

Healthy Air, Healthy Brains: Advancing Air Pollution Policy to Protect Children's Health

Evidence is growing on the adverse neurodevelopmental effects of exposure to combustion-related air pollution.

Project TENDR (Targeting Environmental Neurodevelopmental Risks), a unique collaboration of leading scientists, health professionals, and children's and environmental health advocates, has identified combustion-related air pollutants as critical targets for action to protect healthy brain development.

We present policy recommendations for maintaining and strengthening federal environmental health protections, advancing state and local actions, and supporting scientific research to inform effective strategies for reducing children's exposures to combustion-related air pollution. Such actions not only would improve children's neurological development but also would have the important co-benefit of climate change mitigation and further improvements in other health conditions. (*Am J Public Health*. 2019;109:550–554. doi:10.2105/AJPH.2018.304902)

Devon C. Payne-Sturges, DrPH, Melanie A. Marty, PhD, Frederica Perera, DrPH, PhD, Mark D. Miller, MD, Maureen Swanson, MPA, Kristie Ellickson, PhD, Deborah A. Cory-Slechta, PhD, Beate Ritz, MD, PhD, John Balmes, MD, Laura Anderko, RN, PhD, Evelyn O. Talbott, DrPH, Robert Gould, MD, and Iva Hertz-Picciotto, PhD, MPH

Children are exposed prenatally and in early childhood to multiple environmental stressors that can adversely affect their cognitive abilities, academic performance and consequent educational trajectories, adult health, wealth, and social status.^{1,2} Project TENDR (Targeting Environmental Neurodevelopmental Risks), a unique collaboration of leading scientists, health professionals, and children's and environmental health advocates, points to growing scientific evidence linking exposure to toxic chemicals during early brain development with brain disorders and calls on individuals, industries, and policymakers to reduce these exposures.³ Developmental disabilities, such as learning disabilities, developmental delays, autism, and attention-deficit/hyperactivity disorder (ADHD), affect one in six children in the United States, and the rate of these disorders is rising.⁴ The estimated annual cost (medical care, lost economic productivity) of environmentally mediated neurodevelopmental disorders in US children is \$74.3 billion.⁵

Evidence linking combustion-related air pollution with adverse neurodevelopment is mounting. Sources of these pollutants include fossil fuel burning for power generation and transportation, wildfires, and burning of agricultural waste. Project TENDR identified these air

pollutants—polycyclic aromatic hydrocarbons, nitrogen dioxide, fine particulate matter (PM_{2.5}, including ultrafine particulate matter [UFP]; ≤ 100 nm), and other pollutants for which nitrogen dioxide and PM_{2.5} are markers—as exemplary targets for action. The purpose of this commentary is to present Project TENDR's recommendations to reduce combustion-related air pollutant emissions to protect healthy brain development.

NEURODEVELOPMENTAL EFFECTS OF AIR POLLUTION

Air pollution exposure has been linked with preterm birth and low birth weight,^{6,7} known risk factors for many

neurodevelopmental disorders in children.^{8,9} A growing body of human studies associate exposure to combustion-related air pollutants (PM_{2.5}, polycyclic aromatic hydrocarbons, nitrogen dioxide, black carbon) with adverse effects on brain development, including deficits in intelligence, memory, and behavior.^{10–12} Polycyclic aromatic hydrocarbons, a component of PM_{2.5}, have been associated with developmental delay; reduced IQ; symptoms of anxiety, depression, and inattention¹³; ADHD; and reduced size of brain regions important for processing information and impulse control.¹⁴ Other studies have linked roadway proximity,¹⁵ traffic-related PM,¹⁶ elemental carbon, or nitrogen dioxide¹⁷ to decreased cognitive function,

ABOUT THE AUTHORS

Devon C. Payne-Sturges is with the Maryland Institute for Applied Environmental Health, University of Maryland School of Public Health, College Park. Melanie A. Marty is with the Department of Environmental Toxicology, University of California, Davis. Frederica Perera is with the Columbia Center for Children's Environmental Health, Columbia University, New York, NY. Mark D. Miller and John Balmes are with the Division of Occupational Environmental Medicine, University of California, San Francisco. Maureen Swanson is with the Learning Disabilities Association of America, Pittsburgh, PA. Kristie Ellickson is with the Minnesota State Pollution Control Agency, Saint Paul. Deborah A. Cory-Slechta is with the University of Rochester Medical Center, Rochester, NY. Beate Ritz is with the Fielding School of Public Health, University of California, Los Angeles. Laura Anderko is with the School of Nursing and Health Studies, Georgetown University, Washington, DC. Evelyn O. Talbott is with the School of Public Health, University of Pittsburgh, Pittsburgh. Robert Gould is with the School of Medicine, University of California, San Francisco. Iva Hertz-Picciotto is with the Department of Public Health Sciences, School of Medicine; the MIND Institute; and the Children's Center for Environmental Health, University of California, Davis.

