

Prevention of Disability in Children: Elevating the Role of Environment

Stephen A. Rauch and Bruce P. Lanphear

Summary

Much public attention and many resources are focused on medical research to identify risk factors and mitigate symptoms of disability for individual children. But this focus will inevitably fail to *prevent* disabilities. Stephen Rauch and Bruce Lanphear argue for a broader focus on environmental influences that put entire populations at risk. They argue that identifying and eliminating or controlling environmental risk factors that incrementally increase the prevalence of disability is the key to preventing many disorders.

Rauch and Lanphear examine emerging evidence that many disabilities of childhood have their roots in the environment—from toxins in air, water, and soil, to the stressors of poverty, to marketing practices that encourage unhealthy choices or discourage healthy ones. They review research on well-known environmental causes of disability, such as exposures to lead, cigarette smoke, and industrial air pollution. They point to new evidence suggesting that chemicals found in commonly used plastics may have subtle but serious effects on child development, and that many disabilities spring from the complex interplay of environmental risk factors and genetic susceptibility.

Rauch and Lanphear make a case for turning our attention to societal or population-level interventions that would rely less on medical and genetic technology and more on policies and regulations that would reduce children's exposure to ubiquitous environmental risks. Examples include required testing of new chemicals for developmental toxicity before they are put on the market; zoning regulations that separate residential communities from industrial areas; and restrictions on advertising of unhealthy products, such as tobacco, alcohol, and junk foods, to children. Rauch and Lanphear outline and assess the effectiveness of interventions that could be adopted, and suggest what a healthy modern community might look like. Such interventions, they acknowledge, are likely to be highly controversial, require both long-term investments and shifts in societal thinking, and produce less well-defined outcomes than individual medical treatments. But in the long run, the authors contend, such interventions could prevent many of the disabilities that now afflict millions of children and adults.

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Americans have an unwavering belief that advances in biomedical technology and medical care will solve their health problems. With few exceptions, however, the best these can achieve is enhanced treatment of existing diseases or disabilities. It would be far better to *prevent* disabilities from developing in the first place. For most clinicians, “prevention” usually occurs in a clinical setting and seeks to identify signs, symptoms, or risk factors for a disability in an individual child. In contrast, a strategy that focused on prevention would concentrate on reducing environmental influences that put entire populations at risk. Identifying, and either eliminating or controlling, widespread exposures to modifiable environmental risk factors that incrementally increase the prevalence of disability in a population is the key to preventing many disorders in children and adults.

For the most common childhood conditions, primary prevention may best be achieved through universal and nonmedical interventions.

In this article, we examine the emerging evidence showing that many prevalent disabilities of childhood have their roots in environmental influences, and we make a case for devoting more attention to societal or population-level interventions. These interventions would rely less on medical and

genetic technology and more on recommendations, policies, and regulations that would reduce children’s exposure to ubiquitous environmental risks. Such interventions are likely to be highly controversial, require long-term investments as well as shifts in societal thinking, and have less well-defined outcomes than individual medical treatments. But in the long run, they could prevent many of the disabilities that currently afflict millions of children and adults.

Primary versus Secondary Prevention

Prevention occurs at three levels. Primary prevention seeks to keep disabilities from developing in the first place. Secondary prevention consists of methods of screening and early detection to identify problems early, before they can do too much damage (the “nip it in the bud” approach). Tertiary prevention deals with restoring health and function to people who have already developed a disability. Secondary and tertiary prevention efforts—which are the focus of clinic-based prevention—typically involve screening or treatment. Screening and treatments are beneficial for individuals who are sick, but primary prevention is essential to reduce the prevalence of disability in a population.

The medical community is currently devoting considerable attention and resources to personalized predictive medicine—the identification of genetic markers that make a particular individual susceptible to a specific illness or disability, with the ultimate goal of tailoring therapies to individual patients. These efforts have led to early identification and some promising treatments for specific conditions such as cystic fibrosis.¹ Useful clinical applications have thus far been few in number, however. Overreliance on gene discovery and personalized predictive medicine

may disproportionately benefit those in the best position to take advantage of the new innovations and exacerbate the already gaping socioeconomic disparities in health by draining resources away from underfunded population-level interventions that benefit everyone (see also the article by Paul Wise in this volume).²

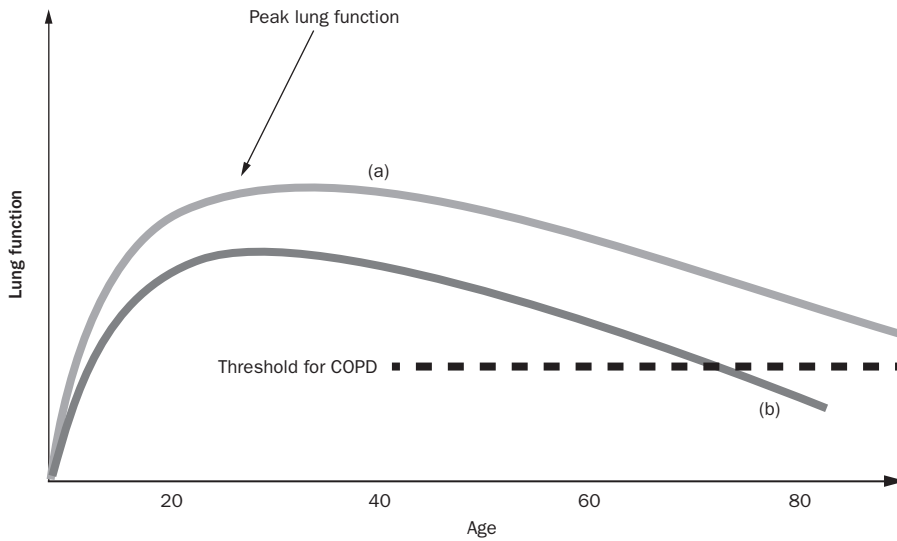
Moreover, the causes of many disabilities in childhood are complex and result from the interplay of environmental risk factors and genetic susceptibility; purely genetic or purely environmental disabilities exist but are rare.³ For the most common childhood conditions, primary prevention may best be achieved through universal and nonmedical interventions. As Geoffrey Rose, a pioneer in the science of prevention, wrote provocatively, “If causes can be removed, susceptibility ceases to matter.”⁴

