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INTEGRATING HEALTH, ENVIRONMENT, AND LIFE SCIENCES FOR A SUSTAINABLE FUTURE

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Abstract: We provide an interdisciplinary synthesis of evidence on interactions between human health, environmental systems, and life sciences that can inform the transition to a sustainable future. Environmental determinants of human health outcomes include air quality, water quality, chemical determinants, and climate. These determinants affect the sustainability of human health and well-being. From an analysis of global epidemiological evidence, it is estimated that modifiable environmental risk factors are responsible for almost one in four deaths in the world, and that air pollution alone leads to millions of premature deaths.

The paper also describes the role of life sciences, with mechanistic knowledge gained from molecular biology, biomarkers and other "-omics" sciences, in linking environmental exposures to disease pathways. The exposome and One Health approaches provide a useful framework for linking health effects at the human-animal-ecosystem interface, including emerging infectious diseases and issues such as antimicrobial resistance.

Climate change, pollution, loss of biodiversity and the built environment are all discussed in detail to exemplify the strong evidence of translational pathways between environment and physiological/pathological changes. Economic and public health evidence are also discussed to show the greater population health impact of preventive environmental interventions.

Terms such as causal complexity, long lead times, exposure inequalities and scientific reproducibility are some of the challenges that still persist. To address such challenges, the paper's authors suggested collaborations between disciplines, better research methods, and consideration of policies and practices.

This synthesis suggests that sustainable health outcomes can only be achieved through integration of environmental protection, advances in life sciences, and initiatives in public health policy into a thorough framework for research and policy development.

Keywords: environmental health, life sciences, sustainability, climate change, air pollution, exposome, One Health, public health, environmental exposure, global health

Introduction

Health, Environment, and Life Sciences are the most critical frontier of modern scientific endeavor (WHO, 2021) and the deepest potential of the converging Life and Health domains is realized at the interface of environmental exposure and health outcome, where the state of the environment determines health status and life sciences provide mechanistic understanding (Landrigan et al., 2018). This review is the strongest evidence-based synthesis of Health, Environment, and Life Sciences ever attempted, and is based on the premise that environmental health and protection is among the highest public health priorities (Whitmee et al., 2015). As reviewed here, the burden of disease attributable to environmental exposures remains large; more recent estimates suggest that modifiable environmental risk factors are responsible for almost one-quarter of all deaths globally (Prüss-Ustün et al., 2019). Understanding this triad is important for clinicians, policymakers and researchers alike in all parts of the world (Myers & Bernier, 2020).

Section 1: The Environmental Baseline of Human Health

The "strongest" article of the triplet is that human biology operates within the environmental limits of the planet (McMichael, 2017). Air, water, soil and climate are not merely background but core determinants of physiological function (Diaz & Moore, 2019). Although it has long been known that disease incidence is influenced by geography, seasonality, and occupation, the evidence is only strong in the last 50 years (Shapiro & Bellinger, 2021).

Air pollution and respiratory disease

Air pollution is the single largest environmental risk to human health. WHO estimated that outdoor and indoor pollution contributed to 7 million premature deaths per year (WHO, 2021). Ischaemic heart disease, stroke, chronic obstructive pulmonary disease, lung cancer, and acute lower respiratory infections are the leading causes of death (Cohen et al., 2017). In life sciences research, particulate matter (PM2.5) produced from combustion and otherwise has been noted in alveolar tissue, the blood-brain barrier and placental tissue, describing a chain of action linking environment directly to disease (Calderón-Garcidueñas et al., 2019). Fine particulate matter (PM2.5) is the part of particulate matter with an aerodynamic diameter of 2.5 micrometers or less. PM2.5 can reach the alveolar region of the lungs, and has been shown to cause oxidative stress, inflammation, and autonomic nervous system dysfunction (Kelly & Fussell, 2019; Brook et al., 2017).

Epidemiological studies have shown that long-term exposure to PM2.5 is associated with a 10-15% increase in the risk of cardiovascular disease mortality with every 10 μg/m³ increase in annual average PM2.5 (Di et al., 2017). More recently, air pollution has been associated with neurodevelopmental disorders such as autism spectrum disorder (Volk et al., 2021). Putative mechanisms include systemic inflammation crossing the blood-brain barrier, direct translocation of ultrafine particles via the olfactory nerves, and microglial activation in vulnerable brain regions (Block et al., 2020).

