Breathless in Los Angeles: The Exhausting Search for Clean Air

Population growth and the proliferation of roadways in Southern California have facilitated a glut of mobile air pollution sources (cars and trucks), resulting in substantial atmospheric pollution.

Despite successful efforts over the past 40 years to reduce pollution, an alarming set of health effects attributable to air pollution have been described in Southern California. The Children's Health Study indicates that reduced lung function growth, increased school absences, asthma exacerbation, and new-onset asthma are occurring at current levels of air pollution, with sizable economic consequences.

We describe these findings and urge a more aggressive effort to reduce air pollution exposures to protect our children's health. Lessons from this "case study" have national implications. (*Am J Public Health*. 2003;93:1494–1499)

Nino Künzli, MD, PhD, Rob McConnell, MD, David Bates, MD, Tracy Bastain, MPH, Andrea Hricko, MPH, Fred Lurmann, MS, Ed Avol, MS, Frank Gilliland, MD, PhD, and John Peters, MD

ROADWAYS ARE AN IMPORTANT feature of the built environment in the United States, one that has developed as a result of massive investment and of public policy heavily influenced by private interests. Los Angeles once had a model public transit system based on an extensive and efficient network of electric trolleys. This system, along with the streetcar systems in 45 other cities, was bought and dismantled in the 1930s by National City Lines, a holding company owned by corporate partners in the automotive industry.[1] In Los Angeles alone, the people who made 280 million passenger trips a year on the mass transit system were forced into other forms of transportation. The automobile controlled the future of Los Angeles. Today, a large proportion of the US population lives in heavily populated "mega-cities," such as the greater Los Angeles region, and depends on automobiles for transportation and diesel trucks and trains for transporting goods.

Truck and automobile emissions are responsible for most of the air pollution in Southern California, with significant additional mobile source contributions from airports and the nation's largest marine port complex. In Southern California, episodic outdoor levels of ozone (O_3), particulate matter less than 10 microns in diameter (PM_{10}), and nitrogen dioxide (NO_2) historically have been among the highest in the United States, and they continue to exceed federal and state clean air guidelines.[2,3] Research conducted in the 1970s and 1980s confirmed acute effects of exposure to ozone and other traffic-related pollutants.[4,5] However, until recently, long-term health consequences were more uncertain, particularly among children, a population with rapidly growing lungs likely to be sensitive to the effects of air pollution.

THE CHILDREN'S HEALTH STUDY

The Children's Health Study (CHS), begun in Southern California in 1993, is one of the largest and most comprehensive investigations of the long-term consequences of air pollution on the respiratory health of children.[2,6] The purpose of this article is to summarize findings and future research strategies of the CHS and to discuss traffic-related regulatory implications. We do not provide a review of the literature on the health effects of air pollution, which can be found elsewhere.[7,8] Although many air pollution studies have been conducted in the Los Angeles area, the CHS is unique in its focus on chronic effects and its repeated evaluations of prospectively followed cohorts of children. Air quality across the CHS communities is comparable to conditions in other areas of the United States (Table 1), and thus the CHS example can be generalized to these regions.

More than 6000 public school children were recruited into the CHS from 12 different communities, which maximized the diversity in air pollution concentrations and mixtures across the region.[9] In total, nearly 4000 children in the 4th, 7th, and 10th grades were recruited at the initiation of the study in 1993, and an additional 2000 4th grade schoolchildren were recruited in 1996.[2,6,10] At study entry, a questionnaire assessed demographic characteristics of the family and the child's history of asthma, hay fever, and early life respiratory illnesses, as well as outdoor and physical activities, environmental tobacco smoke exposure, housing characteristics, and the family's health history. Diet and genetic characteristics have been evaluated in subsequent years.

In addition, yearly questionnaires assess the children's development of respiratory symptoms and their current activity patterns. Furthermore, lung function has been measured each year via spirometry.[11] School absences have been monitored to allow evaluation of the effects of pollution on acute respiratory illnesses.[12]

As a means of characterizing air quality in each of the 12 study communities, ambient concentrations of O_3, $PM_{2.5}$ (particulate matter less than 2.5 microns in diameter), PM_{10}, NO_2, and acid vapors have been measured at central monitoring stations (Table 1). Particle composition has been further characterized according to ion, elemental carbon, and organic carbon mass and sources of particulate pollution.[13] New microenvironmental models were developed to assess within-community variability in children's exposure based on respondent-reported housing characteristics—such as the use of air-conditioning—as well as on

TABLE 1—Annual Means of Major Pollutants Across the 12 Children's Health Study (CHS) Communities and in Other Selected US Cities

