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Global Environmental Change and Noncommunicable Disease Risks

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Abstract

Multiple global environmental changes (GECs) now under way, including climate change, biodiversity loss, freshwater depletion, tropical deforestation, overexploitation of fisheries, ocean acidification, and soil degradation, have substantial, but still imperfectly understood, implications for human health. Noncommunicable diseases (NCDs) make a major contribution to the global burden of disease. Many of the driving forces responsible for GEC also influence NCD risk through a range of mechanisms. This article provides an overview of pathways linking GEC and NCDs, focusing on five pathways: (*a*) energy, air pollution, and climate change; (*b*) urbanization; (*c*) food, nutrition, and agriculture; (*d*) the deposition of persistent chemicals in the environment; and (*e*) biodiversity loss.

INTRODUCTION

Noncommunicable Diseases

Noncommunicable diseases (NCDs), principally cardiovascular diseases, cancers, chronic respiratory diseases, diabetes, and mental health conditions, together with neurologic, endocrine, gastrointestinal, renal, allergic, and autoimmune disorders, have commanded increasing attention in recent years, and with good reason. These diseases kill more than 38 million people each year, accounting for 70% of global deaths (95, 156). Among the victims are more than 14 million adults who, in the words of the World Health Organization (WHO), "die too young"—that is, between the ages of 30 and 70 (155). NCDs also account for 21 of the leading 30 causes of age-standardized years lived with disability (YLDs) in 2016, or 80.6% [95% confidence interval (CI) 78.2–82.5] of YLDs, according to the Global Burden of Disease study (145). Leading contributors to disability include headache, low back pain and osteoarthritis, depression and anxiety, diabetes, asthma, and vitamin A deficiency.

Four aspects of NCDs are especially salient. First, they are not only, or even mostly, a problem of wealthy nations. Low- and middle-income countries (LMICs) account for 86% of the burden of premature deaths from NCDs (19, 155), reflecting what has been known for nearly half a century as the "epidemiologic transition" (101). Second, the economic implications of NCDs are substantial, with a projected cost to the global economy of $47 trillion over the next 20 years (14). Particularly in LMICs, NCDs slow economic development and trap millions of people in poverty. Third, many risk factors for NCDs are environmental in origin or may be influenced by the environment. For example, about 50% of the disease burden from chronic obstructive pulmonary disease and about one-quarter of the ischemic heart disease burden have been attributed to environmental factors, particularly air pollution (79). However, environmental contributions to NCDs are routinely overlooked; for example, the 2013 WHO NCD Global Action Plan largely failed to mention environmental factors (155) [although a later report from WHO did address the issue (158)]. Fourth, NCD research, prevention, and treatment are severely underfunded, particularly in LMICs, relative to their population burden (3).

A debate has arisen concerning the framing of NCDs, and even the term NCD. Some have argued against the name because it starts with "non," defining an entity by what it is not and seemingly making it a "nonissue." Others have pointed to increasing evidence that NCDs are, in fact, communicable, through social networks, viruses, and/or cultural and economic conditions (4). Suggested replacement terms include "biosocial and development diseases" (160) and "life-long disease" (112). These terms, however, may overlook a set of causal factors whose importance is increasingly clear but largely overlooked to date: those related to global environmental changes.

Global Environmental Changes

Since at least the middle of the last century, unprecedented global environmental changes (GECs) have occurred. These changes undermine many of the natural systems that underpin human civilization and have the potential to reverse the progress in health and development achieved in recent decades. These changes include climate change, biodiversity loss, freshwater depletion, tropical deforestation, overexploitation of fisheries, ocean acidification, soil degradation, changes in nutrient (nitrogen, phosphorus, and others) cycling, and environmental loading with persistent toxic substances. **Figure 1** summarizes some of these environmental trends. Because these human-induced impacts on earth systems are so extensive, they have been proposed to signal a new geological epoch, the Anthropocene (131). The far-reaching implications of these changes

