your staffs can provide to our community on these important transportation and environmental issues. There is little time left before Federal DOT Action on the WSDOT proposed June 2011 SR520 FEIS and the Record of Decision is completed. The RBCA looks forward to your responses on the Federal 520 FEIS Review and the SR 520 Record of Decision.

Sincerely,

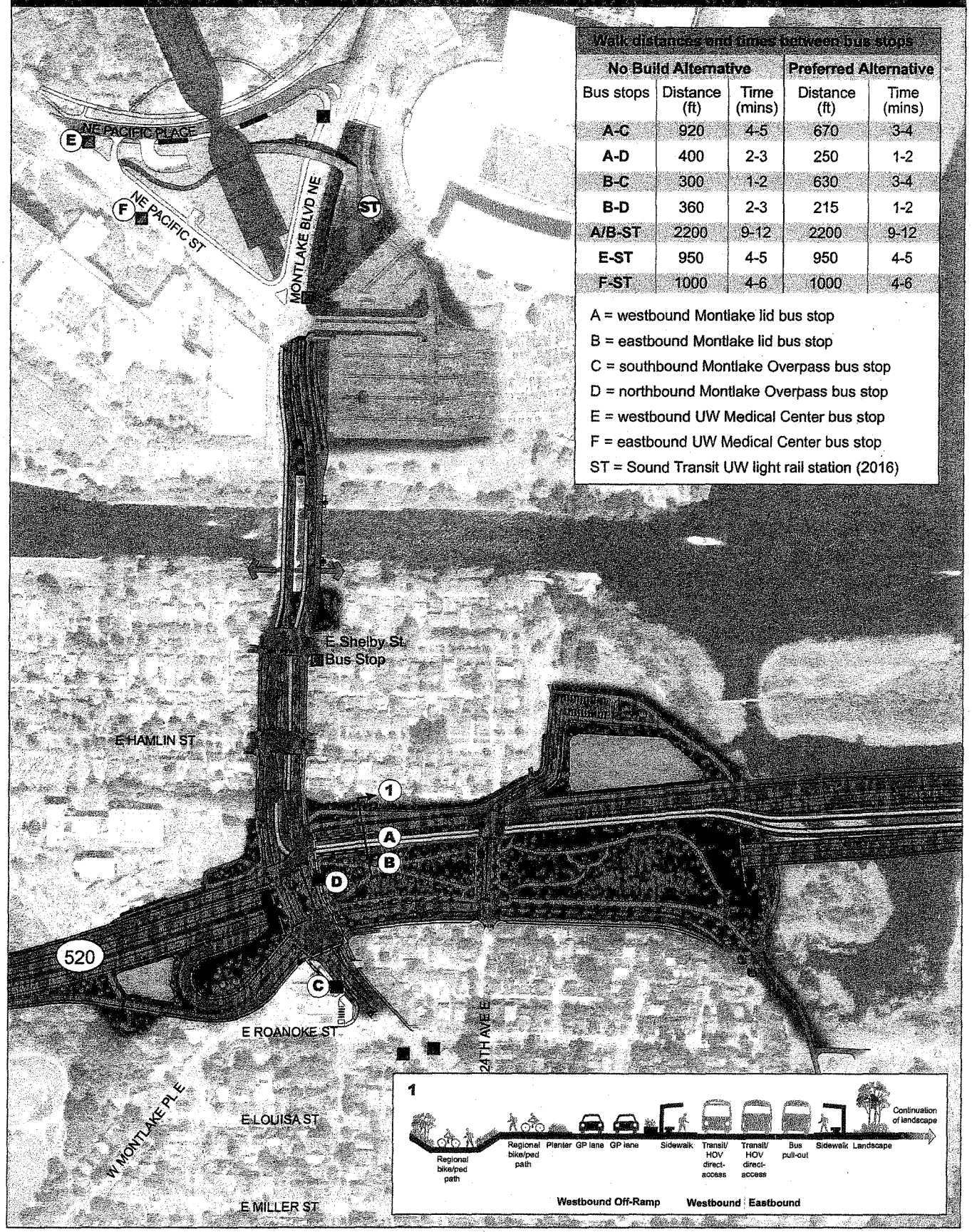
Sarah Swanberg
President, Ravenna-Bryant Community Association
www.ravennabryant.org
rbcasarah@gmail.com

Attachment: 5.1-45 SR 520 June 2011, WSDOT - FEIS,

cc: Governor Christine Gregoire,
PO Box 40002
416 Sid Snyder Ave. SW, Ste.20
Olympia, Washington 98501

File:FHWA FEISR5207511.doc

Exhibit 5.1-28. Preferred Alternative Transit and HOV Facilities within the Montlake Area



Walk distances and times between bus stops				
	No Build Alternative		Preferred Alternative	
Bus stops	Distance (ft)	Time (mins)	Distance (ft)	Time (mins)
A-C	920	4-5	670	3-4
A-D	400	2-3	250	1-2
B-C	300	1-2	630	3-4
B-D	360	2-3	215	1-2
A/B-ST	2200	9-12	2200	9-12
E-ST	950	4-5	950	4-5
F-ST	1000	4-6	1000	4-6

A = westbound Montlake lid bus stop
B = eastbound Montlake lid bus stop
C = southbound Montlake Overpass bus stop
D = northbound Montlake Overpass bus stop
E = westbound UW Medical Center bus stop
F = eastbound UW Medical Center bus stop
ST = Sound Transit UW light rail station (2016)

- Bus Stop (Local Route)
- Bus Stop (SR 520 Route)
- Bus Stop (Local and SR 520 Route)
- Layover Area
- HOV Lanes
- Bicycle/Pedestrian Path
- N

Ravenna-Bryant Community Association
6535 Ravenna Ave. N.E.
Seattle, WA 98115

July 7, 2011

Administrator
Federal Highway Administration
1200 New Jersey Avenue S.E.
Washington, D.C. 20590

Director
Washington Division, FHWA
711 Capitol Way, # 501
Olympia, WA 98501

RE: 4(f) Evaluation for SR 520 Project: I-5 to Medina
Replacement land needed for park land taken

Dear Administrator and Regional Director:

We are very concerned about the damage to the Arboretum as a result of the new Evergreen-Montlake Bridge. The main off setting benefit of the project to the Arboretum is the removal of the R.H. Thomson ramps-to-nowhere and of the Arboretum ramps and restoration of the wetland. Your Record of Decision needs to take the next, critical step: requiring a commitment of the "WSDOT Peninsula" to arboretum use.

The 4(f) Evaluation, contained in the Final Environmental Impact Statement ("FEIS"), pp. 9-116 and 9-117, explains that federal regulations require "all possible planning, as defined in Part 774.17, to minimize harm to Section 4(f) property." Part 774.17 defines the quoted phrase to mean that "all reasonable measures identified in the Section 4(f) evaluation to minimize harm or mitigate for adverse impacts and effects must be included in the project" and that these measures include "... replacement of land or facilities of comparable value and function; or monetary compensation to enhance the remaining property or to mitigate the adverse impacts of the project in other ways." The Section 6(f) Evaluation replaces land taken for the Ship Canal and Arboretum trail. The Section 4(f) Evaluation identifies no other replacement land --- not even making a permanent commitment of the "WSDOT Peninsula" for

arboretum and botanical garden use. The furthest it goes is to "evaluate the possibility" and "explore the feasibility" of its transfer. FEIS, p. 9-124 and 125. Thinking is not doing. It's a first step, but not enough, for environmental mitigation. To have effect, the mitigation must take place on the ground where the damage occurs and replacing the land taken by supplying land in the vicinity that the project renders surplus. That would substitute wildlife habitat for habitat taken.

The project takes park land contiguous to the Arboretum that was long part of it., e.g. parts of Foster and Marsh Islands, McCurdy Park, East Montlake Park, and the University's Canal Reserve open space. Return of the WSDOT Peninsula would make up for park and open space lost and would prevent shrinkage. With natural areas, size matters; larger sizes allow more variety and sustain populations that smaller do not and protect the ambience of the inner areas. Returning the WSDOT Peninsula would certainly be a "reasonable measure." Cash to the City or the University is not at all equivalent here. Cash can be spent anywhere for capital projects, even for indoor museum exhibits or multiple minor projects spread throughout the city-wide park system. It may give a temporary gain, but not the permanence or natural impacts that replacement land will do. It would diminish the patrimony of parks passed on to future generations.

The 4(f) Evaluation is deficient in failing to state that the WSDOT Peninsula will be conveyed as replacement land to the City and/or the University (subject perhaps to recognition as an offsetting special benefit in state eminent domain proceedings). The Evaluation also omits 4(f) properties and understates the adverse environmental impacts of the project on the Arboretum. The reconveyance of the WSDOT Peninsula would cure those deficiencies as well.

This letter was reviewed and authorized by our Board of Directors at its meeting on July 5, 2011. Our community association is located about a mile and a ½ north of the Arboretum. Many of our residents enjoy visiting it and our bird watchers especially cherish it. It is a City treasure that needs all the protection that it can get.

Yours truly

Sarah Swanberg
President

University District Community Council
4534 University Way N.E.
Seattle, WA 98105

July 13, 2011

Victor Mendez
Administrator
Federal Highway Administration
1200 New Jersey Avenue S.E.
Washington, D.C. 20590

Daniel M. Mathis
Director, Washington Division FHWA
711 Capitol Way # 501
Olympia, WA 98501

RE: 4(f) Evaluation, SR 520 Bridge Replacement ... Project

Dear Administrator Mendez and Director Mathis:

The 6(f) Evaluation is solid, but the 4(f) Evaluation is a dud. Compare the 4(f) Evaluation to an electronic toy that comes in a package of parts and requires assembly; if the assembled parts doesn't work when turned on, there's something clearly wrong.

Here, the SR 520 project will take a large amount of park land that lies outside of the Section 6(f) properties, e.g. acres from McCurdy Park, East Montlake Park, and the informal park between Montlake Boulevard and 24th Avenue East just north of the Montlake off-ramp from the westbound lane of SR 520. Yet, the 4(f) Evaluation offers nothing in return south of the Montlake Cut.

WSDOT owns acreage that once was part of the Arboretum and cleaves like a wedge into the Arboretum. It is now used for the arboretum ramps, which WSDOT plans to remove after construction is complete. That acreage needs to be returned to Arboretum use as replacement for the land taken by the SR 520 project outside the 6(f) properties and as mitigation for the adverse impacts of the new structure on the Arboretum.

This is so obvious and so important to the Arboretum that it can't be ignored. The 4(f) Evaluation does not show "... all possible planning to minimize harm to such park[s]..." as required by 23 USC 138 when it lets WSDOT keep that area for other highway uses, lease or sale. The 4(f) Evaluation can't be approved "as is." The Federal Highway Administration has to correct the WSDOT's failure in its record of decision, much like a parent often has to reassemble a toy that was not put together correctly.

Yours truly

Sharon Scully

CC: Paula Hammond, Secretary
WSDOT

Washington State Department of Transportation

OFFICE OF THE SECRETARY
MS: 47316

Referral Slip

Letter Ref No.
2703162

DATE: June 20, 2011

TO: Julie Meredith - SR 520 Bridge Replacement

FROM: Customer Service **E-MAIL:**

NAME: Doug Seaton **PHONE:**

SUBJECT: Difference between new proposed SR 520 bridge plan and current bridge

ACTION NEEDED: Please send a written response directly to the constituent. Return referral email and scanned copy of signed response to HQ Customer Service.

PLEASE CC:

REFERRAL FROM:

PLEASE NOTE:

DUE DATE: **Mon, Jun 27, 2011**

Dear Washington State Department of Transportation:

It is evident that there is a new 520 bridge plan being pushed for at this time. You might have already received some negative feedback on the idea of tolling the 520 to contribute to the lack of funding that you are currently facing.

As someone who has commuted the 520 on a regular basis, I have some thoughts on the proposed bridge plan that is currently accepted. I also read somewhere that the new plan is a result of 14 years of work and analysis.

With this said, I have a few ideas before any further action is taken on the 520 bridge.

The 520 takes a significant and distinguishable beating every time there is a windstorm on Lake Washington. It is distinguishable because the wind and the water beat on one side of the bridge, and leave the other side without any trouble. Because of this, the bridge acts as a wind and storm dam in any case of a storm on Llake Washington. This makes the bridge's wear and tear somewhat abnormal.

The new proposed bridge is basically the exact same design as the old 520. My question is: What is the significant difference between the new bridge plan and the 520 as it already is? Especially for the amount of money that is going into it, and the idea of being tolled every day on the way to a tough day at the office.

Perhaps a cheaper idea would be implementing a ferry terminal to cross the lake at this location. While it would be much slower, the result might be the same as the traffic tolling idea. The majority of people would just take I-90 or go around the lake.

Or, my favorite idea is getting away from the floating bridge and making a larger and stronger bridge that sits above the water, and would last much longer than another 520 floating bridge with a couple of differences that aren't all that noticeable. On top of this, a larger and more majestic bridge could add another feature to the Seattle skyline. Perhaps even a great place for a firework display on the 4th of July.

It sounds like a lot of money and a lot of trouble for no significant difference. Not to mention the winter storms of Lake Washington will beat the new bridge in the same way they did the old one, shortening its lifespan as well. The Washington State Department of Transportation should go big or go home for the kind of money and attention a new 520 draws.

I think the new 520 should resemble the Ed Hendler (Pasco-Kennewick) Cable-stayed Suspension Bridge in Pasco, the Worli Sealink in India, or some other kind of creative cable-stayed bridge design.

Thank You for your consideration,

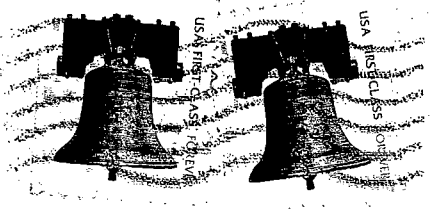
Doug Seaton

Doug Seaton

P.S. Let's not forget the Narrows Bridge incedence in 1940...

11910 83rd PL NE
Kirkland, WA 98034

SEATTLE WA 981
15 JUN 2012 PM 7 L



4326

Washington State Department of Transportation
310 Maple Park Avenue SE
P.O. Box 47300
Olympia, WA 98504-7300

SB504$7300

Seattle City Light

Memorandum

DATE: June 29, 2011

To: Stephanie Brown & Kittie Ford

From: Laurie Geissinger and Mary Junttila

Subject: SR 520 Final EIS - Seattle City Light Comments for City of Seattle Letter

Upon review of the FEIS, following are electric utility considerations that need to be noted in decision making regarding project alternatives/options, mitigation planning, and interagency agreements for the SR 520 Bridge Replacement and HOV Project. These comments reflect planning assumptions made by project engineers, SCL, and discussions between the parties.

- Power to the floating bridge will be provided by PSE.
- For the new bridge, SCL electrical service will only be required for the section of SR 520 from I-5 to the West Transition Bridge.
- Currently, SCL electrical service to the bridge is located near the Museum of History and Industry (MOHAI) parking lot. The service is fed from SCL's University Substation via Feeder No. 2625 at 26 kV. When electrical service is cut from MOHAI, SCL will "relocate" the end of the feeder to outside the project area.
- University Substation is the only SCL substation currently serving this area.
- With assistance from SCL, project engineers are determining and analyzing options for a second service from SCL, in addition to the existing single feeder. Additional service from the East Pine and University Substations has been discussed.
- SCL has noted concern to project engineers about the option of using a second feeder from University Substation to serve as backup service to the project. City Light believes this to be an inadequate means of backup service. In addition to the existing electrical feeder from University Substation that currently serves the area, the remaining feeders from University Substation in the project area (2663, 2665 and 2667) pass through neighborhoods with large trees. Construction and weather can impact trees leading to outages. Also, spanning the Montlake cut with a new feeder may not be feasible, especially given other options for service, from the East Pine Substation. Based on their experience, the Seattle Fire Department seems to concur that a second feeder from University Substation is not an acceptable backup service option.
- In lieu of the above, a second service from East Pine Substation could be built by intercepting and extending Feeder 2752 one half mile to the project area. SCL has provided reliability and other data on feeders from East Pine and University Substations to the project engineers for their analysis.
- The maximum 480 volt transformers SCL stocks are 2,500 KVA. The current estimate provided to SCL for operational electrical energy needs from SCL is 5,000 KVA. This will require supplying the project at a higher voltage to use transformers that are kept in stock.
- SCL intends to serve the project in a manner that will avoid the long lead time associated with ordering transformers that are not in stock.

- SCL will need the design to include sufficient access to electrical equipment as determined by SCL, to maintain service and address critical conditions that may arise including emergency access similar to that provided to Fire and Police, especially in areas with temporary traffic flow revisions.
- Page 5.3-5: The FEIS states, "There would be no operational effects on utilities or utility providers". While there may be no *significant adverse* impacts on SCL anticipated, there are new electrical service requirements for the project requiring additional service capacity and electrical energy, which do have operational effects. Additional electrical service requirements should be clearly noted in utility-related project documents and communications.
- It is anticipated that during construction, SCL will need to coordinate temporary electrical outages with its customers. Mitigation commitments need to include scheduling work in a manner that allows sufficient time for notification and that minimizes effects on customers from temporary service interruptions. Mitigation requirements need to be detailed in inter-agency agreements. In some instances, on-site backup generation may be needed to avoid service interruption for critical end-uses, and SCL is responsible for these determinations.
- There has not been discussion to date that we know of concerning electrical energy requirements for a tunnel boring machine for Option K during construction. SCL needs to plan for a possible decision to serve the construction needs of this Option in advance, starting with preliminary estimates of the electrical requirements and an anticipated need schedule.

Thank you for the opportunity to comment.

Cc: Best
Junttila
Russo
Cooper, Ian
Geissinger
File

City of Seattle

Department of Planning and Development
Diane M. Sugimura, Director

July 1, 2011

To: Stephanie Brown, Regional Project Coordinator

From: Kristian F. Kofoed, Senior Urban Planner, DPD

Subject: DPD Comments on SR 520 Final FEIS

Visual Quality

DPD encourages WSDOT to work with the University of Washington, which manages the arboreal collection in the Arboretum and on Foster Island, to consider replacement plantings that will grow with sufficient vigor to reduce the visual impact of the highway structure as a result of the increased height or other dimensions of the project**.**

City of Seattle
Seattle Department of Transportation
Peter Hahn, Director

Date: July 30, 2011

To: Rebecca Deehr, Office of the Mayor

From: Stephanie Brown, Regional Project Coordinator

Subject: SDOT Comments on SR 520 FEIS

Below are comments from the Seattle Department of Transportation (SDOT) on the Final Environmental Impact Statement (FEIS) for the SR 520, I-5 to Medina: Bridge Replacement and HOV Project. The purpose of this memo is to highlight significant issues that should be addressed by the Washington State Department of Transportation (WSDOT) as the project moves toward a Record of Decision this summer.

In addition, Seattle Public Utilities, Seattle City Light, Seattle Parks and Recreation, and the Seattle Department of Planning and Development have submitted FEIS comments to me; those comments are attached to this memo.

Project Scope

The scope of the Preferred Alternative (PA) for the SR 520 project lacks WSDOT commitments to fund implementation of key bicycle and pedestrian improvements between the new regional shared use path and the Burke Gilman Trail. As noted in the FEIS, bicycle and pedestrian improvements along Montlake Boulevard, Shelby Street, Hamlin Street, and 24th Avenue East were identified through the ESSB 6392 Workgroup. However, responsibility for design and construction of these improvements has been left to SDOT. These connections are a critical element of the PA for non-motorized users, and SDOT is concerned by their exclusion from the project scope.

Range of Alternatives

SDOT disagrees with WSDOT's conclusion that the 4-lane alternative does not meet the project purpose of improving the movement of people and goods across SR 520. The updated analysis included in Appendix 19 of the FEIS shows that the movement of people and goods increases under the 4-lane alternative.

Project Phasing

The phased construction plan described in the FEIS does not include an interim Westside connection for the new pedestrian and bicycle path identified in the PA. This connection must be completed at the same time as an interim connection for vehicles and will impact the timing of other pedestrian and bicycle improvements that connect the SR 520 regional path with the Burke Gilman Trail and other non-motorized facilities in Seattle. SDOT would appreciate the opportunity to work with WSDOT to better define and document the interim connection.

Mitigation Commitments – Local Traffic

The FEIS states that under the PA, "travel patterns on local streets in the area would change due to the direct-access HOV ramp from SR 520, the removal of the Lake Washington Boulevard ramps, and the addition of a new bascule bridge adjacent to the existing bridge on Montlake Boulevard." SDOT believes that these operational changes to city streets require mitigation. To begin addressing the anticipated operational impacts, SDOT and WSDOT are jointly developing a neighborhood traffic management plan. SDOT appreciates WSDOT's involvement in this effort, but requests a funding commitment from WSDOT to implement this plan and appropriately mitigate changing travel patterns on local streets.

Additionally, the description of mitigation of transportation impacts is not specific enough for SDOT to determine whether the impacts of the project on Seattle streets, transit, pedestrians, and bicyclists have been appropriately addressed. SDOT requests additional description of the potential design modifications that WSDOT plans to explore with SDOT.

Mitigation Commitments – Construction

SDOT believes that the length and intensity of construction for the SR 520 project requires the use of demand management and alternative transportation systems to reduce the construction-related impacts. WSDOT should make a stronger commitment to transportation demand management efforts during construction—moving beyond communication and coordination with existing successful programs—and potentially include funding for additional transit service in mitigation commitments.

Additionally, SDOT requests that WSDOT commit to develop (in coordination with SDOT staff), fund, and implement an intelligent transportation system (ITS) program on Montlake Boulevard and 23rd Avenue prior to construction of the Westside improvements.

Mitigation is also needed for the temporary closure of key bicycle and pedestrian routes (e.g., 24th Avenue East bridge, Bill Dawson Trail), particularly due to the fact that pedestrian and bicycle access may be restricted to one side of Montlake Boulevard during construction. SDOT will work with WSDOT to identify appropriate mitigation for pedestrians and bicyclists.

Mike McGinn, Mayor
City of Seattle

July 15, 2011

Julie Meredith, PE
SR 520 Program Director
Washington State Department of Transportation
600 Stewart Street, Suite 520
Seattle, WA 98101

Dear Ms. Meredith,

Thank you for the opportunity to comment on the Final Environmental Impact Statement (FEIS) for the SR 520, I-5 to Medina Bridge Replacement and HOV Project.

I appreciate the hard work done by your staff and the resulting changes that have been made over the course of the last year to accommodate light rail on SR 520 in the future. While there is much work to do before light rail can be implemented, our region will be well served by these changes. Nevertheless, it remains clear to me that the preferred alternative has far more impact than other tenable alternatives.

Range of Alternatives and Tolling Analysis
The FEIS states, "The 4-Lane Alternative evaluated in the Draft EIS was not evaluated further because it was determined not to meet the project purpose of improving mobility on SR 520."[1] The SDOT memo to this office regarding the SR 520 FEIS (see attached) disagrees with your conclusion that the four-lane alternative does not meet the project purpose. I agree with SDOT's assessment.

The FEIS includes analysis for a tolled four-lane alternative in Attachment 19 ("Transit Optimizing Sensitivity Analysis"), which outlines vehicle to capacity ratios (V/C) for the SR 520 corridor under different tolling scenarios. The current V/C ratio for SR 520 is 1.2 and your analysis states a goal of free-flow conditions (represented by a .85 V/C ratio). At a $4.00 toll rate, the V/C ratios for the four-lane tolled alternative are dramatically improved, to 1.06 and .89 in the eastbound and westbound directions, respectively. This $4.00 rate is not much greater than the $3.50 peak rate tolls that are scheduled to begin this year. To say that a tolled four-lane alternative does not meet the project purpose of improving mobility on SR 520 runs counter to your analysis.

Because of the smaller footprint of a four-lane alternative, fewer takings of parkland and habitat, the reduced affect on recreation, the increased visual quality, and the fact that the vehicle to capacity ratio can still be dramatically improved with tolling, I disagree with your choice to exclude the four-lane tolled alternative as an official alternative in the FEIS. A four-lane alternative combined with real investments in high capacity transit has the potential to substantially improve the movement of people and goods while reducing the environmental impact associated with your preferred alternative.

Release of Substantial New Information
WSDOT has included a substantial amount of new information in the FEIS, including new traffic analysis for the preferred alternative and the different phases of construction. Ideally, WSDOT would have made

[1] SR 520, I-5 To Medina: Bridge Replacement and HOV Project Final EIS and Final Section 4(f) and 6(f) Evaluations, Section 2, page 21

Office of the Mayor
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600 Fourth Avenue, PO Box 94749
Seattle, WA 98124-4749

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E-mail mike.mcginn@seattle.gov

this significant information available to the public prior to its inclusion in the FEIS or would have allowed adequate review time in the form of a final comment period after the release of the FEIS.

Mitigation of Local Traffic Impacts
I remain concerned about the lack of commitment to fund mitigation for local traffic changes that result from the preferred alternative, a concern that was expressed by SDOT director Peter Hahn in his April 27, 2011, letter to you. Without specific mitigation commitments, there is no guaranteed plan or funding for implementation of projects to reduce the project's impact on neighborhoods and streets in Seattle. The FEIS does not discuss or commit to future funding for traffic calming, traffic management, or intelligent transportation systems—with the exception of minor traffic calming in the Arboretum—despite the fact that the FEIS acknowledges that travel patterns on local streets will change. The lack of specific mitigation for these changes leaves me unable to determine if the state plans to address these impacts.

Commitment to High Capacity Transit
SDOT's memo recommends the use of transportation demand management strategies during construction, including funding additional transit service to mitigate construction impacts. I support this recommendation and feel that WSDOT has not made a sufficient commitment to increase transit service—especially high capacity transit—both during and after construction. The SR 520 High Capacity Transit Plan calls for bus rapid transit (BRT) on the corridor. Implementation timelines and funding for BRT is unspecified at best and is in jeopardy of being delayed for many years due to regional transit funding crises. While I understand that light rail across I-90 will play an important role in improving east-west transit in the region, BRT is still needed to move more people in fewer vehicles throughout the SR 520 corridor. Further work is also needed to ensure the project is adequate for light rail at the western landing.

Impacts of Project Phasing
The impacts of phased implementation of the SR 520 project are not adequately addressed in the FEIS. As you know, the $2 billion funding gap is not closed, the Seattle portion of the project will remain unfunded and unconstructed for a significant period of time. Throughout this "interim" condition, the new six-lane bridge will transition to the existing four-lane structure at the Western high-rise, which may have impacts on Seattle-area travel and air quality. The FEIS qualitatively describes these impacts, but they have not been quantitatively evaluated to determine their true significance. This lack of analysis could have negative consequences for the city of Seattle.

Thank you for considering these comments. I look forward to WSDOT's continued collaboration with the City of Seattle.

Sincerely,

Mike McGinn
Mayor of Seattle

Attachments: Seattle Department of Transportation Comments on SR 520 Final Environmental Impact Statement;
Seattle Parks and Recreation Comments: SR 520 Final Environmental Impact Statement;
Seattle Department of Planning and Development Comments on SR 520 Final Environmental Impact Statement;
Seattle City Light Comments: SR 520 Final Environmental Impact Statement;
Seattle Public Utilities SR 520 Interests and Concerns

DATE: **June 29, 2011**

TO: **Stephanie Brown, Seattle Department of Transportation**

FROM: **David Graves, AICP, Senior Planner**

SUBJECT: **SR 520 Final Environmental Impact Statement**

Attached are comments of Seattle Parks and Recreation on the SR 520, I-5 to Medina Bridge Replacement and HOV Project Final Environmental Impact Statement. The purpose of this memo is to highlight issues of substantial significance which should be addressed as the project moves forward.

Background

If implemented, proposed upgrades to State Route 520 will have significant impacts to a number of Seattle parks over a span of years, and a base set of impacts for the life of the freeway corridor. There will be impacts to park resources protected under Section 4(f) of the Federal Highway Administration legislation associated with the SR 520 project. There will be impacts to park resources protected under Section 6(f) of the Land and Water Conservation Fund legislation associated with the SR 520 project. There will be a loss of park property, both temporarily during construction and permanently as the right-of-way is expanded to accommodate the expanded highway. Finally, there will be a loss of to park resources protected under the City's Initiative 42, Ordinance No. 118477.

City of Seattle park resources under the jurisdiction of the Superintendant of Seattle Parks and Recreation that will be directly impacted by the SR 520 project include Bagley Viewpoint, Montlake Playfield and the associated submerged lands, Lake Washington Boulevard, East Montlake and McCurdy Parks and the Washington Park Arboretum (Arboretum). City of Seattle park resources under the jurisdiction of the Superintendant of Seattle Parks & Recreation that will be indirectly impacted by the SR 520 project include Roanoke Park, West Montlake Park and Lake Washington Boulevard. As such, appropriate mitigation of the project impacts is warranted and necessary.

Comments

Seattle Parks and Recreation (Parks) respectfully submits the following comments in response to the Final Environmental Impact Statement (FEIS) for the I-5 to Medina: Bridge Replacement and HOV Project issued on June 17, 2011:

- **WSDOT Peninsula** – With the removal of the Lake Washington Boulevard access ramps, the opportunity is presented to return this area to City ownership and restore the north entrance to the Arboretum in the area commonly referred to as the "WSDOT Peninsula". WSDOT has identified in the FEIS that this area could be returned to the City. Prior to the original construction of SR 520, it was part of the Arboretum. This land must remain in public ownership and should be transferred to the City to be reincorporated into the Arboretum.

- **Construction Impacts** – Construction of the segment from Montlake Boulevard to the west highrise will take many years. During this time access to the north part of the Arboretum, Foster and Marsh Islands and East Montlake Park will be limited and through travel will be constrained by temporary work bridges. All efforts to safe ensure public access to and between the Arboretum and East Montlake park during construction must made.

  Visitors to parks within the Westside corridor will be subject to construction traffic, noise and dust. Every effort must be made to ensure that visitors to Roanoke Park, Interlaken Park, Montlake Playfield, West Montlake Park, East Montlake Park and the Washington Park Arboretum are protected to the maximum extent possible from having the proposed highway project intrude on their recreational experience.

- **Loss of park lands** – The FEIS indicates a permanent loss of 5.0 acres of upland and 1.0 acre of submerged land of City-owned park property and temporary use of an additional 3.3 acres of upland and 2.9 acres of submerged land of City-owned park property. Permanent acquisition will require payment for the fair market value of the property; temporary use will require compensation equal to the value of the construction easement. Note also that a revocable use permit will be required for all work on Parks' property not covered under a temporary construction easement.

- **General Mitigation** – Sections 4f and 6f each have specific mitigation requirements which must be met. Additional Federal, State and local regulations also have specific mitigation requirements. Discussions are ongoing between the City and WSDOT on a variety of mitigation opportunities and proposals. Full and complete mitigation for all project impacts must be provided.

Thank you for the opportunity to review and comment on the Final Environmental Impact Statement for the I-5 to Medina: Bridge Replacement and HOV Project.

Additional Information

If you any questions regarding the SR520 project, please contact David Graves at 684-7048 or e-mail to david.graves@seattle.gov.

Seattle Public Utilities

Memorandum

Date: June 29, 2011

To: Stephanie Brown, SR 520 Project Manager, SDOT

From: Nancy Ahern, Deputy Director, Utility Systems Management Branch

Re: SR 520 Interests and Concerns

Seattle Public Utilities (SPU) has reviewed the FEIS for the proposed SR 520 Project and per your request is summarizing its interests and concerns on this project. The purpose of this memorandum is to provide SPU's FEIS review for input into a combined City response. Our interests and concerns fall into several categories as presented in the following:

Concerns for Existing Utilities:
SPU owns several water and wastewater pipelines that cross SR 520, including:

- **The Maple Leaf Pipeline** – is a 54-inch water transmission pipeline that crosses SR 520 about 100 feet east of the existing Montlake Bridge and was relocated in the early 1960's to accommodate SR 520. Dependent upon the SR 520 project alternative that is selected for design and construction, portions of this pipeline may need to be lowered/relocated.
- **The 430 Pipeline** – a 42-inch water transmission pipeline that crosses under SR 520 between the 10th Ave. E. and Delmar Ave. E. overpasses and was also relocated and replaced in the early 1960's to accommodate SR 520. This pipeline may need to be relocated or lowered for SR 520 Project.
- **The Boylston Avenue Feeder** – a 20-inch pipeline located in Boylston Ave. E. west of I-5 and the SR 520 interchange area. Portions of this pipeline may need to be relocated (dependent upon the alternative selected) due to construction impacts and conflict with the new interchange lid.
- **The Roanoke Street Feeder** – a 12-inch water line located in E. Roanoke Street and extending from the Boylston Avenue Feeder (located west of I-5, see above) to 11th Ave. E. Portions of this water feeder main may need to be relocated due to potential impacts from construction and/or the new interchange lid at SR 520 and I-5.

- **The Boyer Avenue Feeder**- a 20-inch water main that crosses under SR 520 at the Boyer Avenue underpass and predates SR 520. This water main will need to be protected in place or replaced/relocated dependent upon the selected alternative and impacts that develop in the SR 520 design process.
- **The Montlake Boulevard Feeder** – a 12-inch water main that crosses SR 520 in the Montlake overpass and supplies the area between SR 520 and the Ship Canal. This water main may need to be replaced if SR 520 is expanded and may need replacement/relocation to near Pacific Street depending on the project alternative selected. Distribution mains in E. Shelby and E. Hamlin Streets crossing Montlake Boulevard may also be impacted.
- **A 24 –inch combined sewer** that carries flows under SR 520 in the vicinity of the Museum of History and Industry to a pump station for conveyance out of the Montlake area. This pipeline was installed in 1961 and may need to be lowered or relocated if SR 520 is lowered or expanded.
- **An 8-inch combined sewer** that carries flows under SR 520 in the vicinity of the Seattle Yacht Club to another pump station for conveyance out of the Montlake area. This pipeline may be impacted by the SR 520 projects if supports for the new freeway need to be placed on or near the pipeline.
- **A 24-inch combined sewer** that carries flows under I-5, north of the I-5/SR 520 interchange near Boylston Avenue. The portion of the pipeline under I-5 was constructed in 1959 and a small portion in the City right of way was constructed in 1906. This pipeline may be affected by the treatment facilities for managing stormwater from the proposed interchange lid.
- In the two proposed lid areas (Montlake and the I-5 interchange) there are water mains and sewer lines in addition to those described above that could be affected requiring either relocating or replacement depending on the extent of the project area in the lid areas.

SPU has several utilities across and along the SR 520 and I-5 corridors that pre-date these freeway and SPU's research of real property records for the Montlake area (the 54-inch water pipeline and the 24-inch combined sewer across SR 520) has indicated that SPU has property rights to require WSDOT to bear the costs for relocations of these facilities.

Other areas described above for possible impacts of SPU facilities continue to be researched. The real property in some areas is not clear or fully resolved and may come up again in resolving some utility conflicts that may arise.

Since the SPU facilities have existed before I-5 and SR 520 were constructed and parts of the SR 520 Project may affect utilities in Seattle streets right of ways, SPU requests that the City position would recognize that **WSDOT should bear the costs of any water and wastewater pipeline relocations that are needed.**

The estimated cost range of these impacts is anticipated to be $5-7M.

Construction Concerns:

- In the project area the construction tasks can have adverse impacts on existing utilities through activities such as dewatering, excavation, settlement and vibration impacts. These types of impacts may trigger protective measures for utilities or relocations. These impacts are often addressed in the design process.
- Certain portions of SPU's utilities will need to be accessible to SPU personnel at all times for emergency response or billing needs and not be blocked by construction activities or storage of materials.
- In the event of construction easements needed for the SR 520 Project, the protection or relocation of utilities may be needed if utilities are adversely impacted.

Protection or Enhancement of Water Resources:

SPU requests that the City express the following interests in the area of water resources and water quality:

1. The City expects that the SR 520 Project will have no impact on the routing or the amounts of stormwater between the City's combined and separated drainage systems, unless it is possible to reduce the amount of flow to the City's combined system through on-site infiltration of stormwater;
2. The City expects that WSDOT will be responsible for constructing, operating and maintaining any water quality or flow control facilities associated with the stormwater treatment requirements for the SR 520 project;
3. The City expects that the stormwater treatment for any SR 520 runoff entering the City's separated or combined drainage systems will meet the City's 2009 stormwater code requirements for water quality and flow;
4. Protecting the water quality of Lake Washington is a shared concern of many jurisdictions, including Seattle. Stormwater runoff from roadways is a major source of pollutants entering receiving water bodies, and the City supports the proposed use of street sweeping, if done frequently with high efficiency sweepers, as an appropriate method for decreasing pollutants discharged to Lake Washington from the SR 520 bridge deck.
5. The City is interested in working with WSDOT on site selection and design of aquatic and wetland mitigation associated with this Project; and
6. The SR 520 Project should be designed and constructed in a manner that avoids, minimizes or mitigates impacts to salmonids. Among the more important considerations include shielding the water surface from artificial lighting on overwater structures, avoiding impacts to adult migration through the SR 520 Project area and minimizing the number and size of pilings.

Thank you for requesting SPU's input. Please call Charlie Madden at (206) 684-5977 if you have any questions or comments about the interests and concerns described in this memorandum or require additional information.

cc: Betty Meyer, Special Projects, Utility Systems Management Branch (USM)
Dave Hilmoe, Drinking Water Division Director, USM
Trish Rhay, Drainage and Wastewater Systems Management Director, USM
Bruce Bachen, Drainage and Wastewater Quality Division Director, USM
Ingrid Wertz, Water Quality Program Manager, USM
Eugene Mantchev, Drinking Water Transmission Manager, USM
Frank E. McDonald, Drainage and Wastewater Manager, USM
Linda DeBoldt, Deputy Director, Project Delivery Branch (PDB)
Liz Kelly, Project Management & Engineering Division Director, PDB

**Note: The formatting issues within this letter existed in the email sent by the commenter. We've kept them as is, so the comment retains its original form.

From: jonstall [mailto:jonstall@wafirst.com]
Sent: Thursday, June 23, 2011 2:16 PM
To: SR 520 Bridge Replacement & HOV Project
Subject: Comments on SR 520 Final EIS

June 23, 2011

Congratuations on completing the Final EIS for the SR 520, I-5 to Medina: Bridge Replacement and HOV Project. This is an important accomplishment and overall a well-done analysis. As a NEPA practitioner working in Washington and across the country I appreciate the effort expended and impressive scope of your analysis. Overall the Final EIS manages to wrap up what has been a somewhat convoluted NEPA process. However, I would like to point out two deficiencies that should be addressed in the Record of Decision.

1 â€" The No Action alternative is incorrectly conceived and evaluated.
The Final EIS clearly explains the perilous condition of the current bridge. Some excertps from the Final EIS on this subject include:

1.3 â€œThe probability that the bridge will sustain serious structural damage (i.e., sink or become impassable to traffic) over the next 15 years is extremely highâ€•

â€œWSDOT estimates that over the next 50 years, there is a 20 percent chance of serious damage to these structures in an earthquake.â€•

Exhibit 1-2

1.5 â€œWhat would happen if the project were not built? If the project were not built, the section of SR 520 between I-5 and Evergreen Point Road would not be improved, and these critical needs would not be met:

â–ª The risk of bridge failure in a storm or earthquake would increase as the structures continued to age, with consequences ranging from severe traffic congestion to loss of life. As the floating bridge becomes more fragile, it would require more frequent closures to protect its components from damage.â€•

Togethe r these statements demonstrate the bridge is in bad shape and a risky proposition. The CEQ reguations on the no action alternative a quite brief (see 1502.14). The clearest direction from CEQ on the no action alternative is given by the third of the â€œ40 most asked questionsâ€• . With regards to projects this says â€œthe proposed activity would not take place, and the resulting environmental effects from taking no action would be compared with the effects of permitting the proposed activity or an alternative activity to go forward.â€ This guidance continues to state â€œThis analysis provides a benchmark, enabling decisionmakers to compare the magnitude of environmental effects of the action alternatives.â€• Thatâ€™s pretty straightforward. However, hereâ€™s how the Final EIS described the No Action (called No Build) alternative:

â€œ2.7 What is the No Build Alternative?
The No Build Alternative assumes that, other than normal maintenance and repair activities, the SR 520 corridor between I-5 and Evergreen Point Road would remain exactly the same as it is today.â€•

â€œAs described in Chapter 1, the remaining design life of the Evergreen Point Bridge is currently estimated at just 10 to 15 years, and a severe storm could cause it to fail even sooner. The Portage Bay

and west approach bridges are also vulnerable to collapse in a severe earthquake. For these reasons, the No Build Alternative is inconsistent with WSDOTâ€™s standards for safety and reliability. Given the vulnerabilities of the existing bridges, the No Build Alternative is not a likely scenario; however, it provides a set of baseline conditions to which the expected effects of the project can be compared.â€•

This interpretation is clearly contrary to the CEQ guidance â€" it is establishing the WRONG baseline for comparison. So what would happen if the SR 520 Bridge fails? I thi nk would have pretty severe impacts, but I donâ€™t know becase this wasnâ€™t evaluated. This deprives the decsion makers and public of clear understanding of the consequences of not acting. Thatâ€™s important.

Finally, it is interesting to note that another large project in the Seattle area with the same lead agencies has defined the no action alternative differently. The Alaskan Way Viadcut Replacement Project 2010 Supplementatl Draft EIS defines the no action alternative in accordance with the CEQ guidance. The result is a pretty clear picture of how important the project is.

Conclusion: The Record of Decision should evalute the effects of the TRUE no action alternative where the SR 520 Bridge is lost without advance planning.

2 â€" The effects of reasonable foreseeable tolling are not properly evaluated.

I take issue with a very brief but very imporant statement on page 1-23 of the Final EIS:

â€œRegion-wide tolling of major corridors, as recommended in the Pu get Sound Regional Councilâ€™s Transportation 2040 plan (PSRC 2010a), might also be implemented by that time. Since it is not possible to say whether or how these tolls would be implemented, WSDOT did not include them in its baseline assumptions.â€•

Other actions WSDOT is taking on I-405, SR 99, and SR 167 and is considering for I-5 Express lines show implementation of the Transporation 2040 Plan is not speculative but reasonably foreseeable. Since timing of tolling is unknown, this is not an entirely fatal flaw in the Final EIS analysis. However, since tolling of nearby facilities could have substantial effects on SR 520 operations it should be at least considered and described. This points to a larger issue â€" the overall effects of implementing the Transporation 2040 plan are not being adequately considered by FHWA and WSDOT. This is a transporatation SYSTEM, and tolling is arguably a set of connected actions (see 1508.18(b)(3))) that should be considered together. Th e system-wide effects, especially to low-income populations covered by E.O. 12898 on environmental justice, could be significant.

Conclusion: The Record of Decion should evaluate the effects on SR 520 operation of tolled operations described in the Transporation 2040 Plan. Further, FHWA and WSDOT should consider a non-project evaluation of tolling in the Puget Sound region.

In closing let me once again congratuate you on an impressive accomplishment. My focus on these deficiencies should not dimish the magnitude and quality of your work in other regards.

Sincerely,

Jon Stall

Response to and questions raised regarding the FEIS of SR 520

Submitted to the Washington State Department of Transportation

July 15, 2011

by

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An Excellent resource on the risks associated with fine particular matter is the Puget Sound Clean Air Agency Web Site.

http://www.pscleanair.org/news/library/factsheets/next10/FineParticulateMatter.pdf

Page 1, paragraph 2 states:

1. We are currently in compliance with current federal standards at traditionally monitored sites for fine particulate matter except in a segment of Pierce County
2. We do not meet the more strict goals of the Puget Sound Clean Air Agency. *Will the Washington State Department of Transportation explain why they consistently ignore State agency guidelines that are more strict than Federal Guidelines?*

Page 1, paragraph 4

1. Fine particulate matter is associated with serious health defects
2. Ultrafine particles not currently measured in Puget Sound may be more harmful. They are not currently measured in the Puget Sound Region

Page 2, paragraph 1

Fine particles act as a mode of transportation into the body for other pollutants.

Page 2, paragraph 5

"Based on monitoring data, the Puget Sound region would currently violate the proposed EPA standard" *Note that this statement refers to data from traditional monitoring stations often located far from high volume highway traffic.*

Page 2, paragraph 6

The Health Committee of the Puget Sound Clean Air Agency, comprised of local health professionals, ". . . did not consider the federal standard to be sufficiently protective of human health. *Why is the Washington State Department of Transportation using the federal standard while not mentioning the local agency's discussion?*

Page 3, paragraph 2

"We typically exceed the health goal for an entire two months out of each year."

Page 3, paragraph 4

"Concentrations (of PM2.5) have not decreased as significantly in recent years as they did in the early 1990s."

Page 4, paragraph 2

"Some monitoring locations with our highest fine particulate levels are not shown because they don't have a long monitoring history. " *Why is this?* "Three additional monitoring locations have a very slim 'margin of safety' (less than 5 ug/m3) that places them at risk of violating the proposed standard." *These comments are all made without recognition of near highway "hot spots" that exposure residents, students, and employees to even higher levels of fine particles.*

Page 6 Figure entitled Fine Particle Matter Emissions Summer Source Categories.

Mobile sources are the greatest source of fine particle matter emissions in the summer.

Discussion of this Fact Sheet

The Washington State Department of Transportation "Final" Environmental Impact Statement does not acknowledge that the Puget Sound Clean Air Agency recommends more stringent standards be applied to protect the health of Puget Sound citizens.

The Washington State Department of Transportation "Final" Environmental Impact Statement does not acknowledge compelling scientific evidence throughout the world that persons living, attending school, working and driving on high volume highways are subject to levels of air pollution that damage health and increase mortality.

The Washington State Department of Transportation as expressed in the "Final" Environmental Impact Statement has made no effort to measure health impairing pollutants in areas adjacent to SR 520. Where is the data assessing pollution in the Eastlake District, North Capitol Hill, Roanoke Park, Montlake, Madison Park, Laurelhurst, and districts adjacent to SR SR 520 on the Eastside of the bridge? Where is the data monitoring air pollution at Seward School, Seattle Preparatory Academy, local daycare centers, the University of Washington campus including the Health Science Campus, and similar campuses on the Eastside of the bridge?

The Washington State Department of Transportation does an inadequate job of estimating the effects of population increases on the traffic pattern on SR 520 particularly on the combined effects of traffic associated air pollution on the neighborhoods adjacent of the confluence of several highways. While the population within 500 meters of SR 520 is the current topic of discussion, it is highly likely that 10-30% of the Puget Sound Population of Puget Sound is at risk for health effects due to similar exposure. The absence of recognition and study of this issue makes current Washington State Department of Transportation plans for SR 520 and other regional highway planning inadequate to meet the transportation needs of the 21st century.

Douglas K Stewart, MD

Seattle, WA

Douglas Stewart MD
University of Washington School of Medicine

Puget Sound Clean Air Agency
pscleanair.org

Next Ten Years Fact Sheet

Working Together For Clean Air

Fine Particulate Matter

Particles smaller than 2.5 micrometers in diameter suspended in outdoor air are called "fine" particulate matter, or $PM_{2.5}$. These particles are approximately $1/30^{th}$ the width of a human hair.

While we've made progress with $PM_{2.5}$ reductions in the last 15 years, we still don't meet our health goal in three of our four counties. Additionally, current monitored levels in two counties would violate recently-proposed federal standards.[1] Unless more aggressive reduction strategies are successfully implemented, we will continue to fall short of our health goal and will likely violate a new federal standard.

$PM_{2.5}$ is the most important criteria air pollutant challenge facing our region, both now and in the future. Serious current health risks, no change in measured levels, and the likelihood of violating new federal standards combine to make $PM_{2.5}$ reductions a priority.

Health and Environmental Impacts

Of the criteria pollutants (those pollutants with federal outdoor air quality standards) and at the levels monitored in the Puget Sound area, fine particulate is associated with the most serious health effects. $PM_{2.5}$ exposure is linked with respiratory disease, decreased lung function, asthma attacks, heart attacks, and premature death.[2,3,4,5] Children, older adults, and people with some illnesses are more sensitive and susceptible to $PM_{2.5}$ health effects.[6,7] Recent research has raised additional concern about the health effects of the smallest "ultrafine" particles (less than 0.1 micrometers) may be even more harmful because of their very small size.[8]

As well as being harmful themselves, fine particles often act as a mode of transportation into the body for other pollutants that adsorb to them. Many of these pollutants are carcinogenic and have other health effects.

In addition to health effects, $PM_{2.5}$ affects visibility. Fine particulate matter can remain suspended for a long time, diminishing views and contributing to regional haze.[9]

Federal Fine Particulate Standards

The Environmental Protection Agency (EPA) set a national ambient air quality standard for $PM_{2.5}$ in 1997. The Puget Sound area has not violated this standard and is currently in compliance for fine particulate matter. The federal standard includes both a daily (65 micrograms per cubic meter [$\mu g/m^3$]) and annual (15 $\mu g/m^3$) standard.

EPA is in the process of revising the fine particulate national ambient air quality standard. EPA released proposed fine particle standards on December 20th, 2005 of 35 $\mu g/m^3$ (daily) and 15 $\mu g/m^3$ (annual). The proposed standards are based on preliminary recommendations made by EPA staff and the Clean Air Science Advisory Committee. [10,11,12] Preliminary recommendations ranged from 25 $\mu g/m^3$ to 40 $\mu g/m^3$ for the daily standard and 12 to 15 $\mu g/m^3$ for the annual standard.[13] EPA will be taking comment on these ranges (as well as proposals for no change to the current standard) and is scheduled to finalize a new standard in September 2006.