A key example of Rose’s dictum is the dramatic decline in infant and child mortality and the subsequent rise in life expectancy in the United States over the past century. One explanation for this shift, often touted to support investments in biomedical research, credits the development of vaccines, antibiotics, and other advances in medical technology. The greatest progress in reducing deaths from many infectious diseases and extending life expectancy, however, occurred decades before the discovery or introduction of effective medical treatments.⁵ John and Sonja McKinlay, among others, have shown that clean water, sanitation, and changes in living conditions led to the initial improvements in public health. Especially in cramped and unsanitary urban slums, which spawned epidemics of typhoid, cholera, and tuberculosis, it was social reform and environmental engineering, not medical advances, that reduced poor health and increased life

expectancy. Thus, while vaccines, antibiotics, and the development of neonatal intensive care have played a significant role in the continuing decline in infant and child mortality, the overall decline has had more to do with establishing a clean water supply than with any “medical” factor.⁶

Notably, few of the early “sanitarians” or bacteriologists understood the exact mechanisms by which disease was transmitted. Rather, they drew conclusions after observing the patterns of disease, which gave them sufficient information to act, even in the absence of conclusive knowledge of a mechanism. Knowing the mechanism through which environmental influences cause disease can enhance prevention and public health, however, and genetic research can be helpful in this regard.⁷ For example, being the victim of maltreatment (or child abuse) has been shown to be a risk factor for antisocial behavior, but questions about the causal relationship persisted because the underlying mechanism was unclear.⁸ One study found that males who were maltreated in childhood were more likely to exhibit violent or antisocial behavior in adolescence and young adulthood. But the risk was primarily observed in men who had a particular variant of the gene coding for monoamine oxidase A (MAOA), an enzyme that breaks down neurotransmitters.⁹ While several studies have confirmed the role of MAOA in conferring susceptibility, there already was, of course, sufficient evidence of the adverse consequences of maltreatment to prevent it *without* understanding the mechanism. Similarly, while it might be desirable and useful to understand the exact way that exposure to recognized hazards such as air pollution leads to disease and disability before regulating that exposure, it is not essential.

Figure 1. Schematic Curve of Lung Function over a Person's Lifetime, with Normal and Impaired Growth



Source: Adapted from Lei Wang and Kent E. Pinkerton, "Detrimental Effects of Tobacco Smoke Exposure during Development on Postnatal Lung Function and Asthma," *Birth Defects Research (Part C)* 84 (2008): 54–60.

Note: Curve (a) represents normal growth and decline in lung function. Curve (b) represents a person with impaired growth caused by exposure to environmental pollutants such as prenatal tobacco smoke or airborne fine particulates. This person has a lower peak lung function, leading to an earlier onset of chronic obstructive pulmonary disease symptoms in old age.

Why Focus on Preventing Disabilities in Children?

Children are particularly vulnerable to environmental stressors; they pass through several delicate developmental stages and, pound for pound, they eat and breathe more environmental contaminants than adults.¹⁰ An exposure that is innocuous in adults can have a dramatic effect when it occurs during fetal development or early childhood. For example, in the mid-1900s thalidomide was prescribed to treat morning sickness in thousands of pregnant women, at doses that were nontoxic for adults. But the drug had devastating effects on their fetuses, especially when administered between twenty-seven and forty days after conception, when limb development occurs. While thalidomide causes gross deformities, many other environmental exposures that occur during fetal

development and childhood can have substantial lifelong implications among a population of children, even if the effects are subtle for an individual child. Unfortunately, these effects are much less likely to be recognized and addressed; David Rall, former director of the National Institute of Environmental Health Sciences, once remarked, "If thalidomide had caused a ten-point loss of IQ instead of obvious birth defects of the limbs, it would probably still be on the market."¹¹

Exposures that occur during fetal development or early childhood can obstruct or retard normal function. Children's lungs, for example, continue to develop from birth throughout adolescence, and lung function increases throughout childhood, reaching a peak in the late teens or early twenties (figure 1). Then it plateaus for several years before it begins to gradually decline in older

adulthood. Several recognized risk factors can alter lung function over the life span. One risk is prenatal exposure to tobacco smoke, which lowers lung function in childhood as well as peak lung function.¹² Similarly, exposure to air pollution has been associated with reduced lung function.¹³ Children who face multiple risk factors that diminish their lung function are at higher risk of meeting a threshold associated with chronic respiratory disease. Put another way, insults in early development can impair or obstruct function throughout the life span, leading to diminished function and accelerated disability at older ages. This relationship between early-life insults and later disability occurs in other chronic conditions, such as lead-associated cognitive deficits and dementia, or lower bone mineral density and earlier-onset osteoporosis associated with calcium-deficient diets.¹⁴

Although Americans' life expectancy has increased, so have the years many of them live with a disability or chronic disease.¹⁵ Many factors undoubtedly account for this increase in disability, but environmentally induced conditions incurred during childhood can compound throughout a person's lifetime and express themselves as chronic diseases in adulthood or old age. Today's increases in childhood obesity will lead to tomorrow's epidemics of diabetes in young adults and to cardiovascular disease in middle-aged or older adults.¹⁶ The emerging evidence thus suggests that preventing the development of chronic disease in adults requires improving the health of children. That, in turn, will require dramatic shifts of resources for a country that spends the vast majority of its health care dollars for medical treatment of the elderly.¹⁷

Another reason to focus on prevention in children is because disabilities in children

are on the rise (see the article in this volume by Neal Halfon and others).¹⁸ The definition of disability varies depending on the survey used, but the number of children diagnosed with an activity limitation stemming from a chronic health condition rose from 1.8 percent in 1960 to 7.3 percent in 2006, while the prevalence of diagnosed developmental disabilities rose from 12.8 percent in 1997–99 to 15 percent in 2006–08.¹⁹ Many of the most common disabilities, including asthma, premature birth, autism, attention-deficit/hyperactivity disorder (ADHD), and obesity, appear to be on the rise.²⁰