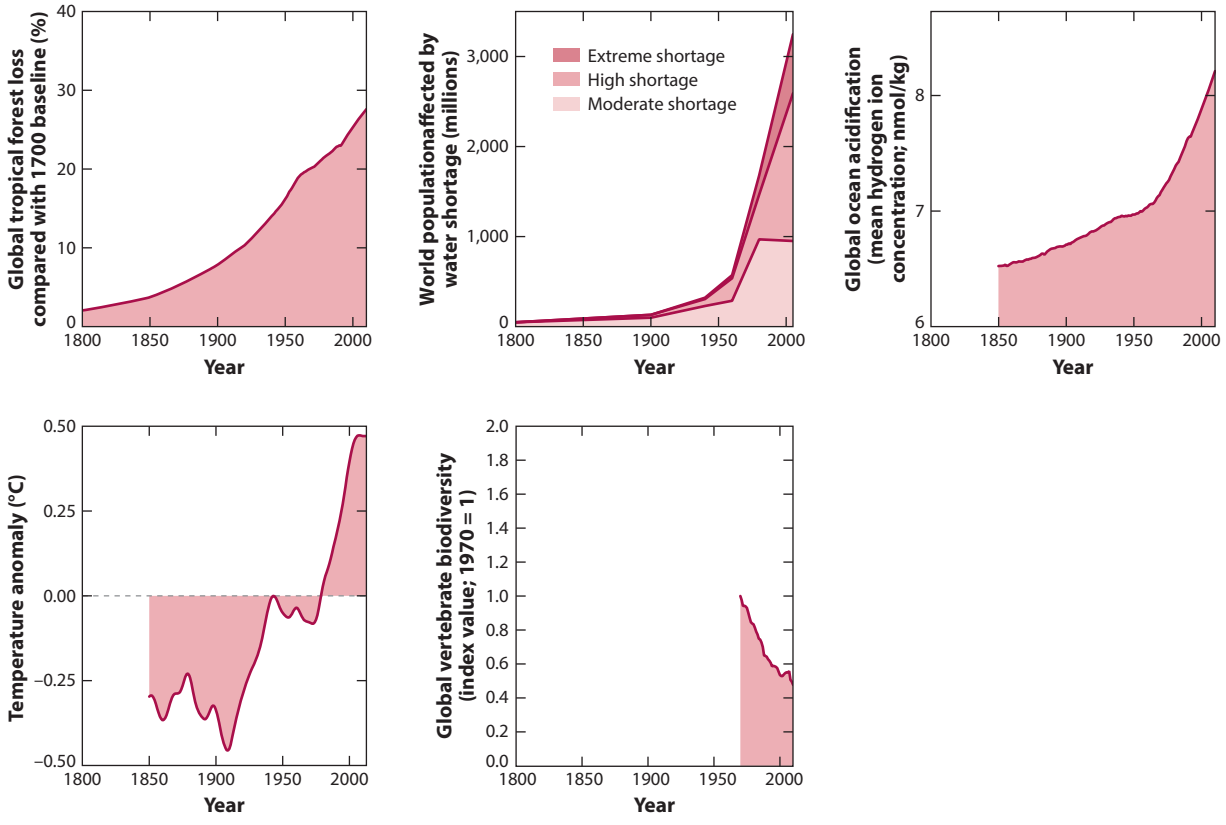

Figure 1

Selected global environmental trends. Figure adapted from Reference 154.

for human health have been described by The Rockefeller Foundation/Lancet Commission on Planetary Health (154).

GECs can affect a range of health outcomes through a complex web of pathways. **Figure 2** shows a subset of these pathways, focused on climate change and related processes (152). A diagram showing other processes, from changes in nitrogen cycling to biodiversity loss, would be considerably more complex. Key pathways to health impacts include extreme events (floods, droughts, intense storms, wildfires, and heat waves), changes in the incidence and distribution of both vector-borne and water-related diseases, reduction in food availability and quality, and socially mediated effects such as increased poverty, conflict, and population displacement (152, 154).

The pathways from GECs to NCD risk can also be represented through the DPSEEA (driving forces, pressures, states, exposures, effects, actions) framework (**Figure 3**) (46). Strategies to prevent and treat NCDs are often narrowly focused on clinical interventions or behavior changes. These approaches are essential but not sufficient; the failure to address many of the key driving forces increasing the risks of NCDs will likely limit the impacts of these preventive strategies (103).

This article outlines pathways by which GECs, and the driving forces responsible for them, influence the risks of NCDs. It considers five pathways: (*a*) energy, air pollution, and climate change; (*b*) urbanization; (*c*) food, nutrition, and agriculture; (*d*) persistent environmental chemicals; and

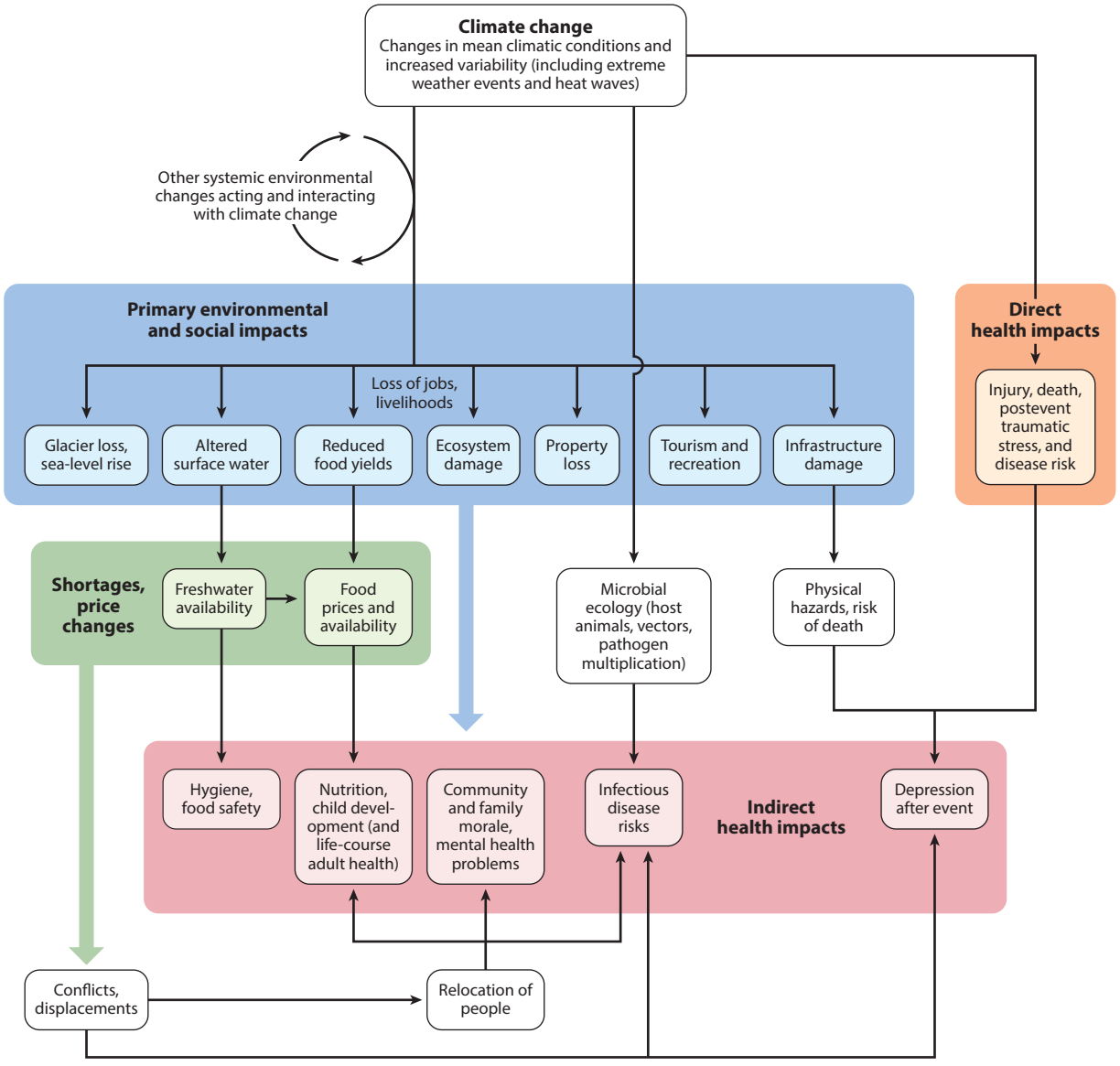

Figure 2

Pathways from climate change to health outcomes. The orange field shows direct health impacts, such as the effects of severe weather events. The blue field shows social impacts that follow climate-related environmental changes; many of these relate to reduced access to needed resources such as water, to infrastructure damage, and to loss of livelihoods. Less direct health impacts are shown in the lower part of the figure. These are mediated by the primary environmental and social impacts of climate change. The red field shows five categories of indirect health impacts. Tertiary effects, indicated by upward-pointing arrows, arise from more diffuse disruptions, dislocations, and conflicts, which are likely to increase with advancing climate change. The figure is not intended to be comprehensive. Figure adapted with permission from Reference 92.