The proposed daily standard is more protective of human health than the current one and brings the federal standard closer to the Puget Sound region's fine particulate daily health goal of 25 $\mu g/m^3$. ***Based on monitoring data, the Puget Sound region would currently violate the proposed EPA standard.***[14,15]

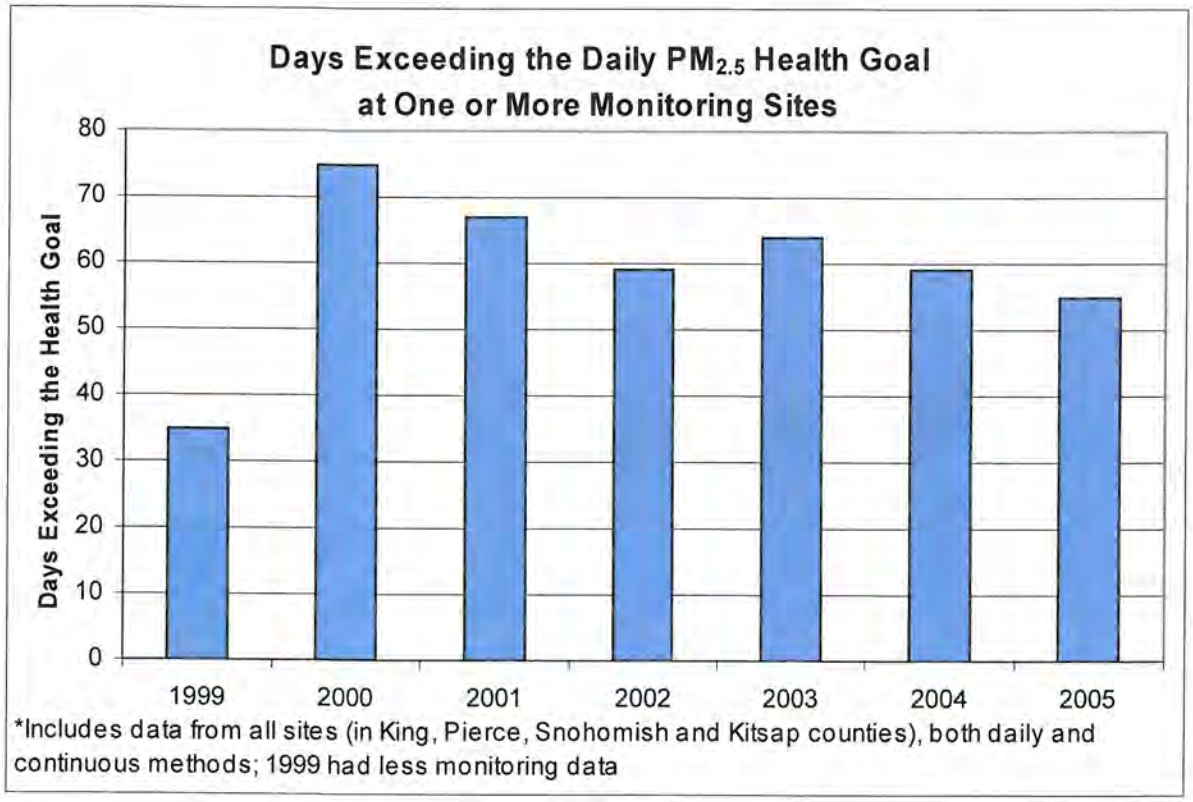
Local Fine Particulate Health Goal

The Agency has developed an air quality health goal for $PM_{2.5}$. The Agency convened a Particulate Matter Health Committee, comprised of local health professionals, who extensively examined the fine particulate standard.[16] The Health Committee did not consider the federal standard to be sufficiently protective of human health.

Based on the committee's recommendations, the Agency adopted a health goal of 25 $\mu g/m^3$ in 1999 for a daily average, well below the current 65 $\mu g/m^3$. The committee recommended a health goal of 15 $\mu g/m^3$ for the annual average,

consistent with the federal standard. ***Measured daily concentrations of $PM_{2.5}$ do not meet the local health goal at monitoring stations in three of the Agency's four counties.*** Kitsap County's two monitoring sites meet the goal.

Our local daily health goal was written and adopted with the intent that it is never exceeded. The graph below shows that we're falling far short of that intent. ***We typically exceed the health goal for an entire two months out of each year.*** Significant emission reductions are clearly needed to get levels firmly below the health goal, with no days exceeding the health goal each year.



$PM_{2.5}$ Monitoring Network and Trends

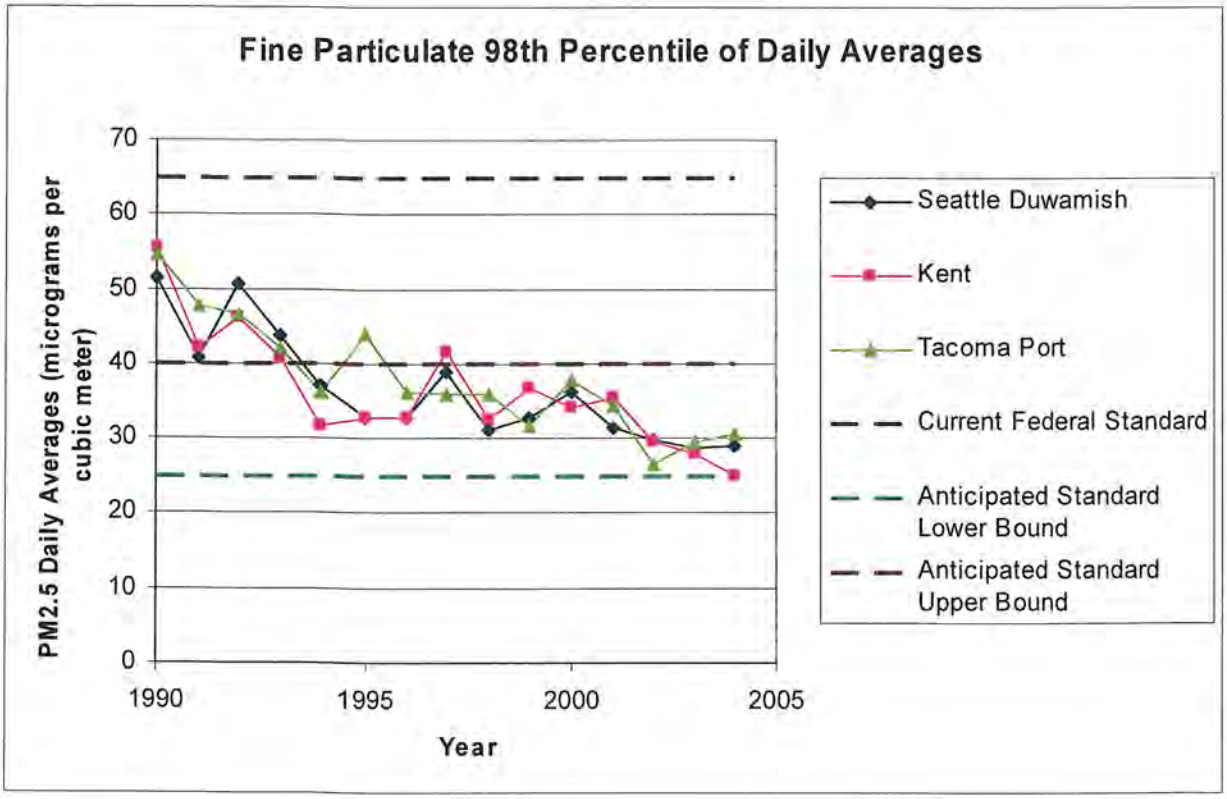
The Agency maintains a particulate monitoring network in its four counties. [17] This network goes well beyond national ambient air quality standard monitoring requirements, and gathers valuable information used to educate people about health risks, guide emissions reduction strategies, and reinforce emissions inventories.

The following graph shows the history of $PM_{2.5}$ concentrations at three long-term monitoring locations. [18] Concentrations have not decreased as significantly in recent years as they did in the early 1990s

The 98th percentile of the daily averages is shown, as this is the statistic required to compare to the federal standard.[19] The current federal standard (implemented in

1999) is shown as the dashed black line. The upper bound and lower bounds showing the ranges for comment on the proposed federal standard are shown in dashed brown and green lines, respectively.

Some monitoring locations with our highest fine particulate levels are not shown because they don't have a long monitoring history. These locations are typically close to wood-burning areas and include two locations that would currently violate the proposed standard of 35 $\mu g/m^3$. Three additional monitoring locations that have a very slim "margin of safety" (less than 5 $\mu g/m^3$) that places them at risk of violating the proposed standard.



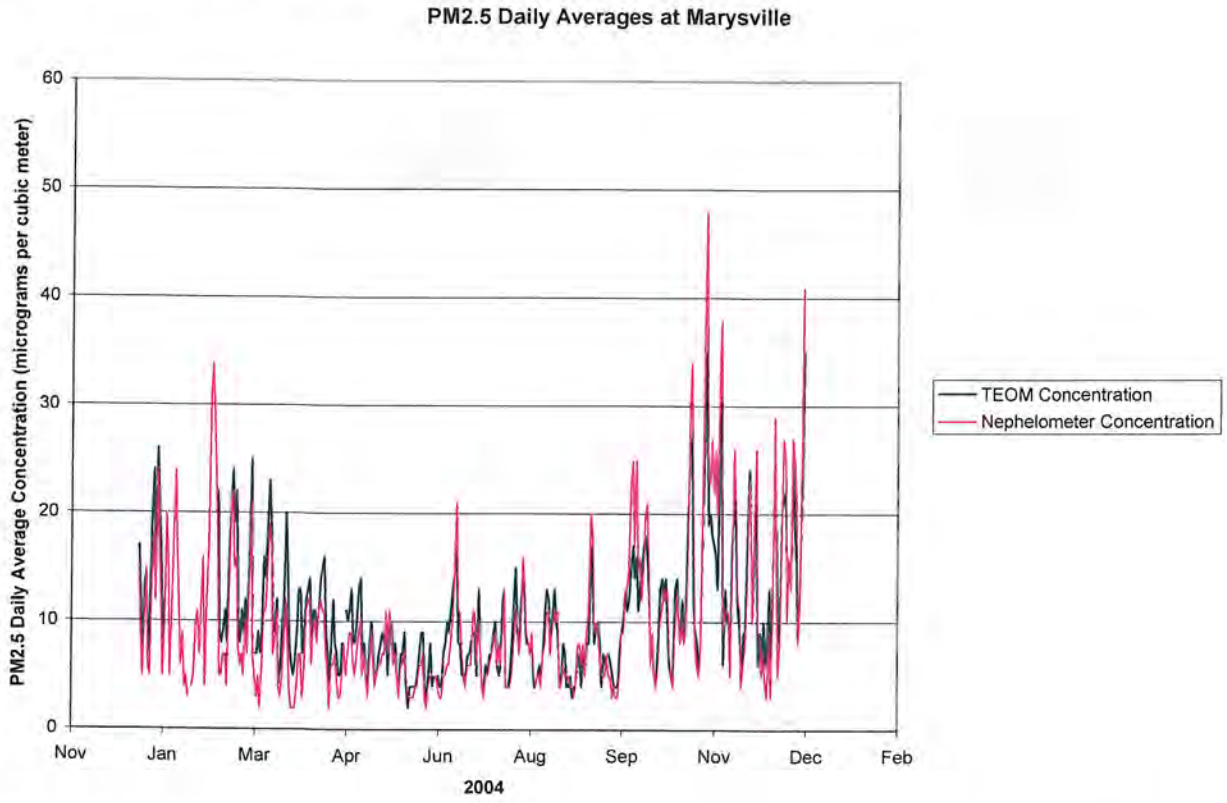
$PM_{2.5}$ Concentrations – Seasonal Variation

Fine particulate matter levels in the Puget Sound area are often higher in the winter months due to meteorology (stagnant air inversions) and wood burning in fireplaces and wood stoves.

The graph below is an example of a monitoring site influenced by wintertime wood stove and fireplace burning. The red and blue lines both show $PM_{2.5}$ concentrations, measured with two different instruments.

During the spring and summer months, outdoor burning and motor vehicles contribute more heavily to fine particulate levels. Although summer months

generally have lower $PM_{2.5}$ concentrations, this "baseline" level is important as it contributes to both daily concentrations and annual averages. Monitors placed by roadways reflect the influence of traffic on $PM_{2.5}$ concentrations; concentrations closely mirror traffic volumes.

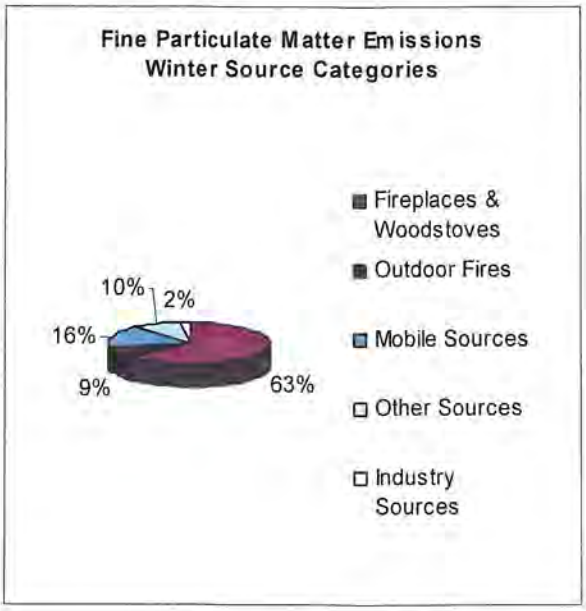


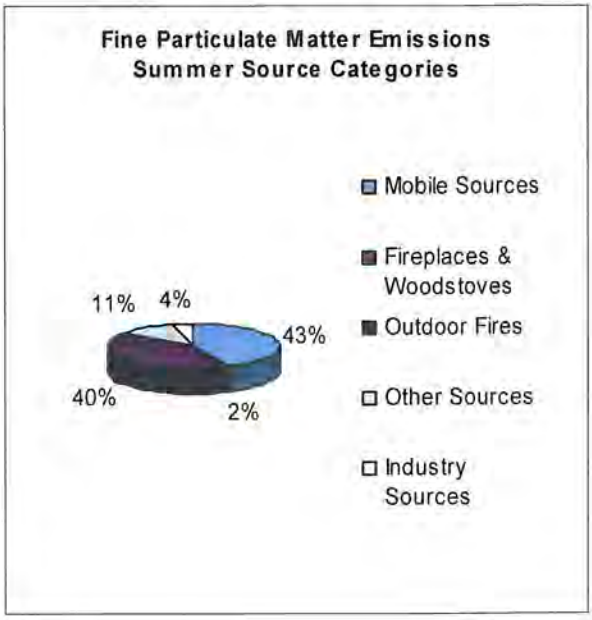
$PM_{2.5}$ Sources

$PM_{2.5}$ comes primarily from wood burning in fireplaces and woodstoves, land-clearing burning, backyard burning of yard waste, vehicle exhaust including cars, trucks, and buses and other combustion sources. $PM_{2.5}$ can also be formed secondarily in the atmosphere through chemical reactions of pollutant gases.

The graphs below reinforce the seasonal differences in $PM_{2.5}$ sources that were noted earlier. During many months burning wood is the largest contributor to fine particulate matter in the Puget Sound area, especially at neighborhood sites such as Lake Forest Park, Marysville, and South Tacoma [20]

Mobile sources, particularly diesel-powered vehicles and equipment, are important year-round sources. In the summer mobile sources are the largest contributor to fine particulate emissions.





Current Emission Reduction Strategies

In the late 1990s, the Agency evaluated, through a stakeholder process, local strategies to ensure maintaining $PM_{2.5}$ "attainment" status and to meet our local health goal. Based on emissions inventory and extensive monitoring information, the stakeholder group recommended a broad suite of local control strategies to reduce $PM_{2.5}$ in the Puget Sound area.[21]

Strategies that are currently implemented include:

- Several measures to reduce diesel emissions ("Diesel Solutions" program)
- Encouraging cleaner winter burning and heating practices through partnerships
- Enforcing winter burn bans (burn ban trigger recently changed to $PM_{2.5}$)
- Woodstove "buy-back" programs
- Expanding zones where outdoor burning is prohibited

In addition to these strategies, implementation of Washington's stricter vehicle standards will also result in lower $PM_{2.5}$ mobile emissions. The Puget Sound Climate Protection Advisory Committee recommended adopting this strategy in its final climate protection report.[22] The 2005 Legislature adopted these standards and the Washington State Department of Ecology is developing implementing regulations.

Future Challenges

Strategies will need to be more aggressive to reduce emissions to meet our health goal and ensure that the Puget Sound region doesn't lose its fine particulate attainment status. Reductions will need to be large enough to offset predicted population growth in this region, as well as possible changes in heating behavior linked with rising costs of non-wood energy.[23] Unless efforts are increased to reduce $PM_{2.5}$, we

- will continue to fall short of our local health goal,
- will likely violate new federal standards,
- could backslide to higher, more harmful $PM_{2.5}$ levels.

Additional Resources

- Final Report of the Puget Sound Clean Air Agency $PM_{2.5}$ Stakeholder group http://www.pscleanair.org/news/other/pm2_5_report.pdf.
- 2004 Air Quality Data Summary. July 2005. http://www.pscleanair.org/ds04/docs/2004AQDSFINAL.pdf.
- Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information. OAQPS Staff Paper. July 1st, 2005. EPA-452/R-05-005. http://www.epa.gov/ttn/naaqs/standards/pm/data/pmstaffpaper_20050630.pdf
- Puget Sound Clean Air Agency's Burning Web Page http://www.pscleanair.org/burning/.
- Puget Sound Clean Air Agency's Diesel Solutions Program http://www.pscleanair.org/dieselsolutions/index.shtml.

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[1] Environmental Protection Agency. Fact Sheet: Proposal to Revise the National Ambient Air Quality Standards for Particulate Matter. December 20th, 2005. http://www.epa.gov/oar/particlepollution/fs20051220pm.html. Monitors in Pierce and Snohomish County exceed and would violate the proposed daily standard.

[2] Pope et al. Lung Cancer, Cardiopulmonary Mortality, and Long–Term Exposure to Fine Particulate Air Pollution. *Journal of the American Medical Association.* 287: 1132-1141. March 6, 2002.

[3] Gauderman et al. The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age. *The New England Journal of Medicine.* Volume 351: 1057–1067. Number 11. September 9, 2004.

[4] Kunzli et al. Ambient Air Pollution and Atherosclerosis in Los Angeles. *Environmental Health Perspectives.* Volume 113,2: 201-206. February 2005. http://ehp.niehs.nih.gov/members/2004/7523/7523.pdf

[5] California Air Resources Board. Staff Report: Public Hearing to Consider Amendments ot the Ambient Air Quality Standards for Particulate Matter and Sulfates. May 3, 2002. http://www.arb.ca.gov/research/aaqs/std-rs/pm-final/pm-final.htm.

[6] Park et al. Effects of Air Pollution on Heart Rate Variability: The VA Normative Aging Study. *Environmental Health Perspectives.* Volume 113, 3. pp 304–309. March 2005. http://ehp.niehs.nih.gov/members/2004/7447/7447.pdf.

[7] Goss et al. Effect of Ambient Air Pollution on Pulmonary Exacerbations and Lung Function in Cystic Fibrosis. *American Journal of Respiratory Critical Care Medicine.* Volume 169: pp 816-821. January 12, 2004.

[8] Sioutas et al. Exposure Assessment for Atmospheric Ultrafine Particles (UFPs) and Implications in Epidemiologic Research. Environmental Health Perspectives. Volume 113, 8: 947-955.

[9] For more information on visibility, see the 2004 Air Quality Data Summary. July 2005.

[10] EPA Draft Fine Particulate Staff Paper Fact Sheet. January 2005. http://www.epa.gov/airlinks/pdfs/pmstaff2_fact.pdf.

[11] Clean Air Science Advisory Committee (CASAC) DRAFT Review of Staff Paper. http://www.epa.gov/sab/pdf/casac_pmrp_mtg_april_6-7_2005_2nd_draft_pm_staff_paper-ra_draft_report_v2.pdf.

[12] Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information. OAQPS Staff Paper. July 1st, 2005. EPA-452/R-05-005. http://www.epa.gov/ttn/naaqs/standards/pm/data/pmstaffpaper_20050630.pdf

[13] The daily and annual standards were often linked together in the preliminary recommendations; for example a more stringent annual range was linked with a less stringent daily range, and vice versa.

[14] Two monitoring stations (Marysville and South Tacoma L Street)violate the standard based on 2004 monitoring – 3-year average of 98th percentile of daily averages for 2002, 2003, and 2004.

[15] Three additional stations are at risk of violating the proposed standard, with slim "safety margins" of 5 $\mu g/m^3$ or less. Lynwood, Tacoma Alexander Avenue, and Puyallup South Hill monitoring stations have 98th percentile 3-year averages of 33, 33, and 30 ug/m3, respectively (based on 2002, 2003, and 2004). These are based in part on continuous monitoring.

[16] Final Report of the Puget Sound Clean Air Agency $PM_{2.5}$ Stakeholder group. http://www.pscleanair.org/news/other/pm2_5_report.pdf.

[17] For more information on monitoring, see the 2004 Air Quality Data Summary. July 2005.

[18] Duwamish data is all CE site – dichot 1990-1998, FRM 1999-2004. Kent data is all CW data – dichot 1990-1998, FRM 1999-2003, TEOM 2004. Tacoma is EA site 1990-1998, EQ site 1999-2004 – dichot 1990-1998, FRM 1999-2002, TEOM 2003 and 2004.

[19] The 24-hour daily average national ambient air quality standard requires comparison of the 3-year average of the 98th percentile.

[20] Emissions inventory graphs are from the Final Report of the Puget Sound Clean Air Agency Stakeholder Group. October 15, 1999.

[21] For more information, see the $PM_{2.5}$ Stakeholder Report. October 1999. http://www.pscleanair.org/news/other/pm2_5_report.pdf.

[22] For more information, see the final climate change report. http://www.pscleanair.org/specprog/globclim/cpsp/pdf/rptexecsum.pdf.

[23] Projected growth based on Puget Sound Regional Council's growth target of almost 1 million more residents by 2022 http://www.psrc.org/projects/monitoring/growthtargets/cover-ch1.pdf.

Centers for Disease Control and Prevention Morbidity and Mortality Weekly Report (MMWR)

This article, whose first author if Fuyen Y Yip, PhD is an excellent summary of current knowledge of Unhealthy Air Quality.

"A growing body of research demonstrates that proximity to sources such as traffic can have adverse health effects, especially with respect to vehicular emissions, including PM2.5" page 3, paragraph 4

Discussion: Where is the recognition and plans for local measurement and consideration of traffic associated air pollution in the Washington State Department of Transportation "Final" Environmental Impact statement? This question has been asked before and the Washington State Department of Transportation has to date failed to address the issue in a satisfactory manner.

Douglas K Stewart - MD

Centers for Disease Control and Prevention
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Morbidity and Mortality Weekly Report (*MMWR*)

Unhealthy Air Quality --- United States, 2006--2009

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Particulate matter and ozone are two well-characterized air pollutants that can affect health and are monitored by the U.S. Environmental Protection Agency (EPA). Particulate matter (solid or liquid particles suspended in the air) varies widely in size and chemical composition and can include smoke, fumes, soot, and combustion by-products, as well as natural particles (e.g., windblown dust, pollen, and sea salt) (*1,2*). Particulate matter therefore represents a complex class of air pollutants that differ from other gaseous air pollutants (e.g., ozone). The transport and effect of particulate matter, both in the atmosphere and in the human respiratory tract, are governed principally by particulate size, shape, and density. Individual particles are characterized by their equivalent aerodynamic diameter: coarse particulate matter (2.5--10 μm); fine particulate matter, or PM2.5 (≤2.5--2.5 μm); and ultrafine particulate matter (<0.1 μm). Ozone is a gas that occurs naturally in the stratosphere, approximately 10--30 miles above the earth's surface, protecting the earth from the sun's ultraviolet rays. Ozone also exists at ground level and is the primary component of smog. At ground level, ozone is created when specific pollutants react in the presence of sunlight. In urban areas, vehicular and industrial emissions are chief contributors to ozone production. Ground-level ozone adversely affects health and damages the environment.

The association between outdoor particulate matter concentrations and acute and chronic adverse health outcomes includes premature death, lung cancer, exacerbation of respiratory and cardiovascular disease, and increased risks for cardiovascular morbidity (e.g., myocardial infarction and arrhythmia) (*1--6*). Data indicate that fine particulate matter is the size fraction most strongly associated with these observed health effects (*1--7*). Populations most susceptible to these exposures include older adults and children, as well as persons with heart and lung disease. National Ambient Air Quality Standards (NAAQS) were set forth in the Clean Air Act Amendments of 1970* requiring EPA to set air quality standards for specific pollutants, such as PM2.5 and ozone, to protect the health of the general public, as well as that of sensitive populations. States that do not meet the standards are subject to additional regulatory requirements and must develop a state implementation plan to meet the standards. State implementation plans might include control requirements and limits on emissions. In 2006, on the basis of increasing evidence of the effects of PM2.5 on human health, EPA revised its 24-hour NAAQS from 65 $\mu g/m^3$ to 35 $\mu g/m^3$. Throughout the United States, PM2.5 concentrations have been decreasing; more counties were in compliance with national pollution standards as of 2008 compared with previous years (*8*). During 2001--2008, the average annual and 24-hour PM2.5 concentrations declined by 17% and 19%, respectively (*8*).

Short-term exposures to ozone have been associated with an increase in mortality as well as

cardiovascular- and respiratory-related hospitalizations (*9--11*). Ozone exposure can result in lung and throat irritation, lung inflammation, wheezing, and difficulty breathing (*11--13*). Exposure to ozone also exacerbates bronchitis, emphysema, and asthma (*11,14,15*). Populations at risk for ozone-ted health effects have been characterized as those that typically spend long periods outdoors (e.g., persons with outdoor occupations and athletes), as well as sensitive groups, including infants and children, older persons, and persons with respiratory or cardiovascular disease (including asthma) (*16--21*).

In 2008, EPA decreased NAAQS for ozone to an 8-hour standard of 75 parts per billion (ppb) (0.075 parts per million [ppm]) from the level of 80 ppb (0.08 ppm) set in 1997. In 2008, the national average concentration of ozone was 14% lower than in 1990. This reduction has been attributed to decreasing levels of summer-time emissions of nitrogen oxides and volatile organic compounds (two key precursors to the formation of ozone) from transportation and fuel combustion sources (*8*).

To characterize the populations who live in areas with PM2.5 and ozone levels that exceed those allowed by the NAAQS (i.e., nonattainment areas), CDC categorized the proportions of populations who live in these areas by race/ethnicity, level of education attainment, and household income, as represented by the poverty to income ratio (PIR). Other factors, including disability status, were not examined because of limited data. Relative difference (percentage difference) was calculated by dividing the difference between the referent category and each category in the classifying variable by the value in the referent category and multiplying by 100.The referent groups selected were consistent with referent groups used in national survey summaries.

Three data sources were used for this assessment: 1) population estimates for 2007 and 2008 from the U.S. Census Bureau Population Estimates Program for U.S. counties, 2) 3-year education and income estimates from the American Community Survey (2006–2008), and 3) data on nattainment counties for PM2.5 (2006–2008) and ozone (2007–2009) obtained from EPA (http://www.epa.gov/airtrends/values.html). EPA areas are designated as nonattainment on the basis of EPA design values (i.e., statistics derived from multiple years of data that describe the ambient air quality status of a given area relative to the level of the NAAQS). In this report, counties in nonattainment represent a location that had a design value for the 24-hour PM2.5 standard that exceeded the 2006 24-hour PM2.5 standard of 35 $\mu g/m^3$; the design value was calculated as the 3-year average (2006–2008) of annual 98th percentile PM2.5 concentrations that were valid (i.e., 24-hour averages that were available for at least 75% of possible hours in a day). For ozone, counties of nonattainment had a design value (calculated as the 3-year average [2007–2009] of the annual fourth-highest daily maximum 8-hour ozone concentration) that was greater than the 2008 8-hour ozone standard of 75 ppb.

The percentage of the population living in 24-hour PM2.5 or in 8-hour ozone nonattainment counties were summarized for each category of each demographic group. Relative differences between categories were calculated as the percentage difference of each category compared with a referent category.

A total of 53 (23.3%) of 227 counties were nonattainment counties for the 2006 24-hour PM2.5 standard (Figure 1), representing 13.6% of the U.S population in 2007 (2007 U.S. population: 301,621,157). Twenty-six (49.1%) of these counties were classified as large central metropolitan counties or large fringe metropolitan counties (i.e., counties in a metropolitan statistical area [MSA] with a population of ≥1 million). Four counties (7.5%) were classified as nonmetropolitan (*22*), and the remaining 23 counties were classified as small to medium metropolitan counties (MSA ulation of 50,000--999,000). During 2007--2009, a total of 201 counties, or 36.2% of the U.S. population in 2008 (2008 U.S. population: 304,059,724), lived in nonattainment counties for 2008

8-hour ozone standard (Figure 2). The majority of counties (158 [78.6%]) in nonattainment for ozone were classified as counties in MSAs with populations of at least 250,000, of which 111 (31.5%) represented counties classified as large central metro or large fringe metro, consisting of ≥1 million ulation. Twenty-seven counties (13.4%) were classified as nonmetropolitan, and the remaining 16 counties were classified as small metropolitan counties (MSA population of 50,000--249,999).

Assessment of the data by race/ethnicity indicates that Asians (26.2%) and Hispanics (26.6%) had the greatest percentage of residence in 2006 24-hour PM2.5 nonattainment counties (Table). The relative difference between non-Hispanic whites and several racial/ethnic groups living in the areas was >100%: Asians (169%), Native Hawaiians/other Pacific Islanders (125%), and Hispanics (165%).

A similar pattern was observed in the proportion of Asian (50.2%) and Hispanic (48.4%) populations living in 2008 8-hour ozone nonattainment counties (Table). Compared with non-Hispanic whites, Asians were 57% more likely and Hispanics were 51% more likely to live in a nonattainment county.

The percentages of populations living in nonattainment counties also varied by household income and education level. The highest income population (PIR = 3 to ≥5) had the greatest percentage of persons in 24-hour PM2.5 nonattainment counties (13.3%) and 8-hour ozone nonattainment counties (41.1%). Similarly, compared with the referent group (PIR ≤1 [poor]), the highest income population was 2% more likely to live in a PM2.5 nonattainment county and 34% more likely to live in an ozone nonattainment county.

The greatest percentage of persons living in PM2.5 nonattainment counties were those with less than a high school education (16.4%). In contrast, the greatest percentage of persons in ozone nonattainment counties was college graduates or those who received advanced degrees (39.6%). Compared with persons who received less than a high school education, persons who received a high school diploma were 28% less likely to live in a nonattainment PM2.5 county, and persons who eived an advanced degree were 10% more likely to live in an ozone nonattainment county. No differences were observed between the percentages of males and females living in PM2.5 nonattainment counties or ozone nonattainment counties.

The findings in this report are subject to at least two limitations. First, residence in a nonattainment county does not necessarily indicate a person's exposure or potential severity of exposure to air pollution. Second, ambient air monitoring sites often are located in counties that are more populated and as a result tend be more representative of air quality in urban areas and might not reflect conditions outside the urban monitoring areas. Likewise, demographic trends observed in this analysis are more likely to closely reflect residents in urban areas than those in rural areas.

The results in this report indicate that minority groups, including Asians and Hispanics, were more likely to live in PM2.5 and ozone nonattainment counties. This finding might be a result of the larger percentage of these populations that live in urban areas (*23*). In 2007, an estimated 55% of all Asians lived in the 10 metropolitan areas with the largest Asian populations, and 49% of all Hispanics lived in the 10 metro areas with the largest Hispanic populations. The majority of these metropolitan areas (e.g., Los Angeles, California, and New York City, New York) were also designated as nonattainment areas for PM2.5 and ozone (*24*). In addition, 15.4% of the nonattainment counties for either or both pollutants were in California, where Asians represented 12.5%, and Hispanics represented 36.6% of the total population in California 2008 (*25*).

Study of other demographic characteristics indicates that persons in the highest income category and in the highest and lowest categories of education attainment have the largest percentages of persons M2.5 and ozone nonattainment counties. These results also likely reflect the demographic distribution of persons who live in predominantly urban areas. The populations in urban centers and

metropolitan areas tend to be diverse, with areas of wealth integrated with those in poverty (*26--30*).

Nonattainment areas often are affected by pollution sources such as heavy traffic and other environmental hazards (e.g., industrial emissions) that can affect health. A growing body of research demonstrates that proximity to sources such as traffic can have adverse health effects, especially with respect to vehicular emissions, including PM2.5. In a recent review, the Health Effects Institute concluded that sufficient evidence exists to indicate that traffic exposure exacerbates asthma among children, and suggestive but not sufficient evidence indicates that these exposures cause other adverse health effects (e.g., impaired lung function and increased cardiovascular morbidity and mortality) (*31*). Zones most affected by traffic-related exposures are estimated to be 300--500 m away from major roads (i.e., roads with intermediate levels of traffic volumes that are less than highways, freeways, and motorways); calculated for large cities, >33% of the U.S. population live in these affected areas (*31*).

Certain segments of the population, such as very young children and older adults, are particularly susceptible to the effects of air pollution. Because industrial facilities and motor vehicles are key contributors to the levels of PM2.5 and ozone production (*7,11*), public health efforts should continue to focus on measures to reduce sources of pollution (e.g., promotion of mass transit and development of technology to reduce mobile and stationary source emissions), which in turn should reduce population exposures to unhealthy air quality (*32--35*).

References

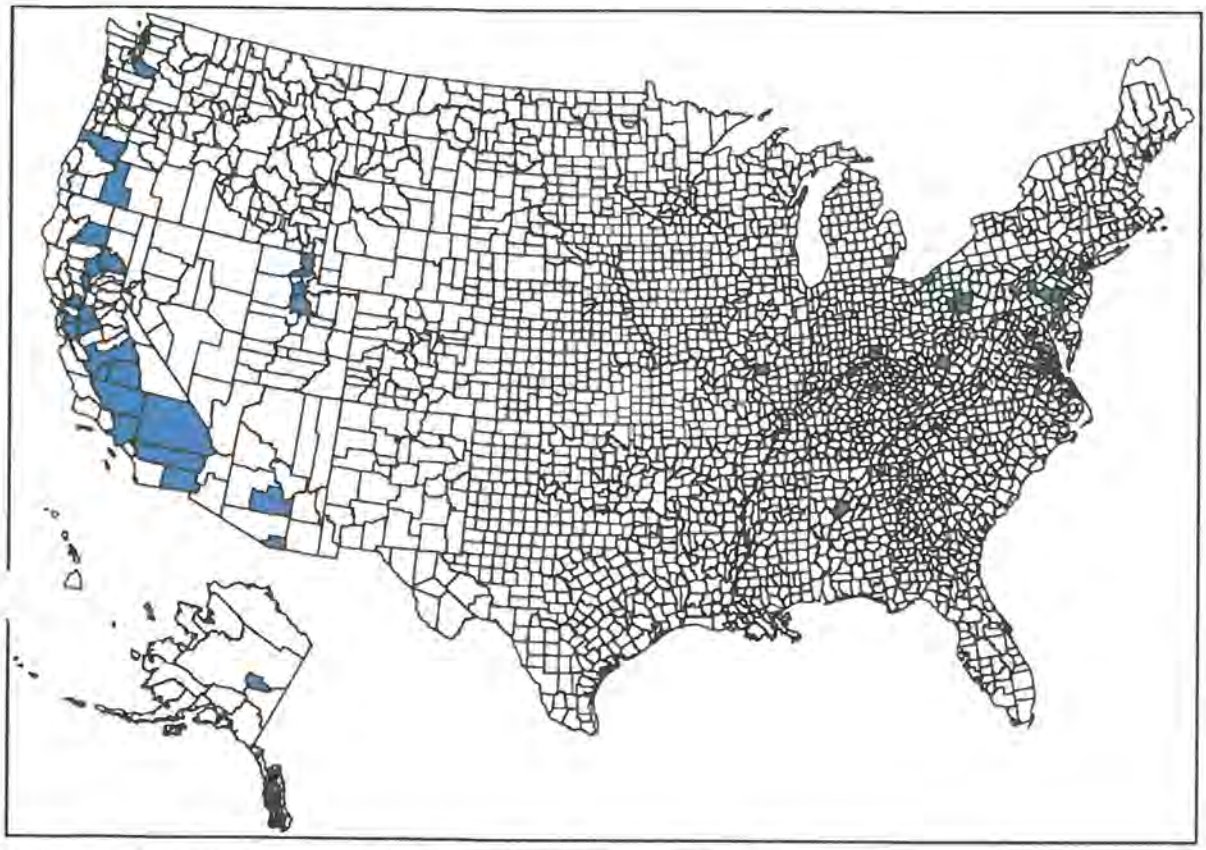
1. Dockery D. Health effects of particulate pollution. Ann Epidemiol 2009;19:257--63.
2. Pope CA 3rd, Dockery DW. Health effects of fine particulate air pollution: lines that connect. J Air Waste Manag Assoc 2006;56:709--42.
3. Brook RD, Rajagopalan S, Pope CA 3rd, et al. Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. Circulation 2010;121:2331--78.
4. Pope CA 3rd, Ezzati M, Dockery DW. Fine-particulate air pollution and life expectancy in the United States. N Engl J Med 2009;360:376--86.
5. Pope CA 3rd, Burnett RT, Thun MJ, et al. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA 2002;287:1132--41.
6. Dockery DW, Pope CA 3rd, Xu X, et al. An association between air pollution and mortality in six U.S. cities. N Engl J Med 1993;329:1753--9.
7. US Environmental Protection Agency (EPA). Integrated science assessment for particulate matter (final report). Washington, DC: US EPA; 2009. Publication no. EPA/600/R-08/139F. Available at http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=216546.
8. US Environmental Protection Agency (EPA). National air quality: status and trends through 2008. Research Triangle Park, NC: US EPA; 2010. Publication no. EPA-454/R-09-002. Available at http://www.epa.gov/airtrends/2010.
9. Bell ML, McDermott A, Zeger SL, Samet JM, Dominici F. Ozone and short-term mortality in 95 U.S. urban communities, 1987--2000. JAMA 2004;292:2372--8.
10. Kinney PL, Ozkaynak H. Associations of daily mortality and air pollution in Los Angeles County. Environ Res 1991;54:99--120.
11. US Environmental Protection Agency (EPA). Air quality criteria for ozone and related photochemical oxidants (2006 final). Washington, DC: US EPA; 2006. Publication no. EPA/600/R-05/004aF-cF. Available at http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=149923.
12. Adams WC. Human pulmonary responses with 30-minute time intervals of exercise and rest when exposed for 8 hours to 0.12 ppm ozone via square-wave and acute triangular profiles. Inhal Toxicol 2006;18:413--22.
13. McDonnell WF, Stewart PW, Andreoni S, Smith MV. Proportion of moderately exercising

individuals responding to low-level, multi-hour ozone exposure. Am J Respir Crit Care Med 1995;152:589--96.
14. Anderson HR, Atkinson RW, Peacock JL, Marston L, Konstantinou K. Meta-analysis of time-series studies and panel studies of particulate matter (PM) and ozone (O3): report of a WHO task group. Geneva, Switzerland: World Health Organization, Europe; 2004. Available at http://www.euro.who.int/__data/assets/pdf_file/0004/74731/e82792.pdf.
15. Gent JF, Triche EW, Holford TR, et al. Association of low-level ozone and fine particles with respiratory symptoms in children with asthma. JAMA 2003;290:1915--67.
16. Giardot SP, Ryan PB, Smith SM, et al. Ozone and PM2.5 exposure and acute pulmonary health effects: a study of hikers in the Great Smoky Mountains National Park. Environ Health Perspect 2006;114:1044--52.
17. Triche EW, Gent JF, Holford TR, et al. Low-level ozone exposure and respiratory symptoms in infants. Environ Health Perspect 2006;114:911--6.
18. Höppe P, Peters A, Rabe G, et al. Environmental ozone effects in different population subgroups. Int J Hyg Environ Health 2003;206:505--16.
19. McConnell R, Berhane K, Gilliland F, et al. Asthma in exercising children exposed to ozone: a cohort study. Lancet 2002;359:386--91.
20. Korrick SA, Neas LM, Dockery DW, et al. Effects of ozone and other pollutants on the pulmonary function of adult hikers. Environ Health Perspect 1998;106:93--9.
21. Spektor DM, Lippmann M, Thurston GD, et al. Effects of ambient ozone on respiratory function in healthy adults exercising outdoors. Am Rev Respir Dis 1988;138:821--8.
22. CDC, National Center for Health Statistics (NCHS). 2006 NCHS urban-rural classification scheme for counties. Hyattsville, MD: NCHS. Available at http://www.cdc.gov/nchs/data_access/urban_rural.htm.
23. US Department of Agriculture, Economic Research Service. Minorities represent growing share of tomorrow's work force. Rural Conditions and Trends 1999;9(2):9--13. Available at http://www.ers.usda.gov/publications/rcat/rcat92/rcat92b.pdf.
24. Frey WH, Berube A, Singer A, Wilson JH. Getting current: recent demographic trends in metropolitan America. Washington DC: Brookings Institution; 2009. http://www.brookings.edu/reports/2009/03_metro_demographic_trends.aspx.
25. US Census Bureau. American factfinder. 2008 population estimates. Available at http://factfinder.census.gov.
26. DeNavas-Walt C, Proctor B, Smith J. Income, poverty, and health insurance coverage in the United States: 2008. Current population reports (P60-236[RV]). Washington DC: US Census Bureau; 2009. Available at http://www.census.gov/prod/2009pubs/p60-236.pdf.
27. Byun S, Meece J, Irvin, M. Rural-nonrural differences in educational attainment: results from the National Educational Longitudinal Study of 1988-2000. Paper presented at the annual meeting of the American Educational Research Association, Denver, CO; May 2010. Available at www.nrcres.org/Byun%20et%20al_rural_nonrural_differences_in_educational_attainment_2010_AERA.doc.
28. McLaughlin D. Income inequality in America. Rural America 2002;17:14--20.
29. UN Habitat: State of the world's cities 2010/2011. Bridging the urban divide. Nairobi, Kenya: United Nations Human Settlements Programme; 2010. Available at http://www.unhabitat.org/documents/SOWC10/L8.pdf.
30. Bauman KJ, Graf NL. Educational attainment: 2000. Census 2000 brief. Washington DC: US Census Bureau; 2003. Publication no. C2KBR-24. Available at http://www.census.gov/prod/2003pubs/c2kbr-24.pdf.
31. Health Effects Institute. Traffic-related air pollution: a critical review of the literature on emissions, exposure, and health effects. Boston, MA: Health Effects Institute; 2010. Publication no. HEI special report 17.
32. Cirera L, Rodriguez M, Gimenez, J, et al. Effects of public health interventions on industrial emissions and ambient air in Cartagena, Spain. Environ Sci Pollut Res 2009;16:152--61.
33. Johansson C, Burman L, Forsberg B. The effects of congestions tax on air quality and health. Atmospheric Environment 2009;43:4843--54.
34. Pope CA 3rd. Respiratory hospital admissions associated with PM10 pollution in Utah, Salt

Lake, and Cache Valleys. Arch Environ Health 1991;46:90--7.
35. Wang S, Zhao M, Xing J, et al. Quantifying the air pollutants emission reduction during the 2008 Olympic games in Beijing. Environ Sci Technol 2010;44:2490--6.

*Clean Air Act Amendments of 1970. Pub. L. No. 91-604, 81 Stat. 486 (December 31, 1970).

FIGURE 1. Counties in 24-hour fine particulate matter (PM2.5) nonattainment areas* --- United States, 2006--2008

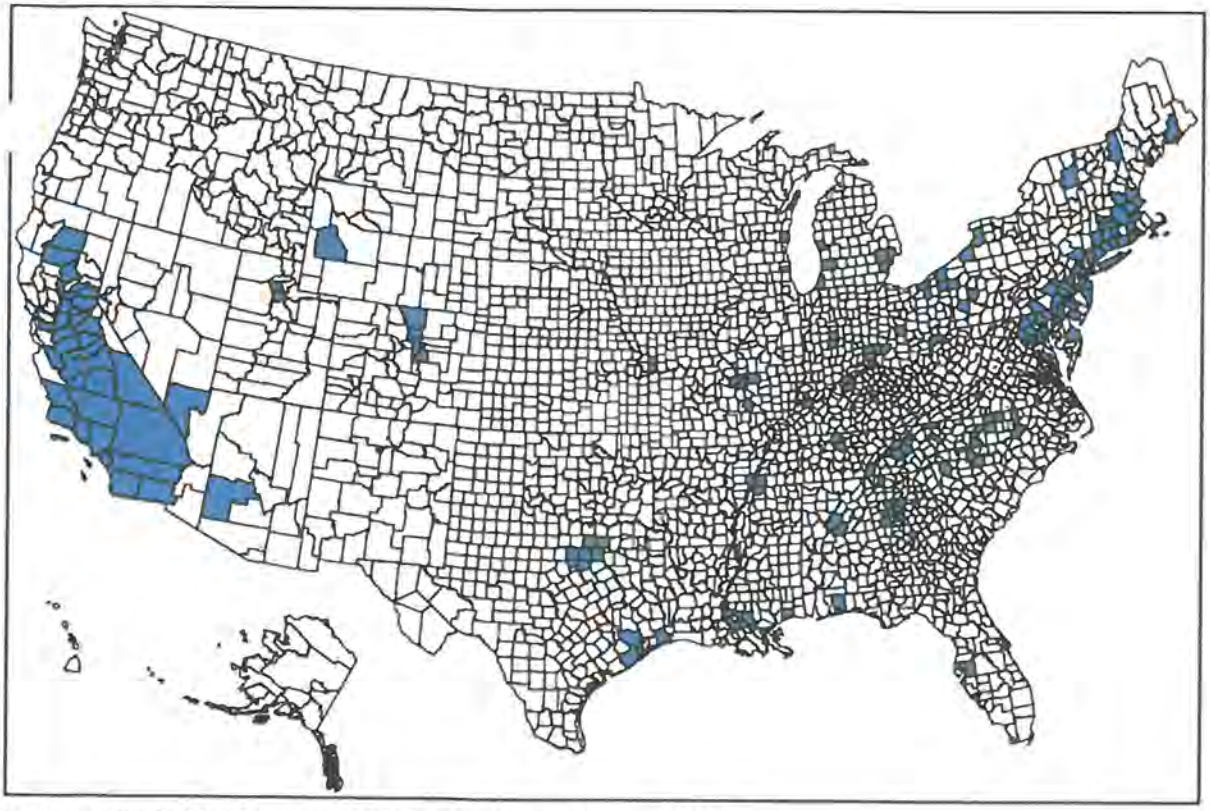


Source: U.S. Environmental Protection Agency (EPA).

* Counties that did not meet the EPA 2006 PM2.5 standard of 35 $\mu g/m^3$ from 2006--2008.

Alternate Text: The figure shows a county-level map of the United States, indicating the 53 counties that did not meet the EPA 2006 PM2.5 standard of 35 μg/m3 from 2006-2008. Clusters of counties appear in the western region, especially, California and the northeastern region.

FIGURE 2. Counties in 8-hour ozone nonattainment areas* --- United States, 2007--2009



Source: U.S. Environmental Protection Agency (EPA).

* Counties that did not meet the EPA 2008 ozone standard of 75 ppb from 2007--2009.

ernate Text: The figure shows a county-level map of the United States, indicating the 201 counties that did not meet the EPA 2008 ozone standard of 75 ppb from 2007-2009. Clusters of counties appear in California and northeastern coast.

TABLE. Percentage and relative difference of populations living in fine particulate matter (PM2.5) and ozone nonattainment* counties, by selected characteristics --- United States, 2006--2008 and 2007--2009

Characteristic	PM2.5 (2006–2008)		Ozone (2007–2009)	
	Population† in nonattainment counties (%)	Relative difference§ (%)	Population¶ in nonattainment counties (%)	Relative difference§ (%)
Race/Ethnicity				
White, non-Hispanic	9.7	Ref.	32.0	Ref.
Black, non-	15.2	56	40.0	25

Hispanic				
American lian/Alaska Native	8.2	--16	18.6	--42
Asian	26.2	169	50.2	57
Native Hawaiian/other Pacific Islander	22.0	125	36.5	14
Non-Hispanic, multiple races	15.2	56	36.1	13
Hispanic	26.6	163	48.4	51
Household income (PIR)**				
<1 (poor)	13.1	Ref.	30.6	Ref.
1 to <2 (near-poor)	12.2	--7	32.2	5
to <3 (middle income)	12.2	--6	34.1	12
3 to ≥5 (high income)	13.3	2	41.1	34
Education level**				
Less than high school	16.4	Ref.	36.1	Ref.
High school diploma (including GED)	11.9	--28	31.9	--12
Some college education	12.9	--22	35.2	--3
College graduate or higher	13.2	--20	39.6	10

Abbreviation: GED = general equivalency diploma; PIR = poverty to income ratio.

* Counties that did not meet the National Ambient Air Quality Standards set forth in the Clean Air Act Amendments of 1970 for the 2006 24-hour PM2.5 standard of 35 μg/m3 from 2006--2008 and the 2008 8-hour ozone standard of 75 ppb from 2007--2009.

† Based on 2007 population (U.S. Census).

elative difference (percentage difference) was calculated by dividing the difference between the referent category and each category in the classifying variable by the value in the referent category and multiplying by 100.

¶ Based on 2008 population (U.S. Census).

** Based on data from 2006–2008.

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Brook RD et all including David Siscovick and Joel D Kaufman of the University of Washington: Particulate Matter Air Pollution and Cardiovascular Disease. An Update to the Scientific Statement From the American ~~Health~~ Heart Association. Circulation 2010;121:2331-2378

This is an authoritative review of the current evidence implicating adverse health effects, particularly cardiovascular, on citizens. The paper should be part of the discussion. The State of Washington and particularly the Department of Transportation and other planning agencies should be using this information ~~at~~ in discussing the implications with a wide range of experts on air pollution including those available locally.