Correspondence should be sent to Devon C. Payne-Sturges, DrPH, MPH, Maryland Institute for Applied Environmental Health, University of Maryland School of Public Health, 2234 L SPH, 255 Valley Dr, College Park, MD 20742 (e-mail: dps1@umd.edu). Reprints can be ordered at <http://www.ajph.org> by clicking the "Reprints" link.

This article was accepted November 24, 2018.

doi: 10.2105/AJPH.2018.304902

including deficits in memory and attention. The effect of polycyclic aromatic hydrocarbon exposures during fetal development on cognitive and behavioral outcomes is magnified by material hardship or maternal demoralization.¹⁸ Low-income communities are thus disproportionately exposed and uniquely vulnerable because of family and community economic hardship. Increasing evidence links prenatal exposure to traffic-related air pollutants¹⁹ and PM_{2.5}²⁰ to autism spectrum disorder.

Laboratory studies provide support for the neurotoxic effects of exposure to air pollutants. In mice, prenatal exposures to fine and ultrafine particles caused enlarged lateral ventricles, an early and excessive myelination pattern, an increase in the size of the corpus callosum (the bridge connecting the two brain hemispheres), and a decrease in the hippocampal area (involved in emotional regulation, spatial navigation, and memory).²¹ Prenatal diesel exhaust exposure produced inflammation in fetal brain, decreased activity, increased anxiety, and brain microglial activation (indicating a pathological process) in males as adults.²² Prenatal diesel exhaust exposure in mice reduced locomotor activity and altered levels of neurotransmitters (dopamine, norepinephrine) in a region-specific manner.²³

Chronic exposure of young adult mice to UFP produced depressive-like behaviors and impaired spatial learning and memory.²⁴ In a series of studies, postnatal UFP exposures of mice produced a pattern of developmental neurotoxicity notably similar to the hypothesized mechanistic underpinnings of autism spectrum disorder. Both sexes exposed during early

postnatal life, a period considered equivalent to the human third trimester, to concentrated ambient UFP showed disrupted development of the corpus callosum and persistent elevation of brain glutamate levels—an excitatory neurotransmitter—with effects more pronounced in males and persistent through adulthood. UFP is likely the most toxic fraction of particulate air pollution and once inhaled can migrate to the central nervous system via the nasal cavity, circulating blood, or sensory nerves found in the gastrointestinal tract.²⁵ Exposures in these studies were at levels consistent with high-traffic areas of major US cities and thus highly relevant. Although specific autism spectrum disorder-defining behaviors were not examined in this series of studies, impaired learning and short-term memory and increased impulsivity were observed.²⁶

Potential cellular mechanisms responsible for air pollution-induced neurological damage include persistent glial activation with concomitant neuroinflammation and oxidative stress.^{22,27–29} The findings in controlled laboratory studies are consistent with and provide mechanistic evidence for air pollutant effects on neurocognitive and neurobehavioral outcomes observed in humans.

RECOMMENDATIONS

Managing ambient air quality in the United States requires a joint effort between the federal and state governments as outlined in the Clean Air Act (1970; amended 1990; 42 USC §7401-7671q [2017]). After passage of the Clean Air Act, levels of six common air pollutants—PM, ozone, lead, carbon

monoxide, oxides of nitrogen and sulfur (known as criteria air pollutants)—and numerous other toxic pollutants dramatically declined while the US economy grew.^{30,31} Yet children and pregnant women across the United States are still exposed to air pollution levels above current standards.³² Regulatory actions to reduce air pollution, specifically PM, ozone, and nitrogen oxide, were spurred by abundant evidence linking air pollution to cardiovascular and respiratory disease, including premature mortality. Given the growing scientific evidence of neurodevelopmental effects, we must continue to pursue reductions in air pollution not only to reduce cardiovascular and respiratory disease but also to improve children's neurodevelopment.