	County or Location	$PM_{2.5}$ Mass, $\mu g/m^3$	PM_{10}, $\mu g/m^3$	O_3, ppb	NO_2, ppb
CHS community[a]					
Lompoc	Santa Barbara	5	15	28	3
Lake Arrowhead	San Bernardino	6	19	71	10
Santa Maria	Santa Barbara	7	23	22	11
Lancaster	Los Angeles	7	29	50	16
Alpine	San Diego	7	27	42	15
Atascadero	San Luis Obispo	8	19	29	12
Lake Elsinore	Riverside	12	33	36	17
San Dimas	Los Angeles	16	32	26	32
Long Beach	Los Angeles	17	37	26	31
Upland	San Bernardino	19	37	28	37
Riverside	Riverside	20	48	37	28
Mira Loma	Los Angeles	27	67	32	28
Select US cities[b]					
Honolulu	Two-county average	4	15	11	...
Miami	Two-county average	11	24	23	11
Phoenix	Four-county average	11	41	33	28
Seattle	Three-county average	12	20	17	21
Houston	Three-county average	14	33	25	18
Sacramento	Two-county average	14	23	27	17
Philadelphia	Three-county average	15	38	28	27
New York City	Two-county average	16	22	19	34
Chicago	Three-county average	17	30	24	22
Atlanta	Two-county average	20	36	35	23
AAQS[c]	United States	15	50	...	53
AAQS[c]	California	12[d]	20[d,e]	...	...[f]

[a]Average PM and NO_2 concentrations based on data collected during all months of 1999 or 2000. Average ozone concentrations were based on data collected in May–September 1999 or 2000.
[b]Data from the Environmental Protection Agency's Aerometric Information and Retrieval System database.[71]
[c]Ambient air quality standards (no annual average standard exists for ozone).
[d]New California standard (June 2002).
[e]Annual geometric mean.
[f]One-hour maximum standard only (250 ppb).

TABLE 2—Associations Between Pollutants and Respiratory Health Outcomes From the Children's Health Study

Respiratory Health Outcome	Associated Pollutants[a]	Study
Slowed lung growth	NO_2, PM_{10}, $PM_{2.5}$, HNO_3	Gauderman et al.[10,15]; Avol et al.[18]
Asthma causation	O_3	McConnell et al.[21]
Asthma exacerbation	NO_2, PM_{10}	McConnell et al.[19]
Acute respiratory illness	O_3	Gilliland et al.[12]

[a]Main pollutants provided in the cited analyses. Pollutants were usually highly correlated; thus, effects may be due to mixtures.

patterns of time spent outside and physical activity patterns that might modify ambient exposures and individual doses.[9,14]

MAIN FINDINGS

In addition to the cross-sectional findings published in 1999,[2,6] the ongoing CHS project has yielded a wealth of data from the cohort follow-up, with a major focus on the chronic effects of air pollution. Chronic effects not previously reported were observed with respect to lung function growth and asthma, and short-term effects were observed with respect to school absences (Table 2).

Lung function growth was approximately 10% slower among children living in communities with higher NO_2 levels and other traffic-related pollutants, including nitric acid vapor and particulate matter.[15] This result was replicated in the second cohort of 4th-grade schoolchildren enrolled in 1996,[10] and the effect was observed among both normal and asthmatic children. These findings are consistent with longitudinal and cross-sectional findings of other investigations.[16,17] An improvement was seen in lung function growth rates among children who moved away from the more polluted communities to areas of lower PM_{10} concentrations, and growth rate retardation was observed among those moving to areas with higher concentrations.[18]

School absence rates increased with daily fluctuations in O_3 levels, particularly when levels rose in communities with low concentrations of PM_{10} and NO_2.[12] A modest increase of 20 parts per billion in 8-hour average ozone was associated with an 83% increase in school absences resulting from acute respiratory illnesses. Children with asthma experienced more bronchitis and persistent phlegm production if they lived in communities with more NO_2 or particulate pollution.[19] This finding accords with results from the Harvard Six Cities Study.[20] Given the fact that people with asthma have more bouts of bronchitis than those without asthma, even a modest increased risk in bronchitis rates due to air pollution may result in a considerable burden in terms of increased asthma symptoms in children.[13]

Children who played team sports and spent more time outside in communities with high ozone levels had a higher incidence of newly diagnosed asthma.[21] In communities with low ozone levels, playing team sports was not associated with an increased risk of asthma. Because exercising children exhibit increased rates of ventilation, playing team sports increases doses of ozone and other lung pollutants. This finding is noteworthy, because it was previously believed that air pollution exacerbated asthma among children who already have the disease rather than causing new-onset asthma. A recent Dutch cohort study of newborn children also revealed increased asthma incidence rates among children living in more polluted communities.[22]

FUTURE RESEARCH STRATEGIES

Ongoing components of the CHS aim to determine whether deficits in lung function growth from air pollution in childhood

result in diminished maximum attained lung function (which occurs in early adulthood) and to evaluate factors, such as asthma, that may modify the effect of air pollution on attained lung function. For example, children reporting recent respiratory illnesses exhibited measurable and significant decrements in pulmonary function, decrements that were most marked in the small airways.[23] By following the cohorts into adult life and repeatedly measuring lung function, it should be possible to distinguish the main effects of acute and cumulative exposures.