Douglas K. Stewart MD

Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION

Particulate Matter Air Pollution and Cardiovascular Disease. An Update to the Scientific Statement From the American Heart Association

Robert D. Brook, Sanjay Rajagopalan, C. Arden Pope, III, Jeffrey R. Brook, Aruni Bhatnagar, Ana V. Diez-Roux, Fernando Holguin, Yuling Hong, Russell V. Luepker, Murray A. Mittleman, Annette Peters, David Siscovick, Sidney C. Smith, Jr, Laurie Whitsel, Joel D. Kaufman and on behalf of the American Heart Association Council on Epidemiology and Prevention, Council on the Kidney in Cardiovascular Disease, and Council on Nutrition, Physical Activity and Metabolism

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AHA Scientific Statement

Particulate Matter Air Pollution and Cardiovascular Disease

An Update to the Scientific Statement From the American Heart Association

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***Abstract*—**In 2004, the first American Heart Association scientific statement on "Air Pollution and Cardiovascular Disease" concluded that exposure to particulate matter (PM) air pollution contributes to cardiovascular morbidity and mortality. In the interim, numerous studies have expanded our understanding of this association and further elucidated the physiological and molecular mechanisms involved. The main objective of this updated American Heart Association scientific statement is to provide a comprehensive review of the new evidence linking PM exposure with cardiovascular disease, with a specific focus on highlighting the clinical implications for researchers and healthcare providers. The writing group also sought to provide expert consensus opinions on many aspects of the current state of science and updated suggestions for areas of future research. On the basis of the findings of this review, several new conclusions were reached, including the following: Exposure to PM <2.5 μm in diameter ($PM_{2.5}$) over a few hours to weeks can trigger cardiovascular disease–related mortality and nonfatal events; longer-term exposure (eg, a few years) increases the risk for cardiovascular mortality to an even greater extent than exposures over a few days and reduces life expectancy within more highly exposed segments of the population by several months to a few years; reductions in PM levels are associated with decreases in cardiovascular mortality within a time frame as short as a few years; and many credible pathological mechanisms have been elucidated that lend biological plausibility to these findings. It is the opinion of the writing group that the overall evidence is consistent with a causal relationship between $PM_{2.5}$ exposure and cardiovascular morbidity and mortality. This body of evidence has grown and been strengthened substantially since the first American Heart Association scientific statement was published. Finally, $PM_{2.5}$ exposure is deemed a modifiable factor that contributes to cardiovascular morbidity and mortality. **(*Circulation*. 2010;121:2331-2378.)**

Key Words: AHA Scientific Statements ■ atherosclerosis ■ epidemiology ■ prevention ■ air pollution ■ public policy

In 2004, the American Heart Association (AHA) published its first scientific statement regarding air pollution and cardiovascular disease (CVD).[1] The rationale was to provide researchers, healthcare providers, and regulatory agencies with a comprehensive review of the evidence linking air pollution exposure with cardiovascular morbidity and mor-

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tality. There was also an explicit aim to educate clinicians about the importance of this issue, because the cardiovascular health consequences of air pollution generally equal or exceed those due to pulmonary diseases.[1–4] Finally, a list of key remaining scientific questions and strategic avenues for investigation were provided to help foster and guide future research.

The first AHA writing group concluded that short-term exposure to particulate matter (PM) air pollution contributes to acute cardiovascular morbidity and mortality[1] and that exposure to elevated PM levels over the long term can reduce life expectancy by a few years. Although some mechanistic details remained incompletely described, the existing science was deemed adequate to substantiate several plausible biological pathways whereby PM could instigate acute cardiovascular events and promote chronic disease.

There is mounting evidence from a rapid growth of published data since the previous statement related to the harmful cardiovascular effects of air pollution.[3,4] Most, but not all, epidemiological studies corroborate the elevated risk for cardiovascular events associated with exposure to fine PM <2.5 μm in aerodynamic diameter ($PM_{2.5}$). $PM_{2.5}$ generally has been associated with increased risks of myocardial infarction (MI), stroke, arrhythmia, and heart failure exacerbation within hours to days of exposure in susceptible individuals. Several new studies have also demonstrated that residing in locations with higher long-term average PM levels elevates the risk for cardiovascular morbidity and mortality. Some recent evidence also implicates other size fractions, such as ultrafine particles (UFPs) <0.1 μm, gaseous copollutants (eg, ozone and nitrogen oxides [NO_x]), and specific sources of pollution (eg, traffic). In addition, there have been many insights into the mechanisms whereby PM could prove capable of promoting CVDs.[2–4] Air pollutants have been linked with endothelial dysfunction and vasoconstriction, increased blood pressure (BP), prothrombotic and coagulant changes, systemic inflammatory and oxidative stress responses, autonomic imbalance and arrhythmias, and the progression of atherosclerosis. In the interim, the US Environmental Protection Agency (EPA) completed its updated "Air Quality Criteria for Particulate Matter"[5] and afterward strengthened the National Ambient Air Quality Standards (NAAQS) for daily $PM_{2.5}$ levels starting in 2006 (down from 65 to 35 μg/m^3).[6] The most recent scientific review coordinated by the EPA, the final report of the Integrated Science Assessment for Particulate Matter (http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=216546), has also been made available publicly. These numerous changes and advances provide the rationale for the present updated AHA scientific statement on PM air pollution and CVD. This updated statement is similar in scope, content, and overall structure to the first document; however, it provides many additional conclusions and recommendations that can now be made because of the expanded number and quality of studies.

Objectives and Methods

The primary objective of this scientific statement is to provide a comprehensive updated evaluation of the evidence linking PM exposure with CVDs. The focus of this review is explicitly on PM because the majority of air pollution studies have centered on its cardiovascular effects, and the strength of the evidence makes it possible to provide consensus opinions and recommendations. Except for in a few circumstances, such as when copollutants have been shown to (or not to) modify the responses to PM exposure or to have independent cardiovascular effects in epidemiological studies of major importance, a detailed discussion of other air pollutants (eg, ozone and NO_2) is beyond the scope of this document. Additional objectives are to provide expert consensus opinions on aspects related to the current state of science, to specifically highlight the health and clinical implications of the reviewed findings, and to provide prudent and practical recommendations for measures to reduce PM exposure that might thereby lower the associated cardiovascular risk. This updated scientific statement is structured to first provide a clinical perspective on the cardiovascular risks posed by PM exposure and then briefly review the components of air pollution. The following sections highlight the major findings from epidemiological studies, including mortality, morbidity, and surrogate outcome results. Next, the animal and human mechanistic studies are reviewed, and an overall framework whereby PM exposure could cause CVDs is outlined. Finally, updated consensus opinions and conclusions are provided, followed by suggestions for areas of future research and policy considerations.

Members of the current writing group were selected from across a broad range of disciplines, including cardiovascular and environmental epidemiology and statistics, atmospheric sciences, cardiovascular and pulmonary medicine, basic science research, and public policy. The writing group identified studies published in the English language between January 1, 2004, and March 31, 2009, by a World Wide Web–based literature search using Medline, PubMed, and Google search engines. Key terms included *air pollution* or *particulate matter* plus any of the following: *cardiovascular, myocardial, heart, cardiac, stroke, heart failure, arrhythmia, heart rate variability, autonomic, sympathetic, atherosclerosis, vascular, blood pressure, hypertension, diabetes, metabolic, thrombosis,* and *coagulation*. Additional studies were identified within the references of these publications and by the personal knowledge of the writing group members. A few studies published after March 31, 2009, were added during the review process. All of the identified epidemiological studies that provided mortality data or hard cardiovascular outcomes (eg, MIs) and controlled human exposure protocols were included. In a few circumstances, studies before 2004 were included briefly in the discussion or tables when it was believed that they provided contextual background and/or relevant findings from earlier analyses of ongoing studies (eg, Harvard Six Cities and American Cancer Society [ACS] cohorts) from which new results after 2004 have been published. It is a limitation of the present review that it was not possible to cite all surrogate outcome human studies because of the enormous number of publications. Some were not included, without intentional bias with regard to results, when multiple referenced studies demonstrated similar findings. In such a situation (eg, heart rate variability [HRV]), this

limitation was noted within the specific section. A main theme of the present statement is to provide clinical context and recommendations for healthcare providers, and thus, it was beyond the scope and not the intent of this document to include all animal, ex vivo, or toxicological studies. A number of these publications were also not included, without intentional bias with regard to results. The writing group included publications that were believed to have relevant implications for human cardiovascular health, those that formed the foundation of the mechanistic hypotheses, and studies that were deemed of major importance. Finally, the "evidence summary" statements and all points in the conclusions and recommendations represent consensus expert opinions agreed on by all members of the writing group during formal discussions. It is explicitly stated when no such agreement was reached. These statements and the points within Tables 6 and 7 do not represent the result of applying the standard AHA criteria (ie, level and class) to the sum findings of the present review, because those do not apply, but rather the qualitative consensus opinions agreed on by the writing group. The purpose is to provide expert opinions on the comparative relative ranking and the strength of the overall evidence regarding different areas within this field of science.

Perspective on the Air Pollution–Cardiovascular Risk Association

Traditional cardiovascular risk factors account for the major portion of the risk for ischemic cardiac events within a population.[7] Individuals with optimal levels of all risk factors have been shown to have a low lifetime cardiovascular event rate.[8] Thus, control of the traditional risk factors is recognized to be of paramount importance to prevent CVDs. In this context, there has been some debate about the overall clinical relevance and utility of adding novel risk factors to risk-prediction models to incrementally improve their overall predictive value, even when assessed by multiple methodologies.[9] On the other hand, the ability to predict future events by existing models remains imperfect. In addition to several mathematical and statistical explanations for this shortcoming,[10,11] it is important to recognize that the development of vascular or atherosclerotic disease (the factor predicted by most statistical models) is usually a necessary but insufficient cause of future ischemic events in and of itself. Cardiovascular events must also be triggered by an additional factor at some unknowable future time, and therefore, they transpire as a stochastic process within a population.[12] This is one of several reasons why PM air pollution is a uniquely important public health issue among the list of novel risk factors; PM inhalation is an established trigger of cardiovascular events that occur within hours to days after exposure.[12] Because of the ubiquitous and involuntary nature of PM exposure, it may continuously enhance acute cardiovascular risk among millions of susceptible people worldwide in an often inconspicuous manner. Moreover, beyond serving as a simple trigger, PM elicits numerous adverse biological responses (eg, systemic inflammation) that, in premise, may further augment future cardiovascular risk over the long term after months to years of exposure.

Effects of Short-Term Exposure

Time-series studies estimate that a 10-$\mu g/m^3$ increase in mean 24-hour $PM_{2.5}$ concentration increases the relative risk (RR) for daily cardiovascular mortality by approximately 0.4% to 1.0%.[3] Despite theoretical statistical risks ascribed to all individuals, this elevated risk from exposure is not equally distributed within a population. At present-day levels, $PM_{2.5}$ likely poses an acute threat principally to susceptible people, even if seemingly healthy, such as the elderly and those with (unrecognized) existing coronary artery or structural heart disease.[13] Therefore, the absolute risk rather than the RR of exposure may more effectively convey the tangible health burden within a population. A 10-$\mu g/m^3$ increase during the preceding day contributes on average to the premature death of approximately 1 susceptible person per day in a region of 5 million people (based on annual US death rates in 2005).[3,14] Although the dangers to 1 individual at any single time point may be small, the public health burden derived from this ubiquitous risk is enormous. Short-term increases in $PM_{2.5}$ levels lead to the early mortality of tens of thousands of individuals per year in the United States alone.[1,3,5]

Effects of Long-Term Exposure

Cohort studies estimate that the RR associated with living in areas with higher PM levels over the long term is of greater magnitude than that observed from short-term exposure increases (RR between 1.06 and 1.76 per 10 $\mu g/m^3$ $PM_{2.5}$).[3] In this context, the World Health Organization estimated that $PM_{2.5}$ contributes to approximately 800 000 premature deaths per year, ranking it as the 13th leading cause of worldwide mortality.[15] Hence, PM air pollution appears to be an important modifiable factor that affects the public health on a global scale.

Air Pollution

The first AHA statement on air pollution reviewed the size fractions, sources, and chemical constituents of PM and the main gaseous air pollutants: Nitrogen oxides (NO_x; ie, $NO+NO_2$), carbon monoxide (CO), sulfur dioxide (SO_2), and ozone (O_3).[1] Therefore, this section within the updated statement focuses on several other contemporary aspects of air pollution characterization and exposure assessment, particularly in relation to their potential influences on cardiovascular health. In brief, PM is broadly categorized by aerodynamic diameter: All particles <10 μm (thoracic particles [PM_{10}]), all particles <2.5 μm (fine particles [$PM_{2.5}$]), all particles <0.1 μm (UFP), and particles between 2.5 and 10 μm (coarse particles [$PM_{10-2.5}$]). Hence, PM_{10} contains within it the coarse and $PM_{2.5}$ fractions, and $PM_{2.5}$ includes UFP particles. The concentrations of PM_{10} and $PM_{2.5}$ are typically measured in their mass per volume of air ($\mu g/m^3$), whereas UFPs are often measured by their number per cubic centimeter (Table 1). The major source of $PM_{2.5}$ throughout

Table 1. Ambient Air Pollutants

Pollutant	US Average Range	US Typical Peak*	Most Recent NAAQS for Criteria Pollutants (Averaging Time)
O_3†	0–125 ppb	200 ppb	75 ppb (8 h)‡
NO_2†	0.5–50 ppb	200 ppb	100 ppb (1 h)§ 53 ppb (Annual mean)
NO†	0–100 ppb	200 ppb	
SO_2†	0.1–50 ppb	150 ppb	140 ppb (24 h)‖ 30 ppb (Annual mean)
CO†	0.1–5 ppm	20 ppm	35 ppm (1 h)‖ 9 ppm (8 h)‖
PM_{10}¶	10–100 $\mu g/m^3$	300 $\mu g/m^3$	150 $\mu g/m^3$ (24 h)#
$PM_{2.5}$¶	5–50 $\mu g/m^3$ (Mean=13.4±5.6)	100 $\mu g/m^3$	15 $\mu g/m^3$ (Annual mean) 35 $\mu g/m^3$ (24 h)**
$PM_{2.5}$ lead¶	0.5–5 ng/m^3	150 ng/m^3	0.15 $\mu g/m^3$ (Rolling 3-month average)††
NH_3†	0.1–20 ppb	100 ppb	
HNO_3†	0–5 ppb	10 ppb	
Methane†	1–2 ppm	5 ppm	
Formaldehyde†	0.1–10 ppb	40 ppb	
Acetaldehyde†	0.1–5 ppb	20 ppb	
NMHC (VOC)¶	20–100 $\mu g/m^3$	250 $\mu g/m^3$	
Propane¶	2–20 $\mu g/m^3$	500 $\mu g/m^3$	
Benzene¶	0.5–10 $\mu g/m^3$	100 $\mu g/m^3$	
1,3-Butadiene¶	0.1–2 $\mu g/m^3$	10 $\mu g/m^3$	
Total suspended particles¶	20–300 $\mu g/m^3$	1000 $\mu g/m^3$	
$PM_{10-2.5}$¶	5–50 $\mu g/m^3$	200 $\mu g/m^3$	
Sulfate¶	0.5–10 $\mu g/m^3$	30 $\mu g/m^3$	
Nitrate¶	0.1–5 $\mu g/m^3$	20 $\mu g/m^3$	
Organic carbon¶	1–20 $\mu g/m^3$	30 $\mu g/m^3$	
Elemental carbon¶	0.1–3 $\mu g/m^3$	10 $\mu g/m^3$	
PAH¶	2–50 ng/m^3	200 ng/m^3	
UFP†	1000–20 000/cm^3	100 000/cm^3	

ppb Indicates parts per billion; ppm, parts per million; and PAH, polycyclic aromatic hydrocarbon.

*Generally not in concentrated plumes or locations of direct source emission impact.

†Typical hourly average concentrations reached in US cities.

‡The 8-hour standard is met when the 3-year average of the 4th highest daily maximum 8-hour average is less than or equal to the indicated number. In January 2010, the EPA proposed a more stringent 8-hour standard within the range of 60 to 70 ppb (http://www.epa.gov/air/ozonepollution/actions.html).

§To attain this standard, the 3-year average of the 98th percentile of the daily maximum 1-hour average at each monitor within an area must not exceed this value.

‖The level is not to be exceeded more than once per year.

¶Typical 24-hour average concentrations.

#The level is not to be exceeded more than once per year on average over 3 years.

**The daily standard is met when the 3-year average of the 98th percentile of 24-hour PM level is less than or equal to the indicated number.

††Although the typical concentrations shown in the table are for $PM_{2.5}$, the lead standard continues to be based on measurements in total suspended particulate.

the world today is the human combustion of fossil fuels from a variety of activities (eg, industry, traffic, and power generation). Biomass burning, heating, cooking, indoor activities, and nonhuman sources (eg, fires) may also be relevant sources, particularly in certain regions.

Common air pollutants and those designated as EPA criteria pollutants (ie, specifically targeted in regulations through limits on emissions or government standards such as the NAAQS) are listed in Table 1. The World Health Organization also provides ambient guidelines (http://www.euro.who.int/Document/E90038.pdf). As a result, many pollutant concentrations are tracked in the United States by nationwide monitoring networks, with up to approximately 1200 sites for O_3 and $PM_{2.5}$. Data are archived by the EPA and are available to the public (http://www.epa.gov/ttn/airs/airsaqs/). O_3 levels exceed the national standard in many areas, and thus, daily information is provided to assist the public in reducing their exposure. A lower standard for ozone concentrations was proposed recently, which will lead to more frequent occurrences of outdoor exposures deemed to be excessive (Table 1). The reporting of $PM_{2.5}$ is also becoming common because of its impact on public health and frequent violations of standards. Current and forecast air quality indices and information on both $PM_{2.5}$ and ozone are available (http://airnow.gov/). At the end of 2008, 211 US counties (or portions of counties) were in nonattainment of the 2006 daily $PM_{2.5}$ NAAQS (http://www.epa.gov/pmdesignations/2006standards/state.htm). On a positive note, the various regulations that have been established have led to substantial reductions in PM and other pollutant levels over the past 40 years in the United States and contributed toward similar improvements in other countries. However, reducing the levels of some pollutants, such as O_3, remains a challenge because of the complex chemical processes that lead to their formation in the atmosphere.[16] The population of many developing nations (China, India, Middle Eastern countries) continues to be exposed to high levels, particularly of PM, which can routinely exceed 100 $\mu g/m^3$ for prolonged periods (http://siteresources.worldbank.org/DATASTATISTICS/Resources/table 3_13.pdf).

Air Pollution Mixtures, Chemistry, and Sources

Detailed information regarding PM sizes, composition, chemistry, sources, and atmospheric interactions is beyond the scope of this document but can be found in the 2004 US EPA Air Quality Criteria for Particulate Matter final report (http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=87903). The source for much of the information provided in this brief summary is this document, unless otherwise specifically referenced. The typical range of ambient concentrations for several air pollutants in the United States, including the latest US NAAQS for the criteria pollutants, is given in Table 1. Classification of air quality according to 1 single pollutant and by size or mass provides an incomplete picture, because ambient air pollution is a complex mixture of gases, particles, and liquids that are continually changing and interacting with each other and natural atmospheric gases. Although $PM_{2.5}$ mass has rightfully attracted considerable attention as a target for regulation and epidemiological study, more than 98% of

the air pollutant mass in the mixture we breathe in urban settings is from gases or vapor-phase compounds such as CO, nonmethane hydrocarbons or volatile organic carbons (VOCs), NO_2, NO, O_3, and SO_2. Each of these can have independent and potentially synergistic or antagonistic effects with each other and with PM; however, at present, the cardiovascular health impact of exposure to combinations of air pollutants is not well understood.

Most of the studies linking CVDs with PM exposures have focused on particle mass; thus, this association is evaluated and reported in the majority of epidemiological and toxicological studies reviewed. Although PM is regulated by mass concentration, the aspect of PM most harmful to cardiovascular health may not be best quantified by mass measurement alone. The sum effect of many features related to chemical composition and size/morphology (eg, oxidative stress potential, solubility, charge, surface area, particle count, lung deposition, and stability within the atmosphere and biological tissues) is important to consider. With regard to specific "toxic" compounds within PM, several lines of existing evidence support the idea that transition metals, organic compounds, semiquinones, and endotoxin are likely relevant in relation to promoting CVDs. In addition, certain characteristics of UFPs (eg, high surface area, particle number, metal and organic carbon content) suggest that they may pose a particularly high cardiovascular risk after short-term exposure.[17] Both the additional characterization of "criteria" pollutants and the measurement of several other pollutants (discussed below) are important to inform air quality management practices that involve air quality modeling, as well as epidemiological studies and risk assessment, which ultimately aim to improve risk-reduction strategies.

In addition to their mass concentration, pollutants can be characterized on the basis of their origin or chemical and physical properties. In terms of origin, nitrogen oxides ($NO+NO_2$), CO, SO_2, and $PM_{2.5}$, as well as carbon dioxide (CO_2), are mainly associated with combustion of fuel or other high-temperature industrial processes. Combustion PM is composed of many chemical compounds, including organic carbon species, elemental or black carbon, and trace metals (eg, lead and arsenic). They range in size from molecular clusters a few nanometers in diameter to light-scattering particles that peak on a mass contribution basis in the diameter range of 200 to 1000 nm (0.2 to 1 μm). UFP numbers are also strongly linked to fresh combustion and traffic-related pollution. Ammonia, methane, pesticides (persistent organic pollutants), reduced sulfur compounds, resuspended dust, and natural coarse particles ($PM_{10-2.5}$) are associated with noncombustion surface or fugitive releases that arise from a variety of human (eg, agriculture) and natural (eg, erosion) activities. Agricultural emissions and releases from a range of industrial processes and waste management are also important sources. Road and wind-blown dust from agricultural practices and from certain industrial facilities (eg, mineral industry) also contribute to these particles, which are typically in the coarse ($PM_{10-2.5}$) or even larger ($>PM_{10}$) range.

In addition to pollutants formed directly by combustion, many others are produced primarily through chemical reactions in the atmosphere among directly emitted pollutants. These are known as secondary pollutants. Sunlight, water vapor, and clouds are often involved in this atmospheric chemistry, which leads to greater oxidation of the pollutants. Examples include PM-associated sulfate, nitrate, and ammonium and many of the organic compounds within $PM_{2.5}$. Besides O_3, which is the most prevalent secondary gaseous oxidant, a number of inorganic and organic acids and VOCs form in the atmosphere. Examples are the hydroxyl radical, peroxyacetyl nitrate, nitric acid, formic and acetic acid, formaldehyde, and acrolein.

VOCs and semivolatile organic compounds (SVOCs), the latter of which are found in both the gas and particle phase, are an additional large class of pollutants. They are associated with both combustion and fugitive emissions, as well as with secondary formation. Key examples are benzene, toluene, xylene, 1,3-butadiene, and polycyclic aromatic hydrocarbons. VOCs are among the 188 hazardous air pollutants listed by the EPA, and their main emission sources have been identified and are regulated (http://www.epa.gov/ttn/atw/mactfnlalph.html). VOCs can undergo reactions that convert toxic substances to less toxic products or vice versa. Many VOCs contribute to the formation of O_3 and are oxidized in the atmosphere, becoming SVOCs, and subsequently partition within particles and contribute to the composition of $PM_{2.5}$, as well as to its mass. A great deal of research has focused on $PM_{2.5}$ in the past decade, which has led to advances in measurement technologies[18] and greater understanding of its chemistry and atmospheric behavior.[19] Nonetheless, understanding is incomplete, particularly with regard to formation of the secondary organic fraction, the relative role of anthropogenic and biogenic emissions to organics, surface chemistry, oxidative potential,[20] and gas-to-particle partitioning.

An alternative to attempting to identify one by one which pollutant(s) or chemical compounds are most harmful is to focus on identifying the sources, which typically emit mixtures of pollutants, of greatest concern. It may be the mixture of pollutants (along with the source from which it is derived, which determines its characteristics) that is most pertinent to human health outcomes. Such information may actually be more relevant for aiding the development of effective air quality policies. One important example reviewed in the epidemiology section is that the evidence continues to grow regarding the harmful cardiovascular effects of traffic-related pollution. Traffic is ubiquitous in modern society, with a sizeable proportion of the population, particularly persons disadvantaged by low socioeconomic status, living close enough (within 500 m) to a major road or a freeway to be chronically exposed to elevated concentrations. Additionally, daily behavior brings most people close to this source, with the average US citizen over 15 years of age spending 55 minutes each day traveling in motor vehicles.[21] However, despite the consistent epidemiological findings, these studies have yet to elucidate which of the many pollutants or other associated risks (ie, noise) produced by traffic are responsible for the increase in risk for CVD. Until the most harmful agents are identified, the only practical manner to potentially reduce health consequences would be to reduce overall traffic and related emissions and to configure cities and lifestyles

such that there is greater separation between the people and the source, so that we could spend less time in traffic (a major source of personal exposures in our society). There are also a myriad of other important pollutant sources of known toxic pollutants that have been implicated in health-effect studies (eg, power generation, industrial sources, steel mills, and wood smoke). A better understanding of the factors that influence population exposure to these sources, of how their emissions and mixtures of different sources affect health, and about the factors that make individuals more susceptible will aid in the development of more effective environmental health policies.

Determinants of Air Pollution Exposure

Many aspects of air pollution play a role in the characteristics of population- and individual-level exposures. Pollutants vary on multiple time scales, with emission rates, weather patterns, and diurnal/seasonal cycles in solar radiation and temperature having the greatest impact on concentrations. The temporal behavior of a pollutant is also governed by its formation rate and the length of time it remains in the atmosphere. As such, the concentrations of many air pollutants tend to co-vary. For example, NO_x and CO are emitted during combustion, as are some particle constituents (eg, elemental carbon) and VOCs, and thus, their concentrations peak during rush hour. On the other hand, O_3 and other photochemical oxidants, including secondary $PM_{2.5}$ and secondary VOCs, peak in the afternoon, particularly given certain meteorologic conditions (eg, more sunshine). Among the common air pollutants, O_3 and $PM_{2.5}$ have the longest atmospheric lifetime and thus can build up over multiple days and spread, by the prevailing winds, over large geographic regions. This can lead to similarities in their temporal and spatial patterns over broad regions and to greater numbers of people being exposed to similar levels, thus lessening interindividual variability in exposure.

Periods of suppressed horizontal and vertical mixing in the lower atmosphere lead to the buildup of multiple pollutants. These situations are most common under slow-moving or stationary high-pressure systems, which bring light winds, a stable atmosphere, and more sunshine. The frequency and seasonality of these meteorologic conditions and how they affect concentrations vary geographically, which leads to differences in the characteristics of pollution episodes from the western to the eastern United States, as well as within these regions.

The commonality of meteorology and emission sources leads to covariation in pollutant concentrations on multiple temporal and spatial scales, which makes it more challenging for epidemiological studies to identify the health effects of individual pollutants and the effects of copollutants or mixtures. Studies that depend on daily counts of mortality or morbidity events have difficulties separating the effects of the different pollutants in the urban mix. Even prospective panel studies measuring specific end points on a subdaily time scale are hindered by pollutant covariation. Some of these challenges could potentially be addressed by undertaking studies covering multiple geographic locations with differences in the structure of pollutant covariation due to different meteorology and source mixes. Indeed, this has been done, at least in part, by several existing multicity studies. Consistency in the findings in individual studies conducted in different cities also helps isolate the pollutants that may be more responsible for the health effects. The consistent positive findings with certain pollutants (eg, PM mass concentration) have helped strengthen the evidence regarding PM_{10} and $PM_{2.5}$ effects, but regardless of location, there remains the strong underlying commonality of fossil fuel combustion for many pollutants.

A final issue to consider is the cardiovascular health effects of exposures that occur at the personal level because of the different microenvironments or activities an individual experiences (eg, time in traffic, indoor sources, secondhand tobacco smoke, occupational exposure, and degree of indoor penetration of ambient PM into homes) versus the effects of exposures from less variable urban- to regional-scale ambient concentrations (ie, background pollution that most individuals encounter more uniformly). Personal monitoring demonstrates substantial variations among individual pollution exposures or characteristics among those living within the same metropolitan area and even the same neighborhood.[22,23] However, the differing additive, synergistic, and/or confounding effects on cardiovascular health of these 2 contrasting components of a person's overall exposure have not been well described. For the most part, the magnitude of the findings reported by the major epidemiological studies (see next section) are indicative of the effects of the urban- to regional-scale ambient concentrations. Actual exposures to all pollutants also vary at the personal level. The cardiovascular health importance of these individual-level variations (above and beyond the effect of urban/regional levels) remains largely unknown, in part because it has been difficult to quantify. The degree to which measurement of personal exposures or more precise exposure assessment (eg, use of geographic information systems, land-use regression models, spatial-temporal models, and adjustments for indoor penetration) can reduce the effects of exposure misclassification in epidemiological studies also remains to be fully elucidated.[24–26]

Epidemiological Studies of Air Pollution

Epidemiological studies of air pollution have examined the health effects of exposures observed in real-world settings at ambient levels. Associations between relevant health end points and measures of air pollution are evaluated while attempting to control for effects of other pertinent factors (eg, patient and environmental characteristics). Despite substantial study and statistical improvements and the relative consistency of results, some potential for residual confounding of variables and publication bias[27] of positive studies are limitations to acknowledge. Probably the most relevant, well-defined, and extensively studied health end points include mortality (all-cause and cause-specific), hospitalizations, and clinical cardiovascular events. This section reviews the results of the epidemiological research with a focus on new studies since the first AHA statement was published,[1] as well as on the cardiovascular health implications. In sum, numerous studies of varied design have been published in the interim that significantly add to the overall weight of evi-

Table 2. Comparison of Pooled Estimated of Percent Increase (and 95% CI or Posterior Interval or *t* Value) in RR of Mortality Estimated Across Meta-Analyses and Multicity Studies of Daily Changes in Exposure

			Percent Increases in Mortality (95% CI)		
	Primary Source	Exposure Increment	All-Cause	Cardiovascular	Respiratory
Meta-estimate with and without adjustment for publication bias	Anderson et al[27] 2005	20 $\mu g/m^3$ PM_{10}	1.0 (0.8–1.2) 1.2 (1.0–1.4)	...	...
Meta-estimates from COMEAP report to the UK Department of Health on CVD and air pollution	COMEAP[31] 2006	20 $\mu g/m^3$ PM_{10} 10 $\mu g/m^3$ $PM_{2.5}$	... ...	1.8 (1.4–2.4) 1.4 (0.7–2.2)	... ...
NMMAPS, 20 to 100 US cities	Dominici et al[34] 2003	20 $\mu g/m^3$ PM_{10}	0.4 (0.2–0.8)	0.6 (0.3–1.0)*	...
APHEA-2, 15 to 29 European cities	Katsouyanni et al[35] 2003 Analitis et al[36] 2006	20 $\mu g/m^3$ PM_{10}	1.2 (0.8–1.4)	1.5 (0.9–2.1)	1.2 (0.4–1.9)
US, 6 cities	Klemm and Mason[37] 2003	10 $\mu g/m^3$ $PM_{2.5}$	1.2 (0.8–1.6)	1.3 (0.3–2.4)†	0.6 (−2.9, 4.2)‡
US, 27 cities, case-crossover	Franklin et al[38] 2007	10 $\mu g/m^3$ $PM_{2.5}$	1.2 (0.3–2.1)	0.9 (−.1, 2.0)	1.8 (0.2, 3.4)
California, 9 cities	Ostro et al[39] 2006	10 $\mu g/m^3$ $PM_{2.5}$	0.6 (0.2–1.0)	0.6 (0.0, 1.1)	2.2 (0.6, 3.9)
France, 9 cities	Le Tertre et al[40] 2002	20 $\mu g/m^3$ BS	1.2 (0.5–1.8)§	1.2 (0.2–2.2)§	1.1 (−1.4, 3.2)§
Japan, 13 cities, age >65 y	Omori et al[41] 2003	20 $\mu g/m^3$ SPM	1.0 (0.8–1.3)	1.1 (0.7–1.5)	1.4 (0.9–2.1)
Asia, 4 cities	Wong et al[42] 2008	10 $\mu g/m^3$ PM_{10}	0.55 (0.26–0.85)	0.59 (0.22–0.93)	0.62 (0.16–1.04)
US, 112 cities	Zanobetti et al[43] 2009	10 $\mu g/m^3$ $PM_{2.5}$	0.98 (0.75–1.22)	0.85 (0.46–1.24)	1.68 (1.04–2.33)
		10 $\mu g/m^3$ $PM_{10-2.5}$‖	0.46 (0.21–0.71)	0.32 (0.00–0.64)	1.16 (0.43–1.89)
		10 $\mu g/m^3$ $PM_{2.5}$¶	0.77 (0.43–1.12)	0.61 (0.05–1.17)	1.63 (0.69–2.59)
		10 $\mu g/m^3$ $PM_{10-2.5}$¶	0.47 (0.21–0.73)	0.29 (−0.04, 0.61)	1.14 (0.043–1.85)

CI indicates confidence interval or posterior interval.
*Cardiovascular and respiratory deaths combined.
†Ischemic heart disease deaths.
‡Chronic obstructive pulmonary disease deaths.
§Includes general additive model–based analyses with potentially inadequate convergence.
‖Results for $PM_{10-2.5}$ are from 47 cities.
¶Results of 2 pollutant models controlling for alternate PM size in 47 cities.

dence that exposure to air pollutants at present-day levels contributes to cardiovascular morbidity and mortality.

Mortality and Air Pollution

Time-Series and Related Studies

Time-series and case-crossover studies explore associations between short-term changes in air pollution and daily changes in death counts. The sum of current evidence supports the findings of an earlier review[28] that demonstrated that short-term elevations in daily PM levels lead to a greater absolute risk for CVD-related mortality than for all other causes. Even if similar acute RR elevations (≈1.01) are estimated between cardiovascular and pulmonary mortality, CVDs account for 69% of the increase in absolute mortality rates compared with 28% for pulmonary diseases attributable to short-term PM exposure. Recently, more rigorous modeling techniques have been used in attempts to better estimate pollution-mortality associations while controlling for other time-dependent confounding covariables.[29,30] There have been well over 100 published daily time-series studies reporting small but statistically significant PM-mortality associations that have been the subject of quantitative reviews or meta-analyses.[3,27,31–33] Table 2 summarizes recent multicity analyses and studies published since 2004.

To address concerns about city selection bias, publication bias, and influences of copollutants, several large, multicity, daily time-series studies have been conducted worldwide. One of the largest was the National Morbidity, Mortality, and Air Pollution Study (NMMAPS). Published reports from this study included as few as 20 US cities,[44,45] as many as 100 cities,[46,47] and more recently, data for hundreds of counties (Table 2).[48] The observed relationship between PM exposure and excess mortality remained independent of several gaseous copollutants (NO_2, CO, or SO_2). Recent analyses suggest that O_3 may also independently contribute to cardiopulmonary mortality risk[49,50]; however, coexposures to secondary particle pollutants may be responsible in part for this latter association.[51]

Several studies have also been conducted outside the United States, including the Air Pollution and Health: A European Approach (APHEA and APHEA-2) projects, which examined daily PM-related mortality effects in multiple cities.[36,52] PM air pollution was significantly associated with daily mortality counts for all-cause, cardiovascular, and respiratory mortality (Table 2). Further analyses of the European data suggest that CVD deaths are also associated with exposure to NO_2[53] and CO.[54] A few new time-series studies have also confirmed similar increases in cardiovascular mortality related to short-term PM exposure in China[55–57] and Bangkok, Thailand.[42] Additional multicity studies have been conducted worldwide with analyses of CVD deaths (Table 2).[38–42,58–60] Finally, in a recent analysis that included several Asian

cities, SO_2, NO_2, O_3, and PM_{10} were all associated with excess cardiovascular mortality.[42]

In an attempt to evaluate the coherence of multicity studies across continents, the Air Pollution and Health: A Combined European and North American Approach (APHENA) study analyzed data from the APHEA, NMMAPS, and Canadian studies.[61] The combined effect on all-cause mortality ranged from 0.2% to 0.6% for a 10-$\mu g/m^3$ elevation in daily ambient PM_{10}, with the largest effects observed in Canada. Among individuals older than 75 years, the effects were greater for cardiovascular mortality than for overall and pulmonary mortality (0.47% to 1.30%). Older age (>75 years) and higher rates of unemployment were related to greater PM mortality risks in both continents. Higher NO_2 levels were associated with larger PM_{10} effects on mortality, particularly in Europe. Finally, there appeared to be no lower-limit threshold below which PM_{10} was not associated with excess mortality across all regions.

Evidence Summary

The overall evidence from time-series analyses conducted worldwide since publication of the first AHA statement[1] confirms the existence of a small, yet consistent association between increased mortality and short-term elevations in PM_{10} and $PM_{2.5}$ approximately equal to a 0.4% to 1.0% increase in daily mortality (and cardiovascular death specifically) due to a 10-$\mu g/m^3$ elevation in $PM_{2.5}$ during the preceding 1 to 5 days (Table 2).

Cohort and Related Studies

Although short-term changes in PM concentrations have deleterious health effects, longer-term exposures may have a more pertinent clinical health effect on cardiovascular morbidity and mortality given that individuals are typically exposed to higher air pollution levels over extended periods of time. An additional source of exposure variability that has been exploited in epidemiological studies is spatial variability, which includes differences in average ambient concentrations over extended periods of time across metropolitan areas or across smaller communities within local areas. Recent emphasis has been on prospective cohort studies that control for individual differences in multiple confounding variables and cardiovascular risk factors. A summary of these studies is presented in Table 3 and Figure 1. These cohort studies generally demonstrate larger overall mortality effects than the results of time-series analyses.

Harvard Six Cities and ACS Studies

Two landmark cohort-based mortality studies, the Harvard Six Cities[62] and the ACS studies,[66] were reported in the mid 1990s and were discussed previously.[1] In both, $PM_{2.5}$ and sulfate particulate pollution were associated with increases in all-cause and cardiopulmonary disease (Table 3). In addition, intensive independent reanalyses[63] corroborated the original findings of both studies and resulted in innovative methodological contributions that demonstrated the robustness of the results to alternative modeling approaches. In both the Harvard Six Cities[62,64] and the ACS[67] studies, PM air pollution–related mortality was substantially higher for cardiovascular- than for pulmonary-related causes.

Since 2004, there have been further analyses of both studies. Laden et al[64] extended the mortality follow-up of the Harvard Six Cities cohort for an additional 8 years. $PM_{2.5}$ associations, similar to those found in the original analysis, were observed for all-cause and CVD mortality (Table 3). Furthermore, reductions in $PM_{2.5}$ concentrations for the extended follow-up period were associated with reduced mortality risk. Further analysis suggested that the health effects of changes in exposure were seen primarily within 2 years.[84] In addition to confirming the earlier mortality relationship, the recent observations suggest that the adverse health effects mediated by longer-term PM air pollution exposure can be estimated reasonably accurately by the previous few years of particle levels.

Extended analyses of the ACS cohort that emphasize efforts to control for the effects of other covariates and risk factors have corroborated the previously reported mortality associations with particulate and sulfur oxide pollution.[68] Elevated mortality risks were most strongly associated with $PM_{2.5}$. Coarse particles ($PM_{10-2.5}$) and gaseous pollutants, except for SO_2, were generally not significantly related to mortality. In another extended analysis,[67] the death certificate classifications of underlying causes of death due to $PM_{2.5}$ exposures were observed to be principally ischemic heart disease, arrhythmias, heart failure, and cardiac arrest. Finally, recent additional analyses attempted to control for the fact that variations in exposure to air pollution across cities or within cities may correlate with socioeconomic or demographic gradients that influence health and susceptibility to environmental exposures.[85,86] When controlled for individual risk factor data, the mortality associations for intrametropolitan $PM_{2.5}$ concentration differences within the Los Angeles, Calif, area were generally larger than those observed in the full cohort across metropolitan areas.[69] However, the results were somewhat sensitive to the inclusion of zip code–level ecological variables, which suggests potential contextual neighborhood confounding. Krewski et al[70] subsequently observed that full adjustments for multiple ecological covariates did not reduce the estimated $PM_{2.5}$-related mortality effect. The association for ischemic heart disease mortality in particular was highly robust across various study areas and modeling strategies and after controlling for both individual and ecological covariates.

An additional recent analysis of the ACS cohort evaluated the health effects of ozone compared with $PM_{2.5}$.[87] The findings reconfirmed the independent cardiovascular mortality increase related to fine-particle exposure. However, after adjustment for $PM_{2.5}$, ozone was associated solely with an elevated risk of death due to respiratory causes; there was no independent risk of ozone exposure on CVD-related mortality. This suggests that the positive findings reported in NMMAPS[50] regarding cardiopulmonary mortality and short-term ozone exposure could be explained at least in part by the enhanced risk of mortality due to lung disease categories.

Table 3. Summary of Cohort Study Results

Study	Size of Cohort (000s)	Follow-Up Period	Covariates Controlled for	Percent Increases in Mortality (95% CI) Associated With 10 $\mu g/m^3$ $PM_{2.5}$ (or Other When Indicated)			
				All-Cause	Cardiopulmonary	Cardiovascular	Ischemic Heart Disease
Harvard Six Cities, original (Dockery et al[62] 1993)	≈8	1974–1991	Individual (smoking+others)	13 (4.2–23)	18 (6.0–32)	…	…
Harvard Six-Cities, HEI reanalysis, Krewski et al[63] 2004	≈8	1974–1991	Individual (smoking+others)	14 (5.4–23)	19 (6.5–33)	…	…
Harvard Six-Cities, extended, Laden et al[64] 2006	≈8	1974–1998	Individual (smoking+others)	16 (7–26)	…	28 (13–44)	…
Six-Cities Medicare cohort, Eftim et al[65] 2008	≈340	2000–2002	Individual (age, sex)	21 (15–27)	…	…	…
ACS, Original, Pope et al[66] 1995	≈500	1982–1989	Individual (smoking+others)	6.6 (3.5–9.8)	12 (6.7–17)	…	…
ACS, HEI reanalysis, Krewski et al[63] 2004	≈500	1982–1989	Individual (smoking+others) +ecological	7.0 (3.9 10)	12 (7.4–17)	13 (8.1–18)	…
ACS, extended I, Pope et al[67,68] 2002, 2004	≈500	1982–1998	Individual (smoking+others)	6.2 (1.6–11)	9.3 (3.3–16)	12 (8–15)	18 (14–23)
ACS, intrametro Los Angeles, Jerrett et al[69] 2005	≈23	1982–2000	Individual (smoking+others) +ecological	17 (5–30)	12 (−3–30)	…	39 (12–73)
ACS, extended II, Krewski et al[70] 2009	≈500	1982–2000	Individual (smoking+others) +ecological	5.6 (3.5–7.8)	13 (9.5–16)	…	24 (20–29)
ACS, Medicare cohort, Eftim et al[65] 2008	7333	2000–2002	Individual (age, sex)+ecological +COPD	11 (9–13)	…	…	…
US Medicare cohort, east/central/west, Zeger et al[71] 2008	13 200	2000–2005	Individual (age, sex)+ecological +COPD	6.8 (4.9–8.7),* 13 (9.5–17) −1.1 (−3 to 0.8)	…	…	…
Women's Health Initiative, Miller et al[72] 2007	≈66	1994–2002	Individual (smoking+others)	…	…	76 (25–147), 24 (9–41)†	…
Nurses' Health Study, Puett et al[73] 2008	≈66	1992–2002	Individual (smoking+others) ecological	7.0 (−3.0 to 18)‡	…	30 (0–71)‡	…
AHSMOG, males only, McDonnell et al[74] 2000	≈4	1977–1992	Individual (smoking+others)	8.5 (−2.3 to 21)	23 (−3 to 55)	…	…
AHSMOG, females only, Chen et al[75] 2005	≈4	1977–2000	Individual (smoking+others)	…	…	42 (6–90)	…
VA hypertensive male I study, Lipfert et al[76] 2006	≈42	1989–1996	Individual (smoking+others) +ecological	15 (5–26)§	…	…	…
VA hypertensive male II study, Lipfert et al[77] 2006	≈30	1997–2001	Individual (smoking+others) +ecological	6 (−6 to 22)	…	…	…
11 CA county, elderly, Enstrom[78] 2005	≈36	1973–2002	Individual (smoking+others) +ecological	4 (1–7)‖, 1 (−0.6 to 2.6)	…	…	…
French PAARC, Filleul et al[79] 2005	≈14	1974–2000	Individual (smoking+others)	7 (3–10)‡	5 (−2 to 12)‡	…	…
German women, Gehring et al[80] 2006	≈5	1980s, 1990s–2003	Individual smoking and socioeconomic status	12 (−8 to 38)	52 (9–115)	…	…

(Continued)

Table 3. Continued

Study	Size of Cohort (000s)	Follow-Up Period	Covariates Controlled for	Percent Increases in Mortality (95% CI) Associated With 10 $\mu g/m^3$ $PM_{2.5}$ (or Other When Indicated)			
				All-Cause	Cardiopulmonary	Cardiovascular	Ischemic Heart Disease
Oslo, Norway, intrametro, Naess et al[81] 2007	≈144	1992–1998	Individual age, occupational class, education	…	…	10 (5–16),¶ 14 (6–21), 5 (1–8), 3 (0–5)	…
Dutch cohort, Beelen et al[82] 2008	≈121	1987–1996	Individual (smoking+others) +ecological	6 (−3 to 16)	…	4 (−10 to 21)	…
Great Britain, Elliott et al[83] 2007	≈660	1966–1998	Socioeconomic status	1.3 (1.0–1.6)‡#	1.7 (1.3–2.2)‡#	1.2 (0.7–1.7)‡#	

HEI indicates Health Effects Institute; VA, Veterans Affairs; COPD, chronic obstructive pulmonary disease; and CA, California.

*Three estimates are for the East, Central, and West regions of the United States, respectively.

†Any cardiovascular event.

‡Associated with 10 $\mu g/m^3$ British Smoke (BS) or PM_{10}.

§Estimates from the single-pollutant model. Effect estimates were smaller and statistically insignificant in analyses restricted to counties with nitrogen dioxide data. County-level traffic density was a strong predictor of survival, and stronger than $PM_{2.5}$ when included with $PM_{2.5}$ in joint regressions.

‖Two estimates are for the follow-up period 1973–1982 and the follow-up period 1983–2002, respectively.

¶Four estimates are for men 51–70 y old, women 51–70 y old, men 71–90 y old, and women 71–90 y old, respectively.

#Using last 0- to 4-year exposure window.

Additional Cohort Studies

Several additional cohort studies have been published in the past few years (Table 3). Eftim and colleagues[65] studied 2 very large "cohorts" of US Medicare participants who lived in locations included in the Harvard Six Cities and ACS studies. Effects of $PM_{2.5}$ exposure on mortality for the period 2000 to 2002 were estimated after controlling for multiple factors, although not at the individual patient level. For all-cause mortality, the $PM_{2.5}$-mortality associations were larger than those observed in the Harvard Six Cities or ACS cohorts. In an additional analysis of 13.2 million US Medicare participants for the time period 2000 to 2005,[71] $PM_{2.5}$-mortality associations were shown to be similar to those observed in the Harvard Six Cities and ACS studies in the East and Central regions of the United States (and when the data were pooled for the entire United States). However, $PM_{2.5}$ was not associated with mortality in the Western United States or for the oldest age group (>85 years old). These findings generally corroborate the earlier cohort studies and add evidence that aspects of exposure (PM sources or composition) and patient susceptibility might play important roles in determining the health risks.

In a cohort of postmenopausal women without prior CVD from the Women's Health Initiative Observational Study,[72] an association between longer-term $PM_{2.5}$ exposure (median follow-up of 6 years) and cardiovascular events (primary end point) was observed. After adjustment for age and other risk factors, an incremental difference of 10 $\mu g/m^3$ $PM_{2.5}$ was associated with a 24% (95% confidence interval [CI]9% to 41%) increase in all first cardiovascular events (fatal and nonfatal, with a total of 1816 cases). Notably, an incremental difference of 10 $\mu g/m^3$ $PM_{2.5}$ was also associated with a large 76% (95% CI 25% to 147%) increase in fatal cardiovascular events, based on 261 deaths. The risks for both coronary heart disease and strokes were found to be similarly elevated. Interestingly, within-city $PM_{2.5}$ gradients appeared to have larger cardiovascular effects than those between cities, although this difference was not statistically significant. Finally, overweight women (body mass index >24.8 kg/m^2) were at relatively greater cardiovascular risk due to particulate air pollution than leaner women. Noteworthy aspects of this study were improved assessment of the end points by medical record review (rather than by death certificate) and long-term particle exposure estimation. The control for individual-level confounding variables was also superior to that of previous cohort studies.

In another cohort of women, a subset of the Nurses' Health Study from the northeastern United States,[73] an increase of 10 $\mu g/m^3$ modeled estimates of PM_{10} exposures was associated with an approximately 7% to 16% increased risk of all-cause mortality and a 30% to 40% increase in fatal coronary heart disease, depending on the level of adjustment for covariates. This study found that the strongest health risks for all-cause and cardiovascular mortality were seen in association with the average PM_{10} exposure during the previous 24 months before death. Similar to the findings of the Women's Health Initiative, the cardiovascular mortality risk estimates were larger than those of previous cohort studies. In addition, obese women (body mass index >30 kg/m^2) were at greater relative risk, and the increases in mortality (all-cause and cardiovascular) were larger than the effects on nonfatal events. The results were also in accordance with the latest Harvard Six Cities analyses[64] that show that exposure over the most recent preceding 1 to 2 years can accurately estimate the majority of the health risks due to longer-term PM air pollution exposures.