The composition of pollutants differs somewhat by combustion source, but it is far more effective to focus on reducing exposure to combustion-related pollutants as a group rather than to address them one by one. Reducing combustion-related emissions would have the important co-benefit of reducing greenhouse gases. Climate change is increasing the frequency and intensity of wildfires, which cause short-term, high-level exposure to combustion-related pollutants, and is predicted to increase ozone concentrations during warm months of the year and increase combustion-related air pollution from increased energy production for air conditioning. Exposure to excessive ambient heat increases risk for premature delivery,³³ itself a risk factor for developmental delay. Given the disproportionate effects of air pollution and climate change on children, Project TENDR recommends the following actions.

Maintain and Strengthen Health Protections

Recommendation 1: The US Environmental Protection Agency (EPA) should give greater consideration to the evidence on the effects of air pollutants on neurodevelopment when setting standards for combustion-related air pollutants and when assessing the full cost of the health effects of air pollution. The EPA sets the National Ambient Air Quality Standards (NAAQS) for the criteria air pollutants, which include PM, and other combustion-related air pollutants. These standards are the driving force for reducing exposure to the criteria air pollutants in the United States. As part of the standard setting process, the EPA conducts a cost-benefit assessment. The EPA prefers to base the NAAQS and accompanying cost-benefit assessment on the health outcomes that they determine are causally associated with exposure (e.g., mortality in adults for PM_{2.5}).

Because the effects on developing brains can involve large lifelong costs to the individual, families, and society, the EPA should develop an assessment of the health and economic cost of neurodevelopmental disorders associated with PM, nitrogen oxide, and other combustion-related air pollutants, even if the evidence is limited for a particular pollutant. The benefits of reducing exposure and thus decreasing neurodevelopmental disorders should be incorporated into the cost-benefit assessment for the corresponding NAAQS.

Current PM NAAQS review. The EPA has released its draft Integrated Science Assessment on the health effects of PM_{2.5}, which could lead to revisions of the NAAQS.³⁴ The draft Integrated

Science Assessment includes a review of scientific data on neurodevelopmental health outcomes and describes the effects on the nervous system as “likely to be causal” for UFP based on strong evidence in animals of neurotoxicity and altered neurodevelopment and for PM_{2.5} based on both animal and human studies.

It is critical that the EPA include the health and economic cost of neurodevelopmental effects in the cost-benefit assessment for the PM_{2.5} NAAQS.

Recommendation 2: Strengthen and enforce federal fuel efficiency standards. The 2017 to 2025 Light-Duty Vehicle Greenhouse Gas Emission Standards and Corporate Average Fuel Economy Standards issued by the EPA and the National Highway Traffic Safety Administration reduce greenhouse gas emissions and improve fuel economy. When fully implemented, the fuel economy of new vehicles will increase from an average of about 25 miles per gallon today to about 36 miles per gallon—that means in excess of 2000 fewer gallons of gas burned over a typical vehicle lifetime and lower exhaust emissions. These standards are expected to reduce exposures to combustion-related air pollutants linked to neurodevelopmental harm.³⁵

Despite the public health benefits, these standards are under threat³⁶ and may be delayed or rolled back. These public health protections should be maintained, if not strengthened.

Advance State and Local Actions

Recommendation 3: Promote and advance clean energy policies that reduce reliance on fossil fuels, including coal, combusted for energy generation and transportation. Many states have programs in place to move toward renewable

fuels for electricity generation.³⁷ New York has instituted programs to obtain 50% of electricity generation from renewable sources by 2030; Washington, DC, and Oregon must meet that standard by 2032 and 2040, respectively.^{37–39} Hawaii and California have a goal of 100% renewable energy for electricity generation by 2045. These programs increase the share of energy generation from noncombustion sources, reduce air pollutant and greenhouse gas emissions, and push technology innovation. As states with strong programs increase electricity from renewable sources, other states could learn from those successes.