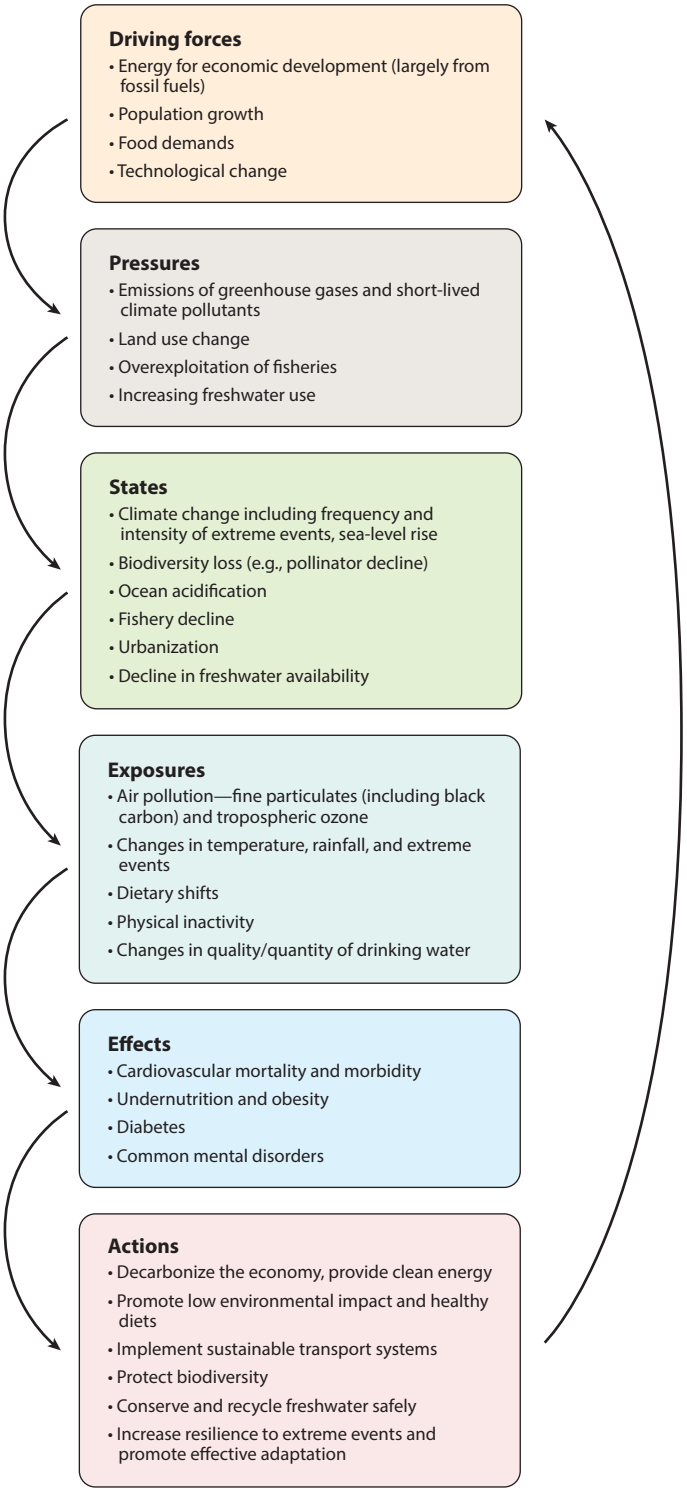

Figure 3

The DPSEEA (driving forces, pressures, states, exposures, effects, actions) framework as applied to global environmental changes. Figure adapted with permission from a figure by James Milner.

(*e*) biodiversity loss (including fisheries depletion). Land use changes, a sixth category, are considered in the context of urbanization, agriculture, and biodiversity loss.

While GECs contribute to the risk of NCDs, the association has potentially positive implications as well. The measures required to limit GECs and to promote sustainable development also affect NCD risk, often in beneficial ways (61, 152). Well-designed policies to reduce environmental change may yield ancillary health benefits (cobenefits) and avoid unanticipated health risks (coharms) (61, 144).

THE IMPACT OF GLOBAL CHANGES ON NCDs

Energy, Air Pollution, and Climate Change

A principal driver of GEC is the combustion of the fossil fuels coal, oil, and gas; of these, coal has the largest effect. The dependence on fossil fuels for energy is a leading source of air pollution (66). It is also the single largest contributor to climate change. Two sets of pathways link energy use to NCDs, one through air pollution, the other through climate change.

Air pollution. Air pollution related to fossil fuel combustion is a major contributor to morbidity and mortality worldwide.[1] Key pollutants include fine particulate matter (PM), ozone, oxides of nitrogen, oxides of sulfur, hydrocarbons, and metals; many of these are related to each other through complex atmospheric chemistry. The Global Burden of Disease (GBD) Study estimates that ambient fine particulate matter ($PM_{2.5}$) alone ranked fifth globally as a mortality risk factor in 2015, accounting for an estimated 4.2 million deaths (7.6% of global deaths) and 103.1 million disability-adjusted life years (DALYs) (4.2% of global DALYs) in 2015 (27, 52). These deaths are not uniformly distributed; the greatest burdens are in China and India (with about 1.1 million annual deaths each), Russia (137,000 deaths), Pakistan (135,000 deaths), and Bangladesh (122,000 deaths). Of note, the WHO estimates of the burden of ambient air pollution are somewhat lower than those of the GBD Study: 3 million deaths and 85 million DALYs (157). The reasons for the different estimates have been reviewed by the Lancet Commission on Pollution (79). A more recent study, on the other hand, estimated a substantially larger burden: 8.9 million deaths globally in 2015 (18), in part because new evidence suggests that even low levels of PM air pollution confer risks to health. Unless there is a rapid transition toward clean renewable sources of energy, economic growth is projected to increase this burden substantially in some regions, such as southeast Asia, over the coming decades (77).