A shift to prevention of disabilities should be attractive to policy makers because prevention reduces health care and societal costs, as well as alleviating human suffering. For example, lead in house paint is known to be associated with lower IQ and ADHD in children and with criminal behavior in adulthood.²¹ Policy makers may balk at requiring homeowners, landlords, and others to undergo the expense and effort of removing the hazard, yet a cost-benefit analysis concluded that every \$1 spent to reduce lead hazards in housing would produce between \$17 and \$221 in benefits by reducing expenditures on screening and treatment for lead toxicity, ADHD treatment, and special education; increasing income and tax revenue; and reducing crime. The analysis estimated a total potential net savings from the elimination of lead hazards of \$118 billion to \$269 billion.²² Another study estimated the cost of disease from exposure to pollutants linked with asthma, cancer, and neurobehavioral disorders at \$76.6 billion in a single year (2008).²³

The Epidemiologic Transition and the Emergence of the New Morbidities

To understand the causes of the “new morbidities,” or disabilities, of childhood, it is

useful to examine trends in patterns of disease and disability over the past century. As noted, in the early 1900s public health concerns were dominated by epidemics of infectious disease, overt nutritional deficiencies, and infant mortality associated with poor urban living conditions. With the advent of public water and sanitation systems, pasteurization of milk, and housing safety codes, death rates fell sharply, especially among infants and children, and life expectancy in the United States increased from forty-seven years in 1900 to sixty-eight years by 1950, and to seventy-eight years in 2007.²⁴ Moreover, the pattern of mortality and morbidity shifted from infectious diseases to chronic conditions such as cardiovascular disease and cancer, a shift commonly known as the epidemiologic transition.²⁵ In recent years, a similar transition has been taking place for children, as the burden of illness and disability shifts from infectious disease to chronic conditions including asthma, obesity, and mental health and neurobehavioral problems such as autism and ADHD.

Risk Factors for Disabilities in Children

Many harmful exposures, from toxic exposures to marketing practices to social inequities, have been recognized as contributing to the rise in child disability.

Poverty

Poverty is one of the most significant risk factors for disabilities and is especially troubling because one-fifth of all children in the United States were living in poverty in 2010.²⁶ Linking disability with poverty is hardly new, but the relationship is just as powerful with chronic conditions as with infectious disease.²⁷ Poverty affects health on several levels: directly, through the psychological stress and social stigmatization that accompany

living in poverty, and indirectly, through increased exposure to a wide range of environmental stressors such as pollution, crime, and lack of access to healthful food.²⁸ People living in poor neighborhoods, especially racial or ethnic minorities, also face disproportionately high exposures to toxic and hazardous wastes, air pollution, contaminated water, and unsafe housing.²⁹

Even without the deprivations of poverty, people may still suffer from being on the lower rungs of the social ladder. Michael Marmot described the phenomenon of a “social gradient,” a direct, linear relationship between health and position in the social hierarchy, while examining members of the British civil service.³⁰ These effects have been found elsewhere, including among children; moreover, the gradient appears to grow sharper (that is, the health of rich and poor diverges further) as children age, and “the adverse health effects of lower income accumulate over children’s lives.”³¹

In addition to the harmful effects of poverty, it has been argued that the overall *level* of inequality in a society also affects health. Richard Wilkinson and Kate Pickett have shown that countries with greater social inequities experience poorer health than more egalitarian countries on almost all available measures, including life expectancy, infant mortality and child health, obesity, and mental health; the United States, with its wide gaps between rich and poor, fares worse than most other developed countries, a difference that persists even when only wealthy individuals are considered.³² In other words, poor Americans fare much worse than wealthy Americans, but even wealthy Americans fare worse than wealthy (and even middle-class) residents of many other countries.³³

Eliminating poverty would likely dramatically improve the overall health of the nation's population, but the changes in the structure of society required to significantly reduce poverty appear to be beyond the typical range of public policies. Indeed, efforts in the United States to address poverty on a national scale have stalled or lost ground in recent decades. Another approach to mitigating the negative health effects of poverty would ask *how* being poor leads to worse health (toxic exposures, psychological stress, lack of medical care) and then develop interventions that address those specific risks.

Airborne and Other Environmental Pollutants

Just as the deplorable conditions of Victorian-era slums led to insights into the causes and control of infectious disease, environmental disasters and epidemics over the past century have linked exposures to industrial pollutants and environmental chemicals with overt toxicity. In Queensland, Australia, an epidemic of childhood lead poisoning in the early 1900s was traced to lead in house paint, establishing the link that still haunts residents of older housing in many countries around the world.³⁴ In December 1952, a dense fog of sulfurous particles from burning coal enveloped London for five days, leading to an estimated 12,000 deaths, mostly from respiratory or cardiovascular disease; children and older adults were especially vulnerable to the sulfur-laden coal smoke.³⁵ This disaster—and a similar one in Donora, Pennsylvania, in 1957—began to focus people's attention on the harmful effects of air pollution, ultimately spurring the development of environmental regulations regarding levels of particulate matter. In the 1950s and 1960s, cases of severe congenital defects in the Japanese town of Minamata Bay were traced to mercury emissions from a local plastics factory.³⁶

Scientists also have taken advantage of other “natural experiments” to test associations between health and air pollution. In the late 1980s, for example, C. Arden Pope and his colleagues showed that the closing of a Utah steel mill led to lower levels of airborne particles and lower mortality and hospitalizations.³⁷ In 1996 the summer Olympic Games in Atlanta reduced traffic there, which led to lower air pollution and fewer hospitalizations.³⁸ More recently, the introduction of E-ZPass, an electronic highway toll collection method, reduced traffic congestion and lowered the incidence of preterm birth and low birth weight by an estimated 6 to 9 percent among babies living within two kilometers of toll plazas along three major roadways in New Jersey and Pennsylvania.³⁹