State and local governments should move toward alternatives to fossil fuels for transportation. New regional transportation plans should include zero-emission transportation technologies for passenger vehicles and goods movement (e.g., within ports and rail yards and to distribution centers). Although major automakers are now producing electric and fuel cell vehicles, supporting infrastructure is lagging. Policies that support construction of charging and hydrogen fueling stations (for fuel cells) are greatly needed. Dramatic reductions in transportation sources of air pollution are feasible and can be accelerated with public policies, as evidenced by reductions in air pollution in California from transportation sources.⁴⁰

Recommendation 4: Target existing large sources of combustion-related air pollutants for emissions reductions, dramatically reducing exposures in neighboring communities. State and regional agencies should develop best practices to guide efforts across the United States toward reducing combustion-related pollutants from large sources near residential neighborhoods, such

as major roadways, ports, and rail yards.⁴¹ Regional planning efforts involving community participants, university investigators, pollution control agencies, and industry and union representatives have resulted in emissions reductions from the ports of Los Angeles and Long Beach, California. Recommendations included replacement of older polluting trucks, clean and zero-emission truck technologies (e.g., all-electric, liquefied natural gas, and hydrogen fuel cell trucks), retrofitting older trucks with pollution controls, and managing traffic routes.⁴²

The New Jersey Department of Environmental Protection worked with a community advisory group and local industries to produce emissions inventories and assess health risk around Camden, New Jersey, an area with large industries, a port, and major roadways in close proximity to residences.⁴³ Risk reduction activities followed, including applying PM control technologies, rerouting truck traffic away from residential areas, implementing diesel idling restrictions, and planting vegetation in dusty areas.

Recommendation 5: Regional air pollution control agencies across the United States should restrict permitting new sources of combustion-related air pollutants in close proximity to residential areas and other sensitive receptors. Siting high-pollution sources near neighborhoods often affects communities with the fewest resources that are already heavily burdened, particularly communities of color. This long-documented practice gave rise to the call for environmental justice.⁴⁴

Land use and siting guidelines would be one approach states could implement to avoid poor land use choices. The California

Air Resources Board’s land use guidelines recommend against locating new residences, schools, day care centers, and playgrounds near sources of combustion-related air pollutants, such as freeways, busy roads, facilities with heavy truck traffic, or downwind of large ports or rail yards.⁴⁵ The adoption of such guidelines in all states would reduce exposures of pregnant women and children to combustion-related pollutants.

Expand Research to Inform Policies

Recommendation 6: Expand air monitoring near locations where children spend time. Near-source monitoring of combustion-related pollutants is important to accurately characterize community exposure and identify highly affected communities. Federal funds, such as EPA’s Community Air Toxics Monitoring grants, are needed to support such efforts at the state and local level. Model community-based approaches exist or are under development. Examples include UFP monitoring efforts at Seattle-Tacoma International Airport to address community concerns and provide much-needed local data on UFP concentrations.⁴⁶ New California legislation (AB617) mandates community-level monitoring of toxic pollutants in disadvantaged communities throughout the state.

More information on exposures and sources will lead to better and more cost-efficient air pollution reduction strategies.

Recommendation 7: Expand research on effectiveness of strategies to mitigate exposures near large sources of combustion-related air pollution that could guide implementation in neighborhoods close to such sources. Highways, neighborhoods, and schools are in close

proximity to one another in our nation's cities, resulting in exposures of pregnant women and children to combustion-related air pollutants. Mitigation measures that can reduce such exposures are needed. This is particularly important for low-income families and underserved racial/ethnic groups that disproportionately reside near major roadways and other sources of air pollution.⁴⁷ Various promising measures include indoor air filtration, placement of building air intake away from sources of air pollution, and vegetative or physical barriers between roadways and residences or schools.

Some evidence indicates that these measures are effective,^{48,49} but more research into optimizing such measures is needed. Furthermore, policies that implement effective measures and reduce community exposures to combustion-related air pollutants must be developed.

Recommendation 8: Increase research on the human health effects of ultrafine particles. Evidence in animals indicates that the ultrafine fraction of airborne PM is associated with serious adverse health effects, including neurological effects. Human studies to date also have found associations,^{50,51} but the lack of a monitoring network for UFP in the United States is hindering health effects research that could inform policy decisions.

Efforts are needed at the federal level to develop a UFP monitoring network. This would facilitate studies of the neurodevelopmental health effects of UFP exposures during pregnancy and childhood. Furthermore, funding is needed to support prospective epidemiological studies in birth or pregnancy cohorts to elucidate further the effects of UFP (and other

combustion-related air pollution) on neurodevelopment.