The excess mortality from ambient PM exposure is attributable primarily to NCDs: ischemic heart disease, cerebrovascular disease, chronic obstructive pulmonary disease, and lung cancer, with a small proportion of excess deaths—perhaps one in ten—due to lower respiratory infection (64). Black carbon, a short-lived climate pollutant (SLCP) and an important component of PM, appears to be a better indicator of harmful exposure than undifferentiated PM in short-term studies, but the evidence from long-term studies is inconclusive (68). Air pollution exposure, especially to PM, may aggravate cardiac arrhythmias (44) and heart failure (123). Systematic reviews suggest an association between air pollution exposure and diabetes risk (37, 63), and while the evidence is not conclusive, a recent analysis calculated that in 2016, ambient $PM_{2.5}$ contributed to approximately 3.2 million incident cases of diabetes, 8.2 million DALYs caused by diabetes, and 206,000 deaths from diabetes (16). Evidence indicates that PM exposure may be neurotoxic across the life span, contributing to neurodevelopmental delays in children and to cognitive decline in older adults (25, 33), although these associations are inconsistent across studies and cannot be considered definitive.

[1]Household air pollution is also a major global health hazard, but it is not discussed here because its link to planetary changes is less direct.

Ozone is formed from atmospheric precursors—hydrocarbons (methane and volatile organic compounds) and oxides of nitrogen—many of which are combustion products. Like PM, tropospheric ozone is also associated with excess mortality, although lesser in magnitude than PM (141). In the GBD data, exposure to ozone caused an additional 254,000 (95% CI 97,000–422,000) deaths and a loss of 4.1 million (95% CI 1.6 million–6.8 million) DALYs from chronic obstructive pulmonary disease in 2015 (27). More recent estimates using updated exposure–response relationships suggest at least a fourfold higher burden, with 1.04–1.23 million respiratory deaths in adults attributable to long-term ozone exposures. The largest increases in estimated attributable mortality were in northern India, southeast China, and Pakistan (90). The association between ozone exposure and mortality seems to relate both to short-term high exposures and to long-term exposure (7, 90). Short-term ozone exposure also triggers exacerbations of airway disease (asthma and chronic obstructive pulmonary disease), accounting for substantial numbers of emergency room visits and hospitalizations (69).

Climate change. Climate change presents another, overlapping set of pathways through which energy use and resulting GECs affect health (126), and NCDs in particular (46). Multiple direct and indirect mechanisms operate.

One pathway operates through the effects of climate-related disasters on health care. Climate change increases the intensity and frequency of extreme weather events. A systematic review of the impacts of cyclone, flood, and storm-related disasters on health care for people with NCDs, based on 48 studies from developed countries, found that people with cancer, diabetes, and cardiovascular diseases sustained increased risks of exacerbation of their health problems following such disasters. These outcomes were due to a range of factors including disruption of transport, weakened health systems including drug supply chains, loss of power, and evacuations of populations (115).

Another pathway operates through heat. The increasing hot weather that comes with climate change is associated with increases in mortality, in emergency presentations, and in hospital admissions due to cardiovascular, respiratory, and renal disease (126). Increased heat-related mortality with advancing climate change will likely outweigh any declines in cold-related mortality, particularly in tropical and subtropical regions and in Southern Europe (51, 126).

Climate change may increase the risk of cardiovascular disease through other mechanisms than the direct effects of heat. First, warmer weather is associated with sleep disturbance (100), which in turn is a risk factor for cardiovascular disease (21). Second, hot weather is associated with a reduction in physical activity (99), and reduced physical activity is a risk factor for cardiovascular disease (13). On the other hand, cold weather also reduces physical activity, so, in cold regions that become warmer, physical activity may increase. The net effect globally is likely to be negative (99). Third, rising sea levels, together with excessive groundwater withdrawals, land use changes, agricultural practices, and other factors, can lead to saline intrusion of groundwater in coastal areas (138). This, in turn, increases the salt intake of affected populations. A study in coastal Bangladesh suggested that pregnant women were exposed to more than double the limits recommended by the WHO/FAO (Food and Agriculture Organization of the United Nations) for salt intake in drinking water and that this exposure increased the risk of preeclampsia and hypertension in pregnancy (73). A further study of nonpregnant adults in coastal Bangladesh showed that sodium concentrations in drinking water were strongly associated with blood pressures after controlling for personal, lifestyle, and environmental factors and that changing to a less saline drinking water source reduced blood pressure (119). On the other hand, some evidence has linked warmer weather with lower blood pressure, which may act to reduce cardiovascular disease risk (147).

Climate change may also aggravate cancer risk. First, as noted above, physical activity declines with heat; sedentariness increases risk of some cancers. Second, evidence indicates that climate

change increases the production of aflatoxin, a liver carcinogen that contaminates some foods such as peanuts (11).

Climate change may increase the risk of kidney disease. Kidney stone formation seems to vary with temperature, perhaps a result of relative dehydration and resulting urinary concentration (136). Chronic kidney disease of unknown origin has been observed in working populations in hot places, such as among sugarcane cutters in Nicaragua (153), although a recent systematic review found inconsistent associations between heat and chronic kidney disease (88).

Climate change may affect NCD risk through a variety of nutritional pathways. These are discussed below in the section on food and nutrition.