Limitations of the CHS are discussed in the articles listed in Table 2. A major limitation involved the exposure assignment of community-based mean values; long-term average exposures to nitrogen oxides, acids, and particulate matter were highly correlated across the 12 CHS communities. New statistical methods and exposure models under development may help to disentangle these co-pollutant effects (K. Berhane, D.O. Stram, W.J. Gauderman, and D.C. Thomas, unpublished data, 2003) and to determine whether source-specific exposures (e.g., exposures to traffic, refineries, power plants, port activities, diesel trains, construction equipment, and wood smoke) are also important.[24,25] Pollutants that were of little concern at the time the CHS began have now been identified as important respiratory hazards and could be incorporated into future exposure assignment approaches (e.g., polycyclic aromatic hydrocarbons associated with particles from diesel exhaust[26] and ultrafine particles [less than 0.1 micron in aerodynamic diameter]).[27]

The association between ozone exposures among children playing team sports and new-onset asthma requires further study. Because asthma prevalence rates vary widely between communities for reasons that are not well understood,[28] examining within-community variability in air pollution may be an important strategy for clarifying the effects of air pollution on asthma. Preliminary results from the CHS suggest that residential proximity to traffic is associated with asthma prevalence rates.[29]

In 2002, the CHS began recruitment of a new cohort of 6000 children aged 5 to 7 years, and this cohort provides an opportunity to evaluate the laboratory observation that co-exposure to ozone or to particulate matter in diesel exhaust enhances the effect of allergens in producing asthma and allergies in animal models.[30,31] Improved techniques for modeling lung function, developed for the CHS, have demonstrated reduced lung function in asthmatic children, even before diagnosis,[32] and these methods are now being applied in an examination of the joint effects of air pollution and asthma on lung function and lung function growth at different ages (K. Berhane, D.O. Stram, W.J. Gauderman, and D.C. Thomas, unpublished data, 2003).

The evidence emerging from the CHS supports the hypothesis that genetics and diet are important for respiratory health, and the hypothesis that they may modify the effect of oxidant pollutants is under active investigation.[33,34] The observed interaction in the CHS between in utero tobacco smoke exposure and asthma prevalence and lung function is a model for similar interactions that might occur with air pollution.[35,36] The effect of in utero tobacco smoke exposure on asthma risk was observed primarily in children with a null genotype for glutathione S-transferase M1; (the null genotype results in a lack of this antioxidant enzyme).[37] Observed protective relationships of lung function with dietary magnesium and potassium[38] and with vitamin C[39] suggest potential avenues for primary prevention.[40]

REGULATORY IMPLICATIONS

The development of good public health policy is based on evaluating overall scientific evidence[7] rather than relying on findings from a single study. However, the effects of air pollution on health observed in the CHS provide an example of evidence that improvements in air quality would lessen both acute and chronic respiratory illnesses among children. According to the CHS results, the successful reductions in ozone levels in Southern California have prevented more than 2.8 million school absences involving an economic cost of more than $220 million.[41,42] The observation that lung function increased in CHS children who moved to cleaner communities (and decreased in children who moved to more polluted communities)[18] strongly suggests that chronic lung function effects are caused by air pollution. Thus, both better compliance with existing standards and further improvements in air quality are needed to protect children's health.

We distinguish 2 approaches to reducing exposure to air pollution. "Primary strategies" that reduce ambient concentrations of air pollutants must be the main focus of regulatory action, and "secondary strategies" that reduce children's exposure to air pollution without improving ambient air quality may have a complementary and temporary role (Table 3). Given traffic's dominant role in Southern California, and the fact that the CHS revealed respiratory health effects associated with a number of traffic-related pollutants, we have chosen to focus on traffic-related emissions. Mobile sources are generally the dominant national contributor to ambient urban air pollution.[43,44]