CONCLUSIONS

Public health policies that reduce combustion-related air pollution will improve not only cardiovascular and respiratory function but also neurodevelopment. This can lead to fewer children with neurodevelopmental disorders, less special education spending required, and more people participating fully in society across their life spans. Reduction of exposure to combustion-related air pollution will provide the cognitive resilience to mitigate neurodegenerative disorders during aging, including Alzheimer's disease.^{52,53} Finally, policies that reduce fossil fuel-based energy generation and transportation will reduce greenhouse gas emissions and mitigate the health effects of climate change. **AJPH**

CONTRIBUTORS

D. C. Payne-Sturges conceptualized and supervised the analysis and led the writing of the article. M. A. Marty contributed to the analysis and assisted with the writing of the article. F. Perera identified literature and assisted with the writing of the article. M. D. Miller, K. Ellickson, and J. Balmes provided guidance on policy recommendations and edits. D. A. Cory-Slechta, B. Ritz, L. Anderko, E. O. Talbot, and R. Gould contributed to the literature summary. M. Swanson and I. Hertz-Picciotto, as co-conveners of Project TENDR (Targeting Environmental Neurodevelopmental Risks), provided overall guidance on policy recommendations.

ACKNOWLEDGMENTS

This work was supported by Project TENDR (Targeting Environmental Neurodevelopmental Risks), which has received grants from John Merck Fund, Ceres Trust Fund, Passport Foundation, Pediatric Epilepsy Research Foundation, and the National Institute of Environmental Health Sciences (R13ES026504). D. C. P.-S. is supported by the National Institute of Environmental Health Sciences (award K01ES028266).

Note. The content of this article is solely the responsibility of the authors and

does not necessarily represent the official views of the funding agencies.

CONFLICTS OF INTEREST

The authors have no conflicts of interest to disclose.

REFERENCES

- Evans GW, English K. The environment of poverty: multiple stressor exposure, psychophysiological stress, and socioemotional adjustment. *Child Dev.* 2002;73(4):1238–1248.
- Landrigan PJ, Rauh VA, Galvez MP. Environmental justice and the health of children. *Mt Sinai J Med.* 2010;77(2):178–187.
- Bennett D, Bellinger DC, Birnbaum LS, et al. Project TENDR: Targeting Environmental Neuro-Developmental Risks The TENDR Consensus Statement. *Environ Health Perspect.* 2016;124(7):A118–A122.
- Boyle CA, Boulet S, Schieve LA, et al. Trends in the prevalence of developmental disabilities in US children, 1997–2008. *Pediatrics.* 2011;127(6):1034–1042.
- Trasande L, Liu Y. Reducing the staggering costs of environmental disease in children, estimated at \$76.6 billion in 2008. *Health Aff (Millwood).* 2011;30(5):863–870.
- Stieb DM, Chen L, Eshoul M, Judek S. Ambient air pollution, birth weight and preterm birth: a systematic review and meta-analysis. *Environ Res.* 2012;117:100–111.
- Lamichhane DK, Leem JH, Lee JY, Kim HC. A meta-analysis of exposure to particulate matter and adverse birth outcomes. *Environ Health Toxicol.* 2015;30:e2015011.