Mental illness and distress are substantial contributors to the global burden of disease, and climate change may contribute in several ways (24). One way is a mechanism already mentioned: reduced physical activity due to heat. Physical activity is associated with reduced depression (91) and anxiety (133). Another pathway is the deleterious effect of disasters—which are increasingly common because of climate change—on mental health. Mental health problems in some circumstances dominate the health burden following disasters (56). Hurricane Katrina provides an example. Although not definitively attributed to climate change, this disaster exemplified the mental health burden that can follow climate-related disasters. Research conducted several months after Hurricane Katrina showed that approximately 49% of those surveyed in New Orleans, and approximately 26% in other hurricane-affected areas, suffered from a DSM-IV (*Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*) anxiety or mood disorder, of which half or more was post-traumatic stress disorder (PTSD) (48). By two years after the hurricane, the prevalence of PTSD and depression had actually increased (72). Similar disease burdens have been documented following other kinds of disasters, which are predicted to become more frequent with climate change, such as severe storms (1), floods (42), heat waves (62), and wildfires (35). Risk factors for postdisaster depression and PTSD across the life span have been studied in both wealthy and resource-poor settings; these include predisaster mental and physical health, personality structure and beliefs, features of the disaster experience itself, postdisaster social support and services, postdisaster displacement, and others (135). Slow-moving climate-related disasters may also threaten mental health. For example, research in Australia during the recent decade-long drought revealed an increase in anxiety, depression, and possibly suicidality among rural populations (98).

Of note, the two sets of pathways discussed here that link energy generation and NCDs—through air pollution and through climate change—are not independent. Certain air pollutants function as SLCPs and promote climate change while also threatening health via direct toxic effects (e.g., black carbon, tropospheric ozone) or by giving rise to ozone (methane) (124).

Urbanization

The city has become the prototypical human habitat, representing a global shift in both demographic and land use patterns. More than half of humanity now lives in urban areas, and with nearly all global population growth occurring in cities, that proportion is expected to reach two-thirds by 2050 (143). Much attention focuses on megacities of more than 10 million people, such as Mexico City and São Paulo, Cairo and Lagos, Karachi and Delhi, and Manila and Jakarta, but nearly half the world's urban dwellers live in a growing number of relatively small cities with fewer than 500,000 inhabitants and where population increase is faster (143).

Urbanization has emerged with different trajectories and in different ways, in different places. Europe and the Americas urbanized relatively early and have plateaued, whereas Asia and Africa continue to undergo rapid urbanization. Fast-growing cities of the Global South confront a range

of health and environmental challenges, including deficiencies in basic infrastructure (piped water, sewage, solid waste management, electricity, transportation, housing) and hazardous exposures (extremely poor air quality, noise, and unsafe roadways), which compounds problems of poverty, poor governance, and inadequate social services (117). In wealthy settings, especially in North America, Europe, and Australia, but increasingly in other regions, urban environmental health challenges reflect excessive automobile dependence, with associated urban sprawl, and resulting problems such as poor air quality, sedentary lifestyles, and injury risk (47). The burden is economically significant; the externalized costs of urban sprawl are about $400 billion annually in the United States alone, owing to the increased cost of providing public services, the higher capital investment needed for infrastructure, and the costs of increased traffic congestion, crashes, and air pollution, a substantial portion of these being health costs (54). Finally, some problems are common to cities in both wealthy and poor nations; these include extreme social stratification, neighborhoods of concentrated poverty, insufficient green space, food deserts, and vulnerability to disasters.

The relationship between GEC and urbanization is bidirectional. Poorly designed cities contribute to GEC. Conversely, GEC imposes burdens on cities, and many of these burdens directly threaten the health and well-being of urban residents.

What is the impact of urbanization on GEC (and on NCD risk)? Economic activity in cities generates between 70% and 85% of global gross domestic product and about 75% of energy-related greenhouse gas (GHG) emissions (54, 142). Cities offer major opportunities to reduce per capita environmental impacts relative to rural areas, due to short travel distances, reduced per capita living space, and efficiencies in delivering goods and services, but poorly designed cities forfeit these potential advantages. Moreover, because cities source most of their energy and goods from outside the city limits, a full accounting of the impact of cities on GEC extends well beyond city boundaries (105). Several pathways are well documented, and they often feature contributions both to GEC and to NCDs.

First, the combination of automobile-dependent transportation systems and urban sprawl, and the concentration of industrial and domestic energy needs, means that large quantities of fossil fuels are burned in metropolitan areas. This practice contributes to climate change, as well as to regional air pollution.

Urban air quality is often poor, especially in cities in LMICs. According to the WHO (**http://www.who.int/phe/health_topics/outdoorair/databases/cities/en/**), more than 80% of people living in urban areas that monitor air pollution are exposed to air-quality levels above WHO guidelines, and 98% of cities in LMICs with more than 100,000 inhabitants do not meet WHO air-quality standards. A systematic review found that, globally, 25% of urban ambient PM air pollution comes from traffic, 15% from industrial activities, 20% from domestic fuel burning, 22% from unspecified sources of human origin, and 18% from natural dust and salt (71). In cities where indoor solid fuels are commonly used, exposure to ambient air pollution is compounded by household air pollution exposure, an important source of black carbon that is a powerful SLCP (85). The contributions of air pollution to NCDs have been discussed above.

Second, automobile-dependent transportation systems contribute both to GHG emissions and to sedentary lifestyles, as walking, cycling, and mass transit give way to private vehicular travel. A recent study of 14 cities in 10 countries identified 4 environmental attributes significantly associated with physical activity: net residential density, intersection density, public transport density, and number of parks. (In contrast with some other studies, land use mix and distance to transit did not show this association.) The difference in physical activity levels between the most and least activity-friendly neighborhoods ranged from 68 to 89 minutes per week (of a recommended total of 150 minutes per week) (116). A recent prospective study in Perth, Australia, confirmed the role

of street connectivity in predicting walking and also found associations for land use mix, nearby public transit stops, and a variety of local destinations (76). Sedentary lifestyles increase the risk of, and physical activity is protective against, a range of NCDs, including cardiovascular disease, some cancers, hypertension, obesity, depression and anxiety, osteoporosis, gall bladder disease, and others (81, 150). The links among automobile dependency, sedentary behaviors, and GHG emissions may be bidirectional, as increasing obesity makes walking more difficult and may be associated with higher transport-related GHG emissions (57).