PRIMARY STRATEGIES: CUTTING EMISSIONS

Ambient air quality standards for major air pollutants are set to protect public health, and vigorous enforcement of compliance with these standards is a principal regulatory tool in the United States. The standards themselves have been based largely on acute effect studies. The California Environmental Protection Agency, for example, estimates that 400 000 episodes of upper and lower respiratory symptoms in children could be prevented each year in California alone if the new $PM_{2.5}$ standard of 12 $\mu g/m^3$ (annual mean) were met.[13] Results from the CHS and other recent studies suggest that long-term effects have been underestimated and that the benefits of meeting current standards would be even larger than the state's estimates.[13,45,46]

Compelling evidence from the CHS that lung function is impaired by air pollution is directly relevant to the current debate over the regulation of particulate pollutants. In addition, the emerging evidence that air pollution is a factor in the development of asthma is relevant to the new federal ozone standards under consideration. Nearly 70 million Americans live in areas that exceed existing ozone standards, nearly 10 million live in areas exceeding NO_2 standards, and more

TABLE 3—Examples of Primary and Secondary Policy Strategies to Reduce Children's Exposure to Traffic-Related Air Pollution

Type of Strategy	Policy Target	Intervention
Primary (reduce air pollution)	Technology	Reduce emissions in new vehicles
		Retrofit school buses and diesel trucks
		Inspect vehicle emissions of all engines
		Increase fuel economy
		Use clean fuels
		Develop zero emission vehicles
	Urban design	Invest in public transport
		Limit urban sprawl
		Build bicycle and walking paths
	Behavior	Use carpools
		Take the bus to work
		Walk/bicycle
		Use school buses or walk to school instead of driving
		Forbid idling of school buses
Secondary (reduce exposure or susceptibility)	Technology	Condition or filter air in schools
	Urban design	Limit vehicles near schools
		Separate schools from roadways
	Behavior	Avoid streets with heavy traffic
		Review guidelines for children with asthma
		Reduce outdoor activity when pollution is high
		Consider antioxidant supplements in high-pollution areas

than 20 million live in areas exceeding standards set for PM_{10}.[4] Clearly, complying with current air quality standards would benefit children's health, and the new evidence strongly endorses the strategy of the California Environmental Protection Agency, which recently set stricter standards (Table 1).[13]

Examples of interventions that would reduce pollution and help achieve compliance with air quality standards are presented in Table 3. A more extensive review of relevant vehicle technology, urban design, and behavioral changes is available elsewhere.[47] Better engine technology has dramatically reduced emissions, and new policies continue to promote this trend[46,48]; new fuel-efficient automobiles currently on the market travel 40 to 50 miles per gallon and have very low emissions, but the average new car sold in the United States is only half as efficient.[49] In 1999, fuel economy levels in the United States reached their lowest value in 15 years, a trend in large part due to an increase in sales of sport utility vehicles.[50] In 2002, automakers pushed Congress to reject any substantial legislated increase in fuel economy standards.[51] Without this regulatory pressure, there is little incentive for companies to promote more fuel-efficient cars.

There is an urgent need for incentives that lead to faster implementation of the "best available technology." However, this goal is hampered by stalled or failed regulatory policy. Delays due to prolonged legal challenges to new air quality standards, long phase-in periods for cleaner diesel engines, and exemptions and delays in holding sport utility vehicles and other larger vehicles (e.g., trucks, ships, school buses) to the same standards as smaller cars create disincentives in regard to the overall reduction of air pollution.

No single policy tool is likely to be sufficient to achieve marked reductions in air pollution. A long-term, integrated set of policies to rebuild communities to make them less dependent on fossil fuels for transportation would yield benefits that go far beyond improved health. For example, policies that promote the rapid development and implementation of very low– or zero-emission vehicles, combined with strong incentives such as emission-related taxes, road tolls, and fuel prices that would cover all direct and indirect costs of traffic (including costs related to health damage), could strongly influence consumer choice.[52,53] Such a strategy would improve children's respiratory health, mitigate the long-term threats posed by greenhouse gas emissions from mobile sources, and reduce the current heavy dependence on foreign oil.[8,54–56]

Prioritizing policies that lead to zero emission vehicle fleets would also avoid the trade-offs between health and the environment inherent in the promotion of diesel automobiles as a solution to the problem of greenhouse gas production.[57] In fact, diesel cars are associated with very little savings of energy or reduction in carbon dioxide levels,[53] and they are associated with much higher emission levels of unhealthy particulates.