Airborne pollutants are known to contribute to other debilitating illnesses in both children and adults, including asthma. The most common childhood chronic condition in the United States, asthma affected an estimated 9.7 percent of American children in 2009.⁴⁰ The disease, which is characterized by airway inflammation, difficulty breathing, and reduced respiratory function, takes a heavy physical and psychological toll on those affected. Its prevalence has risen steadily in most Western countries since the 1980s, although it seems to have leveled off in the past decade.⁴¹ The reasons for this pattern are not entirely clear, but airborne particles smaller than 2.5 microns (also called PM 2.5 or fine particles) have been associated with impaired lung function and asthma exacerbations.⁴² Exposure to prenatal smoking and secondhand smoke is also associated with impaired lung development, reduced lung function, and asthma, and other studies have linked airborne pollutants to preterm birth and lower birth weight as well as to chronic cough and bronchitis.⁴³

These harmful effects of air pollution on respiratory function are well established. More recent studies are now finding links between exposure to air pollutants and reduced cognitive development. Black carbon (an airborne product of combustion from fossil fuels and other sources) has been associated with lower verbal and nonverbal intelligence and poorer memory performance in a Boston-based birth cohort of children aged eight to eleven.⁴⁴ Frederica Perera and others, using polycyclic aromatic hydrocarbons (PAHs) as a biomarker, found that children with higher exposures to combustion products had diminished cognitive abilities.⁴⁵ These links between airborne toxins and cognitive performance are less established, but they fit a larger pattern of toxic exposures interfering with brain development in young children.

The use of exposure biomarkers, which measure the amount or internal dose of a pollutant in the body, has allowed scientists to directly quantify the effects of exposures encountered by the general population.⁴⁶ The increasing use of biomarkers is showing that industrial pollutants and environmental chemicals are not only harmful at the higher levels of exposure but at lower concentrations as well. For example, lead has long been associated with poorer intellectual development in children, but more recently exceedingly low blood lead levels (fewer than five micrograms a deciliter) have been linked with lower IQ scores. Even more troubling, the observed decrements in intellectual abilities are proportionately greater at the lowest blood lead levels, indicating that there is no “safe” level of exposure.⁴⁷ Similarly, pregnant women are at risk of giving birth prematurely not only if they are smokers but if they are exposed to secondhand tobacco smoke.⁴⁸ Scientists looking for “safe” levels of fine particles in air pollution found a steady relationship with

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adult mortality down to the lowest detectable levels.⁴⁹ Thus, for some of the most established pollutants, increasing evidence of toxicity is appearing even at the lowest levels of exposure. Moreover, while it was once thought that only workers and urban dwellers were exposed to these industrial pollutants, it is now realized that these contaminants are ubiquitous: virtually no one is unexposed.⁵⁰

The Rise of Autism: More Questions than Answers

The incidence of autism, one of the most disabling conditions of childhood, has increased dramatically in recent years, although it remains rare in comparison to conditions such as ADHD. An exhaustive study of California’s birth and medical-service records reported an increase in the rate of autism diagnosis before the age of five from 6.2 per 10,000 births in 1990 to 42.5 in 2001. While some of this rise was explained by changes in diagnostic practices and an increased awareness of autism, these factors alone did not account for the dramatic increase in autism.⁵¹

Very little is known about risk factors for autism or autistic behaviors. While autism is believed to have a genetic component, such a rapid increase in prevalence points to an

increase in one or more environmental risk factors. The little evidence available suggests the risk increases for mothers who live near a freeway during the third trimester of pregnancy and decreases for mothers who take prenatal vitamins in the period around conception.⁵² Other suspected causes of autism, such as mercury in childhood vaccines, have not been supported by the evidence.⁵³ Autism may be a “test case” for the ubiquity and variety of man-made chemicals, many of which have never been tested for their health effects in humans, especially children.⁵⁴ While any links between environmental chemicals and autism are speculative, it would not be surprising if a chemical (or combination of chemicals acting synergistically) were contributing to this heightened autism prevalence. It is worth asking whether a revision of the regulatory framework for environmental chemicals might begin to control the autism epidemic, even before the responsible toxicant(s) is identified.

Linking Environmental Toxicants to Psychopathology

Researchers are increasingly finding links between exposures to environmental toxicants and neurobehavioral disorders, one of the most rapidly rising categories of disabilities in children; one such disorder is ADHD, which affects almost one in ten children.⁵⁵ Using a nationally representative sample, for example, Tanya Froehlich and her colleagues estimated that children with blood lead concentrations in the highest tertile—above 1.3 micrograms per deciliter ($\mu\text{g}/\text{dl}$)—were two and a half times as likely as children with the lowest blood lead concentrations (less than 0.8 $\mu\text{g}/\text{dl}$) to meet criteria for ADHD.⁵⁶ This finding is particularly disturbing because blood lead levels above 1.3 micrograms per deciliter are far below the current “level of concern” of 10 $\mu\text{g}/\text{dl}$.

Similarly, children who were prenatally exposed to tobacco were nearly two and a half times more likely to meet criteria for ADHD than children whose mother did not smoke during pregnancy. Furthermore, lead and tobacco exposures interacted synergistically; children in the highest lead category who were also prenatally exposed to tobacco smoke were eight times as likely to meet diagnostic criteria for ADHD as children with neither exposure. Several other lines of evidence link lead exposure with neurobehavioral disorders. Neuroimaging studies, for example, have associated lead exposure with reduction in gray matter volume in the prefrontal cortex, a key area of the brain necessary for executive functions, impulse control, and decision making.⁵⁷ Another study cites declining blood lead levels as the primary reason for the decline in homicides and other criminal behaviors over the past thirty years.⁵⁸

Although the evidence is less definitive, other chemicals, such as organophosphate pesticides, mercury, and polychlorinated biphenyls (PCBs), have also been linked to the development of ADHD.⁵⁹ While the use of biomarkers has allowed scientists to connect environmental exposures to disabilities in children, the long latency between exposure and disability makes it difficult to establish these links with certainty. Still, these studies raise serious questions about the need to revise the existing regulatory framework—which essentially allows children to be exposed to suspected toxicants or chemicals until there is definitive proof of their toxicity.