Finally, several common features of urban life are indirectly linked to GEC but impact NCD risk. Noise is a common exposure in cities, caused by traffic, machinery, and other sources (which are also GEC contributors). Noise is a stressor that contributes to a range of NCDs, including hypertension, cardiovascular disease, anxiety, sleep disturbance, and hearing loss (111). City living may provide fewer opportunities for contact with nature and green space compared with rural living. Systematic reviews suggest that such salutogenic exposure reduces the risk of obesity (78), diabetes, hypertension, and hyperlipidemia (17), depression and anxiety (49), and cardiovascular mortality (50), although the quality and consistency of the evidence vary. Crowding is another common feature of urban life, especially in impoverished parts of cities. While crowding is difficult to define with precision and is context dependent (134), it is associated with similar cardiovascular and mental health effects as found with noise. Children may be especially susceptible to the effects of both noise and crowding (36, 129). [Urban density is associated with reduced transport-related GHG emissions (84), underlining the importance of evidence-based design that achieves the benefits of density while avoiding disbenefits such as noise and subjective crowding.] Inadequate housing is also a problem for many cities, especially those undergoing rapid growth and those in poor nations. Substantial portions of urban populations live in slums—more than half the population of cities such as Nairobi, Mumbai, and Mexico City, totaling an estimated 881 million people globally and rising (38). In such settings, the effects of inadequate housing—chronic stress, contaminated indoor air due to the use of solid fuels, exposure to temperature extremes, injury risks, and more—are amplified by failures in community infrastructure and health services. Respiratory disease and mental disorders are likely results, although few studies have examined the burden of NCDs in slums (38).

Conversely, GEC exerts impacts on cities (and on urban health, including NCD risk). First, droughts, floods, and other environmental disruptions propel rural-to-urban migration, a pattern documented across the Global South (9, 151). The resulting contribution to rapid urban growth intensifies such problems as air pollution, crowding, noise, and strained infrastructure and services. Second, environmental change may particularly threaten cities, through such pathways as heat [amplified by the urban heat island effect (104)], flood risk (60), water scarcity (60), and diminished air quality (102). These vulnerabilities may increase the risk of NCDs such as cardiopulmonary disease (from heat and reduced air quality) and mental illness (from heat and disasters) for people in urban areas. This risk is especially pertinent in poor cities such as those in the Global South, which lack the infrastructure needed for resilience (53), and particularly for people in informal settlements, because of their location (flood plains, steep slopes, etc.) and their lack of infrastructure (120).

There are complex social, economic, cultural, and physical determinants of NCDs in urban environments. Improving understanding of these risks is a priority for research (125). The challenge will be to provide housing and infrastructure that support health and livelihoods, particularly in rapidly growing smaller and midsized cities in LMICs, while avoiding the costly errors of some wealthy cities. Transforming existing cities so that they flourish at much lower levels of environmental impact represents a major policy challenge.

Food, Nutrition, and Agriculture

The relationships among GEC, diet, nutrition, and NCDs are complex and multidirectional. Like cities, food systems both contribute to environmental change and are vulnerable to its effects. Here we discuss three examples of interacting processes. First, the global dietary transition, featuring growing demand for both dietary animal products and processed foods, has direct health consequences, as well as environmental consequences that indirectly affect health. Second, environmental change affects agricultural productivity, threatening nutritional status in some regions, with implications for NCDs. Third, environmental change affects the nutritional content of some foods, with implications for NCDs.

The global dietary transition has been under way for several decades (107). It is marked by the consumption of energy-dense, less diverse, and increasingly processed foods, including animal products, oils and fats, refined carbohydrates, and sugar-sweetened beverages, and by behavioral changes such as increased snacking and increased eating outside the home. This transition is well demonstrated by evidence from LMICs (59), and, with increasing prosperity, population growth, and aging, it is expected that the NCD burden will continue to increase in the absence of strong preventive actions (26). This dietary pattern has direct implications for NCDs, promoting obesity, cardiovascular disease, diabetes, and some cancers (5, 15, 80, 106, 107). It also has environmental impacts that, in turn, may loop back to exert indirect impacts on human health. For example, diets heavy in meat (particularly ruminant meat) require substantially more land, water, and energy throughput to produce than plant-based diets (159). The FAO (39) has estimated that livestock production accounts for 18% of global GHG emissions. In Latin America, conversion of land to pasture for cattle is a major driver of deforestation (particularly in Brazil where it accounts for about 80% of forest loss); this land use change contributes to greenhouse gas emissions, which in turn contribute to NCD risk and to biodiversity loss (30).

A second food-related pathway from GEC to NCDs is the effect of climate change on agricultural productivity. Climate change reduces crop yields particularly at low and mid latitudes. Compared with a scenario without climate change, crop yields (wheat, rice, and maize) could decline by a median of 0.2% per decade for the rest of the century, even as demand increases by an estimated 14% per decade (109). Recent analysis suggests even larger losses, especially in temperate zones, due to the flourishing of insect pests with warmer weather (31). Climate change could therefore substantially increase the risk of undernutrition and stunting, particularly severe stunting (86). Whereas stunting has plateaued in Africa since 1990 at a prevalence of about 40%, evidence has shown a dramatic decrease in Asia from 49% in 1990 to 28% in 2010, suggesting that vulnerability may be highest in Africa (29). Stunting has serious implications, including impaired cognitive development and reduced economic prospects for those affected; these impacts persist in subsequent generations (146). Some evidence suggests that stunting increases the risk of obesity in later life, although this point remains controversial (140).

A recent modeling study of the effects of climate change on crop yield by 2050 suggested a net annual increase of about 529,000 premature deaths (95% CI averaged over all climate scenarios 314,000–736,000) compared with no climate change, mainly as a result of increased NCD mortality (130). Reductions in fruit and vegetable consumption were the major contributor to increased mortality, and most climate-related deaths were projected to occur in South and East Asia. Decreased caloric intake was projected to increase deaths related to undernutrition, but these were approximately balanced by reduced deaths from overweight and obesity.