The pollution-mortality association has also been assessed in several other cohort studies in the United States and Europe (Table 3).[76–83] In a recent analysis of the Adventist Health Study of Smog (AHSMOG) cohort with a much

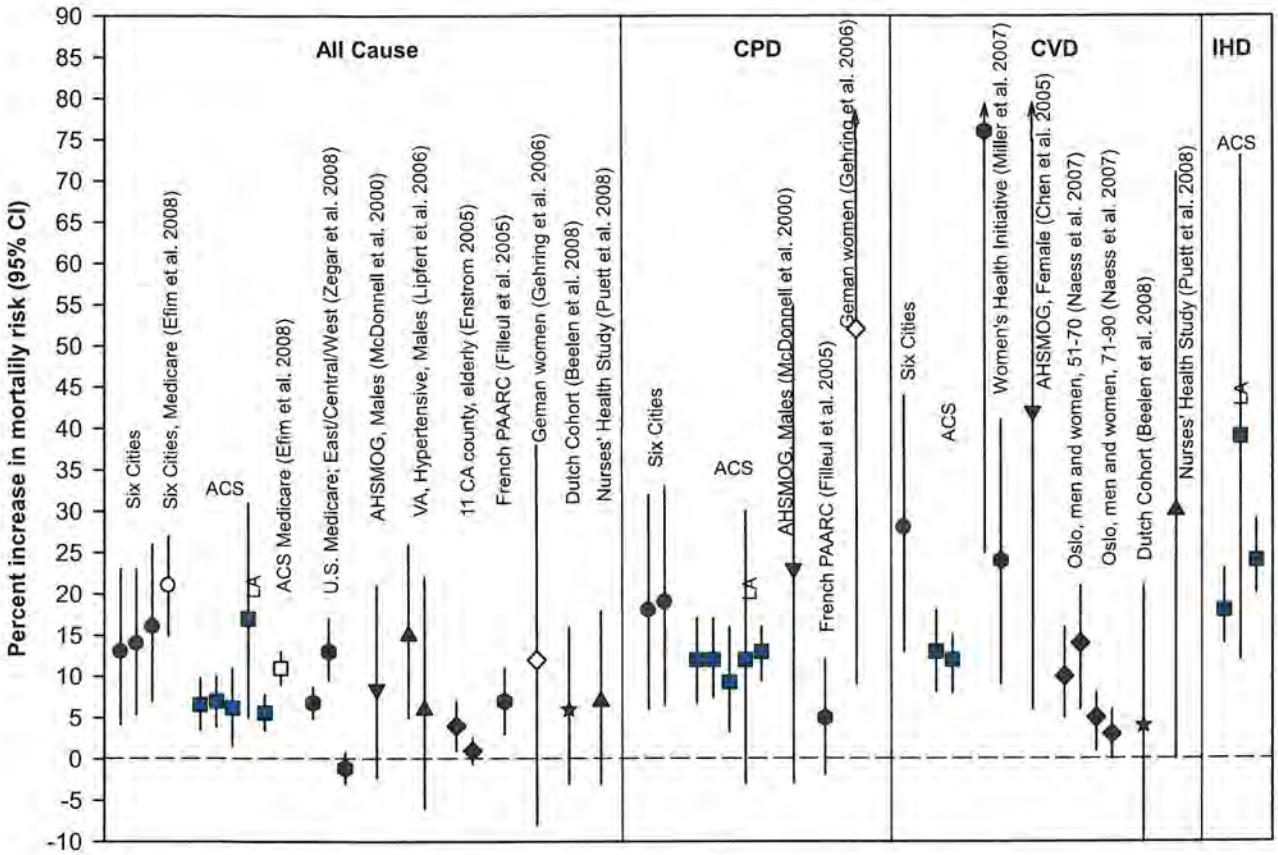

Figure 1. Risk estimates provided by several cohort studies per increment of 10 $\mu g/m^3$ in $PM_{2.5}$ or PM_{10}. CPD indicates cardiopulmonary disease; IHD, ischemic heart disease.

longer follow-up than the original studies,[74,88] fatal coronary heart disease was significantly associated with $PM_{2.5}$ among females but not males.[75] These observations along with the remarkably robust health effects in the Women's Health Initiative Observational Study and Nurses' Health Study suggest that women may be at special risk from PM exposure. The overall cohort study evidence demonstrates that a 10-$\mu g/m^3$ increase in $PM_{2.5}$ exposure is in general positively associated with excess mortality, largely driven by increases in cardiopulmonary or cardiovascular deaths (Figure 1). Independent results from the Women's Health Initiative Study,[72] the US Medicare cohorts,[71] the German women cohort,[80] and the intracity Oslo (Norway) study[81] contribute substantially to this evidence. Although the Dutch cohort,[82] AHSMOG,[74,75] French PAARC (Pollution Atmosphérique et Affections Respiratoires Chroniques [air pollution and chronic respiratory diseases]),[79] Veterans Affairs hypertensive male study,[77] and 11 CA county[78] studies observed increased mortality risks associated with higher $PM_{2.5}$ exposure that were statistically significant in some analyses, the observed health risks were less robust. A finding that is somewhat consistent across the Veterans Affairs hypertensive male study,[77] 11 CA county,[78] Oslo,[81] and US Medicare cohorts[71] is that the $PM_{2.5}$-mortality effect estimates tend to decline with longer periods of follow up or in a substantially older cohort. These studies also often observed elevated mortality risks according to alternative indicators of air pollution exposure, especially metrics of traffic-related exposure.

Evidence Summary

The overall evidence from the cohort studies demonstrates on average an approximate 10% increase in all-cause mortality per 10-$\mu g/m^3$ elevation in long-term average $PM_{2.5}$ exposure. The mortality risk specifically related to CVD appears to be elevated to a similar (or perhaps even greater) extent, ranging from 3% to 76% (Table 3). This broader estimated range in risk compared with the short-term effects observed in time series is due to several recent cohort studies[72,73] that demonstrated larger cardiovascular mortality risks (eg, >30%) than in earlier cohort observations. This may reflect superior aspects of these studies that allowed for a better characterization of the cardiovascular risk of long-term exposure, the fact that these cohorts consisted of only women, or other unclear reasons. Compared with cardiovascular mortality, there is less existing evidence to support an increase in the risk for nonfatal cardiovascular events related to $PM_{2.5}$ exposure among the existing cohort studies, because many of them did not specifically investigate nonfatal outcomes, and several of the more recent studies reported nonsignificant relationships.[72,73]

Natural Experiment and Intervention Studies

Several studies have shown improvements in health outcomes in association with exposures using well-defined natural experiments or interventions, such as abrupt reductions in air pollution[89–91] or changes over a longer period of time.[64,92]

Table 4. Comparison of Pooled Estimated of Percent Increase in Risk of Hospital Admission for CVD Estimated Across Meta-Analyses and Multicity Studies of Daily Changes in Exposure

	Primary Source	Exposure Increment	% Increase (95% CI)
Cardiac admissions, meta-analysis of 51 estimates	COMEAP[31] 2006	20 $\mu g/m^3$ PM_{10}	1.8 (1.4–1.2)
Cardiac admissions, 8 US cities	Schwartz[96] 1999	20 $\mu g/m^3$ PM_{10}	2.0 (1.5–2.5)
Cardiac admissions, 10 US cities	Zanobetti et al[97] 2000	20 $\mu g/m^3$ PM_{10}	2.6 (2.0–3.0)
Cardiac admissions, 14 US cities	Samet et al[98] 2000	20 $\mu g/m^3$ PM_{10}	2.0 (1.5–2.5)
	Schwartz et al[99] 2003		
Cardiac admissions, 8 European cities	Le Tertre et al[40] 2002	20 $\mu g/m^3$ PM_{10}	1.4 (0.8–2.0)
Cardiovascular admissions, 14 Spanish cities	Ballester et al[100] 2006	20 $\mu g/m^3$ PM_{10}	1.8 (7–3.0)
Cardiovascular admission, 8 French cities	Larrieu et al[101] 2007	20 $\mu g/m^3$ PM_{10}	1.6 (0.4–3.0)
Cardiovascular admissions, 202 US counties	Bell et al[102] 2008	20 $\mu g/m^3$ PM_{10}	0.8 (0.6–1.0)
Medicare national claims history files	Dominici et al[103] 2006	10 $\mu g/m^3$ $PM_{2.5}$	
Ischemic heart disease,			0.44 (0.02–0.86)
Cerebrovascular disease			0.81 (0.30–1.32)
Heart failure			1.28 (0.78–1.78)
Heart rhythm			0.57 (−0.01 to 1.15)

Small but statistically significant drops in mortality were associated with an 8½-month copper smelter strike that resulted in sharp reductions in sulfate PM and related air pollutants across 4 Southwest states, even after controlling for other factors.[93] Data from US Medicare enrollment files were used to estimate the association between changes in monthly mortality rates for US counties and average $PM_{2.5}$ concentrations for the previous 12 months.[94] $PM_{2.5}$-mortality associations were observed at the national scale but not the local scale, which raises concerns about possible statistical confounding due to unmeasured individual and ecological variables as a cause for any positive findings in this study. However, a recent large study found that reductions in PM air pollution exposure on a local scale (across US counties) over a 2-decade period (1980s and 1990s) were associated with increased life expectancy even after controlling for changes in socioeconomic, demographic, and proxy smoking variables.[95] Indeed, a decrease of 10 $\mu g/m^3$ in the long-term $PM_{2.5}$ concentration was related to an increase in mean life expectancy of 0.61 ± 0.20 years.

Hospitalization Rates

There are many daily time-series or case-crossover studies that have evaluated associations between cardiovascular hospitalizations and short-term changes in air pollution. Because of the great number of publications, all studies (particularly those focusing on nonparticulate air pollutants) cannot be discussed individually. Nevertheless, Table 4 presents a comparison of pooled estimates of percent increase in RR of hospital admission for general cardiac conditions across a previous meta-analysis of 51 published estimates (COMEAP [Committee on the Medical Effects of Air Pollutants]) and results from many selected multicity studies published after 2004. Several studies before 2004 are included in Table 4 only to demonstrate the consistency of effect.

Because of its comparatively large size and importance, the results of a recent analysis of Medicare files in 204 US urban counties with 11.5 million individuals older than 65 years merit discussion. Daily changes in $PM_{2.5}$ levels were associated with a variety of cardiovascular hospital admission subtypes.[103] A 10-$\mu g/m^3$ increase in $PM_{2.5}$ exposure was related to increases in hospitalizations for cerebrovascular disease by 0.81% (95% CI 0.3% to 1.32%), peripheral vascular disease by 0.86% (95% CI −0.06% to 1.79%), ischemic heart disease by 0.44% (95% CI 0.02% to 0.86%), arrhythmias by 0.57% (95% CI −0.01% to 1.15%), and heart failure by 1.28 (95% CI 0.78% to 1.78%). The most rapid effects, which occurred largely on the same day of $PM_{2.5}$ elevation, were seen for cerebrovascular, arrhythmia, and heart failure admissions. Ischemic heart disease events tended to increase to a greater extent 2 days after exposures. A consistent finding was that the cardiovascular effects of pollution were much stronger in the Northeast than in other regions. In fact, there were few significant associations in Western US regions. It was speculated that these differences reflected variations in particle composition (eg, greater sulfate in the East and nitrate components in the West) and pollution sources (eg, power generation in the East and transportation sources in the West). In a follow-up analysis by Peng et al,[104] $PM_{10-2.5}$ levels were not statistically associated with cardiovascular hospitalizations after adjustment for $PM_{2.5}$. This suggests that the smaller particles (ie, $PM_{2.5}$) are principally responsible for the cardiovascular hospitalizations attributed in prior studies to the combination of both fine and coarse particles (ie, PM_{10}). Given the differences between the size fractions, the results imply that particles and their components derived from combustion sources (ie, $PM_{2.5}$) are more harmful to the cardiovascular system than larger coarse particles. Finally, there is some evidence that gaseous pollutants may also instigate hospitalizations. Hospital admissions for cardiovascular causes, particularly ischemic heart disease, were found to rise in relation to the previous-day and same-day level of SO_2, even after adjustment for PM_{10} levels.[105]

Table 5. Comparisons of Estimated Percent Increase in Risk of Ischemic Heart Disease Events due to Concurrent or Recent Daily PM Exposure

Event/Study Area	Primary Source	Exposure Increment	% Increase (95% CI)
MI events–Boston, Mass	Peters et al[110] 2001	10 $\mu g/m^3$ $PM_{2.5}$	20 (5.4–37)
MI, 1st hospitalization–Rome, Italy	D'Ippoliti et al[112] 2003	30 $\mu g/m^3$ TSP	7.1 (1.2–13.1)
MI, emergency hospitalizations–21 US cities	Zanobetti and Schwartz[113] 2005	20 $\mu g/m^3$ PM_{10}	1.3 (0.2–2.4)
Hospital readmissions for MI, angina, dysrhythmia, or heart failure of MI survivors–5 European cities	Von Klot et al[114] 2005	20 $\mu g/m^3$ PM_{10}	4.2 (0.8–8.0)
MI events–Seattle, Wash	Sullivan et al[115] 2005	10 $\mu g/m^3$ $PM_{2.5}$	4.0 (−4.0–14.5)
MI and unstable angina events–Wasatch Front, Utah	Pope et al[13] 2006	10 $\mu g/m^3$ $PM_{2.5}$	4.8 (1.0–6.6)
Tokyo metropolitan area	Murakami et al[109] 2006	TSP >300 $\mu g/m^3$ for 1 h vs reference periods <99 $\mu g/m^3$	40 (0–97)*
Nonfatal MI, Augsburg, Germany	Peters et al[111] 2004	Exposure to traffic 1 h before MI (note: not PM but self-reported traffic exposure)	292 (222–383)
Nonfatal MI, Augsburg, Germany	Peters et al[116] 2005	Ambient UFP, $PM_{2.5}$, and PM_{10} levels	No association with UFP or $PM_{2.5}$ on same day. Positive associations with $PM_{2.5}$ levels on 2 days prior

TSP indicates total suspended particulate matter.
*Adjusted rate ratio for MI deaths.

Evidence Summary

Excess cardiovascular mortality and increased rates of hospitalizations are similarly associated with day-to-day changes in PM air pollution (Tables 2 and 4). However, significant differences between geographic regions in the risk relationships have been observed, and more investigation is required to explain this heterogeneity.

Specific Cardiovascular Events/Conditions

Ischemic Heart Disease

Among the cohort studies that provided relevant results, the ACS study found a relationship between increased risk for ischemic heart disease death and long-term exposure to elevated $PM_{2.5}$ levels (Table 3).[67,69,106] Indeed, ischemic cardiac events accounted for the largest relative (RR 1.18, 95% CI 1.14 to 1.23) and absolute risk for mortality per 10-$\mu g/m^3$ elevation in $PM_{2.5}$.[67] A survival analysis of US Medicare data for 196 000 survivors of acute MI in 21 cities showed the risk of an adverse post-MI outcome (death, subsequent MI, or first admission for congestive heart failure) was increased with higher exposure to PM_{10}.[107] Data from the Worcester Heart Attack study also found that long-term exposure to traffic-related air pollution was associated with significantly increased risk of acute MI.[108] However, in the Women's Health Initiative[72] and the Nurses' Health Study,[73] only disease categories that included fatal coronary events, but not nonfatal MI alone, were statistically elevated in relation to $PM_{2.5}$. The effect size for cardiovascular mortality was much larger and much more statistically robust than for nonfatal events such as MI in both studies.

Various time-series and case-crossover studies have also reported increased ischemic heart disease hospital admissions associated with short-term elevated concentrations of inhalable and/or fine PM air pollution.[31,40,103] In the US Medicare study, a reduction of $PM_{2.5}$ by 10 $\mu g/m^3$ was estimated to reduce ischemic heart disease admissions in 204 counties by 1523 (95% posterior interval 69 to 2976) cases per year.[103] Several studies have also found positive associations between elevated PM or traffic exposures over a period as brief as a few hours[109–111] or a few days and an elevated risk for MI (Table 5).[13,110,112–115] In general, acute increases in risk for ischemic heart disease events have been observed consistently, even as rapidly as 1 to 2 hours after exposure to elevated PM, in case-crossover analyses.[109–111] Other studies have reported an increased risk for MI shortly after exposure to traffic. Peters et al[111] reported in 691 subjects in Augsburg, Germany, a strong association (odds ratio 2.92, 95% CI 2.22 to 3.83) between onset of MI and traffic exposure within the past hour, although whether this was a result of the air pollution or a combination of other factors (eg, noise and stress) is not certain. Additional analyses did not report an association between recent UFP exposures and MI onset; however, the levels of $PM_{2.5}$ and several gaseous pollutants 2 days earlier were related to MI risk.[116] The lack of relationship between MI and UFPs may be due to the fact that the levels were measured regionally and remote from the localized source and may therefore reflect exposure misclassification. Finally, in the only study in which participating subjects had coronary angiograms performed previously, ischemic cardiac events were found to occur in relation to PM air pollution exposure solely among individuals with obstructive coronary atherosclerosis in at least 1 vessel.[13] This finding suggests the importance of patient susceptibility (eg, the presence of preexisting coronary artery disease) for PM to trigger an acute ischemic event within hours to days after exposure.

Heart Failure

In the ACS cohort study, it appeared that deaths due to arrhythmias, heart failure, and cardiac arrest (RR 1.13, 95% CI 1.05 to 1.21 per 10 $\mu g/m^3$) were also associated with

prolonged exposure to $PM_{2.5}$, although not as strongly as ischemic heart disease mortality,[67] although potential mortality misclassification on death certificates makes the actual cause of death not entirely certain in all circumstances. Heart failure rates or mortality associations were not reported in the other cohort studies.

Daily hospitalizations for heart failure have also been associated with short-term changes in PM exposure.[31] Heart failure associations with PM were observed in a large daily time-series analysis of $PM_{2.5}$ and cardiovascular and respiratory hospitalizations by use of a national database constructed from US Medicare files.[103] A 10-$\mu g/m^3$ increase in concurrent-day $PM_{2.5}$ was associated with a 1.28% (95% CI 0.78% to 1.78%) increase in heart failure admissions, the single largest cause for hospitalization in this cohort. A reduction of $PM_{2.5}$ by 10 $\mu g/m^3$ was estimated to reduce heart failure admissions in 204 counties by 3156 (95% posterior interval 1923 to 4389) cases per year.[103] Another analysis in Medicare recipients in 7 US cities found a 10-$\mu g/m^3$ increase in concurrent-day PM_{10} was associated with a 0.72% (95% CI 0.35% to 1.10%) increase in heart failure admissions.[117] Traffic-related air pollution has also been shown to be significantly associated with increased mortality risk after acute heart failure.[118] Finally, a study from Utah's Wasatch Front area explored longer lagged-exposure periods and found that a 14-day lagged cumulative moving average of 10 $\mu g/m^3$ $PM_{2.5}$ was associated with a 13.1% (95% CI 1.3% to 26.2%) increase in heart failure admissions.[119]

Cerebrovascular Disease

Among the cohort studies that provided pertinent results, the Women's Health Initiative reported significant increases in both nonfatal stroke (hazard ratio 1.28, 95% CI 1.02 to 1.61) and fatal cerebrovascular disease (hazard ratio 1.83, 95% CI 1.11 to 3.00) per 10-$\mu g/m^3$ elevation in prolonged exposure to $PM_{2.5}$.[72] However, no significant association between stroke mortality and PM air pollution was found in the ACS study.[67]

Several studies have also reported small but statistically significant associations between short-term PM exposure and cerebrovascular disease. Daily time-series studies of stroke mortality in Seoul, Korea,[120,121] observed that elevated air pollution (including measures of PM, NO_2, CO, and O_3) was associated with increases in stroke mortality. When analyzed separately by stroke type,[121] the pollution association was associated with ischemic but not hemorrhagic stroke. Risk of stroke mortality was also associated with daily increases in PM_{10} and NO_2 in Shanghai, China.[56] A daily time-series study in Helsinki, Finland,[122] found that $PM_{2.5}$ and CO were associated with stroke mortality in the warm but not the cold seasons. Several studies have also observed increased stroke or cerebrovascular hospital admissions associated with increased exposure to PM or related pollutants.[31,38,40,46,123–125] For example, a study of hospital admissions for Medicare recipients in 9 US cities[125] found that several measures of air pollution (PM_{10}, CO, NO_2, and SO_2) 0 to 2 days before admission were associated with ischemic but not hemorrhagic stroke. Studies of ischemic stroke and transient ischemic attacks based on population-based surveillance have also been conducted in Dijon, France,[126] where O_3 exposure (but not PM_{10}) was associated with ischemic stroke, and in Corpus Christi, Tex,[127] where both $PM_{2.5}$ and O_3 were associated with ischemic strokes and transient ischemic attacks.

Peripheral Arterial and Venous Diseases

There have been only a few studies that have explored a relationship between air pollution and peripheral vascular diseases. Studies using Medicare data for 204 US counties observed nearly statistically significant positive associations between daily changes in measures of PM pollution and hospitalizations for peripheral vascular diseases.[103,104] The ACS cohort found no association between other atherosclerotic and aortic aneurysm deaths and long-term $PM_{2.5}$ exposure.[67]

Recently, a case-control study from the Lombardy region of Italy found a 70% increase in risk of deep vein thrombosis per 10-$\mu g/m^3$ elevation in long-term PM_{10} level.[128] This is the first observation that particulate air pollution can enhance coagulation and thrombosis risk in a manner that adversely affects the venous circulation in addition to the arterial cardiovascular system.

Cardiac Arrhythmias and Arrest

Several studies have observed associations between fine PM and related pollutants and cardiac arrhythmias, often based on data from implanted cardioverter-defibrillators.[129–136] However, no clear pollution-related associations were observed in studies from a relatively clean metropolitan area, Vancouver, British Columbia, Canada,[137,138] or from a relatively large study in Atlanta, Ga.[139] Similarly, pollution-related associations have been observed with cardiac arrest in Rome, Italy,[140] and Indianapolis, Ind,[141] but not in Seattle, Wash.[142,143] The mixed results may reflect different PM compositions due to different sources or variations among the methods used.

Evidence Summary

On the basis of the available epidemiological studies that have reported the associations between PM exposures with specific subsets of cardiovascular outcomes (morbidity, mortality, or hospitalizations), the existing level of overall evidence is strong for an effect of PM on ischemic heart disease, moderate (yet growing) for heart failure and ischemic stroke, and modest or mixed for peripheral vascular and cardiac arrhythmia/arrest (Table 6).

Ambient Air Pollution and Subclinical Pathophysiological Responses in Human Populations

It is likely that many subclinical physiological changes occur in individuals in response to $PM_{2.5}$ exposures that do not become overtly manifest as a cardiovascular event (eg, death or MI). The illustration of these more subtle responses bolsters the plausibility of the observable outcome associations and provides insight into the pathways whereby air

Table 6. Overall Summary of Epidemiological Evidence of the Cardiovascular Effects of $PM_{2.5}$, Traffic-Related, or Combustion-Related Air Pollution Exposure at Ambient Levels

Health Outcomes	Short-Term Exposure (Days)	Longer-Term Exposure (Months to Years)
Clinical cardiovascular end points from epidemiological studies at ambient pollution concentrations		
Cardiovascular mortality	↑ ↑ ↑	↑ ↑ ↑
Cardiovascular hospitalizations	↑ ↑ ↑	↑
Ischemic heart disease*	↑ ↑ ↑	↑ ↑ ↑
Heart failure*	↑ ↑	↑
Ischemic stroke*	↑ ↑	↑
Vascular diseases	↑	↑†
Cardiac arrhythmia/cardiac arrest	↑	↑
Subclinical cardiovascular end points and/or surrogate measures in human studies		
Surrogate markers of atherosclerosis	N/A	↑
Systemic inflammation	↑ ↑	↑
Systemic oxidative stress	↑	
Endothelial cell activation/ blood coagulation	↑ ↑	↑
Vascular/endothelial dysfunction	↑ ↑	
BP	↑ ↑	
Altered HRV	↑ ↑ ↑	↑
Cardiac ischemia	↑	
Arrhythmias	↑	

The arrows are not indicators of the relative size of the association but represent a qualitative assessment based on the consensus of the writing group of the strength of the epidemiological evidence based on the number and/or quality, as well as the consistency, of the relevant epidemiological studies.

↑ ↑ ↑ Indicates strong overall epidemiological evidence.

↑ ↑ Indicates moderate overall epidemiological evidence.

↑ Indicates some but limited or weak available epidemiological evidence.

Blank indicates lack of evidence.

N/A indicates not applicable.

*Categories include fatal and nonfatal events.

†Deep venous thrombosis only.

pollutants mediate CVDs. The "Biological Mechanisms" section discusses the hypothesized global pathways and reviews the studies related to the fundamental cellular/molecular mechanisms elucidated by controlled human and animal exposures and toxicological/basic science experiments. The following section reviews the recent evidence that ambient exposure to air pollution can mediate potentially harmful subclinical cardiovascular effects. In general, many positive associations are found (Table 6). Numerous complex interactions between variations in the characteristics, sources, and chemistry of the particles, coupled with diversity in time frames, mixtures of exposures, and degrees of individual susceptibility, likely explain some of the disparity among findings.

Systemic Inflammation

There is evidence that under some circumstances, exposure to ambient PM can be associated with elevated circulating proinflammatory biomarkers that are indicative of a systemic response after PM air pollution inhalation that is not limited to the confines of the lung. Early reports found associations with day-to-day variation in acute-phase proteins, such as C-reactive protein (CRP), fibrinogen, or white blood cell counts,[144–147] as reviewed previously.[1] Limited evidence on the association between cumulative PM exposures and fibrinogen levels and counts of platelets and white blood cells was also available.[148]

A number of more recent studies have reported positive associations with short-term ambient PM exposure and day-to-day elevations in inflammatory markers. These include increases in CRP in an elderly population[149] and individuals with coronary atherosclerosis[150]; CRP and fibrinogen in young adults[151] and elderly overweight individuals[152]; and CRP, tumor necrosis factor-α (TNF-α), and interleukin (IL)-1β in children.[153] Recent evidence has also been found for an upregulation of circulating soluble adhesion molecules (eg, intercellular adhesion molecule-1) in 92 Boston, Mass–area individuals with diabetes[154] and 57 male subjects with coronary artery disease in Germany.[150] In a larger analysis of 1003 MI survivors, also in Germany, CRP was not related to PM exposure; however, ambient particle number concentration and PM_{10} were associated with increased IL-6 and fibrinogen, respectively.[155] Short-term levels of in-vehicle $PM_{2.5}$ have also been linked to increases in CRP among healthy highway patrol troopers.[156] In a follow-up analysis, elevations in certain particulate components of traffic pollution (eg, chromium) were associated with increased white blood cell counts and increased IL-6 levels.[157] Short-term changes in ambient PM levels have also been linked to acute (1 to 3 days later) alterations in biomarkers of inflammation, oxidative stress, and platelet activation among elderly adults with coronary artery disease living in retirement communities in Los Angeles, Calif.[158,159] Pollutants associated with primary combustion (eg, elemental and black carbon, primary organic carbon) and UFPs rather than $PM_{2.5}$ appeared to be strongly associated with adverse responses in this population.

Regarding more long-term exposures,[160] a positive association between white blood cell count and estimated long-term 1-year exposure to PM_{10} was reported in the Third National Health and Nutrition Examination Survey. Among 4814 adults in Germany, small increases in annual mean $PM_{2.5}$ (3.9 $\mu g/m^3$) were associated with increases in high-sensitivity CRP by 23.9% and in fibrinogen by 3.9% among men only. Estimated long-term traffic exposure was not related to inflammatory changes in either sex.[161]

Several studies, including some with improved exposure assessment,[162] some that included analyses of large population cohorts,[163,164] and a recent evaluation of long-term annual PM_{10} levels in England,[165] have not found a relationship between particulate exposure and inflammation. It is

conceivable that differences in the magnitude or character of the inflammatory response will occur because of variations in the particulate chemistry and duration/intensity of exposures. Certain individuals may also be more susceptible. The evidence suggests that subjects with underlying cardiovascular risk factors and the metabolic syndrome may exhibit stronger associations.[152,160,166] Conversely, antiinflammatory medications such as statins may mitigate the actions of ambient particles.[152,155] All together, there is some evidence for a positive association between recent and long-term PM exposure and a systemic proinflammatory response; nevertheless, there is variation in the strength and consistency of changes among the variety of biomarkers and patient populations evaluated (Table 6).

Systemic Oxidative Stress

A state of oxidative stress refers to a condition in which levels of free radicals or reactive oxygen/nitrogen species (eg, O_2^-, H_2O_2, $ONOO^-$) are higher than normal (eg, healthy individuals in whom they are countered by homeostatic processes such as antioxidants) and thus are capable of exerting many adverse biological effects (eg, lipid/protein/deoxyribonucleic acid [DNA] oxidation, initiation of proinflammatory cascades). Although many biomarkers of differing systemic responses are available (eg, lipid or protein oxidation products), oxidative stress may occur at the local cellular/tissue level and not be directly observable by circulating markers. In addition, oxidative stress is often induced by and elicits inflammatory processes. The 2 processes are biologically linked. Therefore, human studies investigating the effect of PM on oxidative stress per se are difficult to perform. Only a few studies have directly investigated the occurrence of systemic oxidative stress in humans in relation to ambient PM exposure. Three studies of young adults conducted in Denmark demonstrated elevations in biomarkers of protein, lipid, or DNA oxidation in relation to PM exposure from traffic sources.[167–169] In a study of 76 young adults from Taipei, Taiwan,[151] the investigators found evidence of increased levels of 8-hydroxy-2′-deoxyguanosine adducts in DNA in relation to short-term elevations in ambient PM. Two studies have also demonstrated increases in plasma homocysteine, evidence that exposure to ambient PM can elevate this circulating mediator of oxidative stress.[170,171] Finally, Romieu et al[172] found that dietary supplementation with omega-3 polyunsaturated fatty acids might be capable of altering the systemic oxidative stress response (reduction in copper/zinc superoxide dismutase and glutathione) induced by air pollutants among residents living in a nursing home in Mexico City, Mexico. Because of the relatively small number of studies, more investigation is required to make firm conclusions and to understand the nature of the systemic oxidative stress response potentially induced by ambient PM (Table 6).

Thrombosis and Coagulation

Early reports indicated that increased plasma viscosity[144] and elevated concentrations of fibrinogen[146] are associated with short-term changes in ambient PM concentrations. More recent evidence was found for an upregulation of circulating von Willebrand factor in 57 male subjects with coronary artery disease in Germany[150] and 92 Boston-area individuals with diabetes.[154] Riediker[157] found that components of in-vehicle $PM_{2.5}$ were also related to increased von Willebrand factor and decreased protein C among highway patrol troopers. In the Atherosclerosis Risk in Communities study, a 12.8-$\mu g/m^3$ elevation in ambient PM_{10} was associated with a 3.9% higher von Willebrand factor level,[173] but only among those with diabetes. There was no linkage between PM_{10} exposure and fibrinogen or white blood cell levels.

Alterations in other markers that indicate changes in thrombosis, fibrinolysis, and global coagulation have also been reported. An immediate elevation in soluble CD40-ligand concentration, possibly reflecting platelet activation, recently was found to be related to ambient UFP and accumulation-mode particle ($PM_{0.1–1.0}$) levels in patients with coronary artery disease.[155] Ambient PM_{10} levels have also been associated with augmented platelet aggregation 24 to 96 hours after exposure among healthy adults.[174] In this study, there were no concomitant observable changes in thrombin generation, CRP, or fibrinogen induced by PM_{10}. Increases in plasminogen activator inhibitor-1 and fibrinogen levels have been noted in healthy subjects,[151] as well as elevated plasminogen activator inhibitor-1 in patients with coronary artery disease only,[175] in association with ambient PM levels in Taipei. Chronic indoor pollution exposure to biomass cooking in rural India has also been associated with elevated circulating markers of platelet activation.[176] Recently, Baccarelli et al[128,177] demonstrated in healthy subjects and among individuals with deep venous thrombosis living in the Lombardy region of Italy that prothrombin time was shortened in relation to recent and long-term ambient PM_{10} concentrations. Nevertheless, some studies found no effects of ambient pollution,[178] nor have significant changes been reported among all the biomarkers or subgroups of individuals investigated.[150,154,170,173] Similar to the study on systemic inflammation, the results related to thrombosis/coagulation are quite variable given the differences in study designs, patients, biomarkers evaluated, and pollutants; however, these adverse effects appear somewhat more consistent among higher-risk individuals (Table 6).

Systemic and Pulmonary Arterial BP

Several studies have reported that higher daily PM levels are related to acute increases in systemic arterial BP (approximately a 1- to 4-mm Hg increase per 10-$\mu g/m^3$ elevation in PM).[179–184] In a small study of patients with severe heart failure,[185] pulmonary artery and right ventricular diastolic BP were found to increase slightly in relation to same-day levels of PM. Chronic exposure to elevated $PM_{2.5}$ was associated with increased levels of circulating endothelin (ET)-1 and elevated mean pulmonary arterial pressure in children living in Mexico City.[186] These results may explain in part the risk for heart failure exacerbations due to PM

exposure; however, not all studies of systemic arterial BP have been positive.[187-189]

Recently, Dvonch et al[190] demonstrated significant associations between increases in systolic BP and daily elevations in $PM_{2.5}$ across 347 adults living in 3 distinct communities within metropolitan Detroit, Mich. Much larger effects were observed 2 to 5 days after higher $PM_{2.5}$ levels within a specific urban location of southwest Detroit (8.6 mm Hg systolic BP increase per 10-$\mu g/m^3$ $PM_{2.5}$) than throughout the entire region or cohort (3.2 mm Hg). This suggests that specific air pollution sources and components contribute significantly to the potential for PM exposure to raise BP. Interestingly, it was recently reported in a crossover study of 15 healthy individuals that systolic BP was significantly lower (114 versus 121 mm Hg) during a 2-hour walk in Beijing, China, while the subjects were wearing a high-efficiency particulate-filter facemask than when they were not protected.[191] Wearing the facemask was also associated with increased HRV, which suggests that the rapid BP-raising effects of particle inhalation may be mediated through the autonomic nervous system (ANS). In a similar fashion,[192] reducing exposure to particulate pollution from cooking stoves was shown to be associated with lower systolic (3.7 mm Hg, 95% CI −8.1 to 0.6 mm Hg) and diastolic (3.0 mm Hg, 95% CI −5.7 to −0.4 mm Hg) BP among Guatemalan women than among control subjects after an average of 293 days. These findings demonstrate that indoor sources of PM (eg, cooking, biomass) may have important cardiovascular health consequences and that reductions in particulate exposure are capable of lowering BP, and they suggest that chronic exposure to PM air pollution may alter long-term basal BP levels. Even given the rapid variability of BP on a short-term basis and the numerous factors involved in determining individual responses (eg, patient susceptibility, PM composition, and time frames of exposure), overall, it appears that ambient PM can adversely affect systemic hemodynamics, at least under certain circumstances (Table 6).

Vascular Function

In the first ambient PM study related to changes in vascular function, O'Neill et al[193] reported that both endothelium-dependent and -independent vasodilation were blunted in relation to air pollution levels in Boston. The largest changes occurred in association with sulfate and black carbon, suggestive of coal-burning and traffic sources, respectively. Significant adverse responses were observed within 1 day yet were still present and slightly more robust up to 6 days after exposure. Moreover, the adverse responses occurred solely among diabetic individuals and not in patients at risk for diabetes mellitus. Two other studies[184,194] also demonstrated impaired vascular function due to short-term changes in ambient PM among diabetic patients. In the study by Schneider et al,[194] endothelium-dependent vasodilation was blunted during the first day, whereas small-artery compliance was impaired 1 to 3 days after elevated ambient PM levels. Interestingly, higher concentrations of blood myeloperoxidase were related to a greater degree of endothelial dysfunction, which suggests that white blood cell sources of reactive oxygen species (ROS) may be involved.

In healthy adults, very short-term exposure to elevated levels of ambient PM from traffic sources while exercising for 30 minutes near roadways[195] and when resting by bus stops for 2 hours[196] has been related to impaired endothelium-dependent vasodilation. Daily changes in ambient gaseous pollutants (SO_2 and NO_x) in Paris, France, have also been associated with impaired endothelium-dependent vasodilation among nonsmoking men.[197] Finally, indoor particulate air pollution may also be harmful to vascular function. Bräuner and colleagues[198] recently reported that reductions in 48-hour $PM_{2.5}$ levels due to filtering of air in subjects' homes resulted in improved microvascular vascular function among elderly subjects. Nevertheless, changes in short-term ambient PM levels have not been linked with impaired conduit[197] or microvascular[178] endothelial function in all studies. Even when the few negative studies are considered, the overall evidence supports the concept that ambient PM is capable of impairing vascular function, particularly among higher-risk individuals (eg, those with diabetes) and after traffic-related exposure (Table 6).

Atherosclerosis

A few cross-sectional studies have reported an association between measures of atherosclerosis in humans and long-term exposures to ambient air pollution levels. The first study to demonstrate this relationship was an analysis of data from 798 participants in 2 clinical trials conducted in the Los Angeles area. A cross-sectional contrast in exposure of 10 $\mu g/m^3$ $PM_{2.5}$ was associated with an adjusted nonsignificant 4.2% (95% CI −0.2% to 8.9%) increase in common carotid intima-media thickness[199]; however, in certain subgroups of patients, such as women, the effect was much larger (13.8%, 95% CI 4.0% to 24.5%). In a population-based sample of 4494 subjects from Germany,[200] it was found that residential proximity to major roadways was associated with increased coronary artery calcification. A reduction in distance from a major road by half was associated with a 7% (95% CI 0.1% to 14.4%) higher coronary artery calcium score. Proximity to traffic was also related to an increased risk for peripheral artery disease in women but not men.[201] In an analysis of 3 measures of subclinical disease (carotid intima-media thickness, coronary calcium, and ankle-brachial index) among 5172 adults from the Multi-Ethnic Study of Atherosclerosis, only common carotid intima-media thickness was modestly (yet significantly) associated with 20-year exposure to $PM_{2.5}$.[202] In a related study from the same cohort, abdominal aortic calcium was associated with long-term $PM_{2.5}$ exposure, especially for residentially stable participants who resided near a $PM_{2.5}$ monitor.[203] Although it appears that long-term exposure to higher levels of ambient PM might accelerate the progression of atherosclerosis, more investigations are needed (Table 6).

Heart Rate Variability

Numerous studies have continued to explore associations between daily changes in PM air pollution exposure and alterations (typically reductions) in HRV metrics, putative markers of cardiac autonomic balance.[129,149,156,204–242] Recent observations in the Normative Aging Study cohort have shown strong effect modification of the PM-HRV relationship by obesity and genes that modulate endogenous oxidative stress or xenobiotic metabolism, such as glutathione S-transferase M1, methylenetetrahydrofolate reductase, and the hemochromatosis gene.[207,243,244] Additional findings suggest protective effects of statins, dietary antioxidants, and B vitamins, as well as omega-3 polyunsaturated fatty acids.[205,207,215,243,244] These results suggest that pathways that reduce endogenous oxidative stress have a protective effect that mitigates reductions in HRV due to ambient PM exposure.

However, the overall results are not entirely consistent. Some studies have reported increases in HRV mediated by PM, specifically among younger healthy people and patients with chronic obstructive lung disease.[156,208,216] Nevertheless, the general pattern suggests that PM exposure is associated with increased heart rate and reductions in most indices of HRV among older or susceptible individuals, such as those with obesity and the metabolic syndrome. Typically, time-domain measures (eg, standard deviation of normal RR intervals) and total power are reduced within hours after exposure. Most, but not all, pertinent studies have also found that the largest reduction in power is within the high-frequency domain. In sum, these observations provide some evidence that ambient PM air pollution exposure rapidly reduces HRV, a surrogate marker for a worse cardiovascular prognosis (Table 6). Although studies corroborating changes in autonomic activity by other methods (eg, microneurography or norepinephrine kinetics) have not been performed, the HRV findings are perhaps reflective of the instigation of a generalized cardiovascular autonomic imbalance due to relatively greater parasympathetic than sympathetic nervous system withdrawal.

Cardiac Ischemia and Repolarization Abnormalities

There has been limited direct evidence for the actual induction of cardiac ischemia or repolarization abnormalities in the electrocardiogram (ECG) by exposure to ambient levels of PM.[223,245] Recent follow-up analyses from the initial ULTRA study (Exposure and Risk Assessment for Fine and Ultrafine Particles in Ambient Air)[245] suggested that traffic-related combustion pollutants were most strongly related to the promotion of ST-segment depression among elderly nonsmokers during exercise stress testing.[246] Moreover, even very acute $PM_{2.5}$ exposure within the past 1 or 4 hours has been associated with cardiac ischemia during exercise.[247] New findings support these associations in elderly subjects[248] and in patients with coronary artery disease in Boston.[249] In the latter study, traffic-related PM was most strongly related to the incidence of ST-segment depression during 24-hour Holter monitoring, and the risk for ischemia was greatest within the first month after a cardiac event among patients with diabetes. Overall, there is a modest level of evidence that PM exposure can promote cardiac ischemia in susceptible individuals (Table 6).

Epigenetic Changes

There have been relatively few studies examining gene–air pollution exposure interactions, and most have done so while investigating a small number of loci for genetic polymorphisms. Although some studies have suggested greater air pollution susceptibility with one or another genomic polymorphism,[207,243,244] few have evaluated the potential for epigenetic changes after exposures. Reduced levels of DNA methylation have been linked to aging, oxidative stress, and CVD. Recently, Baccarelli et al[250] have shown among 718 elderly participants in the Normative Aging Study that short-term exposures (over 1 to 7 days) to $PM_{2.5}$ and black carbon are associated with decreased "global" DNA methylation in long interspersed nucleotide elements. It was posited that oxidative stress from air pollution exposure could have interfered with the capacity for methyltransferases to interact with DNA or altered the expression of genes involved in the methylation process. This observed effect of pollution exposure was analogous to changes seen with 3.4 years of aging in the cohort. Additional findings among workers in a furnace steel plant support these observations.[251] Nevertheless, the mechanisms involved and the cardiovascular implications of these preliminary, although provocative, epigenetic changes require more investigation.

Traditional Cardiovascular Risk Factors

In addition to the fact that individuals with traditional risk factors are likely to be at higher risk for cardiovascular events due to PM exposure, air pollutants may also promote the development of these risk factors over a prolonged period of time. Few published studies have investigated this possibility. A report from the Multi-Ethnic Study of Atherosclerosis has demonstrated that residential proximity to major roadways was associated with a higher left ventricular mass index as measured by cardiac magnetic resonance imaging.[252] The degree of increase was analogous to a 5.6-mm Hg increase in systolic BP among the study participants. This suggests that traffic-related exposures may have increased left ventricular mass by chronically elevating systemic arterial BP, a common cause of left ventricular hypertrophy. However, other mechanisms cannot be excluded, such as systemic inflammation and oxidative stress, which could potentially activate neurohormonal pathways (eg, ANS imbalance, renin-angiotensin system) that could directly mediate such a finding. In addition, a recent study of adults older than 30 years of age (n=132 224) participating in the National Health Interview Survey reported a significant association between self-reported hypertension and estimated annual $PM_{2.5}$ exposure using US EPA monitoring data.[253] A 10-$\mu g/m^3$ elevation in $PM_{2.5}$ was associated with an

adjusted odds ratio of 1.05 (CI 1.00 to 1.10) for the presence of hypertension. The increase in risk was found only among non-Hispanic whites. These studies provide some initial evidence that longer-term PM exposures may augment the risk for developing chronically elevated BP levels or even overt hypertension.

Brook et al[254] have also demonstrated a novel relationship between a metric of long-term traffic exposure (NO_2 level by residence) and the odds of having the diagnosis of diabetes mellitus among patients in 2 respiratory clinics in Ontario, Canada. In women only, the odds ratio of diabetes was 1.04 (95% CI 1.00 to 1.08) for each increase of 1 parts per billion (ppb) of NO_2. Across the interquartile range (4 ppb NO_2), exposures were associated with nearly a 17% increase in odds for diabetes mellitus. The first biological support for this finding comes from a study in Iran that demonstrated that the previous 7-day-long exposure to PM_{10} was independently associated with worse metabolic insulin sensitivity among 374 children 10 to 18 years of age.[255] These findings suggest that the systemic proinflammatory and oxidative responses due to long-term PM air pollution exposure could potentially increase the risk for developing clinically important aspects of the metabolic syndrome, such as hypertension and diabetes mellitus. Further studies in this regard are warranted.

Evidence Summary

Table 6 provides a consensus qualitative synopsis based on the expert opinions of the writing group members of the overall level of existing support, linking each surrogate or intermediate cardiovascular outcome with exposures to PM at ambient concentrations, based solely on the database of observational studies.

Additional Epidemiological Findings and Areas of Continued Research

Responsible Sources and Pollution Constituents

Although PM concentration (mass per cubic meter) has been associated with cardiovascular events in numerous studies, the specific particulate constituents and the sources responsible remain less clear. Despite the fact that it is a difficult undertaking, several epidemiological studies have attempted to identify the culprit components within the PM mixtures. With regard to PM-associated inorganic ions (nitrate and sulfate), it has been suggested that the overall toxicological data do not clearly implicate these compounds as responsible for mediating the cardiovascular health effects of $PM_{2.5}$.[256] Nevertheless, sulfate particles have been associated with cardiopulmonary mortality in the ACS and Harvard Six Cities studies.[62,68] A recent time-series analysis among 25 US cities found that cardiovascular risk was increased when PM mass contained a higher proportion of sulfate, as well as some metals (aluminum, arsenic, silicon, and nickel).[257] It is possible that these positive findings represent sulfate serving as a marker for an effect mediated by a toxic PM mixture derived from commonly associated sources (eg, coal combustion). Nevertheless, a direct role for particle sulfate in causing cardiovascular events cannot be excluded entirely.[256]

In California, short-term exposures to several different PM constituents that likely reflect combustion-derived particulates, including organic and elemental carbon and nitrates, were most strongly associated with higher cardiovascular mortality.[258] Certain metals (zinc, titanium, potassium, and iron) and sulfate levels in the winter months were also positively related. Similarly, ambient levels of organic and elemental carbon have been most strongly linked among PM constituents with hospitalizations for CVDs in multipollutant models in a study among 119 US cities.[259] Finally, $PM_{2.5}$ composed of higher levels of elemental carbon, along with the metals nickel and vanadium,[48] has also been linked with greater risks for cardiovascular hospitalizations.[260] These results support that the chemistry or composition of the $PM_{2.5}$ (eg, organic/elemental carbon and certain metals) along with the responsible source from which these mixtures are derived (eg, fresh combustion, traffic) may play important roles in determining the risk for cardiovascular events. However, the extent to which these constituents mediate specific responses, alone or together, and their importance beyond the concentration of $PM_{2.5}$ mass alone represent an area of active research that requires more investigation to reach firm conclusions.

Many experiments have demonstrated the especially toxic properties and strong oxidizing potential of the smallest particle sizes (eg, UFP) and of the specific chemical species typically rich within this size fraction (eg, transition metals, organic compounds, and semiquinones).[261] Although some epidemiological evidence suggests that exposure to ultrafine compounds[17] may be associated with higher cardiovascular risk (eg, an elevation of UFP count by 9748/cm^3 has been associated with an increase in cardiovascular mortality of approximately 3% within 4 days in Erfurt, Germany[262]) and adverse responses,[158,159] there have been few such studies because they are challenging to conduct, for numerous reasons. Moreover, there are few UFP monitors, and the levels measured at regional sites may not accurately reflect an individual person's exposure because of marked spatial heterogeneity, because the concentrations are dominated by local point sources of fresh combustion (eg, roadways). This could help explain some of the previously negative study findings.[116]

Similarly, coarse particulates between 0.25 and 1.0 μm in diameter may affect the cardiovascular system,[221,264,265] and although the available data related to hard events and cardiovascular mortality have suggested a relationship,[265,266] recent findings have been less consistent.[104] In the most recent time-series analysis of 112 US cities, coarse PM was independently associated with elevated all-cause, stroke, and pulmonary, but not cardiovascular, mortality after controlling for $PM_{2.5}$.[43] Coarse PM was also not associated with either fatal or nonfatal cardiovascular events after controlling for $PM_{2.5}$ levels in the Nurses' Health Study[267] or the Women's Health Initiative cohort analyses.[72] Additional research is required to establish whether there are independent health effects of the other

particulate size fractions beyond those posed by fine particles. On the other hand, $PM_{2.5}$ mass concentration is the metric most consistently associated with cardiovascular morbidity and mortality. It remains to be determined whether this reflects limitations of available data, the long-lived and regionally homogenous atmospheric nature of $PM_{2.5}$, that few studies have investigated the independent effects of the other sizes, difficulties in performing epidemiology studies with adequate UFP exposure estimates, or that specific constituents within the fine PM fraction (or another unidentified agent correlated with that fraction) are actually responsible for causing cardiovascular events. Although particles <0.1 μm (ie, UFPs) do make up a small fraction of $PM_{2.5}$ mass, the correlation between UFP particle number and total $PM_{2.5}$ mass concentration is often weak. Because of their minute size, UFPs make up only a small portion of the total $PM_{2.5}$ mass, even though they represent the largest actual number of particles within fine PM. They also have the highest surface area and a differing surface chemistry. Therefore, changes in the underlying UFP concentration do not likely account for or explain the linkages between $PM_{2.5}$ mass concentration and cardiovascular events observed in large multicity studies. The overall epidemiological evidence thus indicates that fine PM poses an independent cardiovascular risk and that any putative effects of these other size fractions cannot fully explain the observed $PM_{2.5}$–cardiovascular morbidity/mortality relationship.