The Emergence of Endocrine Disruptors

One emerging area of concern is a class of chemicals known as “endocrine disruptors” because of their ability to mimic natural hormones.⁶⁰ Evidence from several recent studies has linked prenatal exposure to

phthalates and bisphenol A (BPA)—ubiquitous, estrogenic-mimicking chemicals found in plastics—with endocrine-sensitive outcomes such as decreased anogenital distance (a condition linked to infertility), decreased “masculine” play in boys, and externalizing behavioral problems in girls; this link suggests that endocrine disruptors can alter neurological development.⁶¹ While most evidence on the effects of endocrine disruptors concerns sex hormones or the thyroid, some endocrine disruptors (known as “obesogens”) may mimic other hormones, including those involved in the development of obesity. The role of obesogens in the obesity epidemic is still speculative, but a chemical called tributyltin has been identified as a possible obesogen in some animal studies.⁶² In addition, one national cross-sectional study found associations between body mass index—a measure of obesity—and phthalates in adolescent girls.⁶³

The Rise of Obesity and Diabetes

Americans have become steadily heavier over the past thirty years. In a nationally representative sample taken in 2007–08, almost 17 percent of children and adolescents aged two to nineteen were classified as obese, up from 5 percent in 1971–74 and 10 percent in 1988–94.⁶⁴ Obese children are more likely to become obese adults, who are at heightened risk for type II diabetes, cardiovascular disease, and some cancers.⁶⁵ Diabetes is also on the rise in young people, where it can have especially serious health consequences (compared with a later onset).⁶⁶ Being obese can also have profound psychosocial effects on children; one study found that obese children fared as badly or worse on several measures of psychological functioning and stress as children who had cancer.⁶⁷

For conditions such as obesity and diabetes, the dialogue surrounding prevention typically

focuses on individual “lifestyle choices.”⁶⁸ It is easy to blame a person for eating too much, getting too little exercise, or smoking cigarettes. But lifestyle choices depend to a large extent on social context, a point that is too often unacknowledged. For children, whose preferences are still developing and who are open to a wide range of influences, it is easy to see how their “choices” may be manipulated by outside factors.

Marketing and advertising are staples of modern life, affecting how each of us views and interacts with the world. This is especially true for children. Children see an average of fifteen television commercials for food every day (in addition to ads on billboards, online, and elsewhere), the vast majority of which feature foods high in sugar, fat, or sodium.⁶⁹ Food and beverage companies spend upward of \$10 billion annually marketing to children, and several experimental and cross-sectional studies support the thesis that advertising alters children’s taste preferences as well as the amount they eat.⁷⁰ In one study, children given identical food in either a plain bag or a McDonald’s bag rated the food in the branded bag as better tasting; the effect was stronger in children who had more TV sets in their home and who ate at McDonald’s more often.⁷¹ In another study, children who watched cartoons interspersed with food ads ate more than children who watched cartoons with other kinds of commercials.⁷² The increase in consumption was greater for overweight children than for those of normal weight, and greater still in obese children, suggesting that some individuals may be more susceptible than others to these influences.⁷³

Skeptics may dismiss the notion of advertising as “mind control,” but repeated exposure at a very young age can have a profound

effect on a child's later actions, even into adulthood. Children are thought to be capable of some "defense" against persuasion by marketing by age eight, at which point most children are able to recognize advertisements and evaluate their claims accordingly. But there is little evidence that children above age eight are any more resistant to the effects of advertising than younger children.⁷⁴ Ads do not simply make factual claims about their product; they are designed to create emotional associations, often at an unconscious level, and to bypass the "rational" parts of the brain.⁷⁵ Nor is the effect of advertising limited to food and obesity. Repeated studies have linked tobacco marketing to teenagers' decision to start smoking, and several cross-sectional and longitudinal studies have linked exposure to alcohol marketing to adolescents' being more likely to start drinking and to drink more frequently.⁷⁶

The built environment, the physical design of the areas around children's homes, can play a powerful role in determining children's "lifestyle choices." Many children live in neighborhoods with few (if any) sidewalks, bike lanes, parks, and green spaces that encourage exercise. Urban (or suburban) sprawl has created dependence on cars by placing destinations farther apart, while parents' concerns about crime may further reduce the amount of time children spend outside. Conversely, neighborhoods designed to be "walkable" encourage exercise and physical activity.

At the same time, over the past several decades schools faced with budget cuts have been dropping physical education programs to save money, while installing soda machines to raise badly needed sponsorship funds.⁷⁷ Recently, many schools have improved children's nutrition by regulating the offerings in vending machines and providing more

nutritional items in school cafeterias, but such actions have largely taken place on a school-by-school or district-by-district basis.⁷⁸ Several lines of evidence link features of the built environment with obesity or overweight in children and adults.⁷⁹ There is less agreement about the most effective interventions, largely because changing the physical structures of neighborhoods and cities is difficult and costly. But the evidence does suggest that tackling the obesity epidemic will require attention to the built environment as well as to individual behavioral change.

Consumption of healthful or unhealthful foods is typically discussed in terms of lifestyle choices. However, eating a healthful diet is highly dependent on having markets nearby that sell affordable fresh fruits and vegetables, and such places may be scarce or nonexistent in poor neighborhoods, while cheap, highly processed fast food is plentiful—even if, as some have argued, home-cooked food is actually less expensive (in terms of raw ingredients) than the fast-food equivalent.⁸⁰

Tools for Preventing Harmful Exposures

Policy makers and other public health advocates can take several approaches to preventing disabilities that result from harmful environmental exposures. These are often classified into "the Three Es": education, enforcement, and engineering. Education involves giving people information on health risks in an effort to change their behavior. Enforcement uses legislation and regulations to reduce or curtail harmful behaviors. Engineering involves manipulating the environment to passively reduce exposures to a hazard. For example, to prevent childhood obesity or type II diabetes, children might be given lectures or promotional materials about the risks of a diet high in saturated fat and

the benefits of eating more fruits and vegetables (education); fast-food advertising aimed at children might be restricted (enforcement); and neighborhoods might be designed to encourage walking and other physical activity or making healthful snacks and water more easily available to school children than unhealthy ones (engineering).