The World Health Organization[58] has also proposed integrated regulatory approaches. For example, programs promoting bicycling and walking as transportation options for children[59,60] could (1) decrease automobile emissions; (2) reduce the time that children spend in cars, where rates of exposure to certain pollutants and toxic compounds are up to 10 times higher than outdoors[61]; and (3) promote healthy physical activity in the current generation of increasingly sedentary and obese children.[62]

SECONDARY STRATEGIES: REDUCING EXPOSURE, NOT EMISSIONS

Even with the most aggressive efforts to reduce emissions, the current generation of children in the Los Angeles metropolitan area will suffer adverse health effects from air pollution. Thus, policies designed to reduce children's exposure to air pollution should be considered. Examples that merit further discussion include the following:

- In communities with high pollution levels, air-conditioning or filtration in schools would reduce indoor exposure to outdoor pollutants, especially ozone.[14]
- Evidence suggests that fresh traffic exhaust is hazardous, independent of background concentrations.[29,63,64] Prudent policy would dictate that new schools, day-care centers, parks, and sports fields not be sited adjacent to roads with high traffic volumes. Re-siting of schools or changes in traffic regimens around schools with exceptionally high levels of emissions might be considered.
- Children with asthma are a susceptible group. A task force in-

volving health care professionals and air quality regulators could develop clinical guidelines for the care of asthmatic children. These guidelines should include recommendations on how to reduce exposure to ambient air pollution. This is an important public health issue, in that several CHS communities exhibit asthma prevalence rates greater than 20% and high rates of new-onset asthma in schoolchildren.[21]

• In Southern California on high pollution days, warnings are issued to schools with recommendations for children to reduce outdoor exercise. Review of the action levels triggering such warnings might be appropriate. Pollution levels can be forecast up to 5 days in advance in many urban areas, and these forecasts could be used to improve compliance with existing recommendations.

• Evidence is increasing that antioxidant intake protects children from acute oxidative damage due to air pollution exposure.[39,40,65] Consideration should be given to vitamin C supplementation in schools located in areas with high oxidant levels.

TENSIONS BETWEEN DIFFERENT REDUCTION STRATEGIES

In the long term, secondary reduction strategies are limited and have the potential to increase other public health risks. For example, limiting exercise on high pollution days to reduce doses of pollutants entering the lungs may increase the risk of diseases associated with children's increasingly sedentary lifestyles.[62] Walking to school, rather than driving with a parent, may increase children's exposure unless walking routes and traffic patterns around schools are taken into account.[66,67]

Air-conditioning in schools would increase energy consumption and emissions from power plants. Furthermore, air-conditioning may contribute to other health problems, such as sick building syndrome.[68] Although promotion of dietary antioxidant supplements such as vitamin C or E may be a promising intervention, there is some evidence that vitamin C may act as a prooxidant,[69] and further evaluation of such an intervention is required before programs could be implemented.

Finally, people's individual decisions to move to more distant, seemingly less polluted suburban areas may result in overall increased levels of emissions if commuting time increases.[70] In the long term, secondary strategies will fail to protect the public's health unless they are complementary to emission reduction strategies.[8,13,52]

CONCLUSIONS

The CHS and other studies contribute to the strong evidence that air pollution at levels permitted by current standards is harming children's health. In addition, on the basis of emerging evidence of chronic effects, risk assessments used in setting regulatory policy most likely underestimate the harm done by currently permissible levels. Our children deserve a visionary public health regulatory policy that addresses these challenges and protects them from sources of air pollution. A policy framework designed to protect children should focus on reducing emissions in the short term. Long-term policies must accomplish a decisive move toward low- to zero-emission vehicles with high fuel economy ratings. ■

About the Authors

Nino Künzli, Rob McConnell, Tracy Bastain, Andrea Hricko, Ed Avol, Frank Gilliland, and John Peters are with the Department of Preventive Medicine, Keck School of Medicine, University of Southern California, Los Angeles. David Bates is with the Departments of Medicine and Physiology, University of British Columbia, Vancouver. Fred Lurmann is with Sonoma Technology Inc, Petaluma, Calif.

Requests for reprints should be sent to Nino Künzli, MD, PhD, Department of Preventive Medicine, Keck School of Medicine, University of Southern California, 1540 Alcazar St, CHP 236, Los Angeles, CA 90033 (e-mail: kuenzli@usc.edu).

This article was accepted April 18, 2003.

Contributors

N. Künzli led the writing of the article. R. McConnell provided significant writing and technical contributions. D. Bates developed many of the foundations for the article and commented on all versions. T. Bastain contributed to research and to editing. A. Hricko provided policy implications and suggestions and contributed to editing and research. F. Lurmann and E. Avol provided technical expertise and suggestions related to exposure assessments. F. Gilliland provided technical expertise and suggestions regarding genetic epidemiology. J. Peters made significant contributions to writing and editing.