On the other hand, there is mounting evidence for a distinctive role played by motor vehicle traffic–related exposures in elevating cardiovascular risk.[108,111,268,269] Lipfert et al[76,77] interpreted the results of their analysis of the Veterans Affairs hypertensive male cohort as suggesting that traffic density was a more "significant and robust predictor of survival in this cohort" than $PM_{2.5}$. Analyses of the Oslo,[81] Dutch,[82] AHSMOG,[74,75,88] French PAARC,[79] and German women cohorts[80] and related studies from areas in the United Kingdom,[270] Canada,[271] Norway,[272] and Rome[273] found that measures that often indicate traffic-related exposure (NO_2, NO_x, traffic density, and living near major roads) were also associated with increased mortality. Long-term 5-year average traffic-generated air pollution exposure has been associated with an increased risk of fatal MI (odds ratio 1.23, 95% CI 1.15 to 1.32 per 31-$\mu g/m^3$ increase in NO_2) but not nonfatal MI in Stockholm County, Sweden.[274] The results mirror the results of several cohort studies[72,73] that found that air pollution exposures appeared to be more strongly linked with cardiovascular mortality than nonfatal events. Recently, an analysis from a cohort in the Netherlands demonstrated that several metrics of traffic-related air pollution exposure remained significantly associated with increased risk for cardiovascular events even after adjustment for higher levels of traffic noise.[275]

The effect of long-term traffic-related exposure on incidence of fatal and nonfatal coronary heart disease was recently assessed after adjustment for background air pollutants and cardiovascular risk factors in 13 309 adults in the Atherosclerosis Risk in Communities study.[276] Interestingly, background chronic ambient $PM_{2.5}$ concentrations were not related to the interpolated traffic exposure levels or to heart disease outcomes, which supports the highly localized nature of traffic sources of exposure. After 13 years of follow-up in 4 US communities, individuals residing within the highest quartile of traffic density had a relative risk of 1.32 (95% CI 1.06 to 1.65) for fatal and nonfatal heart disease events. Despite multiple statistical adjustments, the investigators also acknowledged the possibility for residual confounding as a potential source of bias. The specific traffic-related pollution components, such as UFP or gaseous-phase chemicals (eg, SVOCs), that are responsible for the positive findings among these studies remain unknown. The close proximity to roadways within these epidemiological studies (eg, 400 m) required to observe an association with elevated cardiovascular risk, however, matches the atmospheric fate of these shorter-lived pollutants. The findings may thus suggest the existence of cardiovascular health effects mediated by specific air pollutants rather than $PM_{2.5}$ per se. There is room for improvement in assessment of traffic exposures in epidemiological research, and better approaches are now being incorporated into research projects, such as accounting for associated factors (eg, noise or spatial autocorrelation with socioeconomic status).[275,277]

Geographic differences in cardiovascular risk due to PM have also been observed across US regions, with more consistent or stronger effects observed in Eastern versus Western states.[71,103,257] Differences between North American and European cities have also been reported.[61] PM exposures are typically, but not always,[258] associated with larger effects during warmer months (spring through fall) than in the winter.[45,103,257] Variations in pollution characteristics (eg, sulfate), time spent outdoors, air conditioning usage and particle penetration indoors, ambient temperature and meteorology, and mobile (eg, diesel) or stationary (eg, coal combustion) sources of exposure may help explain these differences. Finally, variations in the cardiovascular risk posed by PM may also occur because of heterogeneity in the metric of exposure, such as personal versus background regional,[25] indoor versus outdoor sources, and differences in intracity versus intercity gradients.[69] A better understanding of the responsible constituents and sources is important and could potentially lead to more targeted and effective regulations. On the other hand, finding continued evidence that the adverse cardiovascular health effects cannot be linked conclusively to a particular or specific chemical species or source of pollution but rather that they occur in response to a variety of exposure types or mixtures would support the present-day policy of reducing exposure to overall fine particulate mass to achieve public health benefits.

Time Course and Concentration-Response Relationships

Many studies have demonstrated that PM air pollution exposure does not simply advance the mortality by a few days of critically ill individuals who would have otherwise died (eg, mortality displacement or "harvesting").[278,279] There also appears to be a monotonic (eg, linear or log-linear) concentration-response relationship between $PM_{2.5}$ and mor-

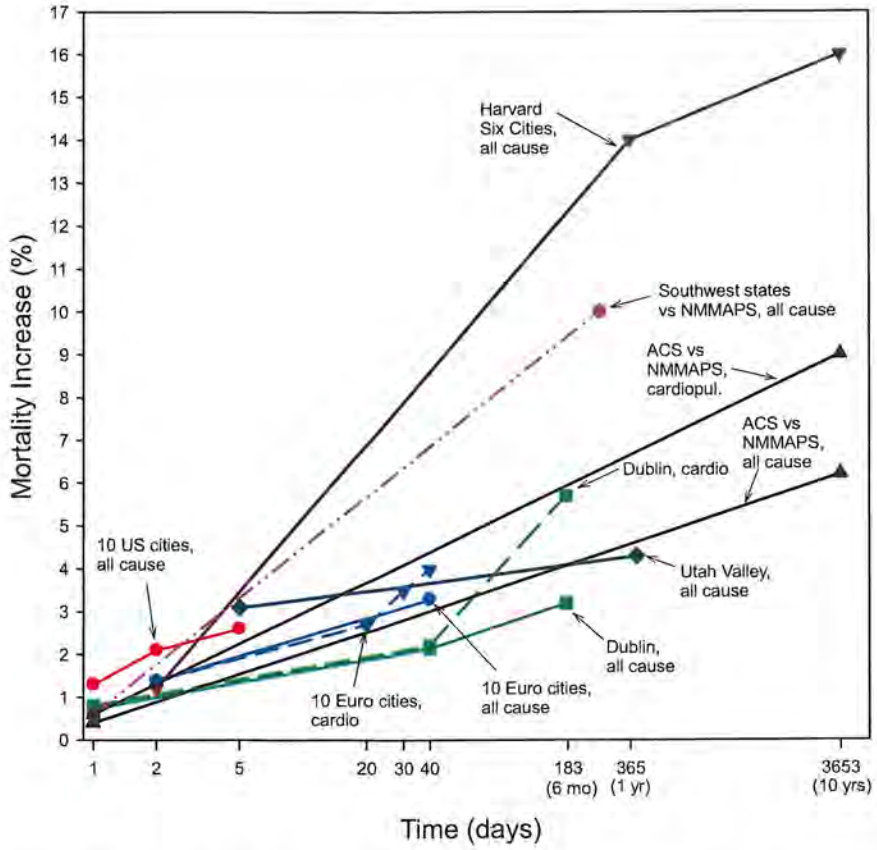

Figure 2. Comparison of estimates of percent change in mortality risk associated with an increment of 10 $\mu g/m^3$ in $PM_{2.5}$ or 20 $\mu g/m^3$ of PM_{10} or British Smoke (BS) for different time scales of exposure (log scale of approximate number of days, updated and adapted from Pope[281a]). Euro indicates European; cardio, cardiovascular disease; and cardiopul, cardiopulmonary.

tality risk observed in cohort studies that extends below present-day regulations of 15 $\mu g/m^3$ for mean annual levels, without a discernable "safe" threshold.[67,70,84] Cardiovascular risk due to particle exposure was also shown to extend below 15 $\mu g/m^3$ in the recent analysis of the Women's Health Initiative Observational Study.[72] This monotonic association supports the idea that any reduction in particulate pollution will translate into health benefits within a population of people, each with their own individual level of susceptibility. It also suggests that a larger decrease in $PM_{2.5}$ exposures will produce a greater reduction in mortality. Finally, a recent analysis of the literature provided important new insights into the nature of the PM exposure-response relationship.[280] The risk for cardiovascular mortality was shown to increase in a linear fashion across a logarithmically increasing dosage of inhaled fine-particle levels that ranged from ambient PM air pollution (≈0.2 mg/d), through secondhand smoke (≈1 mg/d), to active smoking (200 mg/d). This means that the exposure response is extremely steep at very low PM levels (ie, ambient air pollution) and flattens out at higher concentrations (ie, active smoking). This may help explain the seemingly incongruent and comparatively very high degree of cardiovascular risk posed by the much lower levels of PM exposure from ambient pollution and secondhand smoke versus the much higher doses due to active smoking. Thus, the cardiovascular system may be extremely sensitive to very low levels of PM inhalation as encountered with ambient pollution.

At present, the underlying nature and full scope of the temporal-risk relationship posed by longer-term PM exposures remain uncertain.[2,281] The writing group members did concur that the available epidemiological studies demonstrate larger cardiovascular risks posed by more prolonged exposures to higher PM levels than observed over only a few days (Figure 2). Cohort studies using Cox regression survival analyses (over months to years) are capable of evaluating a more complete portion of the temporal-risk relationship than time-series analyses over only a few days that use Poisson regression. However, given the lack of complete information, no conclusions could be drawn on the full magnitude of the augmented risk posed by chronic exposures, the time window (a few months versus decades) required to exhibit this enhanced risk, the underlying biological causes, the extent to which statistical differences between study types explain the variations in risk, and whether clinically relevant chronic CVDs are precipitated by chronic exposures. Some writing group members believe it is important to differentiate as 2 distinct issues the potentially greater effect of long-term exposures on increasing the risk for acute events (eg, cardiovascular mortality) compared with the putative effect on initiating or accelerating the development of chronic CVD processes per se (eg, coronary atherosclerosis). As such, it is possible that the greater risks observed in cohort studies could be capturing the fact that repetitive exposures over months or years augment the risk for sudden cardiovascular events in susceptible people, without actually worsening an underlying "chronic" disease process.

On the one hand, the available studies demonstrate that the majority of the larger risk-effect sizes posed by longer-term versus short-term exposures appear to be manifested within

only 1 to 2 years of follow-up. Extending the duration of follow-up increases cardiovascular risk, but to a progressively smaller degree over time (Figure 2). The discrepancy in the effect sizes among study types (eg, cohort versus time-series studies) could also reflect differences in statistical methodologies or population susceptibilities.[282–284] Recent attempts to investigate this matter[64,84] suggest that the risk for acute events associated with chronic exposures may be reasonably well estimated by only the most proximal 1 to 2 years of PM levels. The most recent time frames of exposure also explain a substantial portion of the excess cardiovascular risk observed in several cohort studies.[70,72,73,83] These findings bolster the argument that relatively rapid and pliable (and potentially reversible) biological responses, such as the instigation of plaque instability or the enhanced thrombotic potential caused by PM-mediated inflammation or endothelial dysfunction (which can occur and abate over only a few weeks to months), could explain the biology responsible for this greater relative risk.

On the other hand, cogent alternative arguments can be made to explain the differences in relative risk between the cohort and time-series studies. The likely high correlation of a recent year's exposure levels with exposures over many years, as well as the uniform rank ordering of exposure severity over time among cities, can explain why only a short period of PM exposure assessment is required to understand the risk of longer-term exposures. In addition, no studies have evaluated the potential risks of exposure over decades or a lifetime. PM augments the ability of traditional risk factors to accelerate the development of atherosclerosis in experimental settings. As such, it is also plausible that long-term exposures may enhance cardiovascular risk to an even greater extent by increasing an individual's susceptibility for future cardiovascular events or acute exposures. In addition, the full extent of this possibility may not be illustrated by the limited follow-up period (4 to 5 years) of the majority of cohort studies. The writing group thus agreed that this important issue requires more investigation.

It is also possible that these 2 explanations are not mutually exclusive. Furthermore, it cannot be concluded from available information that a long period of time is required for reductions in PM levels to translate into a decrease in cardiovascular risk. On the contrary, reductions in secondhand smoke[285] and PM air pollution levels[64,84,90,95] appear to produce fairly rapid decreases in cardiovascular event rates, within a few months to years.[284] At present, the available data do not allow for firm conclusions regarding the underlying biology and the full extent of the potentially nonuniform PM exposure–to–cardiovascular risk temporal relationship.

Susceptibility to Air Pollution Exposure

Susceptibility refers to a heightened risk for a particular cardiovascular end point or event to occur compared with the general population at the same concentration of PM exposure. Typically, this is indicative of an underlying medical condition (eg, diabetes) or personal characteristic (eg, old age) that causes this enhanced risk. This is in contrast to the term "vulnerability," which refers to a population of individuals at greater risk for more frequent or high levels of exposures.

Earlier studies reviewed in the first AHA scientific statement[1] suggested that susceptible populations include the elderly; individuals with diabetes; patients with preexisting coronary heart disease, chronic lung disease, or heart failure; and individuals with low education or socioeconomic status. In the ACS study, current and previous smokers appeared to be at the same or greater degree of risk.[67] Among more recent studies, the Women's Health Initiative also reported positive findings among active smokers and an elevated risk for cardiovascular mortality induced by $PM_{2.5}$.[72] Conversely, current smokers were found to be at no increased risk for cardiovascular mortality in response to $PM_{2.5}$ exposure in the Nurses' Health Study.[73] Thus, the effect modification of smoking status requires more investigation. The APHENA study of European and North American cities recently confirmed that elderly and unemployed individuals are at higher risk of short-term PM exposure.[61] In a multicity time-series study in Asia, women, the elderly, and individuals with lower education and socioeconomic status were also shown to be at elevated risk.[286] A few additional studies have reported some evidence of susceptibility to short-term PM exposures among older individuals, people with diabetes, and those with a lower level of education.[287–289] Finally, a recent study illustrated that present-day levels of $PM_{2.5}$ likely increase the risk for a cardiac event within a few days of exposure principally (or even solely) among individuals with preexisting significant coronary artery disease, even if they are seemingly healthy (eg, without anginal symptoms). Patients without obstructive lesions on heart catheterization were not at any risk for $PM_{2.5}$-induced myocardial events over the short term.[13] This is not surprising, because most acute cardiovascular events occur among individuals with underlying vulnerable substrate (eg, unstable plaques) and not in individuals with normal coronary arteries.

Obesity has been newly recognized as a possible susceptibility factor. Two cohort studies have shown that a greater body mass index enhances the susceptibility for PM-induced cardiovascular mortality, at least among women.[72,73] Although individuals with diabetes showed a trend toward greater risk in the Women's Health Initiative,[72] hypertension, high cholesterol, smoking, elderly age, education, and income did not alter the risk association. Overall, there appears to be little effect modification by race, hypercholesterolemia, or BP among the studies. Finally, sex may also be a risk-effect modifier. The particularly robust risk estimates of the 2 cohort studies that included only women,[72,73] the fact that PM increased cardiovascular risk in female but not male participants of the AHSMOG study,[75] and the multicity time-series findings in Asia[286] suggest that women may be at greater risk for cardiovascular mortality related to PM. Further studies are needed to clarify whether obese individuals and women are indeed susceptible populations.

Biological Mechanisms

There has been substantial improvement in our understanding of the biological mechanisms involved in PM-mediated

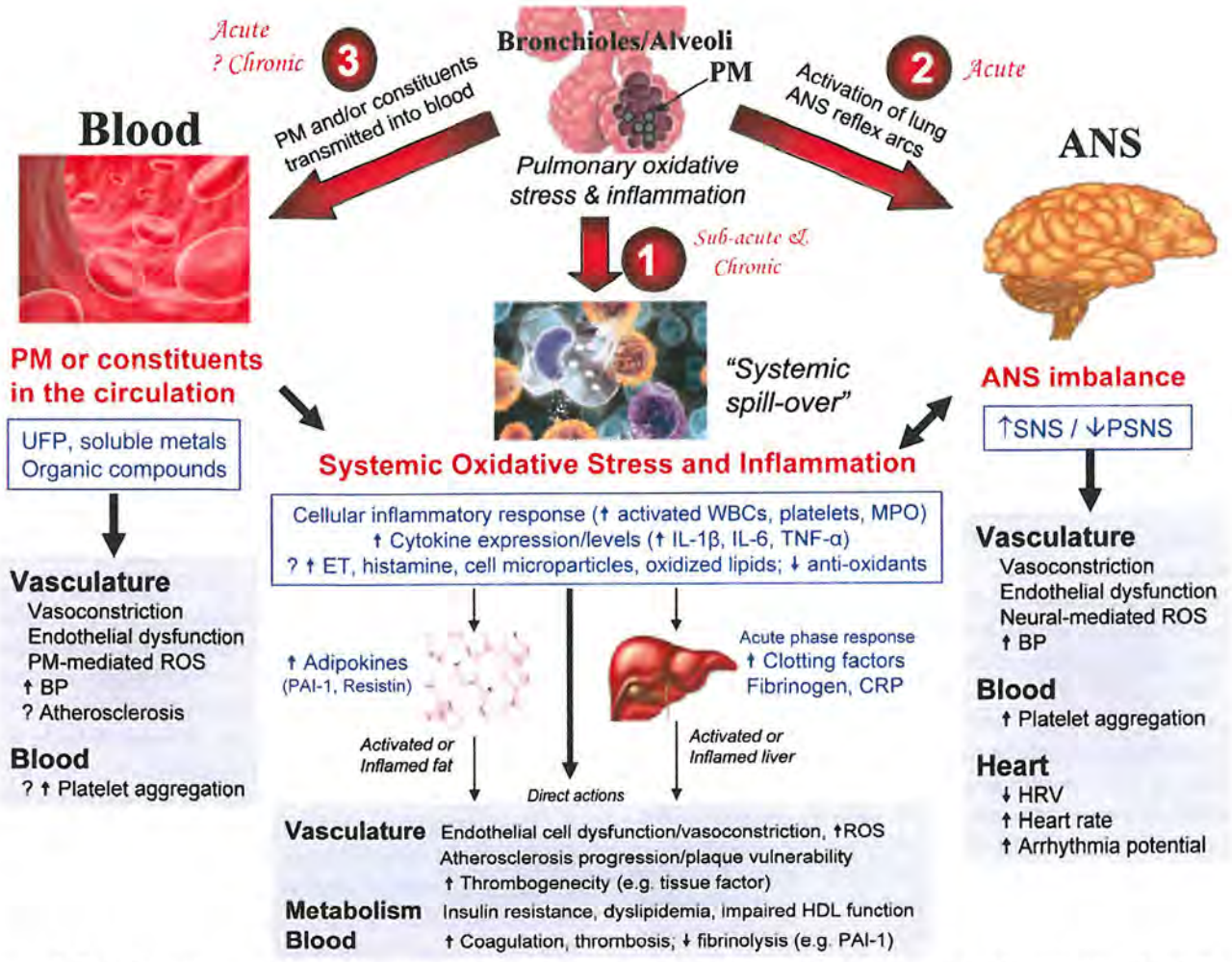

Figure 3. Biological pathways linking PM exposure with CVDs. The 3 generalized intermediary pathways and the subsequent specific biological responses that could be capable of instigating cardiovascular events are shown. MPO indicates myeloperoxidase; PAI, plasminogen activator inhibitor; PSNS, parasympathetic nervous system; SNS, sympathetic nervous system; and WBCs, white blood cells. A question mark (?) indicates a pathway/mechanism with weak or mixed evidence or a mechanism of likely yet primarily theoretical existence based on the literature.

cardiovascular effects. Studies before 2004 were reviewed previously,[1] and only some are again discussed here for contextual background. A number of new experiments have demonstrated very rapid effects of air pollution, such as vascular dysfunction, which argues for the existence of pathways that convey signals systemically within hours of PM inhalation. On the other hand, there is also support for chronic biological effects, such as the promotion of atherosclerosis. At the molecular level, persuasive evidence supports an integral role for ROS-dependent pathways at multiple stages, such as in the instigation of pulmonary oxidative stress, systemic proinflammatory responses, vascular dysfunction, and atherosclerosis. In sum, new studies continue to support the idea that inhalation of PM can instigate extrapulmonary effects on the cardiovascular system by 3 general "intermediary" pathways. These include pathway 1, the release of proinflammatory mediators (eg, cytokines, activated immune cells, or platelets) or vasculoactive molecules (eg, ET, possibly histamine, or microparticles) from lung-based cells; pathway 2, perturbation of systemic ANS balance or heart rhythm by particle interactions with lung receptors or nerves; and pathway 3, potentially the translocation of PM (ie, UFPs) or particle constituents (organic compounds, metals) into the systemic circulation (Figure 3).

Exposure Considerations

Animal and human exposure studies are discussed separately and apart from the effect of ambient PM because their methodologies and clinical relevancies vary widely. Controlled exposure studies involve exposing a subject to various size fractions of PM within a chamber connected to ambient air (concentrated or nonconcentrated) or a source of aerosolized particles. Virtual impactor systems that deliver concentrated ambient particles (CAPs) from "real-world" ambient air are a commonly used approach for mimicking exposures to higher levels of ambient particles without requiring invasive methods or the generation of artificial particles.[3] Both a strength and limitation, however, is that CAPs can vary considerably from day to day in composition. Additionally, only certain particle size ranges are typically concentrated (eg, PM from 0.1 to 2.5 μm in the fine-CAP system), whereas

ambient air contains a mixture of particle sizes, volatile organics, and gases that are not concentrated (and can be lowered). Potential interactions between PM and gaseous copollutants on health end points are therefore excluded, unless the latter are reintroduced in an artificial fashion. Other methods of controlled-inhalation exposures include diesel engine exhaust (diluted and aged mixtures of high numbers of fresh combustion UFPs with vapor-phase components), roadside aerosols, and wood-burning sources. Regarding animal exposures, intratracheal instillation methods may sometimes be required because of the limited availability of inhalation exposure systems. Unfortunately, particle size and surface characteristics—mostly retained in inhalation systems with fresh sources of pollution and which may be important in influencing biological effects—are likely significantly altered in instillation systems or by methods that use previously collected particulate. However, the use of carefully modeled exposures (eg, deposition calculation) and the recognition that areas of "hot spots" containing markedly higher PM levels within the lung may occur even during normal inhalation make the results of these experiments potentially relevant.[2] Further detailed discussions of exposure considerations are reviewed elsewhere.[290]

The protocol details vary considerably among the studies. Many aspects of exposure, including the duration, concentration, PM size ranges and composition, and gaseous copollutants, are important to consider. A wide variety of outcomes may be anticipated depending on the biological pathways evoked by differing exposures. Moreover, there are multiple determinants of the subsequent physiological responses, including the time frames of investigation, preexisting susceptibility, animal models, and the details of the outcomes investigated. All of these factors may explain some of the heterogeneity in the reported study results and must be taken into consideration when interpreting the findings.

Animal Exposure and Toxicological Studies

Studies that investigate the effects of exposure on susceptible animals (eg, those with preexisting cardiovascular or metabolic abnormalities) may be preferable in many circumstances because of the increasing recognition that the pathways underlying the biological effects of PM overlap (ie, modify and/or enhance) those of conventional cardiovascular risk factors. Such factors (eg, hypertension or atherosclerosis) may also be necessary or at least responsible for the evocation of a more readily observable or robust response. For example, in the context of systemic oxidative stress or inflammation, the cellular machinery for the generation of excess ROS and proinflammatory responses (eg, adhesion molecule and cytokine expression) is already primed or operational in susceptible animals.

Pulmonary Oxidative Stress and Inflammation

The molecular events responsible for triggering pulmonary oxidative stress and inflammation, along with the interactions between lung and immune cells, the inhaled PM, and the protective secretions (eg, surfactant, proteins, and antioxidants), are highly complex,[4–6] as reviewed in detail elsewhere.[290a,290b,414] In brief, size, charge, solubility, aggregation, ROS-producing potential, and chemistry play roles in determining the responses. These include the particle fate (eg, lung clearance versus retention rates), the nature of the PM-cell interactions (eg, immune versus lung cell uptake, host cell responses, and intracellular sequestration/location), and the dose (likely typically a small percentage of inhaled PM) and pathways of potential systemic transmission of PM or its constituents, such as in the circulation [free, intracellular within circulating cells, (lipo)protein-bound] or via lymphatic spread.[4,5,290a,290b] Because of their nano-scale size, UFPs may directly enter multiple lung cell types via nonphagocytic pathways and adversely affect organelles, such as mitochondria.[6,290a,290b] Larger unopsonized fine particles are more typically taken up by phagocytes through interactions with innate immunity receptors such as MARCO (macrophage receptor with collagenous structure) or other scavenger receptors.[5,290a,290b] This may in fact be a protective mechanism that sometimes prevents harmful lung inflammation. Certain particle compounds may directly generate ROS in vivo because of their surface chemistry (eg, metals, organic compounds, and semiquinones) or after bioactivation by cytochrome P450 systems (eg, polycyclic aromatic hydrocarbon conversion to quinones).[6,290a,290b] A particle surface or anions present on otherwise more inert particles may disrupt iron homeostasis in the lung and thereby also generate ROS via Fenton reactions.[291] Other PM constituents may do so indirectly by the upregulation of endogenous cellular sources (eg, nicotinamide adenine dinucleotide phosphate [NADPH]) oxidase)[292,293] or by perturbing organelle function (eg, mitochondria) by taken-up PM components.[261] Particle stimulation of irritant and afferent ANS fibers may also play a role in local and systemic oxidative stress formation.[294] Given the rich antioxidant defenses in the lung fluid, secondarily generated oxidization products of endogenous molecules (eg, oxidized phospholipids, proteins) or a reduction in endogenous antioxidants per se may be responsible at least in part for the state of oxidative stress in the lungs (along with instigating the subsequent cellular responses) rather than ROS derived directly from PM and its constituents.

Subsequent to oxidative stress, antioxidant and phase II defenses may be activated (eg, inducible nitric oxide synthase, glutathione) via transcription factor Nrf2-dependent pathways.[261] When inadequate, pathological oxidative stress can initiate a variety of pulmonary inflammatory responses. For example, ROS in the lungs has been shown to augment the signal transduction of membrane ligand (eg, epidermal growth factor by disrupting phosphatases) or pattern-recognition receptors (eg, toll-like receptors [TLR])[295–299] and/or stimulate intracellular pathways (eg, mitogen-activated protein kinases) that lead to the activation of proinflammatory transcription factors (eg, nuclear factor-$\kappa\beta$) that upregulate expression of a variety of cytokines and chemokines.[261] Alteration in lung cell redox status may itself stimulate nuclear factor-$\kappa\beta$. Biological components within coarse PM could also directly trigger inflammation (eg, nuclear factor-$\kappa\beta$ pathways) by binding to TLR2 or TLR4 receptors or other innate immune pattern-recognition receptors.[297] It is also possible that other components of metal-rich

PM could instigate inflammatory pathways via TLR activation directly or via the oxidation of endogenous biological compounds that then serve as TLR ligands.[300] Finally, there is some evidence that PM can activate inflammatory mitogen-activated protein kinase signaling by angiotensin II receptor–dependent pathways.[295] These inflammatory responses can also exacerbate the initial oxidative stress [eg, via upregulation of cellular NAD(P)H oxidase] and thus initiate a positive-feedback cycle.

Available studies support important contributions to pulmonary inflammation from innate immune cells such as neutrophils and macrophages (TNF-α, IL-6), as well as from the adaptive immune system, such as T cells (IL-1, IL-4, IL-6, and IL-10). Although the dominant source of cytokines likely represents the alveolar macrophages and lung epithelial cells, the role of other innate and adaptive immune cells cannot be ruled out.[299,301,302] Recently, myeloperoxidase activity was shown to increase after PM exposure in the same time course of appearance of cellular inflammation (primarily neutrophils) in the lung.[303] Gaseous components such as ozone may also amplify the toxicity of PM.[304]

Systemic Inflammation

In the context of examining the cardiovascular effects of air pollution, it is important to consider the inflammatory mediators that are released from lung cells after contact with PM, because some could conceivably spill over to the general circulation or increase liver production of acute-phase proteins (eg, CRP, fibrinogen). An increase in circulating proinflammatory mediators (eg, activated immune cells, cytokines) could thus serve as a pathway to instigate adverse effects on the heart and vasculature. Numerous experiments have demonstrated increased cellular and inflammatory cytokine content, such as IL-6, IL-1β, TNF-α, interferon-γ, and IL-8, of bronchial fluid and sometimes in circulating blood after acute exposure to a variety of pollutants.[292,305–311]

Critical roles for the elevations in systemic and pulmonary levels of IL-6 and TNF-α have been observed after PM exposure, typically coincident with pulmonary inflammation.[292,302,306,309,311–314] There is at least some evidence that the degree of pulmonary inflammation and systemic inflammation (IL-6) correlates with the elevation of systemic cytokines and systemic vascular dysfunction.[314] In a 4-week inhalation exposure to freshly generated diesel exhaust, IL-6 knockout mice did not demonstrate increased cellular inflammation or TNF-α in bronchial fluid, which implies a role for IL-6.[315] Consistent with these findings, acute intratracheal exposure to PM_{10} resulted in an increase in IL-6, TNF-α, and interferon-γ in the bronchial fluid.[316] However, in this study, IL-$6^{-/-}$ mice showed roughly the same levels of TNF-α in bronchial fluid as wild-type mice, although interferon-γ was decreased to control values.[316] The results also suggested that lung macrophages play an important role, because depletion of these cells abolished the increases in some of the cytokines and systemic cardiovascular responses. Although our understanding of the source of IL-6 and TNF-α and their involvement in the systemic inflammatory response after PM exposure remains incomplete, these and other experiments appear to suggest that at least with PM_{10} particles, alveolar macrophages play a dominant role.[309,314,316]

Among remaining uncertainties, the upstream signaling pathway responsible for the recognition of PM components that in turn produce the systemic inflammation has not been fully elucidated[317]; however, there is some evidence with other particulates and experimental models of lung injury that ROS generated by NADPH oxidase or pattern-recognition receptors may modulate some of these responses.[292,299,318] NADPH-oxidase knockout mice demonstrated significantly lower IL-6 and macrophage inflammatory protein-2 responses to collected PM than wild-type mice.[292] Extrapulmonary sources may also be involved in promulgating the systemic inflammation. $PM_{2.5}$ exposure in a model of diet-induced obesity in C57Bl/6 mice for a duration of 24 weeks resulted in elevations in TNF-α and IL-6. In addition, there were increases in circulating adipokines, such as resistin and plasminogen activator inhibitor-1.[319] The elevation in cytokines, thought to be derived from adipose sources, in addition to findings of adipose inflammation in that study, raises the possibility of additional systemic nonpulmonary sources of such cytokines.

Systemic Oxidative Stress

Numerous in vitro studies have demonstrated activation of ROS-generating pathways by PM incubation, such as NADPH oxidases, mitochondrial sources, cytochrome P450 enzymes, and endothelial nitric oxide synthase in cultured cells or in pulmonary and vascular tissue.[293,311,320–329] Similar to inflammation, the oxidative stress after PM inhalation may not always stay confined within the lungs.[330] The sources of excess ROS within cardiovascular tissue may include circulating immune cells or cytokines, depletion of defense mechanisms (eg, impaired high-density lipoprotein function), oxidation of lipoproteins or other plasma constituents,[331] activation of ANS pathways,[294] or circulating PM constituents (eg, soluble metals, organic compounds) reaching the vasculature.[261] Activation of ROS-dependent pathways modulates diverse responses with far-reaching consequences, including vascular inflammation/activation, atherosclerosis, impaired basal vasomotor balance, enhanced coagulation/thrombosis, and platelet activation.[290b]

Recent experiments have indeed confirmed the existence of footprints or markers of oxidative stress within the cardiovascular system in the in vivo context. Acute-exposure studies[332] have shown a relationship between the vascular dysfunction in spinotrapezius microvessels and the release of myeloperoxidase from leukocytes into the vasculature within only hours after the pulmonary instillation of PM.[332] Interestingly, an insoluble particle (TiO_2) induced very similar effects. More long-term studies[333] have demonstrated that 10 weeks of exposure to $PM_{2.5}$ increased superoxide production in response to angiotensin II and resulted in upregulation of NAD(P)H oxidase subunits and depletion of tetrahydrobiopterin in the vasculature. These effects had functional consequences in terms of increases in systemic vascular resistance and BP. In another investigation that involved apolipoprotein E–deficient (ApoE$^{-/-}$) fed a high-fat diet, chronic exposure

to $PM_{2.5}$ exacerbated vascular oxidant stress and promoted atherosclerosis progression.[334] The proatherogenic effects of ambient UFPs[331] versus $PM_{2.5}$ in genetically susceptible $ApoE^{-/-}$ mice in a mobile facility close to a Los Angeles freeway have also been compared. Exposure to UFPs resulted in an inhibition of the antiinflammatory capacity of plasma high-density lipoprotein and greater systemic oxidative stress, as evidenced by increased hepatic malondialdehyde and upregulation of Nrf2-regulated antioxidant genes.[331]

Other experiments[294] have suggested that ANS imbalance may play an important role in PM-induced cardiac oxidative stress. Pharmacological inhibition of the ANS could significantly reduce chemiluminescence in the heart after exposure.[303] More recently, an upstream modulator, the transient receptor potential vanilloid receptor-1, within the lung was identified as central to the inhaled CAP-mediated induction of cardiac chemiluminescence.[335] In these studies, capsazepine was able to abrogate ECG alterations in rats during the 5-hour exposure, which suggests that neural ANS pathways are crucial.

Thrombosis and Coagulation

Earlier studies using intratracheal instillation of high concentrations of diesel exhaust particles demonstrated the induction of lung inflammation, platelet activation, and increased peripheral vascular thrombosis in both arteries and veins after photochemical injury.[336,337] Thrombosis susceptibility was ascribed to direct passage of the instilled UFPs in the blood, because large polystyrene particles unlikely to cross the lung-blood barrier did not increase peripheral thrombosis. In a subsequent study, a persistent increase in thrombosis susceptibility to diesel exhaust particles was shown after 24 hours, an effect that was mitigated by pretreatment with sodium cromoglycate, which indicates that this response was secondary to histamine release from basophil degranulation.[338] These same effects, however, were mimicked by 400-nm polystyrene particles with a low likelihood of transgressing the pulmonary barrier, which implicates pulmonary release of histamine as a mediator of thrombosis at the later time point. Because histamine was increased in the plasma at 6 and 24 hours after exposure, and diphenhydramine mitigated diesel PM–induced thrombosis at later time points but not at 1 hour, it was hypothesized that additional direct effects of PM constituents reaching the circulation may be responsible for the earliest prothrombotic effects.[339] No increase in circulating von Willebrand factor was observed after instillation of both particles. Finally, pulmonary instillation of carbon nanotubes produced neutrophil lung influx 24 hours later. Circulating platelet-leukocyte conjugates were elevated 6 hours after exposure, whereas procoagulant microvesicular tissue factor activity and peripheral thrombotic potential were increased 24 hours later. Inhibition of P-selectin abrogated these responses, which demonstrates that rapid activation of circulating platelets by the pulmonary deposition of PM plays a vital role.[340] This series of studies suggests that release of lung cell–derived mediators (eg, histamine) after several hours along with the more rapid activation of circulating platelets by lung inflammation via P-selectin–dependent processes may mediate distant system prothrombotic effects without necessarily inducing systemic endothelial damage.

In a study using C57BL/6J mice, intratracheal PM_{10} particles rich in transition metals decreased bleeding, prothrombin, and activated partial thromboplastin times and enhanced the levels of several coagulation factors as well as thrombosis times in response to experimental $FeCl_3$ injury.[316] This prothrombotic effect was mitigated in $IL\text{-}6^{-/-}$ and macrophage-depleted mice, which suggests that IL-6, lung macrophages, and pulmonary inflammation are necessary initial steps. It is possible, however, that coarse-particle components (eg, endotoxin) could have been important mechanistically via TLR activation. The effect of fine PM or UFPs per se requires more investigation. Chronic ambient exposure to $PM_{2.5}$ has also been shown to increase tissue factor expression in macrophages and smooth muscle cells in atherosclerotic lesions. Complementary in vitro studies with cultured human smooth muscle cells and monocytes demonstrate dose-dependent increases in tissue factor in response to collected ambient particles.[341] Other findings also support potential procoagulant and thrombotic effects of PM.[342,343] These collective studies suggest that both short- and long-term PM inhalation can enhance thrombotic and coagulation tendencies, potentially via increases in circulating histamine and inflammatory cytokines and/or activated white cells and platelets. The plausibility of these pathways is supported by the well-recognized cross talk between inflammation and thrombosis.[344] Potential additional roles for UFPs or soluble constituents that reach the circulation and directly enhance platelet aggregation or systemic oxidative stress (thus activating the endothelium and blunting platelet-derived nitric oxide) require more investigation.

Systemic and Pulmonary Hypertension

Early animal studies suggested small or inconsistent effects of PM on BP,[345–347] sometimes dependent on the season[348] of exposures. A potential explanation may be variations in experimental protocols, including differences in the delivery, duration, and composition of exposure and the methods used to measure BP. Moreover, PM by itself may represent a relatively weak stimulus but may act more robustly in concert with other predisposing factors to affect BP. Sun et al[333] recently demonstrated a significant interactive effect of fine-CAP exposure with the vasoconstrictor angiotensin II in rats. Preexposure to $PM_{2.5}$ for a 10-week period resulted in enhancement of its prohypertensive response measured continuously by intra-arterial radiotelemetry. The exaggerated BP elevation was accompanied by endothelial dysfunction, including blunted endothelium-dependent vasodilation and enhanced vasoconstrictor reactivity, along with upregulation of NAPDH oxidase and Rho-kinase–signaling pathways. In vitro exposure to UFPs and $PM_{2.5}$ was also associated with an increase in Rho-kinase activity, phosphorylation of myosin light chain, and myosin phosphatase target subunit. Pretreatment with the nonspecific antioxidant *N*-acetylcysteine and Rho-kinase inhibitors prevented these responses, which suggests an ROS-mediated mechanism for particle-mediated effects on vascular smooth muscle constriction. Further

studies corroborated the role of exaggerated Rho-kinase pathway activity in potentiating the hypertensive response to angiotensin II in mice exposed to $PM_{2.5}$.[349] Moreover, particle exposure augmented angiotensin-mediated cardiac hypertrophy and collagen deposition. Blockade of Rho-kinase abolished these effects. These responses suggest that chronic $PM_{2.5}$ exposure disrupts normal vascular homeostasis and vasoactive mediator balance through ROS-dependent mechanisms in a manner that sensitizes the vessel toward vasoconstrictors. Activation of RhoA/Rho-kinase signaling pathways appears to play an important mechanistic role.

In conscious canines with implanted BP catheters, systemic arterial BP increased and baroreceptor sensitivity was rapidly altered over a few hours during CAP exposure.[350] Interestingly, α-adrenergic antagonism abrogated the responses. The findings support a mechanistic role for acute activation of the sympathetic nervous system by inhaled particles. In a study with Wistar-Kyoto male rats, CAP exposure for 4 days upregulated ET-A receptor expression in the heart. This alteration was also weakly correlated with an increase in BP, which suggests a role for enhance ET activity.[351] PM has also been demonstrated to alter the release of ET-1 and ET-3 from the lungs.[352] Elevation in pulmonary vascular resistance and pulmonary arterial pressure, which suggests constriction of the pulmonary vessels, has also been demonstrated in response to respirable carbon black particles.[353] Recently, ultrafine carbon particles were shown to increase BP in spontaneously hypertensive rats 1 to 3 days after a 24-hour exposure.[354] This response occurred concomitant with increased ET-1 messenger ribonucleic acid levels in lung tissue and small elevations in plasma renin concentration and angiotensin I and II in the systemic circulation. These findings further support the idea that ET may play a role in cardiovascular responses to PM exposure and suggest that activation of the renin-angiotensin system may also be involved. It is not clear whether the elevated circulating ET levels reflect increased release from the lungs and whether this mediates a systemic vasoconstrictor response. Alternatively, the increase may be more indicative of enhanced vascular tissue activity of these systems. Longer-term exposures of carbon black for 4 weeks in Sprague-Dawley rats has also been shown to significantly increase systolic BP concomitant with increases in serum levels of IL-6 and CRP.[355]

Finally, in vitro exposure to soluble and insoluble components of UFPs induces constriction in isolated pulmonary arterial rings and activates intracellular signaling pathways such as phosphorylation of extracellular signal–regulated kinase-1/2 and p38 mitogen-activated protein kinase in pulmonary endothelial cells. These effects were antagonized by losartan, and several metal components (copper and zinc) could replicate the responses.[295] This suggests a possible role for activation of angiotensin II receptor pathways relevant for the maintenance of vasomotor tone and smooth muscle constriction after inhalation of metal constituents within PM.

In sum, the studies demonstrate that long-term PM exposures over a period of weeks are capable of enhancing vasoconstrictive responsiveness of the vasculature (eg, increased Rho-kinase activity and reduced nitric oxide bioavailability) by inflammatory and ROS-dependent cell-signaling pathways. Shorter-term exposures over several hours to days may lead to vasoconstriction and increased pulmonary and systemic BP by pathways dependent on enhanced ET or angiotensin II signaling. Lung cells may release ET into the systemic circulation and thus increase its systemic activity, or the vascular ET system may be relatively upregulated because of increased ROS or reduced nitric oxide. Activation of the renin-angiotensin system may also occur because of systemic oxidative stress or inflammation or as a consequence of ANS imbalance. The very acute increase in BP that occurs concomitant with the inhalation of particles or within only minutes to hours after exposure appears to be mediated by autonomic imbalance that favors a relative activation of the sympathetic nervous system. No study has evaluated the effect of air pollution on renal sodium handling or long-term pressure natriuresis mechanisms, which are fundamental to the generation of chronic hypertension.

Vascular Dysfunction and Atherosclerosis

Many early experiments demonstrated the capacity of PM constituents to blunt nitric oxide–dependent dilation and enhance vasoconstrictor tone in ex vivo vascular studies because of excess ROS formation.[1] The first in vivo experiment demonstrated the proatherosclerotic actions of intratracheal PM_{10} instillation.[356] More recently, the pulmonary instillation of several different PM types was shown to rapidly impair microvascular endothelium-dependent vasodilation within days, likely by proinflammatory or ROS-dependent mechanisms (eg, myeloperoxidase).[332] Several animal studies have now demonstrated that long-term exposure to ambient $PM_{2.5}$, by use of ambient-exposure facilities without direct pulmonary instillation, not only causes endothelial dysfunction but also accelerates the progression of atherosclerosis. Sun et al[334] demonstrated that exposure of atherosclerosis-prone $ApoE^{-/-}$ mice to environmentally relevant levels of CAP, derived from regional northeastern $PM_{2.5}$, for 6 months in conjunction with a high-fat chow diet potentiated plaque development and heightened vascular inflammation (CD68+ macrophage infiltration and inducible nitric oxide synthase expression) and oxidant stress. The atherosclerotic plaque progression was also accompanied by alterations in vasomotor tone, including decreased endothelium-dependent vasodilation and heightened vasoconstriction to adrenergic stimuli. Importantly, the normalized average $PM_{2.5}$ concentration over the entire period was 15.2 $\mu g/m^3$, which approximates the annual NAAQS. Similar findings were reported in other chronic CAP exposures that involved an $ApoE^{-/-}$ model.[357] However, exposures to a double-knockout model of ApoE-deficient and low-density lipoprotein receptor–deficient mice increased plaque cellularity, reflective of inflammation, but did not enhance plaque burden. It is possible that the atherosclerotic severity of this phenotype precluded the observation of more subtle effects of CAP exposures.

Intratracheal instillation of UFP can acutely impair aortic endothelium-dependent vasodilation.[358] Moreover, repeated 10-week-long endotracheal dispersion of UFP carbon black increased atherosclerosis in low-density lipoprotein receptor–

knockout mice.[359] This occurred without evidence of systemic translocation of particles into the cardiovascular tissues. UFP inhalation by use of exposure facilities has also recently been shown to augment atherosclerosis, perhaps to a greater degree than $PM_{2.5}$. When investigating the effects of different PM size fractions, Araujo et al[331] compared the proatherogenic potential of exposure over 40 days to ambient particles <0.18 μm versus $PM_{2.5}$ in $ApoE^{-/-}$ mice. UFPs caused more adverse cardiovascular responses (eg, systemic oxidative stress, impaired high-density lipoprotein function) and greater potency in accelerating atherosclerotic lesion formation, although $PM_{2.5}$ did demonstrate qualitatively similar effects. Recent studies have also demonstrated that PM exposure likely promulgates systemic atherosclerosis by mechanisms that overlap those of other conventional cardiovascular risk factors.[360] Intratracheal instillation of PM_{10} particles caused a rapid impairment in endothelium-dependent vasodilation, stimulation of bone marrow–derived cells, and increased migration of monocytes into atherosclerotic plaques.[361,362] Systemic inflammation (IL-6) was also related to the degree of endothelial dysfunction.[314] Finally, the most compelling evidence for rapid impairment in nitric oxide bioavailability being directly involved in the origin of PM-induced endothelial dysfunction was demonstrated recently. Both fine-PM and UFM inhalation for only a few hours in normal rats blunted agonist-stimulated nitric oxide production within the microvasculature, measured by direct electrochemical sensors, concomitant with an observed impairment in vasomotor relaxation. Inhibition of myeloperoxidase or NADP(H) oxidase partially restored normal nitric oxide bioavailability and endothelial function, which suggests a role of activation of these endogenous radical-generating enzymes in this biological response.[363]

Potentially relevant adverse vascular effects of nonparticulate PM components should not be discounted. There may also exist some synergy between vapor phase, gas, and particle constituents in relation to instigation of cardiovascular responses. Recently,[364] it was demonstrated in $apoE^{-/-}$ mice that whole gasoline engine exhaust over 1 or 7 days increased vascular messenger ribonucleic acid expression of matrix metalloproteinase (MMP)-2 and MMP-9. Levels of ET-1 and ROS were similarly increased. The vascular ROS and MMP-2 elevations were attenuated by tempol. Endothelial receptor antagonism ameliorated the vascular expression of MMP-2, MMP-9, and ROS. In separate experiments, diesel exhaust exposure to rats for 5 hours augmented ET-induced vasoconstriction, potentially via a blunting of ET-B–induced nitric oxide release.[365] The findings suggest that exposure to a fresh mixture of PM, gases, and vapors may play a role in rapidly triggering atherosclerotic plaque vulnerability via ROS and ET-dependent upregulation of MMP levels.

Some studies suggest that predisposed animals may be more susceptible to air pollution–mediated vascular dysfunction. Diesel exhaust particles delivered by intraperitoneal injection impaired nitric oxide–dependent vasodilation only in $apoE^{-/-}$ mice with atherosclerosis and not in healthy control animals.[366] Aortas from prediabetic rats were found to be more susceptible to repeated exposures to oil combustion particles in causing noradrenergic-mediated constriction and impaired endothelium-dependent vasodilation.[367]

Taken together, the available studies suggest that short- and long-term particle exposures (including PM_{10}, $PM_{2.5}$, and UFP) can impair conduit and resistance arterial endothelium-dependent vasodilation. Chronic exposures have been shown to be capable of promoting atherosclerosis progression and enhancing plaque vulnerability. The underlying mechanisms likely involve vascular sequelae of systemic inflammation (due to interactions with innate immune cells and cytokines) or exaggerated oxidative stress pathways. Excess vascular ROS and inflammation will impair endogenous vasodilator bioavailability (eg, nitric oxide), enhance vasoconstrictor tone (eg, ET), and chronically activate multiple intracellular pathways that promote atherosclerosis.[368–370]

Heart Rate Variability

Some of the earliest indications of systemic effects of PM came from ECG studies in rats.[371] In general, reductions in several measures of HRV have been shown.[372–376] Most of the recent research has focused on exploring the roles of susceptibility and exposure characteristics. Decreases in heart rate and HRV indices have been reported to be pronounced in senescent mice, which indicates that aging may be a susceptibility factor.[353] Using an anesthetized model of postinfarction myocardium sensitivity, Wellenius and colleagues[377] did not demonstrate an effect of 1 hour of CAP exposure on heart rate or spontaneous ventricular arrhythmias. In contrast, in a post-MI heart failure model in Sprague-Dawley rats, diesel exhaust emissions reduced HRV in both healthy and heart failure groups and increased the incidence of premature ventricular contractions. Studies in mice have also indicated a potential role for transition metals and nickel in HRV alterations[376] and provide initial clues on the PM components that could influence autonomic tone.[48]

Some beginning insight into the neural pathways involved has been reported recently. PM-induced ECG changes in rats were shown to be prevented by inhibiting the transient receptor potential vanilloid receptor in the lungs. This suggests that the relevant neural mechanism that leads to alterations in HRV or heart rhythm may be induced by activation of receptor-mediated autonomic reflexes in the lung.[335] Circulating particle constituents or inflammatory mediators interacting with myocardial ion channels or electrophysiology did not appear to be a pertinent mechanism, at least in these studies.[335] However, it is unknown whether similar mechanisms can account for the HRV changes observed in humans, and a more detailed understanding of the anatomic pathways involved is required. Finally, it remains unclear whether the changes in cardiac HRV are actually caused by or merely illustrate an underlying alteration in ANS balance. Experiments that clearly define the direct contribution of sympathetic and parasympathetic nervous system activities (eg, microneurography, norepinephrine spillover rates, or autonomic receptor or ganglionic blockade) are needed.

MI and Arrhythmia

PM exposure can increase experimental infarct size and potentiate myocardial ischemia and arrhythmias in experi-

mental MI models. Relatively high concentrations of intratracheal UFP instillation induced pulmonary inflammation and doubled MI size in mice.[358] Conscious dogs exposed to fine CAP for several days experienced greater ST-segment changes during transient coronary artery occlusion.[378] These studies suggested that particulate-related changes in myocardial blood flow may be responsible, a hypothesis recently supported by experiments in chronically instrumented dogs exposed to fine CAP before transient occlusion of the left anterior descending artery. PM exposure was associated with a small but significant decrease in total myocardial flow, especially in the ischemic zone, and increases in coronary vascular resistance without an alteration in rate-pressure product.[379] The abnormalities were inversely related to PM mass, particle number, and black carbon concentration.

Exposure to residual oil fly ash increases arrhythmia frequency in rats with preexisting premature ventricular complexes, which suggests that PM sensitizes ischemic myocardium to abnormal automaticity[372]; however, CAP had no effect in rats.[380] Nevertheless, the data suggest that PM exposure may potentially be capable increasing the sensitivity of the myocardium to ischemia, likely by impairing myocardial blood flow and perfusion. In theory, this could play a role in enhancing the propensity for ventricular arrhythmias.

Insulin Resistance

Recently, Sun et al[319] exposed C57BL/6 mice fed high-fat chow to fine CAP or filtered air for 24 weeks. Mice exposed to $PM_{2.5}$ exhibited marked worsening of whole-body insulin resistance, systemic inflammation (increased IL-6 and TNF-α), and higher levels of adipokines, such as resistin and plasminogen activator inhibitor-1. $PM_{2.5}$ increased visceral adiposity and inflammation ($F4/80^+$ cells), with stromal vascular cells expressing higher TNF-α and IL-6 and lower IL-10 levels. Exposure also induced insulin-signaling abnormalities and reduced phosphorylation of Akt and endothelial nitric oxide synthase in aortic tissue, accompanied by abnormalities in vascular relaxation to insulin. Additionally, there was evidence that $PM_{2.5}$ exaggerated adhesion of monocytes in mesenteric microvessels, culminating in accumulation in visceral adipose. These intriguing findings suggest that longer-term exposure to PM air pollution may promote the chronic development of insulin resistance, obesity, and the metabolic syndrome.