All three methods have strengths and weaknesses. Education is the least invasive, but changing behavior through education is notoriously difficult and often ineffective (smoking-cessation programs and campaigns aimed at increasing fruit and vegetable consumption tend to have low success rates), especially when modifying one's behavior requires acting differently from friends, family, or the larger society. In contrast with education-only efforts, enforcing certain behaviors, by restricting the sales of tobacco products and alcohol to minors, for example, has been more effective. Enforcement can be quite contentious when it involves regulating industries or people's behavior and often leads to accusations of paternalism or heavy-handedness, although paternalism may be more acceptable where children are concerned. From a population-wide perspective, the engineering approach has the greatest potential to improve health: by making more healthful lifestyles the "path of least resistance," it bypasses the difficult process of persuading people to change their behavior. Engineering the environment, such as treating water to reduce diarrheal diseases, phasing out the use of leaded gasoline to prevent lead poisoning, or instituting zoning codes to limit proximity of residential dwellings to industries emitting toxic material, have all proved to be highly effective ways of preventing disease and disability. At the same time, engineering solutions can be costly to implement. This approach also requires the

involvement of professionals outside the typical conception of "health"—engineers and city planners, as well as political leaders—in addition to physicians and public health scientists. Still, as noted, some of the largest increases in life expectancy over the past century have resulted primarily from population-wide engineering solutions.⁸¹

Population-wide approaches to prevention can be effective because they are capable of "shifting the curve." Disabilities exist on a continuum. Thus, a small increase in risk for a common disease or disability affects population health more than a large increase in a rare condition.⁸² For example, children's capacity for attention, hyperactivity, and impulse control varies across a wide spectrum, and it is only to simplify the diagnosis and treatment that health care providers create a clear division between "normal" children and those who have ADHD. Geoffrey Rose used the idea of "shifting the curve" to describe the relationship between individual- and population-level risks. He showed that many diseases or disabilities exist on a continuum; the number of people in the "high-risk" group (in this case, those corresponding to the clinical criteria for ADHD) is closely tied to the overall state of the population as a whole.⁸³ In other words, the number of children diagnosed with ADHD in a population can often be predicted from the average behavioral profile of children in the population. Depending on the shape of the distribution, small shifts in behaviors or exposures associated with ADHD can have a dramatic effect on the number of children who meet clinical criteria for ADHD. And in practice, with the exception of immunizations, population-wide interventions to prevent disabilities are largely limited to modifying environmental risk factors.

Taking the Precautionary Principle with Children's Toxic Exposures

How much evidence is needed before action is taken? The dangers of tobacco and lead were understood for decades before prevention became a priority. Today, however, for a variety of reasons, policy makers are reluctant to act on a hazard unless the precise way that it causes disease or disability is known.⁸⁴ The sanitarians of the early twentieth century understood that demonstrating a pattern of disease was sufficient to take action, often decades before the bacterial causes were discovered. One way to apply that lesson is by reforming the way industrial chemicals are tested and allowed onto the market.

Currently, industrial chemicals are “innocent until proven guilty.” They can be introduced without being fully tested for toxicity: indeed, of more than 200 industrial chemicals known to have neurotoxic effects in adults, only a handful have been tested for neurotoxicity at lower (subclinical) doses.⁸⁵ Moreover, a chemical is taken off the market or a pollutant regulated only when harmful effects are proven definitively; by convention, this means that a chemical has to be proven toxic in laboratory experiments and then in a series of epidemiologic studies, which usually take decades to complete.⁸⁶ In the interim, millions of people, including children and pregnant women, will have been exposed and possibly harmed. Thousands of chemicals are currently in the environment, making it difficult to attribute disability or disease to any one particular chemical. For those chemicals that persist indefinitely in the environment, even when harmful effects are identified, stemming the tide of exposure may be the most that can be accomplished. Although the insecticide DDT was banned in the United States in the early 1970s, one recent study estimated that its metabolite DDE can be

detected in 95 percent of Americans.⁸⁷ It has been linked with diabetes, spontaneous abortion, and impaired neurodevelopment.⁸⁸ PCBs, which have been linked to reduced IQ and immune system and thyroid dysfunction, have been banned for decades; however, they are routinely detected in newborns and children around the world; exposure is nearly universal.⁸⁹

The experience with lead, tobacco, PCBs, mercury, and other toxicants indicates that the United States should adopt the precautionary principle and identify toxicants before they are marketed and widely disseminated in the environment. Other governments have already taken such a step. In 2007 the European Union instituted the REACH Program, which requires manufacturers to prove that chemicals are safe before they are marketed.⁹⁰

Setting Priorities: Population Attributable Fractions

How do we prioritize what environmental influences or risk factors to target? From a prevention perspective, efforts should focus on common and modifiable risk factors associated with high-prevalence disabilities and potentially debilitating conditions, such as ADHD, obesity, or asthma. A tool known as population attributable fraction, a measure of the proportion of disability or disease in a population that can be attributed to a particular risk factor, can help quantify priorities.⁹¹ The population attributable fraction takes into account both the risk posed by an exposure and the frequencies of exposure and disease in the population.