Acknowledgments

This research was supported by the California Air Resources Board (contract 94-331), the National Institute of Environmental Health Sciences (grants 1P01 ESO9581-04, 1P01 ES11627-01, and 5P30 ES07048-07), the Environmental Protection Agency (grant R 826708-01-0), and the Hastings Foundation.

References

1. Newman P, Kenworthy J. *Sustainability and Cities: Overcoming Automobile Dependence.* Washington, DC: Island Press; 1999.

2. Peters JM, Avol E, Navidi W, et al. A study of twelve Southern California communities with differing levels and types of air pollution: I. Prevalence of respiratory morbidity. *Am J Respir Crit Care Med.* 1999;59:760–767.

3. American Lung Association. State of the air 2002: grading the risk. Available at: http://www.lungusa.org/air2001/risk02.html. Accessed December 15, 2002.

4. American Thoracic Society. Health effects of outdoor air pollution. *Am J Respir Crit Care Med.* 1996;153:3–50.

5. American Thoracic Society. Health effects of outdoor air pollution: part 2. *Am J Respir Crit Care Med.* 1996;153: 477–498.

6. Peters JM, Avol E, Gauderman WJ, et al. A study of twelve Southern California communities with differing levels and types of air pollution: II. Effects on pulmonary function. *Am J Respir Crit Care Med.* 1999;159:768–775.

7. Brunekreef B, Holgate ST. Air pollution and health. *Lancet.* 2002;360: 1233–1242.

8. Bates DV, Caton RB. *A Citizen's Guide to Air Pollution.* 2nd ed. Vancouver, British Columbia: David Suzuki Foundation; 2002.

9. Navidi W, Thomas D, Langholz B, Stram D. Statistical methods for epidemiologic studies of the health effects of air pollution. *Res Rep Health Eff Inst.* 1999;86:1–50.

10. Gauderman WJ, Gilliland GF, Vora H, et al. Association between air pollution and lung function growth in Southern California children: results from a second cohort. *Am J Respir Crit Care Med.* 2002;166:76–84.

11. Enright PL, Linn WS, Avol EL, Margolis HG, Gong H Jr, Peters JM. Quality of spirometry test performance in children and adolescents: experience in a large field study. *Chest.* 2000;118:665–671.

12. Gilliland FD, Berhane K, Rappaport EB, et al. The effects of ambient air pollution on school absenteeism due to respiratory illnesses. *Epidemiology.* 2001;12:43–54.

13. *Public Hearing to Consider Amendments to the Ambient Air Quality Standards for Particulate Matter and Sulfates: Staff Report.* Sacramento, Calif: Air Resources Board, California Environmental Protection Agency; 2002.

14. Avol EL, Navidi WC, Colome SD. Modeling ozone levels in and around Southern California homes. *Environ Sci Technol.* 1998;32:463–468.

15. Gauderman WJ, McConnell R, Gilliland F, et al. Association between air pollution and lung function growth in Southern California children. *Am J Respir Crit Care Med.* 2000;162:1383–1390.

16. Frischer T, Studnicka M, Gartner C, et al. Lung function growth and ambient ozone: a three-year population study in school children. *Am J Respir Crit Care Med.* 1999;160:390–396.

17. Raizenne M, Neas LM, Damokosh AI, et al. Health effects of acid aerosols on North American children: pulmonary

function. *Environ Health Perspect.* 1996; 104:506–514.

18. Avol EL, Gauderman WJ, Tan SM, London SJ, Peters JM. Respiratory effects of relocating to areas of differing air pollution levels. *Am J Respir Crit Care Med.* 2001;164:2067–2072.

19. McConnell R, Berhane K, Gilliland F, et al. Air pollution and bronchitic symptoms in Southern California children with asthma. *Environ Health Perspect.* 1999;107:757–760.

20. Dockery DW, Speizer FE, Stram DO, Ware JH, Spengler JD, Ferris BG Jr. Effects of inhalable particles on respiratory health of children. *Am Rev Respir Dis.* 1989;139:587–594.

21. McConnell R, Berhane K, Gilliland F, et al. Asthma in exercising children exposed to ozone: a cohort study. *Lancet.* 2002;359:386–391.

22. Brauer M, Hoek G, Van Vliet P, et al. Air pollution from traffic and the development of respiratory infections and asthmatic and allergic symptoms in children. *Am J Respir Crit Care Med.* 2002; 166:1092–1098.

23. Rappaport EB, Gilliland FD, Linn WS, Gauderman WJ. Impact of respiratory illness on expiratory flow rates in normal, asthmatic, and allergic children. *Pediatr Pulmonol.* 2002;34:112–121.