Controlled-Exposure Studies in Humans

Several new human exposure studies have been published, a few of which have even included patients with CVD or risk factors. Similar to the animal studies, large variations among the exposure protocols, measured outcomes, and subject susceptibilities likely explain much of the differences among findings and must be considered when interpreting the results.

Systemic Inflammation

Controlled human exposure studies have measured the effects on circulating inflammatory markers such as CRP, IL-6, and TNF-α. In many of these single-episode short-term exposures, no overt changes in plasma cytokine levels were observed after CAP[381–383] or diesel exhaust.[345,384–386] Similarly, CRP levels have not consistently been found to increase in the time frame and context of most of these studies.[313,384–386]

However, there have also been some positive findings. Increases in IL-6[313] and TNF-α 24 hours after exposure to diesel exhaust in healthy adults have been reported. High levels of ambient particles can stimulate the bone marrow to enhance the release of neutrophils, band cells, and monocytes into the circulation, which causes a cellular inflammatory response.[387,388] Some controlled-exposure studies corroborate the existence of a cellular proinflammatory response that manifests as increases in circulating white blood cell or immune cell counts. In 1 study, increased peripheral basophils in healthy older adults were noted 4 hours after a 2-hour exposure to fine CAP.[389] In a similar study, increased white blood cell counts were observed in healthy young adults 12 hours after exposure.[381] Recently, investigators observed an increased in total white blood cell and neutrophil levels immediately after a 2-hour exposure to CAP in downtown Toronto, Ontario, Canada.[390] Conversely, decreases in blood monocytes, basophils, eosinophils, and CD54 and CD18 adhesion molecule expression on monocytes after exposure to ultrafine carbon (10 to 50 $\mu g/m^3$) among exercising asthmatic individuals and healthy adults have also been reported.[391] The authors suggested in the latter study that these results may represent the sequestration of these cells in tissue compartments such as the lung or vasculature, where there may be selective expression of the corresponding receptors for these ligands.[362] However, other recent human clinical studies have found no association between peripheral blood cell counts and exposure to fine PM or UFPs such as zinc oxide,[392] ultrafine carbon,[393] or diesel exhaust.[313,384,385]

More subtle, yet physiologically relevant or functional proinflammatory changes may be overlooked by the measurement of circulating cytokines or cell counts alone in human studies. Peretz et al[394] recently evaluated gene expression using an expression array in monocytes after 2 hours of exposure to diesel exhaust. Although initially a small study, 10 genes involved in the inflammatory response were modulated in response to exposure (8 upregulated, 2 downregulated). These findings will need to be reproduced in larger studies and raise the possibility that functional changes in inflammatory cells may occur without discernible changes in their levels in the peripheral circulation.[394]

In sum, the findings from controlled human exposures do not demonstrate a robust inflammatory response; however, they have been limited by the fact that they are, by necessity, of short duration and relatively low concentration. Additionally, the results do not preclude an effect of higher exposures, the presence of more subtle responses, or alterations in other cellular inflammatory pathways not measurable by circulating markers.

Systemic Oxidative Stress

The demonstration of systemic oxidative stress is difficult in human studies. Nonetheless, a few studies have reported positive findings. These include an increase in urinary excre-

tion of free 8-iso-prostaglandin-2α among healthy adults after a 4-hour exposure to concentrated wood smoke[395] and an increase in plasma antioxidant capacity 24 hours after a 1-hour exposure to diesel exhaust in a group of healthy volunteers.[313] The investigators speculated that systemic oxidative stress after exposure may have been responsible for this upregulation in antioxidant defense.[313] Other investigators[394] have observed significant differences in expression of genes involved in oxidative stress pathways due to diesel exhaust exposure. Bräuner et al[167] recently investigated the effect of ultrafine traffic particles on oxidative stress–induced damage to DNA in healthy young adults exposed to low concentrations of ambient urban particles ($PM_{2.5}$ and $PM_{10-2.5}$ mass of 9.7 and 12.6 $\mu g/m^3$, respectively) in an exposure chamber above a busy road with high traffic density. The authors observed increased levels of DNA strand breaks and formamidopyrimidine-DNA glycosylase sites in monocytes after exposure to PM but no changes in the DNA repair enzyme 7,8-dihydro-8-oxoguanine-DNA glycosylase. Similar to their previous findings with ambient levels,[168] the results suggest that short-term exposure to UFPs may result in damage to DNA. This may occur through oxidative stress pathways, although there was no increase in messenger ribonucleic acid levels in heme oxygenase-1, a gene known to be regulated by Nrf2, a transcription factor regulated by oxidative stress.[396] Moreover, more recent observations by the same investigators failed to demonstrate significant biomarker signals for lipid or protein oxidative damage after similar near-roadway exposures.[178] Although not entirely consistent, the available studies demonstrate that acute exposure to PM, perhaps even at ambient levels, may be capable of inducing acute systemic oxidative stress in human subjects under certain circumstances. The assays used to assess the footprint of systemic "oxidative stress" or damage may also play a significant role in the results.

Thrombosis and Coagulation

Several new studies of controlled human exposure have evaluated the effects of PM on hemostatic markers (eg, factor VII, fibrinogen, platelet count, D-dimer, and von Willebrand factor). Although some of these studies have not observed changes after acute exposures,[392] others have reported increases in fibrinogen levels at 8 to 24 hours after exposure to CAP.[381,397] Mills and colleagues[384,385] recently demonstrated a significant effect of diesel exhaust on fibrinolytic function in response to intermittent exercise both in healthy men and in men with coronary heart disease. In both groups of volunteers, bradykinin-induced release of tissue plasminogen activator was observed to decrease compared with filtered air at 6 hours after exposure to diesel exhaust. These perturbations in tissue plasminogen activator release did not persist 24 hours after exposure.[313] In a randomized, controlled crossover study involving "at-risk" metabolic syndrome patients, no changes in plasminogen activator inhibitor-1 were noted over a 24-hour duration; paradoxically, a decrease in von Willebrand factor was noted in this study.[398] In a similar experiment conducted in healthy adults, diesel exhaust had no effect on D-dimer, von Willebrand factor, CRP, or platelet counts compared with filtered air up to 22 hours after exposure.[386] Other investigators[395] recently evaluated the effect of wood smoke on markers of coagulation, inflammation, and lipid peroxidation in young healthy subjects. Serum amyloid A and the ratio of factor VIII to von Willebrand factor, an indicator of an increased risk of venous thromboembolism, were increased at 4 hours after exposure.[395] Samet et al[383] reported an association between various coagulation markers and exposure to ultrafine, fine, and thoracic coarse CAP among healthy young adults. Although exposure to coarse CAP did not result in significant changes in hemostatic variables, the overall trend suggested a prothrombotic effect. Exposure to UFPs increased D-dimer levels, whereas fine-CAP effects tended to increase fibrinogen, similar to previously reported findings.[381]

The measurement of blood levels of coagulation factors or biomarkers of thrombosis could potentially miss a relevant biological effect at the vascular wall. Recently, ex vivo thrombus formation was assessed by use of the Badimon chamber after controlled exposures to dilute diesel exhaust in healthy volunteers.[399] This protocol measures thrombus formation in native (nonanticoagulated) whole blood triggered by exposure to a physiologically relevant substrate, under flow conditions that mimic those found in diseased coronary arteries. It may therefore provide a superior estimate of actual in vivo conditions related to thrombosis potential. Interestingly, dilute diesel exhaust exposure increased thrombus formation within 2 hours, in association with increased platelet activation (ie, increased circulating platelet-monocyte aggregates and soluble CD40 ligand). Taken together, these new studies have provided additional evidence that short-term exposure to PM at near-ambient levels may have small yet potentially significant effects on hemostasis in humans. Whether direct interactions of circulating PM constituents with platelets, activation of platelets due to lung inflammation or secondary to elevated systemic cytokine levels, or an increase in procoagulant factors (eg, fibrinogen) as an acute-phase response to inflammation (or a combination of these pathways) is responsible warrants attention in future studies.

Arterial BP

Although several studies have evaluated the BP response to acute exposures, many inconsistencies in results have been reported.[400] This must be considered in the context that BP was not the primary outcome of interest in most studies, nor was it typically assessed with adequate sophistication. In one of the earliest studies, $PM_{2.5}$ increased systolic BP in healthy subjects but decreased it in asthmatic individuals.[401] Three other controlled studies did not report changes among healthy adults.[345,402,403] However, in a more detailed reanalysis of the changes in BP during the actual period of exposure to CAP plus ozone, Urch et al[404] found a significant increase in diastolic BP of 6 mm Hg. The magnitude of response was associated with the concentration of organic carbon within $PM_{2.5}$.[405] Recent follow-up studies redemonstrated an acute prohypertensive response during the inhalation of CAP in 2 separate cities.[390] The $PM_{2.5}$ mass during exposure and decreases in several HRV metrics were associated with the

magnitude of the short-lived diastolic BP elevation. This suggested that the most plausible mechanism for this acute response was CAP-induced ANS imbalance that favored sympathetic over parasympathetic cardiovascular tone. Whether this reaction occurred because of a generalized stress response, as a consequence of specific soluble PM constituents directly altering central nervous system activity, or via altered ANS reflex arcs due to the interaction of inhaled particles with lung receptors/nerve endings remains to be elucidated.

The effect of inhaled particulates on BP has also been investigated in several other recent controlled human exposure studies. Two new studies assessed BP changes after a 1-hour exposure to diesel exhaust. Mills et al[384] found a 6-mm Hg increase in diastolic BP 2 hours after exposure, which was of marginal statistical significance ($P=0.08$); however, this trend did not persist for 24 hours,[384] nor was it found among patients with coronary artery disease.[385] The available data to date suggest that short-term exposure to $PM_{2.5}$ or diesel exhaust is capable in certain circumstances of rapidly raising BP. The most consistent and largest effects were seen concomitant with the inhalation of particles. Thus far, the most likely mechanism for such rapid hemodynamic responses appears to be ANS imbalance. However, it is possible that reductions in nitric oxide bioavailability that modulate basal arterial tone toward vasoconstriction or increases in ET among other hemodynamically active molecules (eg, angiotensin II) also play a role in some circumstances.

Vascular Dysfunction

The first controlled human exposure study related to vascular function reported that CAP plus ozone exposure caused acute conduit arterial vasoconstriction in healthy adults.[1] Endothelium-dependent and -independent vasodilation remained intact. Recent follow-up experiments determined that $PM_{2.5}$, not ozone, was responsible for the adverse vascular effects. However, in these subsequent and larger experiments, fine-CAP exposure did prove capable of diminishing conduit artery endothelium-dependent vasodilation 24 hours (but not immediately) after exposure.[390] Postexposure $PM_{2.5}$ mass and TNF-α level were both associated with the degree of endothelial dysfunction, which suggests that systemic inflammation induced by higher levels of particles was likely responsible. Finally, the CAP-induced endothelial dysfunction occurred during exposures in Toronto, Canada, but not Ann Arbor, Mich, which suggests that the composition of the particles is probably an important determinant of the vascular responses.

An acute alteration in vascular function/tone after short-term controlled PM air pollution exposure was corroborated recently.[406] In 27 adults (10 healthy adults and 17 with the metabolic syndrome), a 2-hour exposure to dilute diesel exhaust caused a dose-dependent constriction of the brachial artery and elevation in plasma ET level without impairing endothelium-dependent vasodilation. Contrary to the hypothesis that metabolic syndrome patients would show greater effects, vasoconstriction was greater in magnitude among the healthy participants. In an additional study, 2-hour exposure to UFPs composed of elemental carbon impaired peak forearm blood flow response to ischemia 3.5 hours later. There were no other vascular changes or alterations at other time points. BP was also not affected.[407]

Several recent studies have also shown that dilute diesel exhaust can impair peripheral resistance vessel responses to acetylcholine, bradykinin, and nitroprusside 6 hours after exposure.[384] The blunted responses to acetylcholine persisted for 24 hours in healthy adults.[313] In contrast, bradykinin and sodium nitroprusside–mediated vasodilation and bradykinin-induced acute plasma tissue plasminogen activator release were not altered 24 hours later. In subsequent studies, patients with stable coronary artery disease exposed to dilute diesel exhaust for 1 hour during intermittent exercise demonstrated reduced bradykinin-mediated tissue plasminogen activator release; however, microvascular endothelial function was not impaired.[385] This may be related to some degree of preexisting endothelial dysfunction in these patients. However, exercise-induced ST-segment depression and ischemic burden were significantly greater during diesel compared with filtered air exposure. These important findings experimentally highlight that PM air pollution exposure can trigger, or augment existing, myocardial ischemia extremely rapidly (in fact, concomitant with exposure). Reduced coronary flow reserve (that was not observed or resolved at the time of the postexposure brachial artery studies) due to rapid alterations in coronary microvascular function may have contributed to the acute myocardial ischemia. Alternatively, acute ANS imbalance induced by diesel exhaust inhalation may have acutely altered coronary tone and impaired myocardial perfusion.

In a study that exposed healthy young adults to 100 $\mu g/m^3$ of diesel exhaust for 2 hours,[364] it was recently demonstrated that this air pollution mixture acutely raised plasma ET-1 and MMP-9 expression and activity within 30 minutes. These results corroborate the animal data that even short-term exposures can rapidly alter factors, such as MMP activity, that are mechanistically linked with causing atherosclerotic plaque disruption (and thus acute MI). The increase in ET levels also corroborates previous studies[406] that showed that diesel exhaust can acutely affect important endogenous regulators of vasomotor tone.

Controlled air pollution exposures have not always been shown to impair endothelial function or vasomotor tone. Despite an increase in exhaled 8-isoprostane concentrations that suggested pulmonary oxidative stress, fine CAP did not affect brachial flow–mediated dilation or basal diameter in northern Scotland exposures.[382] However, the $PM_{2.5}$ consisted of relatively inert ambient sea-salt particles and was extremely low in combustion-derived sources. This is in contrast to the particle chemistry in the investigators' previous diesel exposure studies that showed positive findings.[408,409] Moreover, 24-hour exposure to ambient pollution shunted into a chamber next to a busy street did not impair microvascular endothelial function in 29 healthy subjects, as assessed by digital tonometry.[178] This exposure to near-roadway ambient air, which consisted of ambient UFP and $PM_{2.5}$, did not alter biomarkers of inflammation, hemostasis,

or protein and lipid oxidation. The authors speculated that the relatively low concentrations of UFP numbers and PM mass or the young, healthy status of the subjects could explain the null findings. Taken together, these studies suggest that brief PM exposure can trigger conduit arterial vasoconstriction, possibly in relation to increased ET activity or augmented sympathetic ANS tone. Under certain circumstances, conduit and resistance arteriole endothelium-dependent vasodilation can also be impaired within a few hours. This abnormality is more likely due to reduced nitric oxide bioavailability as a consequence of systemic proinflammatory and oxidative responses; however, alternative mechanisms and endogenous vasoactive pathways have not been fully explored. It is also apparent that the composition, source, and concentration of pollution, along with the susceptibility of the human subjects, play important roles in determining the vascular effects of acute air pollution exposure.

Heart Rate Variability

The results of several new controlled human exposure studies provide limited evidence to suggest that acute exposure to near-ambient levels of PM may be associated with small changes in HRV. There are at least 4 studies to support this. In the first study, healthy elderly individuals experienced significant decreases in HRV immediately after exposure.[233] Some of these changes persisted for at least 24 hours. Gong et al[410] studied healthy and asthmatic adults exposed to coarse CAPs with intermittent exercise. HRV was not affected immediately after the exposure but decreased in both groups at 4 and 22 hours after the end of the exposure; greater responses were observed in nonasthmatic individuals.[410] In another study, healthy elderly subjects and patients with chronic obstructive pulmonary disease were exposed to approximately 200 $\mu g/m^3$ CAP and filtered air for 2 hours with intermittent mild exercise. HRV over multihour intervals was lower after CAP than after filtered air in healthy elderly subjects but not in subjects with lung disease. A significant negative effect of CAP on ectopic heartbeats during or after CAP exposure relative to filtered air was noted in the healthy subjects, whereas the group with pulmonary disease experienced an improvement during or after CAP relative to filtered air.[389] Other investigators recently compared the effects of 2-hour exposures with intermittent exercise to ultrafine (average concentration 47 $\mu g/m^3$), fine (average concentration 120 $\mu g/m^3$), and coarse (average concentration 89 $\mu g/m^3$) CAP among healthy subjects.[383] In both the ultrafine and coarse studies, a crossover design was used in which each subject was exposed to both PM and filtered air. In the case of the fine-PM study, subjects did not serve as their own control but were exposed to either PM or filtered air. Thoracic coarse fraction CAP produced a statistically significant decrease in the standard deviation of normal-to-normal heart rate 20 hours after exposure compared with filtered air. No statistically significant effects on HRV were observed after exposure to UFPs as measured during controlled 5-minute intervals. However, the authors did observe a significant decrease in the standard deviation of normal-to-normal heart rate after exposure to UFPs based on an analysis of the 24-hour measurements. No differences were reported in HRV with fine-PM exposures. Although some controlled-exposure studies have reported either no acute changes[390] or, on occasion, increases in HRV metrics in subsets of individuals,[208,393,401] these studies generally demonstrate that acute PM exposure is capable of reducing HRV. More consistent reductions have been found among older adults (compared with younger subjects or those with lung diseases, who show mixed responses) and perhaps with exposures to larger particles.[233,389] Whether pulmonary ANS reflex arcs are activated by the deposition of PM within the lung or whether other pathways are responsible for these physiological changes in human exposure studies requires more investigation.

Table 7. Summary of Level of Evidence Supporting Global Biological Pathways and Specific Mechanisms Whereby $PM_{2.5}$, Traffic-Related, or Combustion-Related Air Pollution Exposure Can Affect the Cardiovascular System

	Animal Studies	Human Studies
General "intermediary" pathways whereby PM inhalation can instigate extrapulmonary effects on the cardiovascular system		
Pathway 1: Instigation of systemic proinflammatory responses	↑ ↑ ↑	↑ ↑ ↑
Pathway 2: Alterations in systemic ANS balance/activity	↑	↑ ↑
Pathway 3: PM and/or associated constituents directly reaching the systemic circulation	↑	↑
Specific biological mechanisms directly responsible for triggering cardiovascular events		
Vascular dysfunction or vasoconstriction	↑ ↑ ↑	↑ ↑
Enhanced thrombosis or coagulation potential	↑ ↑	↑ ↑
Elevated arterial BP	↑ ↑	↑ ↑
Enhanced atherosclerosis or plaque vulnerability	↑ ↑	↑
Arrhythmias	↑	↑

The arrows are not indicators of the relative size of the association but represent a qualitative assessment based on the consensus of the writing group of the strength of the mechanistic evidence based on the number and/or quality, as well as the consistency, of the relevant studies.

↑ ↑ ↑ Indicates strong overall mechanistic evidence.

↑ ↑ Indicates moderate overall mechanistic evidence.

↑ Indicates some but limited or weak available mechanistic evidence.

Blank indicates lack of evidence.

Evidence Summary and Contextual Framework for Biological Mechanisms

Table 7 provides an outline of the level of evidence supporting the generalized intermediary pathways and specific mechanisms whereby PM exposures can be capable of eliciting

cardiovascular events. At the molecular level, oxidative stress as a critically important cause and consequence of PM-mediated cardiovascular effects has a sound experimental basis.[261,290b,294,319,333,334,345–349,351,361–364,411] At the integrated physiological level, the collective body of evidence continues to support the existence of 3 general pathways (Figure 3). Some of these responses, such as systemic inflammation (via pathway 1), likely require antecedent pulmonary oxidative stress or inflammation in order to be initiated. Others, including ANS imbalance (via pathway 2) and PM or its constituents reaching the systemic circulation (via pathway 3), may not. Although PM-associated metals[412] and certain UFPs[261,413–415] might be capable of translocating into the blood stream, some studies have been negative in this regard.[355,416] Many issues related to this pathway are controversial and require resolution.[416] These include the relevance of the dosages delivered to cardiovascular organs, the consequences of particle constituent modifications after interactions with lung tissue/fluids and plasma components, the means of transport within the circulation (eg, protein bound or within cells),[417] and the time course and ultimate sites of PM sequestration. It is also possible that increases in some vasoactive mediators or molecules with adverse effects on cardiovascular tissue, such as ET-1,[351–354] may occur in the lung and systemic circulation without the need for antecedent lung inflammation. Moreover, the 3 general pathways represent a simplification of complicated biological processes. They may not be mutually exclusive, may overlap temporally, and likely exhibit synergies in causing manifest cardiovascular disease events. Many of the biological pathways are also known to exhibit mutual interactions (eg, inflammation with thrombosis/coagulation and with autonomic function). The pathways are also likely to be principally active at differing time points (eg, more rapid cardiovascular effects of autonomic imbalance than systemic inflammation) and likely vary in importance in relation to different durations of exposure and in causing different cardiovascular sequelae. The chemical characteristics and sizes of inhaled PM may also determine the pathways activated. As opposed to UFPs or some particulate components or chemicals, larger fine and coarse PM are not likely transported into the circulation to any large degree and therefore are more apt to require intermediary pathways to cause extrapulmonary effects. It may also be that surface-bound components may be delivered into the circulation, whereas larger particles themselves serve as a means to deliver the responsible constituent into the pulmonary tree.

The hyperacute physiological responses that occur minutes to hours after PM inhalation are likely mediated principally via pathways 2 and 3. These include ANS-mediated changes (eg, elevated BP, arrhythmias, and vasoconstriction), along with direct effects of circulating PM constituents on platelets (eg, procoagulant and thrombotic changes) and the endothelium (eg, oxidative stress and vasoconstriction). These responses are liable to be the dominant mechanisms responsible for the actual triggering of acute cardiovascular events. Clinically meaningful effects undoubtedly become manifest only in the context of a susceptible patient, typified by the individual with "vulnerable plaque" in the case of acute coronary syndromes or strokes, "vulnerable myocardium" in the context of arrhythmias, or the "vulnerable circulation" in the context of a heart failure patient at risk for circulatory overload. On the other hand, the biological consequences of systemic inflammation, such as activated white cells and elevated cytokines (via pathway 1), typically require longer periods. Their penultimate effect is the induction of a chronic underlying vulnerable milieu that leads to atherosclerotic plaque vulnerability, enhanced coagulation/thrombotic and arrhythmia potential, and impaired basal vasomotor balance. These actions thereby predispose individuals for future cardiovascular events, particularly when they occur in conjunction with traditional risk factors or prompt susceptibility to the acute biological actions (via pathways 2 and 3) of later air pollution exposures.

This hypothetical segregation of the biological effects of PM exposure as acute or chronic and into the broad pathways is artificial. It is useful in the broad context of understanding potential pathways; however, there is no doubt a large degree of overlap among the mechanisms and the timing of physiological responses. This is most aptly conveyed as the influence of "acute on chronic" actions of exposure. For example, the activation of circulating platelets by the pulmonary deposition of particles or lung inflammation (eg, by P-selectin–dependent pathways, histamine, or IL-6) could occur within hours and more rapidly than typical of the other consequences of inflammation (eg, progression of atherosclerosis). In the presence of a vulnerable or eroded coronary plaque due to long-term air pollution exposure, this sudden prothrombotic tendency could instigate an acute ischemic event (alone or in conjunction with other effects of short-term PM exposure via pathways 2 and 3). Furthermore, the epidemiological cohort studies demonstrate a larger relative risk for increased cardiovascular-related mortality than for morbidity.[72,73,227,274] If this is a true biological response and not simply a statistical phenomenon or a shortcoming of the available data, it not only suggests that exposures are capable of triggering acute cardiovascular events but that PM air pollution may also exaggerate their severity even if they would have otherwise occurred for reasons unrelated to air pollution. Therefore, exposure to PM could also be responsible for promoting fatal over nonfatal events.

Conclusions and Recommendations

A wide array of new studies that range from epidemiology to molecular and toxicological experiments have provided additional persuasive evidence that present-day levels of air pollutants contribute to cardiovascular morbidity and mortality. Although not unexpected given the numerous and heterogeneous nature of the published studies, all findings related to every single cardiovascular end point have not been consistent. However, the overall weight of scientific evidence now supports several new conclusions since the 2004 statement. These consensus points are given below by the AHA writing group after considering the strength, consistency, and coherence of the epidemiological findings, as well as in the context of evaluating the extent of the studies that provided related mechanistic support.

- The preponderance of findings indicate that short-term exposure to $PM_{2.5}$ over a period of a few hours to weeks can trigger CVD-related mortality and nonfatal events, including myocardial ischemia and MIs, heart failure, arrhythmias, and strokes.
- The increase in risk for acute $PM_{2.5}$-associated cardiovascular morbidity and mortality is principally among susceptible, but not necessarily critically ill, individuals. Several studies suggest that susceptible individuals at greater risk may include the elderly, patients with preexisting coronary artery disease, and perhaps those with diabetes. Recent data suggest that women and obese individuals might also be at higher risk.
- Most studies support the idea that longer-term $PM_{2.5}$ exposures increase the risk for cardiovascular mortality to an even greater extent than short-term exposures. Because most studies have focused on mortality data, the effect of long-term exposures on nonfatal cardiovascular events is less consistent and requires more investigation.
- The $PM_{2.5}$ concentration–cardiovascular risk relationships for both short- and long-term exposures appear to be monotonic, extending below 15 $\mu g/m^3$ (the 2006 annual NAAQS level) without a discernable "safe" threshold.
- Long-term exposure to elevated concentrations of ambient $PM_{2.5}$ at levels encountered in the present-day environment (ie, any increase by 10 $\mu g/m^3$) reduces life expectancy within a population probably by several months to a few years. Given that $PM_{2.5}$ is most strongly associated with cardiovascular deaths in the cohort studies, the reduced life expectancy is most likely predominantly due to excess cardiovascular mortality.
- The available studies are suggestive that reductions in PM levels decrease cardiovascular mortality within a time frame as short as a few years.
- Many potential biological mechanisms exist whereby PM exposure could exacerbate existing CVDs and trigger acute cardiovascular events (over the short term) and instigate or accelerate chronic CVDs (over the long run). Experimental support is increasingly strong for several mechanisms, which lends biological plausibility for the epidemiological findings.
- The existing evidence suggests that PM air pollution is capable of augmenting the development and progression of atherosclerosis. There is some support for a potential effect on several other chronic CVDs, including hypertension, heart failure, and diabetes.
- Most recent studies support the conclusion that the overall absolute risk for mortality due to PM exposure is greater for cardiovascular than pulmonary diseases after both short- and long-term exposures.

There are several additional areas worthy of highlighting in which the study results are reasonably consistent but in which the writing group believed further research was required to formulate firm conclusions.

- Although there is only limited epidemiological evidence directly linking UFPs with cardiovascular health problems,[262] the toxicological and experimental exposure evidence is suggestive that this size fraction may pose a particularly high risk to the cardiovascular system. The likelihood of health effects and the causal pathways mediated specifically by UFP exposure have been debated among experts recently.[418] Future research may help to more fully elucidate whether particles within the ultrafine size range (0.001 to 0.1 μm) and/or their constituents are more harmful to the cardiovascular system or pose a relatively greater cardiovascular risk than particles between 0.1 and 2.5 μm in diameter.
- Similarly, many studies have found a strong association between metrics of traffic-related air pollution exposure and elevated cardiovascular risk. Whether this represents the harmful effects of UFPs or diesel exhaust particulates, major components of the traffic mixture, or other pollution components is unclear. Diesel and UFPs possess toxic properties that instigate harmful biological responses in experimental models. However, the particle size fraction(s) and roles played by other copollutants (gases, VOCs, SVOCs) within the traffic-related mixture have not been fully elucidated. Nevertheless, traffic-related pollution as a whole appears to be a specific source associated with cardiovascular risk. It likely poses a major public health burden, regardless of a putative higher toxicity, because of the commonness of exposure in modern society (eg, accounting for ≈60% of daily UFP exposure; http://www.catf.us/projects/diesel/).
- The importance of other specific sources, regional differences in pollution composition, and other specific constituents remains less clear. However, toxicological studies have identified several transition metals (eg, iron, vanadium, nickel, copper, and zinc), organic carbon species, semiquinones, and endotoxin as specific PM-related components capable of prompting oxidative stress and inflammation and thus likely imparting biological harm. Some source-apportionment studies also demonstrate that attention should be given to these compounds as being among the most likely mediators of clinical CVD. More studies are required in this regard to clarify this issue and to better define these and other potentially responsible constituents and sources.
- Although the focus of the present statement is on PM, we recognize that other air pollutants may also pose cardiovascular risk alone or in conjunction with fine-particle exposure. In this context, we believe additional research is necessary to make firm conclusions regarding the independent cardiovascular risks posed by several gaseous pollutants (eg, ozone and NO_2). Although ozone has been linked to increased cardiopulmonary mortality,[50] strokes,[126] and MIs[419] in some short-term studies, long-term exposure was not associated with cardiovascular mortality after accounting for PM in a recent analysis.[87] The recent finding that small changes in low levels of ambient carbon monoxide concentrations are related to cardiovascular hospitalizations also merits further exploration.[420]
- Several secondary aerosols (eg, nitrate and sulfate) are often associated with cardiovascular mortality; however, whether these compounds are directly harmful or are surrogate markers of toxic sources of exposure requires

more investigation. Similarly, the current literature regarding the independent cardiovascular risks posed by coarse particles is mixed, with most recent findings not supporting an association after accounting for the effects of $PM_{2.5}$.[43,72,104]

- Several recent cohort studies and intermediate end-point experiments suggest that obese individuals (and/or those with the metabolic syndrome) may be a susceptible population at greater risk for cardiovascular events due to $PM_{2.5}$ exposure. This is a tremendously important public health issue to corroborate because of the enormous and growing prevalence of obesity worldwide.

This updated review by the AHA writing group corroborates and strengthens the conclusions of the initial scientific statement. In this context, we agree with the concept and continue to support measures based on scientific evidence, such as the US EPA NAAQS, that seek to control PM levels to protect the public health. Because the evidence reviewed supports that there is no safe threshold, it appears that public health benefits would accrue from lowering $PM_{2.5}$ concentrations even below present-day annual (15 $\mu g/m^3$) and 24-hour (35 $\mu g/m^3$) NAAQS, if feasible, to optimally protect the most susceptible populations. Evaluations of the effectiveness of such efforts would be warranted as well. Within the framework of attempting to establish causality between associated variables in epidemiological studies, there are several generally accepted "aspects" that have been evaluated (the following phrases in italics per the Bradford Hill criteria)[421]: With regard to cardiovascular mortality and $PM_{2.5}$ exposure, there is a *consistent association* that satisfies both a *temporal and exposure-response relationship*. There is *coherence of findings* among several fields of science, including toxicology, human and animal exposures, and different types of epidemiological studies and time frames of exposure. Rigorous experiments demonstrate multiple *plausible biological mechanisms*. Finally, natural experiments have confirmed that a change (ie, reduction) in exposure produces a change (ie, decrease) in cardiovascular mortality. In this case, *specificity of outcomes* and *strength of the observation* are less pertinent, because PM exposure could be capable of causing multiple different types of events (eg, MIs, arrhythmias, and heart failure exacerbations), and the overall cardiovascular mortality relative risk posed for any single individual is expected to be small. Nevertheless, given the ubiquity of exposure, the overall public health consequences can be substantial and observable in population- or large cohort–based studies.

It is the opinion of the writing group that the overall evidence is consistent with a causal relationship between $PM_{2.5}$ exposure and cardiovascular morbidity and mortality. This body of evidence has grown and has been strengthened substantially since publication of the first AHA scientific statement.[1] At present, no credible alternative explanation exists. These conclusions of our independent review are broadly similar to those found in the EPA's Integrated Science Assessment for Particulate Matter final report (http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=216546). In summary, the AHA writing group deems that $PM_{2.5}$ exposure is a "modifiable factor contributing to cardiovascular morbidity and mortality."

Clinical Recommendations

Several precautionary recommendations can be made for healthcare providers who interact with individuals who are at risk for CVDs. Although they have not been clinically tested or proven to reduce mortality, they are practical and feasible measures that may help to reduce exposures to air pollution and therefore potentially lower the associated cardiovascular risk. Moreover, a recent observational study found that patient awareness of air quality indices and media alerts along with health professional advice can significantly affect reported changes in outdoor activity to avoid exposure to air pollution.[422]

- Evidence-based appropriate treatment of the traditional cardiovascular risk factors should be emphasized. This may also lessen the susceptibility of patients to air pollution exposures.
- All patients with CVD should be educated about the cardiovascular risks posed by air pollution.
- Consideration should also be given to educating patients without CVD but who are at high risk (eg, the elderly, individuals with the metabolic syndrome or multiple risk factors, and those with diabetes).
- Part of patient education should include the provision of information regarding the available sources (local and national newspapers [*USA Today*], EPA World Wide Web site [http://airnow.gov/], and The Weather Channel and its World Wide Web site [http://www.weather.com/]) that provide a daily EPA Air Quality Index.
- On the basis of the forecast Air Quality Index, prudent recommendations for reducing exposure and limiting activity should be provided based on the patient's level of risk. A list of such recommendations is provided on the EPA World Wide Web site (http://airnow.gov/). For example, when the Air Quality Index for PM is "unhealthy" (151 to 200), then the recommendations are as follows: "People with heart or lung disease, older adults, and children should avoid prolonged or heavy exertion. Everyone else should reduce prolonged or heavy exertion." The action recommendations are as follows: "You can reduce your exposure to particles by 1) planning strenuous activity when particle levels are forecast to be lower, 2) reducing the amount of time spent at vigorous activity, or 3) choosing a less strenuous activity (eg, going for a walk instead of a jog). When particle levels are high outdoors, they also can be high indoors. Certain filters and room air cleaners are available that can help reduce particles indoors."
- Practical recommendations to reduce air pollution exposure should be given to at-risk patients. Although unproven to reduce cardiovascular events, there are a number of prudent and feasible measures, including reducing optional or unnecessary exposures. Additional measures could include eliminating or reducing nonmandatory travel to highly polluted regions and avoiding exposures or outdoor activities (eg, exercising, commut-

ing) during highly polluted times (eg, rush hours) or in proximity to major sources of pollution (eg, roadways, industrial sources). Choosing to exercise indoors with windows closed and using efficient air conditioning and filtering systems may be prudent for certain high-risk patients, particularly during peak pollution periods. Indeed, not only can central air conditioners reduce the indoor exposure level to PM from outdoor sources, there is some evidence that they might reduce the risk for cardiovascular hospitalizations associated with higher ambient pollution levels.[423] If travel/commutes cannot be avoided, maintaining optimal car filter systems, driving with windows closed, and recycling inside vehicle air may help reduce PM exposures (http://www.catf.us/projects/diesel/).[424,425]

However, at present, no specific recommendations regarding the appropriateness of undertaking more aggressive measures, even those shown to provide some benefits in a few studies (eg, wearing facemasks, installing PM filters in households), can be made based on the limited evidence. Similarly, although measures that decrease long-term PM exposures may produce even greater cardiovascular health benefits than the provided recommendations that focus on reducing short-term exposures, no specific recommendations (eg, moving to less polluted regions) can be prudently made at this time given the limited evidence. We acknowledge that occupational and indoor sources along with secondhand tobacco smoke are additional significant sources of personal PM exposures that should be avoided or reduced as much as possible. Finally, in developing nations, reducing exposure to indoor cooking sources of PM and air pollution from biomass combustion is a major issue of concern.[426] Additional suggestions are available on the EPA World Wide Web site.

Finally, although the existing evidence supports a causal relationship between $PM_{2.5}$ and cardiovascular mortality, we acknowledge the importance of continued research in areas of controversy and uncertainty to further understand the full nature of this issue. Although numerous insights have greatly enhanced our understanding of the PM-cardiovascular relationship since the first AHA statement was published,[1] the following list represents broad strategic avenues for future investigation:

Mechanistic Studies

- Better describe the physiological relevance in humans and the fundamental details of the mechanisms underlying the intermediate general mediating pathways (ie, PM or constituent transport into the circulation versus effects of inflammatory cytokines or activated immune cells versus ANS imbalance or other pathways) through which PM inhalation might mediate cardiovascular effects remote from the site of pulmonary deposition.
- Understand the clinical significance and relative importance of the observed biological responses (eg, vascular dysfunction, thrombosis, arrhythmia, ANS imbalance) in relation to the various causes of PM-mediated cardiovascular morbidity and mortality.
- Examine the efficacy of preventive measures (eg, patient education) and treatment modalities (eg, statins, antioxidants, fish oil, treatment of traditional risk factors, and reducing exposures by engineering controls, including filtration, personal protection via facemasks, or behavior modification) on cardiovascular health outcomes.
- Investigate the interaction between preexisting traditional cardiovascular risk factors (eg, diabetes, hypertension) and PM exposure, as well as the potential of air pollutants to exacerbate or worsen these risk factors. Determine the extent to which treatment of such factors (eg, with statins, aspirin, or angiotensin-converting enzyme inhibitors), especially among patients with known CVD, may modify the risk associated with PM exposure.
- Describe the biological effects of acute on top of chronic exposures (eg, synergistic effects versus reduced susceptibility to acute exposures due to augmented protective mechanisms).
- Determine the ability of long-term exposure to precipitate the development of chronic diseases, including clinically relevant atherosclerosis, hypertension, diabetes, and other vascular, metabolic, renal, or neurological diseases.

Epidemiological and Exposure Studies

- Expand our knowledge related to the "responsible" PM pollution constituents (eg, metals, organic compounds, semiquinones, endotoxin, and VOC and SVOC compounds), size fractions (eg, UFPs), sources (eg, traffic, power generation, and biomass combustion), and mixtures of pollutants.
- Investigate the cardiovascular health implications and importance of regional and intracity differences in composition and combinations of pollutants.
- Better understand the effects of mixtures of ambient pollutants (ie, potential synergism between PM and gaseous or vapor-phase pollutants such as ozone).
- Investigate the feasibility and utility of quantifying risk coefficients (concentration-response functions) according to PM source or relevant indices of pollutant mixtures, as a function of susceptibility (eg, age, preexisting disease), for reliable application in integrated, multipollutant risk assessments.
- Investigate the relative importance of various time frames of exposure in relation to PM causing cardiovascular events, including the relevance of epochs not well described, such as ultra-acute peak PM excursions (eg, 1 to 2 hours) and exposures of intermediate duration (eg, 1 to 12 months).
- Better document the time course and specific cardiovascular health benefits induced by reductions in PM.
- Better define susceptible individuals or vulnerable populations.
- Determine whether any "safe" PM threshold concentration exists that eliminates both acute and chronic cardiovascular effects in healthy and susceptible individuals and at a population level.

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Jeffrey R. Brook	University of Toronto, Environment Canada	None	None	None	None	None	None	None
Ana V. Diez-Roux	University of Michigan	EPA†; 1st EPA STAR grant to study the effects of long-term PM exposures on subclinical atherosclerosis and inflammatory markers in MESA; #2 is a subcontract to the University of Washington to participate in a long-term study of air pollution and progression of atherosclerosis, also in MESA	None	None	None	None	None	None
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Yuling Hong	American Heart Association‡	None	None	None	None	None	None	None
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Sanjay Rajagopalan	Ohio State University	None	None	Takeda*	None	None	None	None
David Siscovick	University of Washington	MESA AIR (ancillary study to MESA) funded by EPA†; NIEHS Discovery Center Study focused on air pollution and CVD†; NIH†	None	None	None	None	None	None
Sidney C. Smith, Jr	University of North Carolina	None	None	None	None	None	None	None
Laurie Whitsel	American Heart Association	None	None	None	None	None	None	None

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (1) the person receives $10 000 or more during any 12-month period, or 5% or more of the person's gross income; or (2) the person owns 5% or more of the voting stock or share of the entity, or owns $10 000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

†Significant.

‡Dr Hong is currently with the Centers for Disease Control and Prevention, Atlanta, Ga.

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Doug Dockery	Harvard University	National Institute of Environmental Health Sciences†; Health Effects Institute†	None	None	None	None	Science Advisory Board to MESA Air Study, University of Washington*	None
Mark Frampton	University of Rochester	National Institutes of Health†; American Petroleum Institute†; US EPA†	None	None	None	None	Health Effects Institute*	None
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*Modest.

†Significant.

References

1. Brook RD, Franklin B, Cascio W, Hong Y, Howard G, Lipsett M, Luepker R, Mittleman M, Samet J, Smith SC Jr, Tager I; Expert Panel on Population and Prevention Science of the American Heart Association. Air pollution and cardiovascular disease: a statement for healthcare professionals from the Expert Panel on Population and Prevention Science of the American Heart Association. *Circulation*. 2004;109:2655–2671.
2. Brook RD. Cardiovascular effects of air pollution. *Clin Sci (Lond)*. 2008;115:175–187.
3. Pope CA 3rd, Dockery DW. Health effects of fine particulate air pollution: lines that connect. *J Air Waste Manag Assoc*. 2006;56:709–742.
4. Simkhovich BZ, Kleinman MT, Kloner RA. Air pollution and cardiovascular injury epidemiology, toxicology, and mechanisms. *J Am Coll Cardiol*. 2008;52:719–726.
5. US Environmental Protection Agency. Air Quality Criteria for Particulate Matter (October 2004). Available at: http://www.epa.gov/ttn/naaqs/standards/pm/s_pm_index.html. Accessed March 26, 2010.
6. US Environmental Protection Agency. National Ambient Air Quality Criteria Standards. Available at: http://www.epa.gov/air/criteria.html. Accessed September 1, 2008.
7. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, McQueen M, Budaj A, Pais P, Varigos J, Lisheng L; INTERHEART Study Investigators. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *Lancet*. 2004;364:937–952.
8. Lloyd-Jones DM, Leip EP, Larson MG, D'Agostino RB, Beiser A, Wilson PW, Wolf PA, Levy D. Prediction of lifetime risk for cardiovascular disease by risk factor burden at 50 years of age. *Circulation*. 2006;113:791–798.
9. Wang TJ, Gona P, Larson MG, Tofler GH, Levy D, Newton-Cheh C, Jacques PF, Rifai N, Selhub J, Robins SJ, Benjamin EJ, D'Agostino RB, Vasan RS. Multiple biomarkers for the prediction of first major cardiovascular events and death. *N Engl J Med*. 2006;355:2631–2639.
10. Cook NR. Use and misuse of the receiver operating characteristic curve in risk prediction. *Circulation*. 2007;115:928–935.
11. Stern RH. Evaluating new cardiovascular risk factors for risk stratification. *J Clin Hypertens (Greenwich)*. 2008;10:485–488.
12. Tofler GH, Muller JE. Triggering of acute cardiovascular disease and potential preventive strategies. *Circulation*. 2006;114:1863–1872.
13. Pope CA 3rd, Muhlestein JB, May HT, Renlund DG, Anderson JL, Horne BD. Ischemic heart disease events triggered by short-term exposure to fine particulate air pollution. *Circulation*. 2006;114:2443–2448.
14. Kung H-C, Hoyert DL, Xu J, Murphy SL. Deaths: Final Data for 2005. *National Vital Statistics Reports*. 2005;561–121.
15. World Health Organization. *World Health Report 2002*. Geneva, Switzerland: World Health Organization; 2002.
16. Geddes JA, Murphy JG, Wang DK. Long term changes in nitrogen oxides and volatile organic compounds in Toronto and the challenges facing local ozone control. *Atmos Environ*. 2009;43:3407–3415.
17. Delfino RJ, Sioutas C, Malik S. Potential role of ultrafine particles in associations between airborne particle mass and cardiovascular health. *Environ Health Perspect*. 2005;113:934–946.
18. Wexler AS, Johnston MV. What have we learned from highly time-resolved measurements during EPA's Supersites Program and related studies? *J Air Waste Manag Assoc*. 2008;58:303–319.
19. Solomon PA, Hopke PK, Froines J, Scheffe R. Key scientific findings and policy- and health-relevant insights from the U.S. Environmental Protection Agency's Particulate Matter Supersites Program and related studies: an integration and synthesis of results. *J Air Waste Manag Assoc*. 2008;58(suppl):S3–S92.
20. Hopke PK. New directions: reactive particles as a source of human health effects. *Atmos Environ*. 2008;42:3192–3194.
21. Bureau of Transportation Statistics. *Highlights of the 2001 National Household Travel Survey*. Washington, DC: US Department of Transportation; 2003.

22. Sarnat JA, Wilson WE, Strand M, Brook J, Wyzga R, Lumley T. Panel discussion review: session 1: exposure assessment and related errors in air pollution epidemiologic studies. *J Expo Sci Environ Epidemiol.* 2007;17(suppl):S75–S82.
23. Monn C. Exposure assessment of air pollutants: a review on spatial heterogeneity and indoor/outdoor/personal exposure to suspended particulate matter, nitrogen dioxide and ozone. *Atmos Environ.* 2001;35:1–32.
24. Brauer M, Hoek G, van Vliet P, Meliefste K, Fischer P, Gehring U, Heinrich J, Cyrys J, Bellander T, Lewne M, Brunekreef B. Estimating long-term average particulate air pollution concentrations: application of traffic indicators and geographic information systems. *Epidemiology.* 2003;14:228–239.
25. Ebelt ST, Wilson WE, Brauer M. Exposure to ambient and nonambient components of particulate matter: a comparison of health effects. *Epidemiology.* 2005;16:396–405.
26. Molitor J, Jerrett M, Chang CC, Molitor NT, Gauderman J, Berhane K, McConnell R, Lurmann F, Wu J, Winer A, Thomas D. Assessing uncertainty in spatial exposure models for air pollution health effects assessment. *Environ Health Perspect.* 2007;115:1147–1153.
27. Anderson HR, Atkinson RW, Peacock JL, Sweeting MJ, Marston L. Ambient particulate matter and health effects: publication bias in studies of short-term associations. *Epidemiology.* 2005;16:155–163.
28. Pope CA 3rd. Epidemiology of fine particulate air pollution and human health: biologic mechanisms and who's at risk? *Environ Health Perspect.* 2000;108(suppl):713–723.
29. Dominici F. Time-series analysis of air pollution and mortality: a statistical review. *Res Rep Health Eff Inst.* 2004:3–27.
30. Dominici F, Burnett RT. Risk models for particulate air pollution. *J Toxicol Environ Health A.* 2003;66:1883–1889.
31. Committee on the Medical Effects of Air Pollutants. *Cardiovascular Disease and Air Pollution: A Report by the Committee on the Medical Effects of Air Pollutant's Cardiovascular Sub-Group.* London, UK: Department of Health, National Health Service; 2006.
32. Levy JI, Hammitt JK, Spengler JD. Estimating the mortality impacts of particulate matter: what can be learned from between-study variability? *Environ Health Perspect.* 2000;108:109–117.
33. Stieb DM, Judek S, Burnett RT. Meta-analysis of time-series studies of air pollution and mortality: update in relation to the use of generalized additive models. *J Air Waste Manag Assoc.* 2003;53:258–261.
34. Dominici F, Daniels M, McDermott A, Zeger SL, Samet J. Shape of the exposure-response relation and mortality displacement in the NMMAPS database. In: *Revised Analyses of Time-Series of Air Pollution and Health. Special Report.* Boston, Mass: Health Effects Institute; 2003.
35. Katsouyanni K, Touloumi G, Samolu E, Petasakis Y, Analitis A, Le Tertre A, Rossi G, Zmirou D, Ballester F, Boumghar A, Anderson HR, Wojtyniak B, Paldy A, Braustein R, Pekkanen J, Schindler C, Schwartz J. Sensitivity analysis of various models of short-term effects of ambient particles on total mortality in 29 cities in APHEA2. In: *Revised Analyses of Time-Series of Air Pollution and Health.* Boston, Mass: Health Effects Institute; 2003.
36. Analitis A, Katsouyanni K, Dimakopoulou K, Samoli E, Nikoloulopoulos AK, Petasakis Y, Touloumi G, Schwartz J, Anderson HR, Cambra K, Forastiere F, Zmirou D, Vonk JM, Clancy L, Kriz B, Bobvos J, Pekkanen J. Short-term effects of ambient particles on cardiovascular and respiratory mortality. *Epidemiology.* 2006;17:230–233.
37. Klemm RJ, Mason R. Replication of reanalysis of Harvard six-city mortality study. In: *Revised Analyses of Time-Series of Air Pollution and Health.* Boston, Mass: Health Effects Institute; 2003.
38. Franklin M, Zeka A, Schwartz J. Association between PM2.5 and all-cause and specific-cause mortality in 27 US communities. *J Expo Sci Environ Epidemiol.* 2007;17:279–287.
39. Ostro B, Broadwin R, Green S, Feng WY, Lipsett M. Fine particulate air pollution and mortality in nine California counties: results from CALFINE. *Environ Health Perspect.* 2006;114:29–33.
40. Le Tertre A, Medina S, Samoli E, Forsberg B, Michelozzi P, Boumghar A, Vonk JM, Bellini A, Atkinson R, Ayres JG, Sunyer J, Schwartz J, Katsouyanni K. Short-term effects of particulate air pollution on cardiovascular diseases in eight European cities. *J Epidemiol Community Health.* 2002;56:773–779.
41. Omori T, Fujimoto G, Yoshimura I, Nitta H, Ono M. Effects of particulate matter on daily mortality in 13 Japanese cities. *J Epidemiol.* 2003;13:314–322.
42. Wong CM, Vichit-Vadakan N, Kan H, Qian Z. Public Health and Air Pollution in Asia (PAPA): a multicity study of short-term effects of air pollution on mortality. *Environ Health Perspect.* 2008;116:1195–1202.
43. Zanobetti A, Schwartz J. The effect of fine and coarse particulate air pollution on mortality: a national analysis. *Environ Health Perspect.* 2009;117:898–903.
44. Samet JM, Dominici F, Curriero FC, Coursac I, Zeger SL. Fine particulate air pollution and mortality in 20 U.S. cities, 1987–1994. *N Engl J Med.* 2000;343:1742–1749.
45. Dominici F, Zeger SL, Samet JM. A measurement error model for time-series studies of air pollution and mortality. *Biostatistics.* 2000;1:157–175.
46. Dominici F, McDermott A, Zeger SL, Samet JM. National maps of the effects of particulate matter on mortality: exploring geographical variation. *Environ Health Perspect.* 2003;111:39–44.
47. Peng RD, Dominici F, Pastor-Barriuso R, Zeger SL, Samet JM. Seasonal analyses of air pollution and mortality in 100 US cities. *Am J Epidemiol.* 2005;161:585–594.
48. Dominici F, Peng RD, Ebisu K, Zeger SL, Samet JM, Bell ML. Does the effect of PM10 on mortality depend on PM nickel and vanadium content? A reanalysis of the NMMAPS data. *Environ Health Perspect.* 2007;115:1701–1703.
49. Bell ML, Kim JY, Dominici F. Potential confounding of particulate matter on the short-term association between ozone and mortality in multisite time-series studies. *Environ Health Perspect.* 2007;115:1591–1595.
50. Bell ML, McDermott A, Zeger SL, Samet JM, Dominici F. Ozone and short-term mortality in 95 US urban communities, 1987–2000. *JAMA.* 2004;292:2372–2378.
51. Franklin M, Schwartz J. The impact of secondary particles on the association between ambient ozone and mortality. *Environ Health Perspect.* 2008;116:453–458.
52. Katsouyanni K. Ambient air pollution and health. *Br Med Bull.* 2003;68:143–156.
53. Samoli E, Aga E, Touloumi G, Nisiotis K, Forsberg B, Lefranc A, Pekkanen J, Wojtyniak B, Schindler C, Niciu E, Brunstein R, Dodic Fikfak M, Schwartz J, Katsouyanni K. Short-term effects of nitrogen dioxide on mortality: an analysis within the APHEA project. *Eur Respir J.* 2006;27:1129–1138.
54. Samoli E, Touloumi G, Schwartz J, Anderson HR, Schindler C, Forsberg B, Vigotti MA, Vonk J, Kosnik M, Skorkovsky J, Katsouyanni K. Short-term effects of carbon monoxide on mortality: an analysis within the APHEA project. *Environ Health Perspect.* 2007;115:1578–1583.
55. Wong CM, Ou CQ, Chan KP, Chau YK, Thach TQ, Yang L, Chung RY, Thomas GN, Peiris JS, Wong TW, Hedley AJ, Lam TH. The effects of air pollution on mortality in socially deprived urban areas in Hong Kong, China. *Environ Health Perspect.* 2008;116:1189–1194.
56. Kan H, Jia J, Chen B. Acute stroke mortality and air pollution: new evidence from Shanghai, China. *J Occup Health.* 2003;45:321–323.
57. Qian Z, He Q, Lin HM, Kong L, Bentley CM, Liu W, Zhou D. High temperatures enhanced acute mortality effects of ambient particle pollution in the "oven" city of Wuhan, China. *Environ Health Perspect.* 2008;116:1172–1178.
58. Klemm RJ, Lipfert FW, Wyzga RE, Gust C. Daily mortality and air pollution in Atlanta: two years of data from ARIES. *Inhal Toxicol.* 2004;16(suppl):131–141.
59. Klemm RJ, Mason RM Jr, Heilig CM, Neas LM, Dockery DW. Is daily mortality associated specifically with fine particles? Data reconstruction and replication of analyses. *J Air Waste Manag Assoc.* 2000;50:1215–1222.
60. Laden F, Neas LM, Dockery DW, Schwartz J. Association of fine particulate matter from different sources with daily mortality in six U.S. cities. *Environ Health Perspect.* 2000;108:941–947.
61. Samoli E, Peng R, Ramsay T, Pipikou M, Touloumi G, Dominici F, Burnett R, Cohen A, Krewski D, Samet J, Katsouyanni K. Acute effects of ambient particulate matter on mortality in Europe and North America: results from the APHENA study. *Environ Health Perspect.* 2008;116:1480–1486.
62. Dockery DW, Pope CA 3rd, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG Jr, Speizer FE. An association between air pollution and mortality in six U.S. cities. *N Engl J Med.* 1993;329:1753–1759.
63. Krewski D, Burnett RT, Goldberg MS, Hoover K, Siemiatycki J, Abrahamowicz M, White WH. Validation of the Harvard Six Cities Study of particulate air pollution and mortality. *N Engl J Med.* 2004;350:198–199.
64. Laden F, Schwartz J, Speizer FE, Dockery DW. Reduction in fine particulate air pollution and mortality: Extended follow-up of the Harvard Six Cities study. *Am J Respir Crit Care Med.* 2006;173:667–672.
65. Eftim SE, Samet JM, Janes H, McDermott A, Dominici F. Fine particulate matter and mortality: a comparison of the six cities and American Cancer Society cohorts with a Medicare cohort. *Epidemiology.* 2008;19:209–216.