Tanya Froehlich and her coworkers estimated that exposure to higher levels of lead and prenatal tobacco each accounted for 500,000 additional cases of ADHD in U.S. children;

Table 1. Population Attributable Fractions (PAF) and Number of Attributable Cases for Select Environmental Risk Factors and Childhood Disabilities

Condition	Exposure	PAF (%)	Number of cases
ADHD	Prenatal tobacco smoke	22	510,000
	Blood lead in top tertile	25	598,000
	Prenatal smoke or blood lead	38	900,000
Conduct disorder	Environmental tobacco smoke (cotinine in top quintile)	39.2 ^a	—
	Blood lead in top quartile	38.9 ^a	—
Preterm birth	Maternal smoking during pregnancy	5.3–7.7	—
Low birth weight		13.1–19.0	—
Asthma	Residential exposures (secondhand smoke, pets, allergies)		
	age 0–5	39	533,000
	age 6–16	44	2,000,000
At risk for overweight (85th–95th percentile) ^b	Never breast fed in first 6 months, age 3–5	20.2 ^a	—
Overweight (95th percentile ^b and above)	Breast feeding (mostly formula vs. mostly breast fed), age 9–14	9.2 ^a	—
Metabolic syndrome	Smoking (age 12–19)	27.5	—

Sources: Tanya E. Froehlich and others, "Association of Tobacco and Lead Exposures with Attention-Deficit/Hyperactivity Disorder," *Pediatrics* 124 (2009): e1054–e1063; Joe M. Braun and others, "Association of Environmental Toxicants and Conduct Disorder in U.S. Children: NHANES 2001–2004," *Environmental Health Perspectives* 116, no. 7 (2008): 956–62; Patricia M. Dietz and others, "Infant Morbidity and Mortality Attributable to Prenatal Smoking in the U.S.," *American Journal of Preventive Medicine* 39, no. 1 (2010): 45–52; Bruce P. Lanphear and others, "Residential Exposures Associated with Asthma in U.S. Children," *Pediatrics* 107, no. 3 (2001): 505–11; Bruce P. Lanphear and others, "Contribution of Residential Exposures to Asthma in U.S. Children and Adolescents," *Pediatrics* 107, no. 6 (2001): e98, DOI: 10.1542/peds.107.6.e98; Mary L. Hediger and others, "Association between Infant Breastfeeding and Overweight in Young Children," *Journal of the American Medical Association* 285 (2001): 2453–60; Matthew W. Gillman and others, "Risk of Overweight among Adolescents Who Were Breastfed as Infants," *Journal of the American Medical Association* 285 (2001): 2461–67; Michael Weitzman and others, "Tobacco Smoke Exposure Is Associated with the Metabolic Syndrome in Adolescents," *Circulation* 112 (2005): 862–69.

Notes: PAFs for exposures are not additive, and may sum to over 100 percent.

a. We calculated PAF estimates from figures in paper and using the formula $\frac{\text{exposed cases}}{\text{total cases}} \times \frac{(RR-1)}{RR}$, where RR stands for relative risk.

b. Percentiles are weight-for-height, compared to sex- and age-specific distributions.

— Estimate of attributable cases not given.

using the population attributable fraction, they estimated that 38 percent of cases of ADHD could be prevented if childhood lead exposure and smoking in pregnant women were eliminated.⁹² They also showed that, because lead and tobacco interact synergistically, children who had high blood lead and exposure to prenatal tobacco constituted only 7.7 percent of the population, but they represented nearly 25 percent of the total cases of ADHD.⁹³ It is worth noting that both blood lead levels in children and smoking among pregnant women have decreased significantly in the last few decades, so they cannot explain the increase in ADHD

prevalence.⁹⁴ However, the prevalence of ADHD would undoubtedly be higher if these two environmental factors had not been reduced. There are now several other toxicants, as well as other risk factors, suspected of contributing to the development of ADHD.⁹⁵ However, the current health care system continues to focus almost entirely on identification and treatment of children for ADHD rather than on further reductions in toxicants demonstrated or suspected of elevating the risk for the disorder.

Calculating population attributable estimates for prevalent disabilities is not always feasible

—it requires a representative sample, an estimate of the prevalence of exposure, and a measure of risk—but estimates do exist for several notable risk factors for prevalent childhood disabilities (table 1). Bruce Lanphear and others estimated, for example, that residential exposures—including exposure to secondhand smoke, the presence of pets, use of a gas stove, and allergies to dust mites or cockroaches—accounted for approximately 533,000 cases of asthma (39 percent of all cases) in children under six and 2 million cases (44 percent of the total) in children aged six to sixteen.⁹⁶

Healthy Communities: Challenges and Successes

We have a remarkable opportunity to protect the health of children and prevent the development of disability. While it is not yet definitive, a growing body of evidence shows that prenatal or early-life exposure to chemicals or malnutrition can have severe effects on physical and mental development that persist over the life span and that effects are found at increasingly lower levels of exposure.

Children are routinely exposed to thousands of man-made chemicals, most of which have not been tested for safety, from an early age, and often even before they are born. From a very young age, children are inundated with marketing for fast-food restaurants, sugary cereals, tobacco, and alcohol; these exposures can shape their developing behaviors, food preferences, and decisions to smoke or drink alcohol. While the task may seem daunting, these exposures are all modifiable if we have the will to do so, and taking action would produce considerable benefits.

Reducing toxic exposures is not impossible. C. Arden Pope and his colleagues estimated that as much as 15 percent of the increase in life expectancy from 1980 to 2000 in many

U.S. cities was attributable to environmental regulations that reduced air pollution.⁹⁷ Further reductions in allowable levels of airborne pollutants are likely to result in even greater benefits.⁹⁸ Another promising finding is a reduction in asthma rates brought about by smoking bans. In Scotland and Kentucky, recent bans on smoking in public places have each led to an 18 percent reduction in child asthma hospitalizations and emergency-room visits in the areas affected by the bans.⁹⁹ Through coordinated public health campaigns, social attitudes about smoking are changing, and tobacco use has declined.¹⁰⁰ Regulations lowering the allowable levels of lead in gasoline, paints, and other consumer products led to an 84 percent reduction in children with elevated blood lead (more than 10 micrograms per deciliter) in the United States between 1988–91 and 1998–2004.¹⁰¹ And while efforts to curb childhood obesity have, thus far, been unsuccessful at the societal level, a few school-based programs have had some success in lowering the body mass index for some children or increasing their physical activity.¹⁰²

These success stories demonstrate the potential benefits that could result from wide-scale prevention of disability in children. But it is not enough to address *this* chemical or *that* risk factor when thousands more have not been tested and new ones are introduced every day. Many of the best-known environmental risk factors have been decreasing in recent decades, yet the prevalence of childhood disability is rising. If the established pollutants are not responsible for the increase in disability, those other exposures that are responsible must be identified. If we want to make meaningful progress in preventing disability and promoting health, we must be willing to make fundamental changes to our environment. We must ask ourselves: What

kind of world do we want to live in? What would a healthy city or community really look like?