A third food-related pathway from GEC to NCDs is the effect of environmental change on the nutritional value of crops. A meta-analysis of data on 130 varieties of plants found that elevated carbon dioxide (CO_2) levels reduced the overall concentration of 25 important minerals in plants,

including calcium, potassium, zinc, and iron, by 8% on average (87). An estimated 175 million (95% CI 162–186) more people, mainly in Africa and South Asia, could be placed at new risk of zinc deficiency by 2050 as a result of elevated CO_2 levels (127). Strong evidence indicates that elevated CO_2 levels also reduce protein and increase carbohydrate levels particularly in C3 crops. One study found that elevated CO_2 was associated with a ~6% decrease in protein in wheat grains and an ~8% decrease in rice grains but demonstrated no significant effect in soybeans (93). The health implications of these changes are under investigation and are likely to depend on the nutritional profile of the affected populations. A study in the United States showed that the substitution of dietary carbohydrate for dietary protein increased the risk of hypertension, lipid disorders, and 10-year coronary heart disease risk (6). In low-income countries, if protein intake is low, there may be additional substantial impacts on public health.

Oil palm cultivation exemplifies the complex relationships among GEC, diet, nutrition, and NCDs. Production is increasing as a result of demand for biofuels in Europe and food in India, Indonesia, and China. Oil palm plantations are consistently less biodiverse than primary forests; they contain about 50% of the vertebrate species found in primary forests and show reduced species richness compared with secondary forests (118). To clear tropical forests in Indonesia for palm oil (as well as timber) production, fire is commonly used; the resulting smoke contains fine PM that contributes to a global burden of more than 250,000 deaths annually (70). Clearing tropical forests to produce palm oil is also deleterious in terms of climate change. And dietary palm oil contains highly saturated fatty acids, which have been proposed to be a risk factor for heart disease and other NCDs (10). A systematic review and meta-analysis (41) of short-term dietary intervention studies suggests, however, that both favorable and unfavorable changes in coronary heart disease/cardiovascular disease risk markers occurred when palm oil was substituted for primary dietary fats and that additional evidence is needed to inform firm conclusions.

Two additional pathways from GEC to NCDs are the effects of fisheries depletion and pollinator loss. These are discussed below in the section on biodiversity.

Persistent Chemicals in the Environment

Another feature of the Anthropocene has been widespread contamination of the global environment by chemicals (32, 79). Although many features of this contamination, from its geographic extent and levels in various biota to its full impacts on human health, are not fully understood, the links with NCDs are coming into sharper focus. Two examples are illustrative: endocrine disrupters and metals.

Endocrine disrupters include diverse classes of chemicals that affect endocrine pathways, by either blocking or activating receptors in sex hormone, thyroid, or other pathways. Many endocrine disrupters are synthetic organic chemicals such as polychlorinated biphenyls, bisphenols [e.g., bisphenol A (BPA)], organochlorine pesticides, brominated flame retardants, and perfluorinated substances (perfluorooctanoic acid and perfluorooctane sulfonate). Many such chemicals persist in the environment over long periods of time and are therefore called persistent organic pollutants (POPs). While there is considerable variation among these chemicals, three points are relevant. First, they are widely distributed in global ecosystems. Second, human exposure is widespread. Third, evidence suggests numerous associations with NCD risk.

With regard to environmental distribution, POPs have been found in nearly every ecosystem assessed, including in Arctic and Antarctic regions remote from any sites of manufacturing or use (149). Once these substances enter the environment, global transport is widespread, in both biological and physical media. Fat-soluble chemicals are concentrated (biomagnified) as they move from lower to higher trophic levels of food webs (89). A recently recognized contributor is the

presence of plastics in various ecosystems, from oceans to lakes to coastlines (8). Plastic fragments and microplastics are now commonly found in the gastrointestinal tracts of many marine birds and fish (114). Component monomers such as propylene and ethylene are relatively nontoxic, but associated plasticizers such as BPA and other adsorbed organic chemicals may have substantial biological activity (113). Of note, concomitant global trends affect the distribution of POPs. For example, climate change, operating through such processes as altered ocean currents, changes in temperature, and erosion of soils following severe weather events, may both liberate more pollutants and accelerate their breakdown (94, 148).

When POPs are widely distributed through ecosystems, human exposure is unavoidable. Measurement of population tissue levels of POPs has revealed nearly ubiquitous body burdens (22, 108). While levels of various contaminants vary by location, age, dietary patterns, and other factors, few humans escape exposure to, and accumulation of, at least some POPs.

Evidence suggests that these exposures play a role in several NCDs through both epigenetic and nonepigenetic mechanisms (65). POPs exposure has been associated with metabolic conditions such as adiposity, insulin resistance, and dyslipidemias, although evidence is inconsistent and methodologic shortcomings are common in published studies (67, 82, 137). POPs exposure has also been associated with the risk of some cancers, especially non-Hodgkin's lymphoma (45) and hormone-responsive cancers such as those of the breast, ovaries, and prostate; there is considerable animal evidence, but human epidemiologic evidence is less definitive (58). POPs may also increase the risks of thyroid disease, neurobehavioral disorders, and reproductive dysfunction (58). For each of these outcomes, the role of ambient environmental contamination relative to local exposures, such as in the workplace, remains to be defined.

Metals represent a second example of widespread chemical contamination. Human exposure to metals is typically a local phenomenon; examples include the child who consumes lead from aging paint in substandard housing or the worker exposed to mercury in artisanal gold mining. However, more dispersed mobilization of metals has occurred, with impacts that are regional if not global. For example, in China, industrial activity, mining, and the use of inadequately treated wastewater for irrigation have contaminated soil across large portions of the country with lead, cadmium, chromium, and other metals (74). Approximately 250,000 km^2 of farmland—an area equivalent to the arable farmland of Mexico—is contaminated, and in 2017 Chinese officials reportedly designated 35,000 km^2 of farmland as too polluted to permit any agricultural use (34). As another example, mercury emissions have been a feature of coal combustion since the dawn of the Industrial Revolution. Mercury contamination is concentrated near point sources such as power plants, smelters, and cement, iron, and steel plants; however, this is also a global phenomenon, as the mercury in air emissions travels great distances, even intercontinentally, before settling back to earth (121). The metals, collectively, contribute to many NCDs, including neurobehavioral abnormalities (lead, mercury), cardiovascular disease (lead, cadmium), renal disease (lead, cadmium), and some cancers (arsenic, chromium) (97).

