

STATE-OF-THE-ART REVIEW

# Health Consequences of Environmental Exposures: Causal Thinking in Global Environmental Epidemiology



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## Abstract

The 2010 Global Burden of Disease estimates indicate a trend toward increasing years lived with disability from chronic noncommunicable diseases (NCDs). Risk factors examined included smoking, diet, alcohol, drug abuse, and physical inactivity. By contrast, little consideration was given to accumulating evidence that exposures to environmental chemicals, psychosocial stress, and malnutrition during fetal development and across the life span also increase risk of NCDs. To address this gap, we undertook a narrative review of early-life environmental contributions to disease. We documented numerous etiologic associations. We propose that future GBD estimates use an expanded approach for assessing etiologic contributions of environmental exposures to recognized disease risk factors. We argue that broadening the definition of environmental disease, together with improved methods of assessing early life exposures and their health outcomes across the life span, will allow better understanding of causal associations and provide the incentives required to support strategies to control avoidable exposures and reduce disease risk.

**KEY WORDS** children, pollution, non-communicable disease, burden of disease, public health

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In 2010, the United Nations adopted a global plan of action to reduce poverty and to improve health by launching 8 Millennium Development Goals to be achieved by 2015. Significant progress has been made toward achieving these goals in many countries. At the same time, we have seen a substantial change in the global pattern of disease. Publication of the 2010 estimates of the Global Burden of Disease (GBD) and of 67 risk factors and risk factor clusters has demonstrated a significant shift toward chronic noncommunicable diseases (NCDs).<sup>1,2</sup> Early childhood deaths have

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PDS, M-NB-D, and WS conceived the concept for this article, PDS wrote the initial draft. All authors reviewed and edited the manuscript and PDS produced the final draft, which was approved by all authors.

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declined, but in contrast, years lived with disability (YLD) have increased.<sup>2</sup> Globally, YLD attributable to communicable, maternal, neonatal, and nutritional diseases have decreased by 19.5% between 1990 and 2010, whereas those attributable to chronic disease have increased: cardiovascular diseases by 17.7%; chronic respiratory disease by 8.5%; neurological conditions by 12.2%; diabetes by 30.0%; and mental and behavioral disorders by 5.0%.<sup>3</sup>

These estimates of disease burden and assessments of the hazards posed by their underlying risk factors are based on conservative methodology that requires a very high level of evidence before a risk factor can be included.<sup>1</sup> These estimates, therefore, included careful consideration of the contributions to GBD of such well-established risk factors as alcohol, tobacco, drug use, diet, and physical inactivity.<sup>1</sup> These exposures were considered to be “lifestyle” risk factors. By contrast, the current GBD methodology does not include estimates of the contributions to disease burden of many environmental risk factors. The methodology fails to consider the chemicals present in food and water that have toxic, carcinogenic, or endocrine disruptive actions. It does not consider the substantial body of evidence that chemical exposures and nutritional deficiencies in early life increase risks of chronic diseases such as hypertension, high blood glucose, and increased body mass index in childhood, during adult life, and in the elderly.<sup>4</sup> Furthermore, the current methodology fails to consider emerging evidence that behaviors currently classified as caused by “lifestyle” are, in fact, the result of much more than personal choice and are profoundly shaped by the social and cultural environment, genetics, and parts of the natural and the manmade environment. For example, although obesity is included as a risk factor, chemical exposures in early life that appear likely to contribute to obesity<sup>5,6</sup> are not. In short, these considerations indicate that the GBD analysis at present significantly underestimates the role of environmental exposures in human disease. Ambient noise is another environmental hazard with adverse consequences on health (in addition to hearing loss)<sup>7</sup> that is often omitted from environmental contributions to disease.

Quantifying the disease burden caused by the environment is difficult because evidence on causal links between exposure to many environmental factors and health outcomes is still evolving. Additionally, there is often a lack of reliable exposure data at the population level, especially in regard to

exposures in early life that may have occurred years or decades ago.<sup>8</sup> An expert panel convened by the World Health Organization (WHO) estimated that 24% of the global disease burden and 23% of all deaths could be attributed to environmental exposures, based on data collected in the late 1990s and early 2000s.<sup>9</sup> Among children 0–14 years of age, WHO estimates that the proportion of deaths attributable to the environment could be as high as 36%.<sup>9</sup> The WHO has reported the fraction of disease attributed to the environment for 85 diseases. The WHO definition of the “environment” included a wide range of modifiable physical, chemical, and biological factors external to the human host that directly affect health and also increase unhealthy behaviors (eg, the impact of the structure of the environment on physical inactivity). However, even these estimates almost certainly did not go far enough and also did not include the contributions of early life chemical exposure or ambient noise to other risk factors.

## ENVIRONMENTAL EXPOSURES AND CHRONIC, NONCOMMUNICABLE DISEASES

Recent data from the US Centers for Disease Control and Prevention national biomonitoring program<sup>10</sup> demonstrate that almost all Americans have detectable levels of a wide variety of environmental chemicals in their bodies, including many with known endocrine-disrupting, neurotoxic, and carcinogenic activities. These include organic chemicals that persist in the environment long after their production and use have been stopped, such as polychlorinated biphenyls, and nonpersistent chemicals to which individuals are constantly exposed, such as the plastic components and plasticizers bisphenol A (and other bisphenols) and phthalates. Many of these chemicals may be present at levels that can cause biological or toxicologically relevant effects in animal models. There is strong evidence that such exposures increase the risk of diabetes,<sup>5,6,11</sup> hypertension,<sup>12</sup> cardiovascular disease,<sup>13,14</sup> obesity,<sup>15,16</sup> and cancer.<sup>17</sup> Table 1 gives an overview of the environmental exposures associated with chronic noncommunicable diseases, and Supplementary Tables 1–5 (available in the online version at <http://dx.doi.org/10.1016/j.aogh.2016.01.004>) provide more details.

Understanding is increasing that the risk of developing many chronic NCDs are increased by early-life exposure to toxic chemicals, nutritional imbalances, and psychosocial stress<sup>18</sup> and, as such,

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