Many interventions aimed at mothers with small children have been shown to be effective in giving children a healthy base for development. Breast feeding is known to lower risks of such wide-ranging conditions as asthma, obesity, and diabetes, and it is associated with greater mental development in preterm infants.¹⁰³ Increasing rates of breast feeding will require not only educational campaigns but the removal of structural and institutional barriers for breast-feeding mothers; new federal legislation requiring employers to provide space and break time for mothers provides some support in states without previous legislation, although barriers to acceptance still remain.¹⁰⁴

Another intervention that has gained support is the practice of nurses' visits to low-income first-time mothers in their home to promote care of healthy infants and injury prevention. Evidence for the effectiveness of this intervention is mixed, but randomized trials have shown that one program, the Nurse Family Partnership, which has been tested around the country and now operates in thirty-two states, reduces maltreatment and behavioral problems and increases cognitive performance in children.¹⁰⁵ These successes provide ideas for changes that would work at a larger level, but investments in these interventions must be long term to be effective; it takes years for the benefits to accrue. Such programs are thus often deemphasized in favor of medical treatments that produce more immediate results for the individual but few long-lasting benefits for society.

By their physical design, cities and towns can lend themselves either to a healthy population

or to one with high levels of disability and disease. One aspect of cities that has received much attention is the built environment. As noted, the built environment is linked with obesity, but just as environments can be "obesogenic," they can also promote physical activity and healthful eating. By designing cities with efficient public transportation, greater urban density, mixed land use, and easy access to fresh produce, more healthful choices would become easier to make. As with any engineering solution, however, these large-scale changes will require great effort, leadership, expense, and collective will.

Increased public transportation, in particular, would make cities more healthful on several fronts. Fewer cars on the road, particularly if a greater share of them emitted fewer pollutants, would reduce air pollution levels, which would lead to lower rates of asthma and cardiovascular disease and to longer life expectancy.¹⁰⁶ Greater use of public transportation could also lower levels of obesity; one study found that users of public transit in Atlanta were more than twice as likely to meet the recommended levels of physical activity.¹⁰⁷ Another study after the addition of light rail transit in Charlotte, North Carolina, found that transit users lost weight compared with those who did not use it.¹⁰⁸

Environments are social, as well as physical. It is virtually impossible to shield a child from the marketing that surrounds her at every stage of her life, and research is making it increasingly clear that the repetitive exposures leave a mark. One way to improve children's health would be to restrict certain types of advertising. Tobacco ads are already banned from television, but depictions of smoking in movies still influence children's decisions about whether (or when) to begin smoking. Similar arguments can be made for alcohol

and fast food: one study estimated that a ban on fast-food advertising aimed at children and adolescents would reduce rates of overweight children by 18 percent for children aged three to eleven, and 14 percent for those aged twelve to eighteen.¹⁰⁹ Increasing children's media literacy might also give children some "resistance" against marketing and a healthy suspicion of advertisers' claims. While such media savvy may help counter the most harmful effects of the consumer culture, the only widely effective solution is likely to be regulation of marketing to children. The British government has banned junk food advertisements in programs aimed at children under sixteen; it remains to be seen whether other countries will follow with similar regulations.¹¹⁰

Finally, virtually every health measure available is connected with socioeconomic status. Efforts to reduce poverty will require a high level of coordination and political determination and may require realigning a nation's collective priorities. Some programs, such as instituting a living wage, have sought to address specific aspects of poverty. Limited evidence is available about the feasibility and effectiveness of a living wage, but few would argue that a family's basic needs, such as housing, food, clothing, and health care, should go unmet.

What would a healthy community look like? In many ways, Vancouver, on the west coast of Canada's British Columbia, fits this description. The city has low levels of air pollution and relatively low rates of smoking (15.1 percent of people over age fifteen, lower than the rest of Canada or the United States).¹¹¹ The city is built to encourage walking, bicycling, and use of public transit; the number of major highways that cut through the heart of the city is minimal. And its

inhabitants seem to live longer, healthier lives. In 2005–09, Vancouver's life expectancy at birth was 82.6 years, which—if it were a country—would rank second only to Japan.¹¹² In addition, Vancouver has begun an initiative to become the world's greenest city by 2020, an effort that includes sustainable industries, low levels of air pollution, and a citywide goal of walking, cycling, or using public transportation for at least half of all trips taken.¹¹³ This vision is in stark contrast with cities of the past that aspired to attract industry, only to end up with high levels of air pollution and widespread sprawl centered around cars and highways. One might imagine policies influencing other aspects of life that affect children's health: low-density billboards and restrictions on marketing unhealthy products would create a more healthful media environment. Cities could institute a living wage for workers, following the example of more than 100 U.S. cities, or commit to providing health care for its uninsured residents, as San Francisco has done.¹¹⁴

A nation that committed itself could take actions that would prevent childhood disabilities by greatly reducing exposures to environmental hazards, at a great savings in human capital and health care costs. A strategy for the prevention of disability must prioritize and target prevalent environmental exposures across populations, rather than continue to focus primarily on the treatment of high-risk or susceptible children. Preventing disabilities will require us to change the way we live—how we build our communities, travel, regulate pollutants, and invest our resources. We now have the evidence and tools to profoundly improve the health and functioning of children, but implementing a preventive strategy will take a concerted effort involving parents, pediatricians, public health officials, policy makers, and society at large.

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