24. Schauer JJ, Fraser MP, Cass GR, Simoneit BR. Source reconciliation of atmospheric gas-phase and particle-phase pollutants during a severe photochemical smog episode. *Environ Sci Technol.* 2002;36:3806–3814.

25. Schauer JJ, Cass GR. Source apportionment of wintertime gas-phase and particle-phase air pollutants using organic compounds as tracers. *Environ Sci Technol.* 2000;34:1821–1832.

26. Li N, Wang M, Oberley TD, Sempf JM, Nel AE. Comparison of the pro-oxidative and proinflammatory effects of organic diesel exhaust particle chemicals in bronchial epithelial cells and macrophages. *J Immunol.* 2002; 169:4531–4541.

27. Li N, Sioutas C, Cho A, et al. Ultrafine particulate pollutants induce oxidative stress and mitochondrial damage. *Environ Health Perspect.* In press.

28. International Study of Asthma and Allergies in Childhood Steering Committee. Worldwide variation in prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and atopic eczema: ISAAC. *Lancet.* 1998;351:1225–1232.

29. McConnell R, Berhane K, Lurmann F, et al. Traffic and asthma prevalence in children. *Am J Respir Crit Care Med.* 2002;165:A492.

30. Diaz-Sanchez D. The role of diesel exhaust particles and their associated polyaromatic hydrocarbons in the induction of allergic airway disease. *Allergy.* 1997;52(suppl):52–56.

31. Miller LA, Hyde DM, Gershwin LJ, et al. The effect of house dust mite aeroallergen and air pollutant exposures during infancy. *Chest.* 2003;123(suppl 3):434S.

32. Berhane K, McConnell R, Gilliland F, et al. Sex-specific effects of asthma on pulmonary function in children. *Am J Respir Crit Care Med.* 2000;162:1723–1730.

33. Gilliland FD, Gauderman WJ, Vora H, Rappaport E, Dubeau L. Effects of glutathione-S-transferase M1, T1, and P1 on childhood lung function growth. *Am J Respir Crit Care Med.* 2002;166: 710–716.

34. Gilliland FD, Rappaport EB, Berhane K, et al. Effects of glutathione S-transferase P1, M1, and T1 on acute respiratory illness in school children. *Am J Respir Crit Care Med.* 2002;166:346–351.

35. Gilliland FD, Li YF, Peters JM. Effects of maternal smoking during pregnancy and environmental tobacco smoke on asthma and wheezing in children. *Am J Respir Crit Care Med.* 2001; 163:429–436.

36. Gilliland FD, Berhane K, McConnell R, et al. Maternal smoking during pregnancy, environmental tobacco smoke exposure and childhood lung function. *Thorax.* 2000;55:271–276.

37. Gilliland FD, Li YF, Dubeau L, et al. Effects of glutathione S-transferase M1, maternal smoking during pregnancy, and environmental tobacco smoke on asthma and wheezing in children. *Am J Respir Crit Care Med.* 2002;166:457–463.

38. Gilliland FD, Berhane KT, Li YF, Kim DH, Margolis HG. Dietary magnesium, potassium, sodium, and children's lung function. *Am J Epidemiol.* 2002; 155:125–131.

39. Gilliland F, Li Y, Peters J. Effects of vitamin A, C, and E intake on children's lung function. *Am J Respir Crit Care Med.* 2001;163:A37.

40. Romieu I, Sienra-Monge JJ, Ramirez-Aguilar M, et al. Antioxidant supplementation and lung functions among children with asthma exposed to high levels of air pollutants. *Am J Respir Crit Care Med.* 2002;166:703–709.

41. *Latest Findings on National Air Quality: 2000 Status and Trends.* Research Triangle Park, NC: Office of Planning and Standards, US Environmental Protection Agency; 2001. EPA publication 454/K-01-002.

42. Hall JV, Brajer V, Lurmann F, Wu J. *Economic Valuation of Ozone-Related School Absences in the South Coast Air Basin.* Sacramento, Calif: California Air Resources Board; 2002. Report STI-901250-2174-FR.

43. Commission on Geosciences, Environment, and Resources. *Modeling Mobile-Source Emissions.* Washington, DC: National Academy Press; 2000.

44. *Automobile Emissions: An Overview.* Washington, DC: US Environmental Protection Agency; 1994. EPA publication 400-F-92-007.

45. *Final Report to Congress on Benefits and Costs of the Clean Air Act, 1970-1990.* Washington, DC: US Environmental Protection Agency; 1997. EPA publication 410-R-97-002.

46. *Regulatory Impact Analysis-Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements.* Washington, DC: US Environmental Protection Agency; 2000. EPA publication 420-R-00-026.