66. Pope CA 3rd, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE, Heath CW Jr. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. *Am J Respir Crit Care Med*. 1995;151:669–674.
67. Pope CA 3rd, Burnett RT, Thurston GD, Thun MJ, Calle EE, Krewski D, Godleski JJ. Cardiovascular mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease. *Circulation*. 2004;109:71–77.
68. Pope CA 3rd, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *JAMA*. 2002;287:1132–1141.
69. Jerrett M, Burnett RT, Ma R, Pope CA 3rd, Krewski D, Newbold KB, Thurston G, Shi Y, Finkelstein N, Calle EE, Thun MJ. Spatial analysis of air pollution and mortality in Los Angeles. *Epidemiology*. 2005;16:727–736.
70. Krewski D, Jerrett M, Burnett RT, Ma R, Hughes E, Shi Y, Turner MC, Pope CA 3rd, Thurston G, Calle EE, Thun MJ. *Extended Follow-Up and Spatial Analysis of the American Cancer Society Study Linking Particulate Air Pollution and Mortality: Special Report*. Cambridge, MA: Health Effects Institute; 2009.
71. Zeger SL, Dominici F, McDermott A, Samet JM. Mortality in the Medicare population and chronic exposure to fine particulate air pollution in urban centers (2000–2005). *Environ Health Perspect*. 2008;116:1614–1619.
72. Miller KA, Siscovick DS, Sheppard L, Shepherd K, Sullivan JH, Anderson GL, Kaufman JD. Long-term exposure to air pollution and incidence of cardiovascular events in women. *N Engl J Med*. 2007;356:447–458.
73. Puett RC, Schwartz J, Hart JE, Yanosky JD, Speizer FE, Suh H, Paciorek CJ, Neas LM, Laden F. Chronic particulate exposure, mortality, and coronary heart disease in the nurses' health study. *Am J Epidemiol*. 2008;168:1161–1168.
74. McDonnell WF, Nishino-Ishikawa N, Petersen FF, Chen LH, Abbey DE. Relationships of mortality with the fine and coarse fractions of long-term ambient PM10 concentrations in nonsmokers. *J Expo Anal Environ Epidemiol*. 2000;10:427–436.
75. Chen LH, Knutsen SF, Shavlik D, Beeson WL, Petersen F, Ghamsary M, Abbey D. The association between fatal coronary heart disease and ambient particulate air pollution: are females at greater risk? *Environ Health Perspect*. 2005;113:1723–1729.
76. Lipfert FW RW, JD Baty JD, JP Miller. Traffic density as a surrogate measure of environmental exposures in studies of air pollution health effects: long-term mortality in a cohort of US veterans. *Atmos Environ*. 2006;40:154–169.
77. Lipfert FW, Baty JD, Miller JP, Wyzga RE. PM2.5 constituents and related air quality variables as predictors of survival in a cohort of U.S. military veterans. *Inhal Toxicol*. 2006;18:645–657.
78. Enstrom JE. Fine particulate air pollution and total mortality among elderly Californians, 1973–2002. *Inhal Toxicol*. 2005;17:803–816.
79. Filleul L, Rondeau V, Vandentorren S, Le Moual N, Cantagrel A, Annesi-Maesano I, Charpin D, Declercq C, Neukirch F, Paris C, Vervloet D, Brochard P, Tessier JF, Kauffmann F, Baldi I. Twenty five year mortality and air pollution: results from the French PAARC survey. *Occup Environ Med*. 2005;62:453–460.
80. Gehring U, Heinrich J, Krämer U, Grote V, Hochadel M, Sugiri D, Kraft M, Rauchfuss K, Eberwein HG, Wichmann HE. Long-term exposure to ambient air pollution and cardiopulmonary mortality in women. *Epidemiology*. 2006;17:545–551.
81. Naess Ø, Nafstad P, Aamodt G, Claussen B, Rosland P. Relation between concentration of air pollution and cause-specific mortality: four-year exposures to nitrogen dioxide and particulate matter pollutants in 470 neighborhoods in Oslo, Norway. *Am J Epidemiol*. 2007;165:435–443.
82. Beelen R, Hoek G, van den Brandt PA, Goldbohm RA, Fischer P, Schouten LJ, Jerrett M, Hughes E, Armstrong B, Brunekreef B. Long-term effects of traffic-related air pollution on mortality in a Dutch cohort (NLCS-AIR study). *Environ Health Perspect*. 2008;116:196–202.
83. Elliott P, Shaddick G, Wakefield JC, de Hoogh C, Briggs DJ. Long-term associations of outdoor air pollution with mortality in Great Britain. *Thorax*. 2007;62:1088–1094.
84. Schwartz J, Coull B, Laden F, Ryan L. The effect of dose and timing of dose on the association between airborne particles and survival. *Environ Health Perspect*. 2008;116:64–69.
85. Jerrett M, Burnett RT, Brook J, Kanaroglou P, Giovis C, Finkelstein N, Hutchison B. Do socioeconomic characteristics modify the short term association between air pollution and mortality? Evidence from a zonal time series in Hamilton, Canada. *J Epidemiol Community Health*. 2004;58:31–40.
86. Jerrett M, Burnett RT, Willis A, Krewski D, Goldberg MS, DeLuca P, Finkelstein N. Spatial analysis of the air pollution-mortality relationship in the context of ecologic confounders. *J Toxicol Environ Health A*. 2003;66:1735–1777.
87. Jerrett M, Burnett RT, Pope CA 3rd, Ito K, Thurston G, Krewski D, Shi Y, Calle E, Thun M. Long-term ozone exposure and mortality. *N Engl J Med*. 2009;360:1085–1095.
88. Abbey DE, Nishino N, McDonnell WF, Burchette RJ, Knutsen SF, Lawrence Beeson W, Yang JX. Long-term inhalable particles and other air pollutants related to mortality in nonsmokers. *Am J Respir Crit Care Med*. 1999;159:373–382.
89. Pope CA 3rd. Particulate pollution and health: a review of the Utah valley experience. *J Expo Anal Environ Epidemiol*. 1996;6:23–34.
90. Clancy L, Goodman P, Sinclair H, Dockery DW. Effect of air-pollution control on death rates in Dublin, Ireland: an intervention study. *Lancet*. 2002;360:1210–1214.
91. Hedley AJ, Wong CM, Thach TQ, Ma S, Lam TH, Anderson HR. Cardiorespiratory and all-cause mortality after restrictions on sulphur content of fuel in Hong Kong: an intervention study. *Lancet*. 2002;360:1646–1652.
92. Dominici F, Peng RD, Zeger SL, White RH, Samet JM. Particulate air pollution and mortality in the United States: did the risks change from 1987 to 2000? *Am J Epidemiol*. 2007;166:880–888.
93. Pope CA 3rd, Rodermund DL, Gee MM. Mortality effects of a copper smelter strike and reduced ambient sulfate particulate matter air pollution. *Environ Health Perspect*. 2007;115:679–683.
94. Janes H, Dominici F, Zeger SL. Trends in air pollution and mortality: an approach to the assessment of unmeasured confounding. *Epidemiology*. 2007;18:416–423.
95. Pope CA 3rd, Ezzati M, Dockery DW. Fine-particulate air pollution and life expectancy in the United States. *N Engl J Med*. 2009;360:376–386.
96. Schwartz J. Air pollution and hospital admissions for heart disease in eight U.S. counties. *Epidemiology*. 1999;10:17–22.
97. Zanobetti A, Schwartz J, Dockery DW. Airborne particles are a risk factor for hospital admissions for heart and lung disease. *Environ Health Perspect*. 2000;108:1071–1077.
98. Samet JM, Zeger SL, Dominici F, Curriero F, Coursac I, Dockery DW, Schwartz J, Zanobetti A. The National Morbidity, Mortality, and Air Pollution Study. Part II: Morbidity and mortality from air pollution in the United States. *Res Rep Health Eff Inst*. 2000;94:5–70.
99. Schwartz J, Zanobetti A, Bateson T. Morbidity and mortality among elderly residents of cities with daily PM measurements. In: *Revised Analyses of Time-Series Studies of Air Pollution and Health*. Boston, Mass: Health Effects Institute; 2003.
100. Ballester F, Rodríguez P, Iñíguez C, Saez M, Daponte A, Galán I, Taracido M, Arribas F, Bellido J, Cirarda FB, Cañada A, Guillén JJ, Guillén-Grima F, López E, Pérez-Hoyos S, Lertxundi A, Toro S. Air pollution and cardiovascular admissions association in Spain: results within the EMECAS project. *J Epidemiol Community Health*. 2006;60:328–336.
101. Larrieu S, Jusot JF, Blanchard M, Prouvost H, Declercq C, Fabre P, Pascal L, Tertre AL, Wagner V, Rivière S, Chardon B, Borrelli D, Cassadou S, Eilstein D, Lefranc A. Short term effects of air pollution on hospitalizations for cardiovascular diseases in eight French cities: the PSAS program. *Sci Total Environ*. 2007;387:105–112.
102. Bell ML, Ebisu K, Peng RD, Walker J, Samet JM, Zeger SL, Dominici F. Seasonal and regional short-term effects of fine particles on hospital admissions in 202 US counties, 1999–2005. *Am J Epidemiol*. 2008;168:1301–1310.
103. Dominici F, Peng RD, Bell ML, Pham L, McDermott A, Zeger SL, Samet JM. Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. *JAMA*. 2006;295:1127–1134.
104. Peng RD, Chang HH, Bell ML, McDermott A, Zeger SL, Samet JM, Dominici F. Coarse particulate matter air pollution and hospital admissions for cardiovascular and respiratory diseases among Medicare patients. *JAMA*. 2008;299:2172–2179.
105. Sunyer J, Ballester F, Tertre AL, Atkinson R, Ayres JG, Forastiere F, Forsberg B, Vonk JM, Bisanti L, Tenías JM, Medina S, Schwartz J, Katsouyanni K. The association of daily sulfur dioxide air pollution levels with hospital admissions for cardiovascular diseases in Europe (The Aphea-II study). *Eur Heart J*. 2003;24:752–760.
106. Krewski D. Evaluating the effects of ambient air pollution on life expectancy. *N Engl J Med*. 2009;360:413–415.

107. Zanobetti A, Schwartz J. Particulate air pollution, progression, and survival after myocardial infarction. *Environ Health Perspect*. 2007; 115:769–775.
108. Tonne C, Melly S, Mittleman M, Coull B, Goldberg R, Schwartz J. A case-control analysis of exposure to traffic and acute myocardial infarction. *Environ Health Perspect*. 2007;115:53–57.
109. Murakami Y, Ono M. Myocardial infarction deaths after high level exposure to particulate matter. *J Epidemiol Community Health*. 2006; 60:262–266.
110. Peters A, Dockery DW, Muller JE, Mittleman MA. Increased particulate air pollution and the triggering of myocardial infarction. *Circulation*. 2001;103:2810–2815.
111. Peters A, von Klot S, Heier M, Trentinaglia I, Hörmann A, Wichmann HE, Löwel H; Cooperative Health Research in the Region of Augsburg Study Group. Exposure to traffic and the onset of myocardial infarction. *N Engl J Med*. 2004;351:1721–1730.
112. D'Ippoliti D, Forastiere F, Ancona C, Agabiti N, Fusco D, Michelozzi P, Perucci CA. Air pollution and myocardial infarction in Rome: a case-crossover analysis. *Epidemiology*. 2003;14:528–535.
113. Zanobetti A, Schwartz J. The effect of particulate air pollution on emergency admissions for myocardial infarction: a multicity case-crossover analysis. *Environ Health Perspect*. 2005;113:978–982.
114. von Klot S, Peters A, Aalto P, Bellander T, Berglind N, D'Ippoliti D, Elosua R, Hörmann A, Kulmala M, Lanki T, Löwel H, Pekkanen J, Picciotto S, Sunyer J, Forastiere F; Health Effects of Particles on Susceptible Subpopulations (HEAPSS) Study Group. Ambient air pollution is associated with increased risk of hospital cardiac readmissions of myocardial infarction survivors in five European cities. *Circulation*. 2005;112:3073–3079.
115. Sullivan J, Sheppard L, Schreuder A, Ishikawa N, Siscovick D, Kaufman J. Relation between short-term fine-particulate matter exposure and onset of myocardial infarction. *Epidemiology*. 2005;16:41–48.
116. Peters A, von Klot S, Heier M, Trentinaglia I, Cyrys J, Hörmann A, Hauptmann M, Wichmann HE, Löwel H. Air pollution, personal activities, and onset of myocardial infarction in a case-crossover study, Part I. In: *Particulate Air Pollution and Nonfatal Cardiac Events*. Boston, Mass: Health Effects Institute; 2005:124.
117. Wellenius GA, Schwartz J, Mittleman MA. Particulate air pollution and hospital admissions for congestive heart failure in seven United States cities. *Am J Cardiol*. 2006;97:404–408.
118. Medina-Ramón M, Goldberg R, Melly S, Mittleman MA, Schwartz J. Residential exposure to traffic-related air pollution and survival after heart failure. *Environ Health Perspect*. 2008;116:481–485.
119. Pope CA III, Renlund DG, Kfoury AG, May HT, Horne BD. Relation of heart failure hospitalization to exposure to fine particulate air pollution. *Am J Cardiol*. 2008;102:1230–1234.
120. Hong YC, Lee JT, Kim H, Ha EH, Schwartz J, Christiani DC. Effects of air pollutants on acute stroke mortality. *Environ Health Perspect*. 2002; 110:187–191.
121. Hong YC, Lee JT, Kim H, Kwon HJ. Air pollution: a new risk factor in ischemic stroke mortality. *Stroke*. 2002;33:2165–2169.
122. Kettunen J, Lanki T, Tiittanen P, Aalto PP, Koskentalo T, Kulmala M, Salomaa V, Pekkanen J. Associations of fine and ultrafine particulate air pollution with stroke mortality in an area of low air pollution levels. *Stroke*. 2007;38:918–922.
123. Chan CC, Chuang KJ, Chien LC, Chen WJ, Chang WT. Urban air pollution and emergency admissions for cerebrovascular diseases in Taipei, Taiwan. *Eur Heart J*. 2006;27:1238–1244.
124. Tsai SS, Goggins WB, Chiu HF, Yang CY. Evidence for an association between air pollution and daily stroke admissions in Kaohsiung, Taiwan. *Stroke*. 2003;34:2612–2616.
125. Wellenius GA, Schwartz J, Mittleman MA. Air pollution and hospital admissions for ischemic and hemorrhagic stroke among Medicare beneficiaries. *Stroke*. 2005;36:2549–2553.
126. Henrotin JB, Besancenot JP, Bejot Y, Giroud M. Short-term effects of ozone air pollution on ischaemic stroke occurrence: a case-crossover analysis from a 10-year population-based study in Dijon, France. *Occup Environ Med*. 2007;64:439–445.
127. Lisabeth LD, Escobar JD, Dvonch JT, Sánchez BN, Majersik JJ, Brown DL, Smith MA, Morgenstern LB. Ambient air pollution and risk for ischemic stroke and transient ischemic attack. *Ann Neurol*. 2008;64:53–59.
128. Baccarelli A, Martinelli I, Zanobetti A, Grillo P, Hou LF, Bertazzi PA, Mannucci PM, Schwartz J. Exposure to particulate air pollution and risk of deep vein thrombosis. *Arch Intern Med*. 2008;168:920–927.
129. Peters A, Liu E, Verrier RL, Schwartz J, Gold DR, Mittleman M, Baliff J, Oh JA, Allen G, Monahan K, Dockery DW. Air pollution and incidence of cardiac arrhythmia. *Epidemiology*. 2000;11:11–17.
130. Dockery DW, Luttmann-Gibson H, Rich DQ, Link MS, Mittleman MA, Gold DR, Koutrakis P, Schwartz JD, Verrier RL. Association of air pollution with increased incidence of ventricular tachyarrhythmias recorded by implanted cardioverter defibrillators. *Environ Health Perspect*. 2005;113:670–674.
131. Rich DQ, Schwartz J, Mittleman MA, Link M, Luttmann-Gibson H, Catalano PJ, Speizer FE, Dockery DW. Association of short-term ambient air pollution concentrations and ventricular arrhythmias. *Am J Epidemiol*. 2005;161:1123–1132.
132. Berger A, Zareba W, Schneider A, Rückerl R, Ibald-Mulli A, Cyrys J, Wichmann HE, Peters A. Runs of ventricular and supraventricular tachycardia triggered by air pollution in patients with coronary heart disease. *J Occup Environ Med*. 2006;48:1149–1158.
133. Rich DQ, Kim MH, Turner JR, Mittleman MA, Schwartz J, Catalano PJ, Dockery DW. Association of ventricular arrhythmias detected by implantable cardioverter defibrillator and ambient air pollutants in the St Louis, Missouri metropolitan area. *Occup Environ Med*. 2006;63:591–596.
134. Rich DQ, Mittleman MA, Link MS, Schwartz J, Luttmann-Gibson H, Catalano PJ, Speizer FE, Gold DR, Dockery DW. Increased risk of paroxysmal atrial fibrillation episodes associated with acute increases in ambient air pollution. *Environ Health Perspect*. 2006;114:120–123.
135. Sarnat SE, Suh HH, Coull BA, Schwartz J, Stone PH, Gold DR. Ambient particulate air pollution and cardiac arrhythmia in a panel of older adults in Steubenville, Ohio. *Occup Environ Med*. 2006;63:700–706.
136. Santos UP, Terra-Filho M, Lin CA, Pereira LA, Vieira TC, Saldiva PH, Braga AL. Cardiac arrhythmia emergency room visits and environmental air pollution in Sao Paulo, Brazil. *J Epidemiol Community Health*. 2008;62:267–272.
137. Rich KE, Petkau J, Vedal S, Brauer M. A case-crossover analysis of particulate air pollution and cardiac arrhythmia in patients with implantable cardioverter defibrillators. *Inhal Toxicol*. 2004;16:363–372.
138. Vedal S, Rich K, Brauer M, White R, Petkau J. Air pollution and cardiac arrhythmias in patients with implantable cardioverter defibrillators. *Inhal Toxicol*. 2004;16:353–362.
139. Metzger KB, Klein M, Flanders WD, Peel JL, Mulholland JA, Langberg JJ, Tolbert PE. Ambient air pollution and cardiac arrhythmias in patients with implantable defibrillators. *Epidemiology*. 2007;18:585–592.
140. Forastiere F, Stafoggia M, Picciotto S, Bellander T, D'Ippoliti D, Lanki T, von Klot S, Nyberg F, Paatero P, Peters A, Pekkanen J, Sunyer J, Perucci CA. A case-crossover analysis of out-of-hospital coronary deaths and air pollution in Rome, Italy. *Am J Respir Crit Care Med*. 2005;172:1549–1555.
141. Rosenthal FS, Carney JP, Olinger ML. Out-of-hospital cardiac arrest and airborne fine particulate matter: a case-crossover analysis of emergency medical services data in Indianapolis, Indiana. *Environ Health Perspect*. 2008;116:631–636.
142. Levy D, Sheppard L, Checkoway H, Kaufman J, Lumley T, Koenig J, Siscovick D. A case-crossover analysis of particulate matter air pollution and out-of-hospital primary cardiac arrest. *Epidemiology*. 2001;12:193–199.
143. Sullivan J, Ishikawa N, Sheppard L, Siscovick D, Checkoway H, Kaufman J. Exposure to ambient fine particulate matter and primary cardiac arrest among persons with and without clinically recognized heart disease. *Am J Epidemiol*. 2003;157:501–509.
144. Peters A, Döring A, Wichmann HE, Koenig W. Increased plasma viscosity during an air pollution episode: a link to mortality? *Lancet*. 1997;349:1582–1587.
145. Seaton A, Soutar A, Crawford V, Elton R, McNerlan S, Cherrie J, Watt M, Agius R, Stout R. Particulate air pollution and the blood. *Thorax*. 1999;54:1027–1032.
146. Pekkanen J, Brunner EJ, Anderson HR, Tiittanen P, Atkinson RW. Daily concentrations of air pollution and plasma fibrinogen in London. *Occup Environ Med*. 2000;57:818–822.
147. Peters A, Fröhlich M, Döring A, Immervoll T, Wichmann HE, Hutchinson WL, Pepys MB, Koenig W. Particulate air pollution is associated with an acute phase response in men: results from the MONICA-Augsburg Study. *Eur Heart J*. 2001;22:1198–1204.
148. Schwartz J Air pollution and blood markers of cardiovascular risk. *Environ Health Perspect*. 2001;109(suppl):405–409.
149. Pope CA 3rd, Hansen ML, Long RW, Nielsen KR, Eatough NL, Wilson WE, Eatough DJ. Ambient particulate air pollution, heart rate variability, and blood markers of inflammation in a panel of elderly subjects. *Environ Health Perspect*. 2004;112:339–345.

150. Rückerl R, Ibald-Mulli A, Koenig W, Schneider A, Woelke G, Cyrys J, Heinrich J, Marder V, Frampton M, Wichmann HE, Peters A. Air pollution and markers of inflammation and coagulation in patients with coronary heart disease. *Am J Respir Crit Care Med*. 2006;173:432–441.
151. Chuang KJ, Chan CC, Su TC, Lee CT, Tang CS. The effect of urban air pollution on inflammation, oxidative stress, coagulation, and autonomic dysfunction in young adults. *Am J Respir Crit Care Med*. 2007;176:370–376.
152. Zeka A, Sullivan JR, Vokonas PS, Sparrow D, Schwartz J. Inflammatory markers and particulate air pollution: characterizing the pathway to disease. *Int J Epidemiol*. 2006;35:1347–1354.
153. Calderón-Garcidueñas L, Villarreal-Calderon R, Valencia-Salazar G, Henríquez-Roldán C, Gutiérrez-Castrellón P, Torres-Jardón R, Osnaya-Brizuela N, Romero L, Torres-Jardón R, Solt A, Reed W. Systemic inflammation, endothelial dysfunction, and activation in clinically healthy children exposed to air pollutants. *Inhal Toxicol*. 2008;20:499–506.
154. O'Neill MS, Veves A, Sarnat JA, Zanobetti A, Gold DR, Economides PA, Horton ES, Schwartz J. Air pollution and inflammation in type 2 diabetes: a mechanism for susceptibility. *Occup Environ Med*. 2007;64:373–379.
155. Rückerl R, Phipps RP, Schneider A, Frampton M, Cyrys J, Oberdörster G, Wichmann HE, Peters A. Ultrafine particles and platelet activation in patients with coronary heart disease: results from a prospective panel study. *Part Fibre Toxicol*. 2007;4:1.
156. Riediker M, Cascio WE, Griggs TR, Herbst MC, Bromberg PA, Neas L, Williams RW, Devlin RB. Particulate matter exposure in cars is associated with cardiovascular effects in healthy young men. *Am J Respir Crit Care Med*. 2004;169:934–940.
157. Riediker M. Cardiovascular effects of fine particulate matter components in highway patrol officers. *Inhal Toxicol*. 2007;19(suppl):99–105.
158. Delfino RJ, Staimer N, Tjoa T, Gillen DL, Polidori A, Arhami M, Kleinman MT, Vaziri ND, Longhurst J, Sioutas C. Air pollution exposures and circulating biomarkers of effect in a susceptible population: clues to potential causal component mixtures and mechanisms. *Environ Health Perspect*. 2009;117:1232–1238.
159. Delfino RJ, Staimer N, Tjoa T, Polidori A, Arhami M, Gillen DL, Kleinman MT, Vaziri ND, Longhurst J, Zaldivar F, Sioutas C. Circulating biomarkers of inflammation, antioxidant activity, and platelet activation are associated with primary combustion aerosols in subjects with coronary artery disease. *Environ Health Perspect*. 2008;116:898–906.
160. Chen JC, Schwartz J. Metabolic syndrome and inflammatory responses to long-term particulate air pollutants. *Environ Health Perspect*. 2008;116:612–617.
161. Hoffmann B, Moebus S, Dragano N, Stang A, Möhlenkamp S, Schmermund A, Memmesheimer M, Bröcker-Preuss M, Mann K, Erbel R, Jöckel KH. Chronic residential exposure to particulate matter air pollution and systemic inflammatory markers. *Environ Health Perspect*. 2009;117:1302–1308.
162. Sullivan JH, Hubbard R, Liu SL, Shepherd K, Trenga CA, Koenig JQ, Chandler WL, Kaufman JD. A community study of the effect of particulate matter on blood measures of inflammation and thrombosis in an elderly population. *Environ Health*. 2007;6:3.
163. Steinvil A, Kordova-Biezuner L, Shapira I, Berliner S, Rogowski O. Short-term exposure to air pollution and inflammation-sensitive biomarkers. *Environ Res*. 2008;106:51–61.
164. Diez Roux AV, Auchincloss AH, Astor B, Barr RG, Cushman M, Dvonch T, Jacobs DR Jr, Kaufman J, Lin X, Samson P. Recent exposure to particulate matter and C-reactive protein concentration in the multiethnic study of atherosclerosis. *Am J Epidemiol*. 2006;164:437–448.
165. Forbes LJ, Patel MD, Rudnicka AR, Cook DG, Bush T, Stedman JR, Whincup PH, Strachan DP, Anderson RH. Chronic exposure to outdoor air pollution and markers of systemic inflammation. *Epidemiology*. 2009;20:245–253.
166. Dubowsky SD, Suh H, Schwartz J, Coull BA, Gold DR. Diabetes, obesity, and hypertension may enhance associations between air pollution and markers of systemic inflammation. *Environ Health Perspect*. 2006;114:992–998.
167. Bräuner EV, Forchhammer L, Møller P, Simonsen J, Glasius M, Wåhlin P, Raaschou-Nielsen O, Loft S. Exposure to ultrafine particles from ambient air and oxidative stress-induced DNA damage. *Environ Health Perspect*. 2007;115:1177–1182.
168. Vinzents PS, Møller P, Sørensen M, Knudsen LE, Hertel O, Jensen FP, Schibye B, Loft S. Personal exposure to ultrafine particles and oxidative DNA damage. *Environ Health Perspect*. 2005;113:1485–1490.
169. Sørensen M, Daneshvar B, Hansen M, Dragsted LO, Hertel O, Knudsen L, Loft S. Personal PM2.5 exposure and markers of oxidative stress in blood. *Environ Health Perspect*. 2003;111:161–166.
170. Baccarelli A, Zanobetti A, Martinelli I, Grillo P, Hou L, Lanzani G, Mannucci PM, Bertazzi PA, Schwartz J. Air pollution, smoking, and plasma homocysteine. *Environ Health Perspect*. 2007;115:176–181.
171. Park SK, O'Neill MS, Vokonas PS, Sparrow D, Spiro A 3rd, Tucker KL, Suh H, Hu H, Schwartz J. Traffic-related particles are associated with elevated homocysteine: the VA normative aging study. *Am J Respir Crit Care Med*. 2008;178:283–289.
172. Romieu I, Garcia-Esteban R, Sunyer J, Rios C, Alcaraz-Zubeldia M, Velasco SR, Holguin F. The effect of supplementation with omega-3 polyunsaturated fatty acids on markers of oxidative stress in elderly exposed to PM(2.5). *Environ Health Perspect*. 2008;116:1237–1242.
173. Liao D, Heiss G, Chinchilli VM, Duan Y, Folsom AR, Lin HM, Salomaa V. Association of criteria pollutants with plasma hemostatic/inflammatory markers: a population-based study. *J Expo Anal Environ Epidemiol*. 2005;15:319–328.
174. Rudez G, Janssen NA, Kilinc E, Leebeek FW, Gerlofs-Nijland ME, Spronk HM, ten Cate H, Cassee FR, de Maat MP. Effects of ambient air pollution on hemostasis and inflammation. *Environ Health Perspect*. 2009;117:995–1001.
175. Su TC, Chan CC, Liau CS, Lin LY, Kao HL, Chuang KJ. Urban air pollution increases plasma fibrinogen and plasminogen activator inhibitor-1 levels in susceptible patients. *Eur J Cardiovasc Prev Rehabil*. 2006;13:849–852.
176. Ray MR, Mukherjee S, Roychoudhury S, Bhattacharya P, Banerjee M, Siddique S, Chakraborty S, Lahiri T. Platelet activation, upregulation of CD11b/CD18 expression on leukocytes and increase in circulating leukocyte-platelet aggregates in Indian women chronically exposed to biomass smoke. *Hum Exp Toxicol*. 2006;25:627–635.
177. Baccarelli A, Zanobetti A, Martinelli I, Grillo P, Hou L, Giacomini S, Bonzini M, Lanzani G, Mannucci PM, Bertazzi PA, Schwartz J. Effects of exposure to air pollution on blood coagulation. *J Thromb Haemost*. 2007;5:252–260.
178. Bräuner EV, Møller P, Barregard L, Dragsted LO, Glasius M, Wåhlin P, Vinzents P, Raaschou-Nielsen O, Loft S. Exposure to ambient concentrations of particulate air pollution does not influence vascular function or inflammatory pathways in young healthy individuals. *Part Fibre Toxicol*. 2008;5:13.
179. Chuang KJ, Chan CC, Shiao GM, Su TC. Associations between submicrometer particles exposures and blood pressure and heart rate in patients with lung function impairments. *J Occup Environ Med*. 2005;47:1093–1098.
180. Ibald-Mulli A, Stieber J, Wichmann HE, Koenig W, Peters A. Effects of air pollution on blood pressure: a population-based approach. *Am J Public Health*. 2001;91:571–577.
181. Zanobetti A, Canner MJ, Stone PH, Schwartz J, Sher D, Eagan-Bengston E, Gates KA, Hartley LH, Suh H, Gold DR. Ambient pollution and blood pressure in cardiac rehabilitation patients. *Circulation*. 2004;110:2184–2189.
182. Choi JH, Xu QS, Park SY, Kim JH, Hwang SS, Lee KH, Lee HJ, Hong YC. Seasonal variation of effect of air pollution on blood pressure. *J Epidemiol Community Health*. 2007;61:314–318.
183. Auchincloss AH, Diez Roux AV, Dvonch JT, Brown PL, Barr RG, Daviglus ML, Goff DC, Kaufman JD, O'Neill MS. Associations between recent exposure to ambient fine particulate matter and blood pressure in the Multi-ethnic Study of Atherosclerosis (MESA). *Environ Health Perspect*. 2008;116:486–491.
184. Liu L, Ruddy TD, Dalipaj M, Szyszkowicz M, You H, Poon R, Wheeler A, Dales R. Influence of personal exposure to particulate air pollution on cardiovascular physiology and biomarkers of inflammation and oxidative stress in subjects with diabetes. *J Occup Environ Med*. 2007;49:258–265.
185. Rich DQ, Freudenberger RS, Ohman-Strickland P, Cho Y, Kipen HM. Right heart pressure increases after acute increases in ambient particulate concentration. *Environ Health Perspect*. 2008;116:1167–1171.
186. Calderón-Garcidueñas L, Vincent R, Mora-Tiscareño A, Franco-Lira M, Henríquez-Roldán C, Barragán-Mejía G, Garrido-García L, Camacho-Reyes L, Valencia-Salazar G, Paredes R, Romero L, Osnaya H, Villarreal-Calderón R, Torres-Jardón R, Hazucha MJ, Reed W. Elevated plasma endothelin-1 and pulmonary arterial pressure in children exposed to air pollution. *Environ Health Perspect*. 2007;115:1248–1253.
187. Harrabi I, Rondeau V, Dartigues JF, Tessier JF, Filleul L. Effects of particulate air pollution on systolic blood pressure: a population-based approach. *Environ Res*. 2006;101:89–93.

188. Ibald-Mulli A, Timonen KL, Peters A, Heinrich J, Wölke G, Lanki T, Buzorius G, Kreyling WG, de Hartog J, Hoek G, ten Brink HM, Pekkanen J. Effects of particulate air pollution on blood pressure and heart rate in subjects with cardiovascular disease: a multicenter approach. *Environ Health Perspect*. 2004;112:369–377.
189. Madsen C, Nafstad P. Associations between environmental exposure and blood pressure among participants in the Oslo Health Study (HUBRO). *Eur J Epidemiol*. 2006;21:485–491.
190. Dvonch JT, Kannan S, Schulz AJ, Keeler GJ, Mentz G, House J, Benjamin A, Max P, Bard RL, Brook RD. Acute effects of ambient particulate matter on blood pressure: differential effects across urban communities. *Hypertension*. 2009;53:853–859.
191. Langrish JP, Mills NL, Chan JK, Leseman DL, Aitken RJ, Fokkens PH, Cassee FR, Li J, Donaldson K, Newby DE, Jiang L, Chan JKK. Beneficial cardiovascular effect of reducing exposure to particulate air pollution with a simple facemask. *Part Fibre Toxicol*. 2009;6:8.
192. McCracken JP, Smith KR, Díaz A, Mittleman MA, Schwartz J. Chimney stove intervention to reduce long-term wood smoke exposure lowers blood pressure among Guatemalan women. *Environ Health Perspect*. 2007;115:996–1001.
193. O'Neill MS, Veves A, Zanobetti A, Sarnat JA, Gold DR, Economides PA, Horton ES, Schwartz J. Diabetes enhances vulnerability to particulate air pollution-associated impairment in vascular reactivity and endothelial function. *Circulation*. 2005;111:2913–2920.
194. Schneider A, Neas L, Herbst MC, Case M, Williams RW, Cascio W, Hinderliter A, Holguin F, Buse JB, Dungan K, Styner M, Peters A, Devlin RB. Endothelial dysfunction: associations with exposure to ambient fine particles in diabetic individuals. *Environ Health Perspect*. 2008;116:1666–1674.
195. Rundell KW, Hoffman JR, Caviston R, Bulbulian R, Hollenbach AM. Inhalation of ultrafine and fine particulate matter disrupts systemic vascular function. *Inhal Toxicol*. 2007;19:133–140.
196. Dales R, Liu L, Szyszkowicz M, Dalipaj M, Willey J, Kulka R, Ruddy TD. Particulate air pollution and vascular reactivity: the bus stop study. *Int Arch Occup Environ Health*. 2007;81:159–164.
197. Briet M, Collin C, Laurent S, Tan A, Azizi M, Agharazii M, Jeunemaitre X, Alhenc-Gelas F, Boutouyrie P. Endothelial function and chronic exposure to air pollution in normal male subjects. *Hypertension*. 2007; 50:970–976.
198. Bräuner EV, Forchhammer L, Møller P, Barregard L, Gunnarsen L, Afshari A, Wåhlin P, Glasius M, Dragsted LO, Basu S, Raaschou-Nielsen O, Loft S. Indoor particles affect vascular function in the aged: an air filtration-based intervention study. *Am J Respir Crit Care Med*. 2008;177:419–425.
199. Künzli N, Jerrett M, Mack WJ, Beckerman B, LaBree L, Gilliland F, Thomas D, Peters J, Hodis HN. Ambient air pollution and atherosclerosis in Los Angeles. *Environ Health Perspect*. 2005;113:201–206.
200. Hoffmann B, Moebus S, Möhlenkamp S, Stang A, Lehmann N, Dragano N, Schmermund A, Memmesheimer M, Mann K, Erbel R, Jöckel KH; Heinz Nixdorf Recall Study Investigative Group. Residential exposure to traffic is associated with coronary atherosclerosis. *Circulation*. 2007; 116:489–496.
201. Hoffmann B, Moebus S, Möhlenkamp S, Stang A, Lehmann N, Dragano N, Schmermund A, Memmesheimer M, Mann K, Erbel R, Jöckel KH; Heinz Nixdorf Recall Study Investigative Group. Residential exposure to urban air pollution, ankle-brachial index, and peripheral arterial disease. *Epidemiology*. 2009;20:280–288.
202. Diez Roux AV, Auchincloss AH, Franklin TG, Raghunathan T, Barr RG, Kaufman J, Astor B, Keeler J. Long-term exposure to ambient particulate matter and prevalence of subclinical atherosclerosis in the Multi-Ethnic Study of Atherosclerosis. *Am J Epidemiol*. 2008;167:667–675.
203. Allen RW, Criqui MH, Diez Roux AV, Allison M, Shea S, Detrano R, Sheppard L, Wong ND, Stukovsky KH, Kaufman JD. Fine particulate matter air pollution, proximity to traffic, and aortic atherosclerosis. *Epidemiology*. 2009;20:254–264.
204. Cavallari JM, Fang SC, Eisen EA, Schwartz J, Hauser R, Herrick RF, Christiani DC. Time course of heart rate variability decline following particulate matter exposures in an occupational cohort. *Inhal Toxicol*. 2008;20:415–422.
205. Chahine T, Baccarelli A, Litonjua A, Wright RO, Suh H, Gold DR, Sparrow D, Vokonas P, Schwartz J. Particulate air pollution, oxidative stress genes, and heart rate variability in an elderly cohort. *Environ Health Perspect*. 2007;115:1617–1622.
206. Park SK, O'Neill MS, Vokonas PS, Sparrow D, Wright RO, Coull B, Nie H, Hu H, Schwartz J. Air pollution and heart rate variability: effect modification by chronic lead exposure. *Epidemiology*. 2008;19:111–120.
207. Baccarelli A, Cassano PA, Litonjua A, Park SK, Suh H, Sparrow D, Vokonas P, Schwartz J. Cardiac autonomic dysfunction: effects from particulate air pollution and protection by dietary methyl nutrients and metabolic polymorphisms. *Circulation*. 2008;117:1802–1809.
208. Peretz A, Kaufman JD, Trenga CA, Allen J, Carlsten C, Aulet MR, Adar SD, Sullivan JH. Effects of diesel exhaust inhalation on heart rate variability in human volunteers. *Environ Res*. 2008;107:178–184.
209. Min KB, Min JY, Cho SI, Paek D. The relationship between air pollutants and heart-rate variability among community residents in Korea. *Inhal Toxicol*. 2008;20:435–444.
210. Cavallari JM, Eisen EA, Fang SC, Schwartz J, Hauser R, Herrick RF, Christiani DC. PM2.5 metal exposures and nocturnal heart rate variability: a panel study of boilermaker construction workers. *Environ Health*. 2008;7:36.
211. Cárdenas M, Vallejo M, Romano-Riquer P, Ruiz-Velasco S, Ferreira-Vidal AD, Hermosillo AG. Personal exposure to PM2.5 air pollution and heart rate variability in subjects with positive or negative head-up tilt test. *Environ Res*. 2008;108:1–6.
212. Chen JC, Cavallari JM, Stone PH, Christiani DC. Obesity is a modifier of autonomic cardiac responses to fine metal particulates. *Environ Health Perspect*. 2007;115:1002–1006.
213. Chuang KJ, Chan CC, Su TC, Lin LY, Lee CT. Associations between particulate sulfate and organic carbon exposures and heart rate variability in patients with or at risk for cardiovascular diseases. *J Occup Environ Med*. 2007;49:610–617.
214. Cavallari JM, Eisen EA, Chen JC, Fang SC, Dobson CB, Schwartz J, Christiani DC. Night heart rate variability and particulate exposures among boilermaker construction workers. *Environ Health Perspect*. 2007;115:1046–1051.
215. Adar SD, Gold DR, Coull BA, Schwartz J, Stone PH, Suh H. Focused exposures to airborne traffic particles and heart rate variability in the elderly. *Epidemiology*. 2007;18:95–103.
216. Wheeler A, Zanobetti A, Gold DR, Schwartz J, Stone P, Suh HH. The relationship between ambient air pollution and heart rate variability differs for individuals with heart and pulmonary disease. *Environ Health Perspect*. 2006;114:560–566.
217. Vallejo M, Ruiz S, Hermosillo AG, Borja-Aburto VH, Cárdenas M. Ambient fine particles modify heart rate variability in young healthy adults. *J Expo Sci Environ Epidemiol*. 2006;16:125–130.
218. Timonen KL, Vanninen E, de Hartog J, Ibald-Mulli A, Brunekreef B, Gold DR, Heinrich J, Hoek G, Lanki T, Peters A, Tarkiainen T, Tiittanen P, Kreyling W, Pekkanen J. Effects of ultrafine and fine particulate and gaseous air pollution on cardiac autonomic control in subjects with coronary artery disease: the ULTRA study. *J Expo Sci Environ Epidemiol*. 2006;16:332–341.
219. Riojas-Rodríguez H, Escamilla-Cejudo JA, González-Hermosillo JA, Téllez-Rojo MM, Vallejo M, Santos-Burgoa C, Rojas-Bracho L. Personal PM2.5 and CO exposures and heart rate variability in subjects with known ischemic heart disease in Mexico City. *J Expo Sci Environ Epidemiol*. 2006;16:131–137.
220. Luttmann-Gibson H, Suh HH, Coull BA, Dockery DW, Sarnat SE, Schwartz J, Stone PH, Gold DR. Short-term effects of air pollution on heart rate variability in senior adults in Steubenville, Ohio. *J Occup Environ Med*. 2006;48:780–788.
221. Lipsett MJ, Tsai FC, Roger L, Woo M, Ostro BD. Coarse particles and heart rate variability among older adults with coronary artery disease in the Coachella Valley, California. *Environ Health Perspect*. 2006;114: 1215–1220.
222. Chen JC, Stone PH, Verrier RL, Nearing BD, MacCallum G, Kim JY, Herrick RF, You J, Zhou H, Christiani DC. Personal coronary risk profiles modify autonomic nervous system responses to air pollution. *J Occup Environ Med*. 2006;48:1133–1142.
223. Henneberger A, Zareba W, Ibald-Mulli A, Rückerl R, Cyrys J, Couderc JP, Mykins B, Woelke G, Wichmann HE, Peters A. Repolarization changes induced by air pollution in ischemic heart disease patients. *Environ Health Perspect*. 2005;113:440–446.
224. Sullivan JH, Schreuder AB, Trenga CA, Liu SL, Larson TV, Koenig JQ, Kaufman JD. Association between short term exposure to fine particulate matter and heart rate variability in older subjects with and without heart disease. *Thorax*. 2005;60:462–466.
225. Schwartz J, Litonjua A, Suh H, Verrier M, Zanobetti A, Syring M, Nearing B, Verrier R, Stone P, MacCallum G, Speizer FE, Gold DR.