- Institute of Medicine. *Preterm Birth: Causes, Consequences, and Prevention.* Washington, DC: National Academies Press; 2007.
- Centers for Disease Control and Prevention: Low birth weight and the environment. October 26, 2016. Available at: <https://ephracking.cdc.gov/showRbLBWGrowthRetardationEnv.action>. Accessed February 24, 2018.
- Clifford A, Lang L, Chen R, Anstey KJ, Seaton A. Exposure to air pollution and cognitive functioning across the life course – a systematic literature review. *Environ Res.* 2016;147:383–398.
- Xu X, Ha SU, Basnet R. A review of epidemiological research on adverse neurological effects of exposure to ambient air pollution. *Front Public Health.* 2016;4:157.
- Brockmeyer S, D'Angiulli A. How air pollution alters brain development: the role of neuroinflammation. *Transl Neurosci.* 2016;7(1):24–30.
- Perera FP, Chang HW, Tang D, et al. Early-life exposure to polycyclic aromatic hydrocarbons and ADHD behavior problems. *PLoS One.* 2014;9(11):e111670.
- Peterson BS, Rauh VA, Bansal R, et al. Effects of prenatal exposure to air pollutants (polycyclic aromatic hydrocarbons) on the development of brain white matter, cognition, and behavior in later childhood. *JAMA Psychiatry.* 2015;72(6):531–540.
- Harris MH, Gold DR, Rifas-Shiman SL, et al. Prenatal and childhood traffic-related pollution exposure and childhood cognition in the Project Viva Cohort (Massachusetts, USA). *Environ Health Perspect.* 2015;123(10):1072–1078.
- Suglia SF, Gryparis A, Wright RO, Schwartz J, Wright RJ. Association of black carbon with cognition among children in a prospective birth cohort study. *Am J Epidemiol.* 2008;167(3):280–286.
- Sunyer J, Esnaola M, Alvarez-Pedrerol M, et al. Association between traffic-related air pollution in schools and cognitive development in primary school children: a prospective cohort study. *PLoS Med.* 2015;12(3):e1001792.
- Vishnevetsky J, Tang D, Chang HW, et al. Combined effects of prenatal polycyclic aromatic hydrocarbons and material hardship on child IQ. *Neurotoxicol Teratol.* 2015;49:74–80.
- Becerra TA, Wilhelm M, Olsen J, Cockburn M, Ritz B. Ambient air pollution and autism in Los Angeles County, California. *Environ Health Perspect.* 2013;121(3):380–386.
- Raz R, Roberts AL, Lyall K, et al. Autism spectrum disorder and particulate matter air pollution before, during, and after pregnancy: a nested case-control analysis within the Nurses' Health Study II Cohort. *Environ Health Perspect.* 2015;123(3):264–270.
- Klocke C, Allen JL, Sobolewski M, et al. Neuropathological consequences of gestational exposure to concentrated ambient fine and ultrafine particles in the mouse. *Toxicol Sci.* 2017;156:492–508.
- Bolton JL, Smith SH, Huff NC, et al. Prenatal air pollution exposure induces neuroinflammation and predisposes offspring to weight gain in adulthood in a sex-specific manner. *FASEB J.* 2012;26(11):4743–4754.
- Suzuki T, Oshio S, Iwata M, et al. In utero exposure to a low concentration of diesel exhaust affects spontaneous locomotor activity and monoaminergic system in male mice. *Part Fibre Toxicol.* 2010;7(1):7.
- Fonken LK, Xu X, Weil ZM, et al. Air pollution impairs cognition, provokes depressive-like behaviors and alters hippocampal cytokine expression and