47. Mennell M, Bhattacharya KK. Air quality management. In: Bates DV, Caton RB, eds. *A Citizen's Guide to Air Pollution.* 2nd ed. Vancouver, British Columbia, Canada: David Suzuki Foundation; 2002:277–338.

48. National Research Council. *Estimating the Public Health Benefits of Proposed Air Pollution Regulation.* Washington, DC: National Academy Press; 2002.

49. Hellman KH, Heavenrich RM. *Light Duty Automative Technology and Fuel Economy Trends, 1975-2001.* Washington, DC: US Environmental Protection Agency; 2001. EPA publication 420-R-01-008.

50. *National Air Quality and Emissions Trends Report, 1999.* Research Triangle Park, NC: Office of Air Quality Planning and Standards, US Environmental Protection Agency; 2001. EPA publication 454/R-01-004.

51. Power S. U.S. proposes modest increases in fuel-economy standards. *Wall Street Journal.* December 13, 2002:B2.

52. *Strategies to Reduce Greenhouse Gas Emissions From Road Transport: Analytical Methods.* Paris, France: Organisation for Economic Cooperation and Development; 2002.

53. Schipper L, Marie-Lilliu C, Fulton L. *Diesel in Europe: Analysis of Characteristics, Usage Patterns, Energy Savings, and CO2 Emission Implications.* Paris, France: Energy Efficiency Technology Office, International Energy Agency; 2000.

54. Cifuentes L, Borja-Aburto VH, Gouveia N, Thurston G, Davis DL. Climate change: hidden health benefits of greenhouse gas mitigation. *Science.* 2001;293:1257–1259.

55. Dora C. A different route to health: implications of transport policies. *BMJ.* 1999;318:1686–1689.

56. Davis DL, Krupnick A, McGlynn G. *Ancillary Benefits and Costs of Greenhouse Gas Mitigation: Proceedings of an IPCC Co-Sponsored Workshop.* Paris, France: Organisation for Economic Cooperation and Development; 2000.

57. Ball J. Clean-air czar of California shifts to accept diesel engines. *Wall Street Journal.* October 24, 2002:A1.

58. *Transport, Environment and Health.* Copenhagen, Denmark: World Health Organization; 2000.

59. California Air Resources Board. Improving California air quality through increased bicycling: fact sheet from the California Bicycle Summit, March 5-6, 1998. Available at: http://www.arb.ca.gov/planning/tsaq/bicycle/factsht.htm. Accessed November 15, 2002.

60. Assembly Bill 1475. California State Legislature (1999).

61. Rodes C, Sheldon L, Whitaker D, et al. *Measuring Concentrations of Selected Air Pollutants Inside California Vehicles: Final Report.* Research Triangle Park, NC: Research Triangle Institute; 1998.

62. Kahn EB, Ramsey LT, Brownson RC, et al. The effectiveness of interventions to increase physical activity: a systematic review. *Am J Prev Med.* 2002; 22(suppl 4):73–107.

63. Brunekreef B, Janssen NA, de Hartog J, Harssema H, Knape M, van Vliet P. Air pollution from truck traffic and lung function in children living near motorways. *Epidemiology.* 1997;8:298–303.

64. van Vliet P, Knape M, de Hartog J, Janssen N, Harssema H, Brunekreef B. Motor vehicle exhaust and chronic respiratory symptoms in children living near freeways. *Environ Res.* 1997;74:122–132.

65. Bowler RP, Crapo JD. Oxidative stress in allergic respiratory diseases. *J Allergy Clin Immunol.* 2002;110:349–356.

66. Gulliver J. *Space-Time Modelling of Exposure to Air Pollution Using GIS* [dissertation]. Northampton, England: University of Leicester; 2002.

67. Chan LY, Lau WL, Zou SC, Cao ZX, Lai SC. Exposure level of carbon monoxide and respirable suspended particulate in public transportation modes while commuting in urban area of Guangzhou, China. *Atmospheric Environment.* 2002;36:5831–5840.

68. Wargocki P, Sundell J, Bischof W, et al. Ventilation and health in non-industrial indoor environments: report from a European multidisciplinary scientific consensus meeting (EUROVEN). *Indoor Air.* 2002;12:113–128.

69. Podmore ID, Griffiths HR, Herbert KE, Mistry N, Mistry P, Lunec J. Vitamin C exhibits pro-oxidant properties. *Nature.* 1998;392:559.

70. Frumkin H. Urban sprawl and public health. *Public Health Rep.* 2002; 117:201–217.

71. Environmental Protection Agency. Aerometric Information and Retrieval System database. Available at: http://www.epa.gov/ttn/airs. Accessed December 1, 2002.