Traffic related pollution and heart rate variability in a panel of elderly subjects. *Thorax*. 2005;60:455–461.
226. Romieu I, Téllez-Rojo MM, Lazo M, Manzano-Patiño A, Cortez-Lugo M, Julien P, Bélanger MC, Hernandez-Avila M, Holguin F. Omega-3 fatty acid prevents heart rate variability reductions associated with particulate matter. *Am J Respir Crit Care Med*. 2005;172:1534–1540.
227. Park SK, O'Neill MS, Vokonas PS, Sparrow D, Schwartz J. Effects of air pollution on heart rate variability: the VA normative aging study. *Environ Health Perspect*. 2005;113:304–309.
228. Chuang KJ, Chan CC, Chen NT, Su TC, Lin LY. Effects of particle size fractions on reducing heart rate variability in cardiac and hypertensive patients. *Environ Health Perspect*. 2005;113:1693–1697.
229. Chan CC, Chuang KJ, Shiao GM, Lin LY. Personal exposure to submicrometer particles and heart rate variability in human subjects. *Environ Health Perspect*. 2004;112:1063–1067.
230. Chan CC, Chuang KJ, Su TC, Lin LY. Association between nitrogen dioxide and heart rate variability in a susceptible population. *Eur J Cardiovasc Prev Rehabil*. 2005;12:580–586.
231. Liao D, Duan Y, Whitsel EA, Zheng ZJ, Heiss G, Chinchilli VM, Lin HM. Association of higher levels of ambient criteria pollutants with impaired cardiac autonomic control: a population-based study. *Am J Epidemiol*. 2004;159:768–777.
232. Holguín F, Téllez-Rojo MM, Hernández M, Cortez M, Chow JC, Watson JG, Mannino D, Romieu I. Air pollution and heart rate variability among the elderly in Mexico City. *Epidemiology*. 2003;14:521–527.
233. Devlin RB, Ghio AJ, Kehrl H, Sanders G, Cascio W. Elderly humans exposed to concentrated air pollution particles have decreased heart rate variability. *Eur Respir J Suppl*. 2003;40:76s–80s.
234. Magari SR, Hauser R, Schwartz J, Williams PL, Smith TJ, Christiani DC. Association of heart rate variability with occupational and environmental exposure to particulate air pollution. *Circulation*. 2001;104:986–991.
235. Magari SR, Schwartz J, Williams PL, Hauser R, Smith TJ, Christiani DC. The association between personal measurements of environmental exposure to particulates and heart rate variability. *Epidemiology*. 2002; 13:305–310.
236. Pope CA 3rd, Eatough DJ, Gold DR, Pang Y, Nielsen KR, Nath P, Verrier RL, Kanner RE. Acute exposure to environmental tobacco smoke and heart rate variability. *Environ Health Perspect*. 2001;109:711–716.
237. Pope CA 3rd, Verrier RL, Lovett EG, Larson AC, Raizenne ME, Kanner RE, Schwartz J, Villegas GM, Gold DR, Dockery DW. Heart rate variability associated with particulate air pollution. *Am Heart J*. 1999; 138:890–899.
238. Creason J, Neas L, Walsh D, Williams R, Sheldon L, Liao D, Shy C. Particulate matter and heart rate variability among elderly retirees: the Baltimore 1998 PM study. *J Expo Anal Environ Epidemiol*. 2001;11: 116–122.
239. Peters A, Perz S, Döring A, Stieber J, Koenig W, Wichmann HE. Increases in heart rate during an air pollution episode. *Am J Epidemiol*. 1999;150:1094–1098.
240. Gold DR, Litonjua A, Schwartz J, Lovett E, Larson A, Nearing B, Allen G, Verrier M, Cherry R, Verrier R. Ambient pollution and heart rate variability. *Circulation*. 2000;101:1267–1273.
241. Liao D, Creason J, Shy C, Williams R, Watts R, Zweidinger R. Daily variation of particulate air pollution and poor cardiac autonomic control in the elderly. *Environ Health Perspect*. 1999;107:521–525.
242. Dockery DW, Pope CA 3rd, Kanner RE, Martin Villegas G, Schwartz J. Daily changes in oxygen saturation and pulse rate associated with particulate air pollution and barometric pressure. *Res Rep Health Eff Inst*. 1999;1–19.
243. Park SK, O'Neill MS, Wright RO, Hu H, Vokonas PS, Sparrow D, Suh H, Schwartz J. HFE genotype, particulate air pollution, and heart rate variability: a gene-environment interaction. *Circulation*. 2006;114:2798–2805.
244. Schwartz J, Park SK, O'Neill MS, Vokonas PS, Sparrow D, Weiss S, Kelsey K. Glutathione-S-transferase M1, obesity, statins, and autonomic effects of particles: gene-by-drug-by-environment interaction. *Am J Respir Crit Care Med*. 2005;172:1529–1533.
245. Pekkanen J, Peters A, Hoek G, Tiittanen P, Brunekreef B, de Hartog J, Heinrich J, Ibald-Mulli A, Kreyling WG, Lanki T, Timonen KL, Vanninen E. Particulate air pollution and risk of ST-segment depression during repeated submaximal exercise tests among subjects with coronary heart disease: the Exposure and Risk Assessment for Fine and Ultrafine Particles in Ambient Air (ULTRA) study. *Circulation*. 2002;106:933–938.
246. Lanki T, de Hartog JJ, Heinrich J, Hoek G, Janssen NA, Peters A, Stölzel M, Timonen KL, Vallius M, Vanninen E, Pekkanen J. Can we identify sources of fine particles responsible for exercise-induced ischemia on days with elevated air pollution? The ULTRA study. *Environ Health Perspect*. 2006;114:655–660.
247. Lanki T, Hoek G, Timonen KL, Peters A, Tiittanen P, Vanninen E, Pekkanen J. Hourly variation in fine particle exposure is associated with transiently increased risk of ST segment depression. *Occup Environ Med*. 2008;65:782–786.
248. Gold DR, Litonjua AA, Zanobetti A, Coull BA, Schwartz J, MacCallum G, Verrier RL, Nearing BD, Canner MJ, Suh H, Stone PH. Air pollution and ST-segment depression in elderly subjects. *Environ Health Perspect*. 2005;113:883–887.
249. Chuang KJ, Coull BA, Zanobetti A, Suh H, Schwartz J, Stone PH, Litonjua A, Speizer FE, Gold DR. Particulate air pollution as a risk factor for ST-segment depression in patients with coronary artery disease. *Circulation*. 2008;118:1314–1320.
250. Baccarelli A, Wright RO, Bollati V, Tarantini L, Litonjua AA, Suh HH, Zanobetti A, Sparrow D, Vokonas PS, Schwartz J. Rapid DNA methylation changes after exposure to traffic particles. *Am J Respir Crit Care Med*. 2009;179:572–578.
251. Tarantini L, Bonzini M, Apostoli P, Pegoraro V, Bollati V, Marinelli B, Cantone L, Rizzo G, Hou L, Schwartz J, Bertazzi PA, Baccarelli A. Effects of particulate matter on genomic DNA methylation content and iNOS promoter methylation. *Environ Health Perspect*. 2009;117:217–222.
252. Van Hee VC, Adar SD, Szpiro AA, Barr RG, Bluemke DA, Diez Roux AV, Gill EA, Sheppard L, Kaufman JD. Exposure to traffic and left ventricular mass and function: the Multi-Ethnic Study of Atherosclerosis. *Am J Respir Crit Care Med*. 2009;179:827–834.
253. Johnson D, Parker JD. Air pollution exposure and self-reported cardiovascular disease. *Environ Res*. 2009;109:582–589.
254. Brook RD, Jerrett M, Brook JR, Bard RL, Finkelstein MM. The relationship between diabetes mellitus and traffic-related air pollution. *J Occup Environ Med*. 2008;50:32–38.
255. Kelishadi R, Mirghaffari N, Poursafa P, Gidding SS. Lifestyle and environmental factors associated with inflammation, oxidative stress and insulin resistance in children. *Atherosclerosis*. 2009;203:311–319.
256. Schlesinger RB. The health impact of common inorganic components of fine particulate matter (PM2.5) in ambient air: a critical review. *Inhal Toxicol*. 2007;19:811–832.
257. Franklin M, Koutrakis P, Schwartz P. The role of particle composition on the association between PM2.5 and mortality. *Epidemiology*. 2008; 19:680–689.
258. Ostro B, Feng WY, Broadwin R, Green S, Lipsett M. The effects of components of fine particulate air pollution on mortality in California: results from CALFINE. *Environ Health Perspect*. 2007;115:13–19.
259. Peng RD, Bell ML, Geyh AS, McDermott A, Zeger SL, Samet JM, Dominici F. Emergency admissions for cardiovascular and respiratory diseases and the chemical composition of fine particle air pollution. *Environ Health Perspect*. 2009;117:957–963.
260. Bell ML, Ebisu K, Peng RD, Samet JM, Dominici F. Hospital admissions and chemical composition of fine particle air pollution. *Am J Respir Crit Care Med*. 2009;179:1115–1120.
261. Nel A, Xia T, Mädler L, Li N. Toxic potential of materials at the nanolevel. *Science*. 2006;311:622–627.
262. Stölzel M, Breitner S, Cyrys J, Pitz M, Wölke G, Kreyling W, Heinrich J, Wichmann HE, Peters A. Daily mortality and particulate matter in different size classes in Erfurt, Germany. *J Expo Sci Environ Epidemiol*. 2007;17:458–467.
263. Brunekreef B, Forsberg B. Epidemiological evidence of effects of coarse airborne particles on health. *Eur Respir J*. 2005;26:309–318.
264. Yeatts K, Svendsen E, Creason J, Alexis N, Herbst M, Scott J, Kupper L, Williams R, Neas L, Cascio W, Devlin RB, Peden DB. Coarse particulate matter (PM2.5–10) affects heart rate variability, blood lipids, and circulating eosinophils in adults with asthma. *Environ Health Perspect*. 2007;115:709–714.
265. Host S, Larrieu S, Pascal L, Blanchard M, Declercq C, Fabre P, Jusot JF, Chardon B, Le Tertre A, Wagner V, Prouvost H, Lefranc A. Short-term associations between fine and coarse particles and hospital admissions for cardiorespiratory diseases in six French cities. *Occup Environ Med*. 2008;65:544–551.
266. Kan H, London SJ, Chen G, Zhang Y, Song G, Zhao N, Jiang L, Chen B. Differentiating the effects of fine and coarse particles on daily mortality in Shanghai, China. *Environ Int*. 2007;33:376–384.
267. Puett RC, Hart JE, Yanosky JD, Paciorek C, Schwartz J, Suh H, Speizer FE, Laden F. Chronic fine and coarse particulate exposure, mortality, and coronary heart disease in the Nurses' Health Study. *Environ Health Perspect*. 2009;117:1697–1701.

268. Hoffmann B, Moebus S, Stang A, Beck EM, Dragano N, Möhlenkamp S, Schmermund A, Memmesheimer M, Mann K, Erbel R, Jöckel KH; Heinz Nixdorf RECALL Study Investigative Group. Residence close to high traffic and prevalence of coronary heart disease. *Eur Heart J*. 2006;27:2696–2702.
269. Maynard D, Coull BA, Gryparis A, Schwartz J. Mortality risk associated with short-term exposure to traffic particles and sulfates. *Environ Health Perspect*. 2007;115:751–755.
270. Maheswaran R, Haining RP, Brindley P, Law J, Pearson T, Fryers PR, Wise S, Campbell MJ; Small-area level ecological study. Outdoor air pollution, mortality, and hospital admissions from coronary heart disease in Sheffield, UK: a small-area level ecological study. *Eur Heart J*. 2005;26:2543–2549.
271. Finkelstein MM, Jerrett M, Sears MR. Traffic air pollution and mortality rate advancement periods. *Am J Epidemiol*. 2004;160:173–177.
272. Nafstad P, Håheim LL, Wisløff T, Gram F, Oftedal B, Holme I, Hjermann I, Leren P. Urban air pollution and mortality in a cohort of Norwegian men. *Environ Health Perspect*. 2004;112:610–615.
273. Rosenlund M, Forastiere F, Stafoggia M, Porta D, Perucci M, Ranzi A, Nussio F, Perucci CA. Comparison of regression models with land-use and emissions data to predict the spatial distribution of traffic-related air pollution in Rome. *J Expo Sci Environ Epidemiol*. 2008;18:192–199.
274. Rosenlund M, Bellander T, Nordquist T, Alfredsson L. Traffic-generated air pollution and myocardial infarction. *Epidemiology*. 2009; 20:265–271.
275. Beelen R, Hoek G, Houthuijs D, van den Brandt PA, Goldbohm RA, Fischer P, Schouten LJ, Armstrong B, Brunekreef B. The joint association of air pollution and noise from road traffic with cardiovascular mortality in a cohort study. *Occup Environ Med*. 2009;66:243–250.
276. Kan H, Heiss G, Rose KM, Whitsel EA, Lurmann F, London SJ. Prospective analysis of traffic exposure as a risk factor for incident coronary heart disease: the Atherosclerosis Risk in Communities (ARIC) study. *Environ Health Perspect*. 2008;116:1463–1468.
277. Havard S, Deguen S, Zmirou-Navier D, Schillinger C, Bard D. Traffic-related air pollution and socioeconomic status: a spatial autocorrelation study to assess environmental equity on a small-area scale. *Epidemiology*. 2009;20:223–230.
278. Goodman PG, Dockery DW, Clancy L. Cause-specific mortality and the extended effects of particulate pollution and temperature exposure. *Environ Health Perspect*. 2004;112:179–185.
279. Zanobetti A, Schwartz J, Samoli E, Gryparis A, Touloumi G, Peacock J, Anderson RH, Le Tertre A, Bobros J, Celko M, Goren A, Forsberg B, Michelozzi P, Rabczenko D, Hoyos SP, Wichmann HE, Katsouyanni K. The temporal pattern of respiratory and heart disease mortality in response to air pollution. *Environ Health Perspect*. 2003;111:1188–1193.
280. Pope CA 3rd, Burnett RT, Krewski D, Jerrett M, Shi Y, Calle EE, Thun MJ. Cardiovascular mortality and exposure to airborne fine particulate matter and cigarette smoke: shape of the exposure-response relationship. *Circulation*. 2009;120:941–948.
281. Thomas D. Why do estimates of the acute and chronic effects of air pollution on mortality differ? *J Toxicol Environ Health A*. 2005;68: 1167–1174.
281a. Pope CA 3rd. Mortality effects of longer term exposures to fine particulate air pollution: review of recent epidemiological evidence. *Inhalant Toxicol*. 2007;19(suppl 1):S33–S38.
282. Brook RD, Rajagopalan S. Air pollution and cardiovascular events. *N Engl J Med*. 356:2104–2105, 2007; author reply 2105–2106.
283. Künzli N, Medina S, Kaiser R, Quénel P, Horak F Jr, Studnicka M. Assessment of deaths attributable to air pollution: should we use risk estimates based on time series or on cohort studies? *Am J Epidemiol*. 2001;153:1050–1055.
284. Brook RD. Potential health risks of air pollution beyond triggering acute cardiopulmonary events. *JAMA*. 2008;299:2194–2196.
285. Dinno A, Glantz S. Clean indoor air laws immediately reduce heart attacks. *Prev Med*. 2007;45:9–11.
286. Kan H, London SJ, Chen G, Zhang Y, Song G, Zhao N, Jiang L, Chen B. Season, sex, age, and education as modifiers of the effects of outdoor air pollution on daily mortality in Shanghai, China: The Public Health and Air Pollution in Asia (PAPA) Study. *Environ Health Perspect*. 2008;116:1183–1188.
287. Forastiere F, Stafoggia M, Berti G, Bisanti L, Cernigliaro A, Chiusolo M, Mallone S, Miglio R, Pandolfi P, Rognoni M, Serinelli M, Tessari R, Vigotti M, Perucci CA; SISTI Group. Particulate matter and daily mortality: a case-crossover analysis of individual effect modifiers. *Epidemiology*. 2008;19:571–580.
288. Ostro BD, Feng WY, Broadwin R, Malig BJ, Green RS, Lipsett MJ. The impact of components of fine particulate matter on cardiovascular mortality in susceptible subpopulations. *Occup Environ Med*. 2008;65:750–756.
289. Zeka A, Zanobetti A, Schwartz J. Individual-level modifiers of the effects of particulate matter on daily mortality. *Am J Epidemiol*. 2006; 163:849–859.
290. Frampton. Human clinical studies of airborne pollutants. In: Gardner D, ed. *Toxicology of the Lung*. Boca Raton, Fla: Taylor & Francis; 2006: 29–82.
290a. Mühlfeld C, Rothen-Rutishauser B, Blank F, Vanhecke D, Ochs M, Gehr P. Interactions of nanoparticles with pulmonary structures and cellular responses. *Am J Physiol Lung Cell Mol Physiol*. 2008;294: L817–L829.
290b. Møller P, Jackobsen NR, Folkmann JK, Danielsen PH, Mikkelsen L, Hemmingsen JG, Vesterdal LK, Forchhammer L, Wallin K, Loft S. Role of oxidative damage in toxicity of particulates. *Free Radic Res*. 2010; 44:1–46.
291. Ghio AJ, Cohen MD. Disruption of iron homeostasis as a mechanism of biologic effect by ambient air pollution particles. *Inhal Toxicol*. 2005; 17:709–716.
292. Becher R, Bucht A, Øvrevik J, Hongslo JK, Dahlman HJ, Samuelsen JT, Schwarze PE. Involvement of NADPH oxidase and iNOS in rodent pulmonary cytokine responses to urban air and mineral particles. *Inhal Toxicol*. 2007;19:645–655.
293. Li Z, Hyseni X, Carter JD, Soukup JM, Dailey LA, Huang YC. Pollutant particles enhanced H2O2 production from NAD(P)H oxidase and mitochondria in human pulmonary artery endothelial cells. *Am J Physiol Cell Physiol*. 2006;291:C357–365.
294. Rhoden CR, Wellenius GA, Ghelfi E, Lawrence J, González-Flecha B. PM-induced cardiac oxidative stress and dysfunction are mediated by autonomic stimulation. *Biochim Biophys Acta*. 2005;1725:305–313.
295. Li Z, Carter JD, Dailey LA, Huang YC. Pollutant particles produce vasoconstriction and enhance MAPK signaling via angiotensin type I receptor. *Environ Health Perspect*. 2005;113:1009–1014.
296. Cao D, Tal TL, Graves LM, Gilmour I, Linak W, Reed W, Bromberg PA, Samet JM. Diesel exhaust particulate-induced activation of Stat3 requires activities of EGFR and Src in airway epithelial cells. *Am J Physiol Lung Cell Mol Physiol*. 2007;292:L422–429.
297. Becker S, Dailey L, Soukup JM, Silbajoris R, Devlin RB. TLR-2 is involved in airway epithelial cell response to air pollution particles. *Toxicol Appl Pharmacol*. 2005;203:45–52.
298. Huang YC, Wu W, Ghio AJ, Carter JD, Silbajoris R, Devlin RB, Samet JM. Activation of EGF receptors mediates pulmonary vasoconstriction induced by residual oil fly ash. *Exp Lung Res*. 2002;28:19–38.
299. Hollingsworth JW 2nd, Cook DN, Brass DM, Walker JK, Morgan DL, Foster WM, Schwartz DA. The role of Toll-like receptor 4 in environmental airway injury in mice. *Am J Respir Crit Care Med*. 2004;170:126–132.
300. Cho HY, Jedlicka AE, Clarke R, Kleeberger SR. Role of Toll-like receptor-4 in genetic susceptibility to lung injury induced by residual oil fly ash. *Physiol Genomics*. 2005;22:108–117.
301. Hollingsworth JW, Maruoka S, Li Z, Potts EN, Brass DM, Garantziotis S, Fong A, Foster WM, Schwartz DA. Ambient ozone primes pulmonary innate immunity in mice. *J Immunol*. 2007;179:4367–4375.
302. Fujii T, Hayashi S, Hogg JC, Mukae H, Suwa T, Goto Y, Vincent R, van Eeden SF. Interaction of alveolar macrophages and airway epithelial cells following exposure to particulate matter produces mediators that stimulate the bone marrow. *Am J Respir Cell Mol Biol*. 2002;27:34–41.
303. Rhoden CR, Ghelfi E, González-Flecha B. Pulmonary inflammation by ambient air particles is mediated by superoxide anion. *Inhal Toxicol*. 2008;20:11–15.
304. Bosson J, Pourazar J, Forsberg B, Adelroth E, Sandström T, Blomberg A. Ozone enhances the airway inflammation initiated by diesel exhaust. *Respiratory medicine*. 2007;101:1140–1146.
305. Kennedy T, Ghio AJ, Reed W, Samet J, Zagorski J, Quay J, Carter J, Dailey L, Hoidal JR, Devlin RB. Copper-dependent inflammation and nuclear factor-kappaB activation by particulate air pollution. *Am J Respir Cell Mol Biol*. 1998;19:366–378.
306. Quay JL, Reed W, Samet J, Devlin RB. Air pollution particles induce IL-6 gene expression in human airway epithelial cells via NF-kappa B Activation. *Am J Respir Cell Mol Biol*. 1998;19:98–106.
307. Veronesi B, Oortgiesen M, Carter JD, Devlin RB. Particulate matter initiates inflammatory cytokine release by activation of capsaicin and acid receptors in a human bronchial epithelial cell line. *Toxicol Appl Pharmacol*. 1999;154:106–115.

308. Harder SD, Soukup JM, Ghio AJ, Devlin RB, Becker S. Inhalation of PM2.5 does not modulate host defense or immune parameters in blood or lung of normal human subjects. *Environmental health perspectives.* 2001;109(suppl):599–604.
309. van Eeden SF, Tan WC, Suwa T, Mukae H, Terashima T, Fujii T, Qui D, Vincent R, Hogg JC. Cytokines involved in the systemic inflammatory response induced by exposure to particulate matter air pollutants (PM(10)). *Am J Respir Crit Care Med.* 2001;164:826–830.
310. Hartz AM, Bauer B, Block ML, Hong JS, Miller DS. Diesel exhaust particles induce oxidative stress, proinflammatory signaling, and P-glycoprotein up-regulation at the blood-brain barrier. *FASEB J.* 2008;22:2723–2733.
311. Shukla A, Timblin C, BeruBe K, Gordon T, McKinney W, Driscoll K, Vacek P, Mossman BT. Inhaled particulate matter causes expression of nuclear factor (NF)-kappaB-related genes and oxidant-dependent NF-kappaB activation in vitro. *Am J Respir Cell Mol Biol.* 2000;23:182–187.
312. Boland S, Baeza-Squiban A, Fournier T, Houcine O, Gendron MC, Chévrier M, Jouvenot G, Coste A, Aubier M, Marano F. Diesel exhaust particles are taken up by human airway epithelial cells in vitro and alter cytokine production. *Am J Physiol.* 1999;276:L604–L613.
313. Törnqvist H, Mills NL, Gonzalez M, Miller MR, Robinson SD, Megson IL, Macnee W, Donaldson K, Söderberg S, Newby DE, Sandström T, Blomberg A. Persistent endothelial dysfunction in humans after diesel exhaust inhalation. *Am J Respir Crit Care Med.* 2007;176:395–400.
314. Tamagawa E, Bai N, Morimoto K, Gray C, Mui T, Yatera K, Zhang X, Xing L, Li Y, Laher I, Sin DD, Man SF, van Eeden SF. Particulate matter exposure induces persistent lung inflammation and endothelial dysfunction. *Am J Physiol Lung Cell Mol Physiol.* 2008;295:L79–L85.
315. Fujimaki H, Kurokawa Y, Yamamoto S, Satoh M. Distinct requirements for interleukin-6 in airway inflammation induced by diesel exhaust in mice. *Immunopharmacol Immunotoxicol.* 2006;28:703–714.
316. Mutlu GM, Green D, Bellmeyer A, Baker CM, Burgess Z, Rajamannan N, Christman JW, Foiles N, Kamp DW, Ghio AJ, Chandel NS, Dean DA, Sznajder JI, Budinger GR. Ambient particulate matter accelerates coagulation via an IL-6-dependent pathway. *J Clin Invest.* 2007;117:2952–2961.
317. Becker S, Mundandhara S, Devlin RB, Madden M. Regulation of cytokine production in human alveolar macrophages and airway epithelial cells in response to ambient air pollution particles: further mechanistic studies. *Toxicol Appl Pharmacol.* 2005;207(suppl):269–275.
318. Dostert C, Pétrilli V, Van Bruggen R, Steele C, Mossman BT, Tschopp J. Innate immune activation through Nalp3 inflammasome sensing of asbestos and silica. *Science.* 2008;320:674–677.
319. Sun Q, Yue P, Deiuliis JA, Lumeng CN, Kampfrath T, Mikolaj MB, Cai Y, Ostrowski MC, Lu B, Parthasarathy S, Brook RD, Moffatt-Bruce SD, Chen LC, Rajagopalan S. Ambient air pollution exaggerates adipose inflammation and insulin resistance in a mouse model of diet-induced obesity. *Circulation.* 2009;119:538–546.
320. Han JY, Takeshita K, Utsumi H. Noninvasive detection of hydroxyl radical generation in lung by diesel exhaust particles. *Free Radic Biol Med.* 2001;30:516–525.
321. Squadrito GL, Cueto R, Dellinger B, Pryor WA. Quinoid redox cycling as a mechanism for sustained free radical generation by inhaled airborne particulate matter. *Free Radic Biol Med.* 2001;31:1132–1138.
322. Baulig A, Garlatti M, Bonvallot V, Marchand A, Barouki R, Marano F, Baeza-Squiban A. Involvement of reactive oxygen species in the metabolic pathways triggered by diesel exhaust particles in human airway epithelial cells. *Am J Physiol Lung Cell Mol Physiol.* 2003;285:L671–L679.
323. Donaldson K, Stone V, Borm PJ, Jimenez LA, Gilmour PS, Schins RP, Knaapen AM, Rahman I, Faux SP, Brown DM, MacNee W. Oxidative stress and calcium signaling in the adverse effects of environmental particles (PM10). *Free Radic Biol Med.* 2003;34:1369–1382.
324. Beck-Speier I, Dayal N, Karg E, Maier KL, Schumann G, Schulz H, Semmler M, Takenaka S, Stettmaier K, Bors W, Ghio A, Samet JM, Heyder J. Oxidative stress and lipid mediators induced in alveolar macrophages by ultrafine particles. *Free Radic Biol Med.* 2005;38:1080–1092.
325. Prahalad AK, Soukup JM, Inmon J, Willis R, Ghio AJ, Becker S, Gallagher JE. Ambient air particles: effects on cellular oxidant radical generation in relation to particulate elemental chemistry. *Toxicol Appl Pharmacol.* 1999;158:81–91.
326. Dellinger B, Pryor WA, Cueto R, Squadrito GL, Hegde V, Deutsch WA. Role of free radicals in the toxicity of airborne fine particulate matter. *Chem Res Toxicol.* 2001;14:1371–1377.
327. González-Flecha B. Oxidant mechanisms in response to ambient air particles. *Mol Aspects Med.* 2004;25:169–182.
328. Ikeda M, Watarai K, Suzuki M, Ito T, Yamasaki H, Sagai M, Tomita T. Mechanism of pathophysiological effects of diesel exhaust particles on endothelial cells. *Environ Toxicol Pharmacol.* 1998;6:117–123.
329. Bai Y, Suzuki AK, Sagai M. The cytotoxic effects of diesel exhaust particles on human pulmonary artery endothelial cells in vitro: role of active oxygen species. *Free Radic Biol Med.* 2001;30:555–562.
330. Risom L, Møller P, Loft S. Oxidative stress-induced DNA damage by particulate air pollution. *Mutat Res.* 2005;592:119–137.
331. Araujo JA, Barajas B, Kleinman M, Wang X, Bennett BJ, Gong KW, Navab M, Harkema J, Sioutas C, Lusis AJ, Nel AE. Ambient particulate pollutants in the ultrafine range promote early atherosclerosis and systemic oxidative stress. *Circ Res.* 2008;102:589–596.
332. Nurkiewicz TR, Porter DW, Barger M, Millecchia L, Rao KM, Marvar PJ, Hubbs AF, Castranova V, Boegehold MA. Systemic microvascular dysfunction and inflammation after pulmonary particulate matter exposure. *Environ Health Perspect.* 2006;114:412–419.
333. Sun Q, Yue P, Ying Z, Cardounel AJ, Brook RD, Devlin R, Hwang JS, Zweier JL, Chen LC, Rajagopalan S. Air pollution exposure potentiates hypertension through reactive oxygen species-mediated activation of Rho/ROCK. *Arterioscler Thromb Vasc Biol.* 2008;28:1760–1766.
334. Sun Q, Wang A, Jin X, Natanzon A, Duquaine D, Brook RD, Aguinaldo JG, Fayad ZA, Fuster V, Lippmann M, Chen LC, Rajagopalan S. Long-term air pollution exposure and acceleration of atherosclerosis and vascular inflammation in an animal model. *JAMA.* 2005;294:3003–3010.
335. Ghelfi E, Rhoden CR, Wellenius GA, Lawrence J, Gonzalez-Flecha B. Cardiac oxidative stress and electrophysiological changes in rats exposed to concentrated ambient particles are mediated by TRP-dependent pulmonary reflexes. *Toxicol Sci.* 2008;102:328–336.
336. Nemmar A, Hoylaerts MF, Hoet PH, Dinsdale D, Smith T, Xu H, Vermylen J, Nemery B. Ultrafine particles affect experimental thrombosis in an in vivo hamster model. *Am J Respir Crit Care Med.* 2002;166:998–1004.
337. Nemmar A, Hoet PH, Dinsdale D, Vermylen J, Hoylaerts MF, Nemery B. Diesel exhaust particles in lung acutely enhance experimental peripheral thrombosis. *Circulation.* 2003;107:1202–1208.
338. Nemmar A, Hoet PH, Vermylen J, Nemery B, Hoylaerts MF. Pharmacological stabilization of mast cells abrogates late thrombotic events induced by diesel exhaust particles in hamsters. *Circulation.* 2004;110:1670–1677.
339. Nemmar A, Nemery B, Hoet PH, Vermylen J, Hoylaerts MF. Pulmonary inflammation and thrombogenicity caused by diesel particles in hamsters: role of histamine. *Am J Respir Crit Care Med.* 2003;168:1366–1372.
340. Nemmar A, Hoet PH, Vandervoort P, Dinsdale D, Nemery B, Hoylaerts MF. Enhanced peripheral thrombogenicity after lung inflammation is mediated by platelet-leukocyte activation: role of P-selectin. *J Thromb Haemost.* 2007;5:1217–1226.
341. Sun Q, Yue P, Kirk RI, Wang A, Moatti D, Jin X, Lu B, Schecter AD, Lippmann M, Gordon T, Chen LC, Rajagopalan S. Ambient air particulate matter exposure and tissue factor expression in atherosclerosis. *Inhal Toxicol.* 2008;20:127–137.
342. Cozzi E, Wingard CJ, Cascio WE, Devlin RB, Miles JJ, Bofferding AR, Lust RM, Van Scott MR, Henriksen RA. Effect of ambient particulate matter exposure on hemostasis. *Transl Res.* 2007;149:324–332.
343. Gilmour PS, Morrison ER, Vickers MA, Ford I, Ludlam CA, Greaves M, Donaldson K, MacNee W. The procoagulant potential of environmental particles (PM10). *Occup Environ Med.* 2005;62:164–171.
344. Esmon CT. Inflammation and thrombosis. *J Thromb Haemost.* 2003;1:1343–1348.
345. Nightingale JA, Maggs R, Cullinan P, Donnelly LE, Rogers DF, Kinnersley R, Chung KF, Barnes PJ, Ashmore M, Newman-Taylor A. Airway inflammation after controlled exposure to diesel exhaust particulates. *Am J Respir Crit Care Med.* 2000;162:161–166.
346. Wichers LB, Nolan JP, Winsett DW, Ledbetter AD, Kodavanti UP, Schladweiler MC, Costa DL, Watkinson WP. Effects of instilled combustion-derived particles in spontaneously hypertensive rats. Part I: Cardiovascular responses. *Inhal Toxicol.* 2004;16:391–405.
347. Cheng TJ, Hwang JS, Wang PY, Tsai CF, Chen CY, Lin SH, Chan CC. Effects of concentrated ambient particles on heart rate and blood pressure in pulmonary hypertensive rats. *Environ Health Perspect.* 2003;111:147–150.
348. Chang CC, Hwang JS, Chan CC, Wang PY, Hu TH, Cheng TJ. Effects of concentrated ambient particles on heart rate, blood pressure, and cardiac contractility in spontaneously hypertensive rats. *Inhal Toxicol.* 2004;16:421–429.
349. Ying Z, Yue P, Xu X, Zhong M, Sun Q, Mikolaj M, Wang A, Brook RD, Chen LC, Rajagopalan S. Air pollution and cardiac remodeling: a role

for RhoA/Rho-kinase. *Am J Physiol Heart Circ Physiol.* 2009;296: H1540–1550.
350. Bartoli CR, Wellenius GA, Diaz EA, Lawrence J, Coull BA, Akiyama I, Lee LM, Okabe K, Verrier RL, Godleski JJ. Mechanisms of inhaled fine particulate air pollution- induced arterial blood pressure changes. *Environ Health Perspect.* 2009;117:361–366.
351. Ito T, Suzuki T, Tamura K, Nezu T, Honda K, Kobayashi T. Examination of mRNA expression in rat hearts and lungs for analysis of effects of exposure to concentrated ambient particles on cardiovascular function. *Toxicology.* 2008;243:271–283.
352. Thomson E, Kumarathasan P, Goegan P, Aubin RA, Vincent R. Differential regulation of the lung endothelin system by urban particulate matter and ozone. *Toxicol Sci.* 2005;88:103–113.
353. Tankersley CG, Champion HC, Takimoto E, Gabrielson K, Bedja D, Misra V, El-Haddad H, Rabold R, Mitzner W. Exposure to inhaled particulate matter impairs cardiac function in senescent mice. *Am J Physiol Regul Integr Comp Physiol.* 2008;295:R252–263.
354. Upadhyay S, Stoeger T, Harder V, Thomas RF, Schladweiler MC, Semmler-Behnke M, Takenaka S, Karg E, Reitmeir P, Bader M, Stampfl A, Kodavanti UP, Schulz H. Exposure to ultrafine carbon particles at levels below detectable pulmonary inflammation affects cardiovascular performance in spontaneously hypertensive rats. *Part Fibre Toxicol.* 2008;5:19.
355. Niwa Y, Hiura Y, Sawamura H, Iwai N. Inhalation exposure to carbon black induces inflammatory response in rats. *Circ J.* 2008;72:144–149.
356. Suwa T, Hogg JC, Quinlan KB, Ohgami A, Vincent R, van Eeden SF. Particulate air pollution induces progression of atherosclerosis. *J Am Coll Cardiol.* 2002;39:935–942.
357. Chen LC, Nadziejko C. Effects of subchronic exposures to concentrated ambient particles (CAPs) in mice: V. CAPs exacerbate aortic plaque development in hyperlipidemic mice. *Inhal Toxicol.* 2005;17:217–224.
358. Cozzi E, Hazarika S, Stallings HW 3rd, Cascio WE, Devlin RB, Lust RM, Wingard CJ, Van Scott MR. Ultrafine particulate matter exposure augments ischemia-reperfusion injury in mice. *Am J Physiol Heart Circ Physiol.* 2006;291:H894–H903.
359. Niwa Y, Hiura Y, Murayama T, Yokode M, Iwai N. Nano-sized carbon black exposure exacerbates atherosclerosis in LDL-receptor knockout mice. *Circ J.* 2007;71:1157–1161.
360. Tsou CL, Peters W, Si Y, Slaymaker S, Aslanian AM, Weisberg SP, Mack M, Charo IF. Critical roles for CCR2 and MCP-3 in monocyte mobilization from bone marrow and recruitment to inflammatory sites. *J Clin Invest.* 2007;117:902–909.
361. Goto Y, Ishii H, Hogg JC, Shih CH, Yatera K, Vincent R, van Eeden SF. Particulate matter air pollution stimulates monocyte release from the bone marrow. *Am J Respir Crit Care Med.* 2004;170:891–897.
362. Yatera K, Hsieh J, Hogg JC, Tranfield E, Suzuki H, Shih CH, Behzad AR, Vincent R, van Eeden SF. Particulate matter air pollution exposure promotes recruitment of monocytes into atherosclerotic plaques. *Am J Physiol Heart Circ Physiol.* 2008;294:H944–H953.
363. Nurkiewicz TR, Porter DW, Hubbs AF, Stone S, Chen BT, Frazer DG, Boegehold MA, Castranova V. Pulmonary nanoparticle exposure disrupts systemic microvascular nitric oxide signaling. *Toxicol Sci.* 2009;110:191–203.
364. Lund AK, Lucero J, Lucas S, Madden MC, McDonald JD, Seagrave JC, Knuckles TL, Campen MJ. Vehicular emissions induce vascular MMP-9 expression and activity associated with endothelin-1-mediated pathways. *Arterioscler Thromb Vasc Biol.* 2009;29:511–517.
365. Cherng TW, Campen MJ, Knuckles TL, Gonzalez Bosc L, Kanagy NL. Impairment of coronary endothelial cell ET(B) receptor function after short-term inhalation exposure to whole diesel emissions. *Am J Physiol Regul Integr Comp Physiol.* 2009;297:R640–R647.
366. Hansen CS, Sheykhzade M, Møller P, Folkmann JK, Amtorp O, Jonassen T, Loft S. Diesel exhaust particles induce endothelial dysfunction in apoE−/− mice. *Toxicol Appl Pharmacol.* 2007;219:24–32.
367. Proctor SD, Dreher KL, Kelly SE, Russell JC. Hypersensitivity of prediabetic JCR:LA-cp rats to fine airborne combustion particle-induced direct and noradrenergic-mediated vascular contraction. *Toxicol Sci.* 2006;90:385–391.
368. Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. *N Engl J Med.* 2005;352:1685–1695.
369. Lee MY, Griendling KK. Redox signaling, vascular function, and hypertension. *Antioxid Redox Signal.* 2008;10:1045–1059.
370. Miller MR, Borthwick SJ, Shaw CA, McLean SG, McClure D, Mills NL, Duffin R, Donaldson K, Megson IL, Hadoke PW, Newby DE. Direct impairment of vascular function by diesel exhaust particulate through reduced bioavailability of endothelium-derived nitric oxide induced by superoxide free radicals. *Environ Health Perspect.* 2009;117:611–616.
371. Watkinson WP, Campen MJ, Costa DL. Cardiac arrhythmia induction after exposure to residual oil fly ash particles in a rodent model of pulmonary hypertension. *Toxicol Sci.* 1998;41:209–216.
372. Wellenius GA, Saldiva PH, Batalha JR, Krishna Murthy GG, Coull BA, Verrier RL, Godleski JJ. Electrocardiographic changes during exposure to residual oil fly ash (ROFA) particles in a rat model of myocardial infarction. *Toxicol Sci.* 2002;66:327–335.
373. Chang CC, Hwang JS, Chan CC, Wang PY, Hu TH, Cheng TJ. Effects of concentrated ambient particles on heart rate variability in spontaneously hypertensive rats. *J Occup Health.* 2005;47:471–480.
374. Chang CC, Hwang JS, Chan CC, Cheng TJ. Interaction effects of ultrafine carbon black with iron and nickel on heart rate variability in spontaneously hypertensive rats. *Environ Health Perspect.* 2007;115: 1012–1017.
375. Chen LC, Hwang JS. Effects of subchronic exposures to concentrated ambient particles (CAPs) in mice: IV. Characterization of acute and chronic effects of ambient air fine particulate matter exposures on heart-rate variability. *Inhal Toxicol.* 2005;17:209–216.
376. Anselme F, Loriot S, Henry JP, Dionnet F, Napoleoni JG, Thuillez C, Morin JP. Inhalation of diluted diesel engine emission impacts heart rate variability and arrhythmia occurrence in a rat model of chronic ischemic heart failure. *Arch Toxicol.* 2007;81:299–307.
377. Wellenius GA, Coull BA, Batalha JR, Diaz EA, Lawrence J, Godleski JJ. Effects of ambient particles and carbon monoxide on supraventricular arrhythmias in a rat model of myocardial infarction. *Inhal Toxicol.* 2006;18:1077–1082.
378. Wellenius GA, Coull BA, Godleski JJ, Koutrakis P, Okabe K, Savage ST, Lawrence JE, Murthy GG, Verrier RL. Inhalation of concentrated ambient air particles exacerbates myocardial ischemia in conscious dogs. *Environ Health Perspect.* 2003;111:402–408.
379. Bartoli CR, Wellenius GA, Coull BA, Akiyama I, Diaz EA, Lawrence J, Okabe K, Verrier RL, Godleski JJ. Concentrated ambient particles alter myocardial blood flow during acute ischemia in conscious canines. *Environ Health Perspect.* 2009;117:333–337.
380. Wellenius GA, Batalha JR, Diaz EA, Lawrence J, Coull BA, Katz T, Verrier RL, Godleski JJ. Cardiac effects of carbon monoxide and ambient particles in a rat model of myocardial infarction. *Toxicol Sci.* 2004;80:367–376.
381. Ghio AJ, Hall A, Bassett MA, Cascio WE, Devlin RB. Exposure to concentrated ambient air particles alters hematologic indices in humans. *Inhal Toxicol.* 2003;15:1465–1478.
382. Mills NL, Robinson SD, Fokkens PH, Leseman DL, Miller MR, Anderson D, Freney EJ, Heal MR, Donovan RJ, Blomberg A, Sandström T, MacNee W, Boon NA, Donaldson K, Newby DE, Cassee FR. Exposure to concentrated ambient particles does not affect vascular function in patients with coronary heart disease. *Environ Health Perspect.* 2008;116:709–715.
383. Samet JM, Graff D, Berntsen J, Ghio AJ, Huang YC, Devlin RB. A comparison of studies on the effects of controlled exposure to fine, coarse and ultrafine ambient particulate matter from a single location. *Inhal Toxicol.* 2007;19(suppl):29–32.
384. Mills NL, Törnqvist H, Robinson SD, Gonzalez M, Darnley K, MacNee W, Boon NA, Donaldson K, Blomberg A, Sandstrom T, Newby DE. Diesel exhaust inhalation causes vascular dysfunction and impaired endogenous fibrinolysis. *Circulation.* 2005;112:3930–3936.
385. Mills NL, Törnqvist H, Gonzalez MC, Vink E, Robinson SD, Söderberg S, Boon NA, Donaldson K, Sandström T, Blomberg A, Newby DE. Ischemic and thrombotic effects of dilute diesel-exhaust inhalation in men with coronary heart disease. *N Engl J Med.* 2007;357:1075–1082.
386. Carlsten C, Kaufman JD, Peretz A, Trenga CA, Sheppard L, Sullivan JH. Coagulation markers in healthy human subjects exposed to diesel exhaust. *Thromb Res.* 2007;120:849–855.
387. Tan WC, Qiu D, Liam BL, Ng TP, Lee SH, van Eeden SF, D'Yachkova Y, Hogg JC. The human bone marrow response to acute air pollution caused by forest fires. *Am J Respir Crit Care Med.* 2000;161:1213–1217.
388. van Eeden SF, Yeung A, Quinlam K, Hogg JC. Systemic response to ambient particulate matter: relevance to chronic obstructive pulmonary disease. *Proc Am Thorac Soc.* 2005;2:61–67.
389. Gong H, Linn WS, Terrell SL, Anderson KR, Clark KW, Sioutas C, Cascio WE, Alexis N, Devlin RB. Exposures of elderly volunteers with and without chronic obstructive pulmonary disease (COPD) to concentrated ambient fine particulate pollution. *Inhal Toxicol.* 2004;16:731–744.

390. Brook RD, Urch B, Dvonch JT, Bard RL, Speck M, Keeler G, Morishita M, Marsik FJ, Kamal AS, Kaciroti N, Harkema J, Corey P, Silverman F, Gold DR, Wellenius G, Mittleman MA, Rajagopalan S, Brook JR. Insights into the mechanisms and mediators of the effects of air pollution exposure on blood pressure and vascular function in healthy humans. *Hypertension*. 2009;54:659–667.
391. Frampton MW, Stewart JC, Oberdörster G, Morrow PE, Chalupa D, Pietropaoli AP, Frasier LM, Speers DM, Cox C, Huang LS, Utell MJ. Inhalation of ultrafine particles alters blood leukocyte expression of adhesion molecules in humans. *Environ Health Perspect*. 2006;114:51–58.
392. Beckett WS, Chalupa DF, Pauly-Brown A, Speers DM, Stewart JC, Frampton MW, Utell MJ, Huang LS, Cox C, Zareba W, Oberdörster G. Comparing inhaled ultrafine versus fine zinc oxide particles in healthy adults: a human inhalation study. *Am J Respir Crit Care Med*. 2005; 171:1129–1135.
393. Routledge HC, Manney S, Harrison RM, Ayres JG, Townend JN. Effect of inhaled sulphur dioxide and carbon particles on heart rate variability and markers of inflammation and coagulation in human subjects. *Heart*. 2006;92:220–227.
394. Peretz A, Peck EC, Bammler TK, Beyer RP, Sullivan JH, Trenga CA, Srinouanprachnah S, Farin FM, Kaufman JD. Diesel exhaust inhalation and assessment of peripheral blood mononuclear cell gene transcription effects: an exploratory study of healthy human volunteers. *Inhal Toxicol*. 2007;19:1107–1119.
395. Barregard L, Sällsten G, Gustafson P, Andersson L, Johansson L, Basu S, Stigendal L. Experimental exposure to wood-smoke particles in healthy humans: effects on markers of inflammation, coagulation, and lipid peroxidation. *Inhal Toxicol*. 2006;18:845–853.
396. Rangasamy T, Cho CY, Thimmulappa RK, Zhen L, Srisuma SS, Kensler TW, Yamamoto M, Petrache I, Tuder RM, Biswal S. Genetic ablation of Nrf2 enhances susceptibility to cigarette smoke-induced emphysema in mice. *J Clin Invest*. 2004;114:1248–1259.
397. Ghio AJ, Kim C, Devlin RB. Concentrated ambient air particles induce mild pulmonary inflammation in healthy human volunteers. *Am J Respir Crit Care Med*. 2000;162:981–988.
398. Carlsten C, Kaufman JD, Trenga CA, Allen J, Peretz A, Sullivan JH. Thrombotic markers in metabolic syndrome subjects exposed to diesel exhaust. *Inhal Toxicol*. 2008;20:917–921.
399. Lucking AJ, Lundback M, Mills NL, Faratian D, Barath SL, Pourazar J, Cassee FR, Donaldson K, Boon NA, Badimon JJ, Sandstrom T, Blomberg A, Newby DE. Diesel exhaust inhalation increases thrombus formation in man. *Eur Heart J*. 2008;29:3043–3051.
400. Brook RD, Rajagopalan S. Particulate matter, air pollution, and blood pressure. *J Am Soc Hypertens*. 2009;3:332–350.
401. Gong H Jr, Linn WS, Sioutas C, Terrell SL, Clark KW, Anderson KR, Terrell LL. Controlled exposures of healthy and asthmatic volunteers to concentrated ambient fine particles in Los Angeles. *Inhal Toxicol*. 2003; 15:305–325.
402. Brook RD, Brook JR, Urch B, Vincent R, Rajagopalan S, Silverman F. Inhalation of fine particulate air pollution and ozone causes acute arterial vasoconstriction in healthy adults. *Circulation*. 2002;105:1534–1536.
403. Frampton MW. Systemic and cardiovascular effects of airway injury and inflammation: ultrafine particle exposure in humans. *Environ Health Perspect*. 2001;109(suppl):529–532.
404. Urch B, Silverman F, Corey P, Brook JR, Lukic KZ, Rajagopalan S, Brook RD. Acute blood pressure responses in healthy adults during controlled air pollution exposures. *Environ Health Perspect*. 2005;113:1052–1055.
405. Urch B, Brook JR, Wasserstein D, Brook RD, Rajagopalan S, Corey P, Silverman F. Relative contributions of PM2.5 chemical constituents to acute arterial vasoconstriction in humans. *Inhal Toxicol*. 2004;16:345–352.
406. Peretz A, Sullivan JH, Leotta DF, Trenga CA, Sands FN, Allen J, Carlsten C, Wilkinson CW, Gill EA, Kaufman JD. Diesel exhaust inhalation elicits acute vasoconstriction in vivo. *Environ Health Perspect*. 2008;116:937–942.
407. Shah AP, Pietropaoli AP, Frasier LM, Speers DM, Chalupa DC, Delehanty JM, Huang LS, Utell MJ, Frampton MW. Effect of inhaled carbon ultrafine particles on reactive hyperemia in healthy human subjects. *Environ Health Perspect*. 2008;116:375–380.
408. Duffin R, Tran L, Brown D, Stone V, Donaldson K. Proinflammogenic effects of low-toxicity and metal nanoparticles in vivo and in vitro: highlighting the role of particle surface area and surface reactivity. *Inhal Toxicol*. 2007;19:849–856.
409. Oberdörster G, Oberdörster E, Oberdörster J. Nanotoxicology: an emerging discipline evolving from studies of ultrafine particles. *Environ Health Perspect*. 2005;113:823–839.
410. Gong H Jr, Linn WS, Terrell SL, Clark KW, Geller MD, Anderson KR, Cascio WE, Sioutas C. Altered heart-rate variability in asthmatic and healthy volunteers exposed to concentrated ambient coarse particles. *Inhal Toxicol*. 2004;16:335–343.
411. Bartoli CR, Wellenius GA, Diaz EA, Lawrence J, Coull BA, Akiyama I, Lee LM, Okabe K, Verrier RL, Godleski JJ. Mechanisms of inhaled fine particulate air pollution-induced arterial blood pressure changes. *Environ Health Perspect*. 2009;117:361–366.
412. Wallenborn JG, McGee JK, Schladweiler MC, Ledbetter AD, Kodavanti UP. Systemic translocation of particulate matter-associated metals following a single intratracheal instillation in rats. *Toxicol Sci*. 2007;98: 231–239.
413. Phalen RF, Oldham MJ, Nel AE. Tracheobronchial particle dose considerations for in vitro toxicology studies. *Toxicol Sci*. 2006;92: 126–132.
414. Kreyling WG, Semmler-Behnke M, Möller W. Ultrafine particle-lung interactions: does size matter? *J Aerosol Med*. 2006;19:74–83.
415. Peters A, Veronesi B, Calderón-Garcidueñas L, Gehr P, Chen LC, Geiser M, Reed W, Rothen-Rutishauser B, Schürch S, Schulz H. Translocation and potential neurological effects of fine and ultrafine particles a critical update. *Part Fibre Toxicol*. 2006;3:13.
416. Mills NL, Amin N, Robinson SD, Anand A, Davies J, Patel D, de la Fuente JM, Cassee FR, Boon NA, Macnee W, Millar AM, Donaldson K, Newby DE. Do inhaled carbon nanoparticles translocate directly into the circulation in humans? *Am J Respir Crit Care Med*. 2006;173:426–431.
417. Furuyama A, Kanno S, Kobayashi T, Hirano S. Extrapulmonary translocation of intratracheally instilled fine and ultrafine particles via direct and alveolar macrophage-associated routes. *Arch Toxicol*. 2009;83:429–437.
418. Knol AB, de Hartog JJ, Boogaard H, Slottje P, van der Sluijs JP, Lebret E, Cassee FR, Wardekker JA, Ayres JG, Borm PJ, Brunekreef B, Donaldson K, Forastiere F, Holgate ST, Kreyling WG, Nemery B, Pekkanen J, Stone V, Wichmann HE, Hoek G. Expert elicitation on ultrafine particles: likelihood of health effects and causal pathways. *Part Fibre Toxicol*. 2009;6:19.
419. Ruidavets JB, Cournot M, Cassadou S, Giroux M, Meybeck M, Ferrières J. Ozone air pollution is associated with acute myocardial infarction. *Circulation*. 2005;111:563–569.
420. Bell ML, Peng RD, Dominici F, Samet JM. Emergency hospital admissions for cardiovascular diseases and ambient levels of carbon monoxide: results for 126 United States urban counties, 1999–2005. *Circulation*. 2009;120:949–955.
421. Hill AB. The environment and disease: association or causation? *Proc R Soc Med*. 1965;58:295–300.
422. Wen XJ, Balluz L, Mokdad A. Association between media alerts of air quality index and change of outdoor activity among adult asthma in six states, BRFSS, 2005. *J Community Health*. 2009;34:40–46.
423. Bell ML, Ebisu K, Peng RD, Dominici F. Adverse health effects of particulate air pollution: modification by air conditioning. *Epidemiology*. 2009; 20:682–686.
424. Pui DY, Qi C, Stanley N, Oberdörster G, Maynard A. Recirculating air filtration significantly reduces exposure to airborne nanoparticles. *Environ Health Perspect*. 2008;116:863–866.
425. Zhu Y, Eiguren-Fernandez A, Hinds WC, Miguel AH. In-cabin commuter exposure to ultrafine particles on Los Angeles freeways. *Environ Sci Technol*. 2007;41:2138–2145.
426. Fullerton DG, Bruce N, Gordon SB. Indoor air pollution from biomass fuel smoke is a major health concern in the developing world. *Trans R Soc Trop Med Hyg*. 2008;102:843–851.