Biodiversity Loss

Biodiversity loss has accelerated dramatically during the Anthropocene (96). Although this loss has numerous potential impacts on human health, including on NCDs (12), two examples are especially illustrative: the effects of pollinator loss and of fisheries depletion.

Pollination by insects is an important form of reproduction for more than 35% of the annual global food production by volume. At least 87 major types of food crops, and up to 40% of the world's supply of some micronutrients, such as vitamin A, depend on pollination by insects (75). Pollinators are declining in many parts of the world probably for a combination of reasons,

including habitat loss, pesticide use, and parasitic infestation. Pollinator loss can reduce the amount of fruits, vegetables, and nuts and seeds in the diet and lead to vitamin A and folate deficiencies. A recent analysis projects that a 50% loss of pollination would cause an additional ~0.7 million deaths worldwide, mostly as a result of increased ischemic heart disease and stroke due to reduced fruit and vegetable consumption (128).

Fisheries depletion has emerged as a global problem, with about 90% of fisheries at or beyond maximum sustainable levels of exploitation (40). Climate change will intensify this problem in coming decades (28). For many human populations, fish is a leading dietary source of protein, of micronutrients (often in highly bioavailable forms), and of omega-3 fatty acids (mainly from oily fish). Dietary omega-3 fatty acids may reduce ischemic heart disease risk, although research findings remain inconsistent (110). In addition, through gene regulation, anti-inflammatory effects, or other mechanisms, omega-3 fatty acids may play a role in preventing and/or treating other NCDs such as cancer (83) and arthritis (122). However, reductions in fish stocks could limit these potential benefits. For example, the United Kingdom is unable to meet healthy diet guidelines for its population with its domestic catch, and intake fell to only 19% of the recommended level in 2012 (139). One study projected that more than 10% of the global population could face micronutrient and fatty-acid deficiencies due to fish declines over the coming decades, especially in LMICs near the equator (55). Aquaculture is, to some extent, replacing wild fish catches, but sustainable aquaculture must address a range of challenges including disease and chemical contamination in farmed fish, unsustainable feedstocks, water pollution near fish farms, and genetic contamination of wild fish (20).

CONCLUSIONS

The Importance of Systems Understanding

Although we have presented five aspects of GEC—energy, air pollution, and climate change; urbanization; food, nutrition, and agriculture; chemical contamination; and biodiversity loss—in separate discussions, they are by no means distinct. A complete understanding of their effects on NCDs requires consideration of their interconnections (**Figure 4**). For example, energy, pollution, and biodiversity are linked. Heavy reliance on petroleum as an energy source results

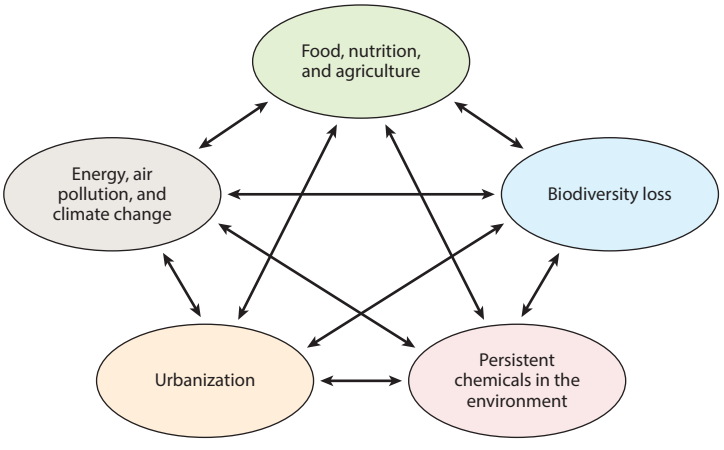

Figure 4

Interactions among various global environmental changes.

in contamination of marine environments by petroleum-derived pollutants, which are toxic to developing fish and which compound threats to some fisheries (23). Similarly, urbanization is linked with climate change in many ways. Cities occupy only 2% of the world's landmass, but as noted above they account for the lion's share of global energy use and global CO_2 emissions. The form of urbanization can, in turn, determine a city's experience of climate change. For example, evidence from the United States suggests that sprawling metropolitan areas are at substantially higher risk of extreme heat events than are more compact urban areas (132). Ample, well-designed urban green space and blue space can not only promote health directly, but also help reduce the heat island effect (104). A third example is the link between urbanization and food systems. Rural-to-urban migration in LMICs is associated with rapid changes in dietary patterns, including the adoption of Western-style processed foods; one study found the obesity prevalence in Accra, Ghana, to be 4–5 times higher, and the diabetes prevalence 2–3 times higher, than in rural areas of Ghana (2). The various pathways through which GECs affect NCD risk are interrelated in complex ways, and solutions must take account of these complexities.

Planetary Health: Reducing NCD Risk and Achieving Environmental Sustainability

The preceding discussion has offered an analysis of links between planetary changes and NCD risk. In each of the five areas discussed, strategies are available that simultaneously promote health and well-being and environmental sustainability (61, 79, 154). Clean, renewable energy sources can replace fossil fuels. The built environment, from the scale of buildings to the scale of entire metropolitan areas, including both stationary assets such as buildings, parks, and utilities, and the mobile components of transportation systems, can be designed in ways that optimize human health as well as environmental performance. The food system, from farm to table, can be shifted toward more sustainable food production, consumption of healthier foods, and less waste. Green chemistry aims to design molecules that are less persistent, less toxic, and as functional as many legacy chemicals, if not more so. And management strategies for land, water, and other ecosystem components can optimize biodiversity conservation, for example through forest conservation, as well as reduce air pollution from landscape fires. In light of the growing evidence that GECs can affect NCD risk through a range of pathways, it is imperative that the evidence base linking the two be strengthened and greater investment be devoted to policies that can reduce both environmental change and NCD risk.

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