- morphology. *Mol Psychiatry*. 2011;16(10):987–995,973.
25. Block ML, Elder A, Auten RL, et al. The outdoor air pollution and brain health workshop. *Neurotoxicology*. 2012;33(5):972–984.
26. Allen JL, Oberdorster G, Morris-Schaffer K, et al. Developmental neurotoxicity of inhaled ambient ultrafine particle air pollution: parallels with neuropathological and behavioral features of autism and other neurodevelopmental disorders. *Neurotoxicology*. 2017;59:140–154.
27. Allen JL, Liu X, Pelkowski S, et al. Early postnatal exposure to ultrafine particulate matter air pollution: persistent ventriculomegaly, neurochemical disruption, and glial activation preferentially in male mice. *Environ Health Perspect*. 2014;122(9):939–945.
28. Allen JL, Liu X, Weston D, et al. Developmental exposure to concentrated ambient ultrafine particulate matter air pollution in mice results in persistent and sex-dependent behavioral neurotoxicity and glial activation. *Toxicol Sci*. 2014;140(1):160–178.
29. Levesque S, Taetzsch T, Lull ME, et al. Diesel exhaust activates and primes microglia: air pollution, neuroinflammation, and regulation of dopaminergic neurotoxicity. *Environ Health Perspect*. 2011;119(8):1149–1155.
30. US Environmental Protection Agency. *The Benefits and Costs of the Clean Air Act From 1990 to 2020*. Washington, DC: Office of Air and Radiation; 2011.
31. Samet JM, Burke TA, Goldstein BD. The Trump Administration and the environment – heed the science. *N Engl J Med*. 2017;376(12):1182–1188.
32. US Environmental Protection Agency. America’s children and the environment: environments and contaminants – criteria air pollutants. January 2018. Available at: <https://www.epa.gov/ace/ace-environments-and-contaminants-criteria-air-pollutants>. Accessed February 24, 2018.
33. Avalos LA, Chen H, Li DK, Basu R. The impact of high apparent temperature on spontaneous preterm delivery: a case-crossover study. *Environ Health*. 2017;16(1):5.
34. US Environmental Protection Agency. *Integrated Science Assessment (ISA) for Particulate Matter (External Review Draft)*. Washington, DC: US Environmental Protection Agency; 2018.
35. US Environmental Protection Agency. *Regulatory Impact Analysis: Final Rulemaking for 2017–2025 Light-Duty Vehicle Greenhouse Gas Emission Standards and Corporate Average Fuel Economy Standards*. Washington, DC: Assessment and Standards Division Office of Transportation and Air Quality; 2012.
36. US Environmental Protection Agency. EPA, DOT open comment period on reconsideration of GHG standards for cars and light trucks. August 10, 2017. Available at: <https://www.epa.gov/newsreleases/epa-dot-open-comment-period-reconsideration-ghg-standards-cars-and-light-trucks>. Accessed August 10, 2017.
37. National Conference of State Legislatures. State renewable portfolio standards and goals. July 20, 2018. Available at: <http://www.ncsl.org/research/energy/renewable-portfolio-standards.aspx>. Accessed August 1, 2018.
38. California Energy Commission. California Renewable Energy Overview and Programs. 2017. Available at: <http://www.energy.ca.gov/renewables>. Accessed August 1, 2018.
39. New York State Energy Research and Development Authority. Renewable portfolio standard. Available at: <https://www.nyserda.ny.gov/All-Programs/Programs/Clean-Energy-Standard/Renewable-Portfolio-Standard>. Accessed November 8, 2018.
40. Office of Environmental Health Hazard Assessment. *Gasoline-Related Air Pollutants in California: Trends in Exposure and Health Risk 1996 to 2014*. Sacramento: California Environmental Protection Agency; 2018.
41. Hricko A, Rowland G, Eckel S, Logan A, Taher M, Wilson J. Global trade, local impacts: lessons from California on health impacts and environmental justice concerns for residents living near freight rail yards. *Int J Environ Res Public Health*. 2014;11(2):1914–1941.
42. Ports of Los Angeles and Long Beach. San Pedro Bay Ports Clean Air Action Plan. Available at: <http://www.cleanairactionplan.org>. Accessed November 8, 2018.
43. New Jersey Department of Environmental Protection. *Camden Waterfront South Air Toxics Pilot Project*. Trenton, NJ: Division of Air Quality; 2005.
44. Bullard RD, Wright BH. Environmental justice for all: community perspectives on health and research needs. *Toxicol Ind Health*. 1993;9(5):821–841.
45. California Air Resources Board. *Air Quality and Land Use Handbook: A Community Health Perspective*. Sacramento: California Air Resources Board; April 2005.
46. Seto E. WA State Airport Community Air Quality Study. July 18, 2017. Available at: <http://setoresearch.dyndns.org/website/researchblog/?p=857>. Accessed April 8, 2018.
47. Brugge D, Patton AP, Bob A, et al. Developing community-level policy and practice to reduce traffic-related air pollution exposure. *Environ Justice*. 2015;8(3):95–104.
48. Chen L, Liu C, Zhang L, Zou R, Zhang Z. Variation in tree species ability to capture and retain airborne fine particulate matter (PM_{2.5}). *Sci Rep*. 2017;7(1):3206.
49. Miller SL, Facciola NA, Toohy D, Zhai J. Ultrafine and fine particulate matter inside and outside of mechanically ventilated buildings. *Int J Environ Res Public Health*. 2017;14(2):pii:E128.
50. Lanzinger S, Schneider A, Breitner S, et al. Associations between ultrafine and fine particles and mortality in five central European cities – results from the UFIREG study. *Environ Int*. 2016;88:44–52.
51. Peters A, Hampel R, Cyrus J, et al. Elevated particle number concentrations induce immediate changes in heart rate variability: a panel study in individuals with impaired glucose metabolism or diabetes. *Part Fibre Toxicol*. 2015;12(1):7.
52. Ritz B, Lee PC, Hansen J, et al. Traffic-related air pollution and Parkinson’s disease in Denmark: a case-control study. *Environ Health Perspect*. 2016;124(3):351–356.
53. Chen H, Kwong JC, Copes R, et al. Exposure to ambient air pollution and the incidence of dementia: a population-based cohort study. *Environ Int*. 2017;108:271–277.