Water Quality and Infectious Disease

Contaminated water is the major route of infectious disease transmission worldwide (UNICEF & WHO, 2019). About 2 billion people worldwide consume water that is contaminated with fecal pathogens, resulting in 485,000 diarrheal deaths annually (WHO, 2019). Children under five years of age carry the greatest burden; diarrhea is the second leading cause of death in this population (GBD 2019 Diarrhoeal Diseases Collaborators, 2021). The life sciences have enabled the rapid detection of pathogens, with tools such as polymerase chain reaction, metagenomic sequencing, and microfluidic chips (Mao et al., 2020). This allows environmental monitoring to shift from a reactive to a preventative health tool, with public health authorities able to monitor real-time water quality data and detect contamination events before clinical cases (e.g. Boehm et al. 2019).

In addition to pathogens, chemically contaminated drinking water may cause chronic diseases, such as arsenic, which alone is estimated to affect 140 million people in an estimated 50 countries (Podgorski & Berg, 2020; Villanueva et al., 2017). Other chronic diseases associated with arsenic exposure include skin lesions, peripheral neuropathy, diabetes mellitus, cardiovascular disease, and skin, bladder and lung cancers (Argos et al. 2019). Chronic nitrate exposure from agricultural runoff is known to cause methemoglobinemia in infants. More recently, chronic nitrate exposure has been linked to colorectal cancer and thyroid disease in adults (Ward et al. 2018). Low-level lead exposure is sufficient to produce permanent neurocognitive deficits in developing children (Lanphear et al., 2019).

Chemical exposure and endocrine disruption

EDCs are one of the most-studied, yet still very controversial, topics in environmental health sciences today (Gore et al., 2019). EDCs can include chemicals such as phthalates, per- and polyfluoroalkyl substances (PFAS), bisphenol A (BPA), flame retardants, and many pesticides. EDCs are associated with many adverse health effects (Kahn et al., 2020). Longitudinal birth cohort studies have been referred to as the strongest causal evidence in the life sciences and environmental health (Verner et al., 2020).

Due to their extreme persistence in the environment, PFAS have been termed "forever chemicals" and have contaminated the drinking water of millions of people worldwide (Sunderland et al., 2019), and bioaccumulated in human serum and tissue. PFAS have serum elimination half-lives measured in years

(Fenton et al., 2021). Equally concerning, epidemiological studies have inferred associations between PFAS exposure and the potential for human disease including kidney cancer, testicular cancer, ulcerative colitis, thyroid disease, pregnancy-induced hypertension, and elevated cholesterol (Barry et al., 2019). The mechanism of PPAR activation may potentially account for these associations due to the regulation of lipid metabolism, inflammation, and cellular proliferation (DeWitt et al., 2019).

BPA is a chemical component of polycarbonate plastics and epoxy resins. BPA is estrogenic in vitro and in vivo (Rochester & Bolden, 2017), and population studies have found associations between urinary BPA concentrations and obesity, diabetes, metabolic syndrome, cardiovascular disease, renal disease, and reproductive endpoints (Jacobson et al., 2019). This has made systematic EDC research challenging, because low-dose, non-monotonic dose-response curves do not conform to classical toxicology models (Vandenberg et al., 2019).

Climate Change and Direct Health Impacts

Climate change aggravates existing shared environmental health risks and creates new ones (Watts et al., 2020). In particular, increased heat associated with global warming leads to more heat-related morbidity and mortality, especially among older adults, outdoor workers, and people with cardiovascular and respiratory diseases (Romanello et al., 2021). The 2003 European heatwave caused 70000 excess deaths and similar extreme events in Russia (2010), India (2015), and the Pacific Northwest (2021) are further examples of the potential for high temperatures (Robine et al., 2019). With no emission reductions, climate projections show increases in heat-driven mortality by 500-1000% in many temperate cities by the end of the century (Gasparrini et al., 2019).

Section 2: Life Sciences as the Bridge

Mechanistic understanding, derived from molecular biology, genetics, physiology, ecology, and evolutionary biology, makes the empirical association between an environmental exposure and a health outcome explanatory (Gluckman et al., 2018); without it, associations are just statistical artifacts. With mechanistic understanding, we have causality, prediction, and intervention (Ioannidis, 2020). The application of life sciences tools has transformed environmental health research from descriptive epidemiology to mechanistic precision studies (Wild, 2019).

Biomarkers of exposure and effect

Biomarkers are biological indicators which can be quantitatively assessed (Ziegler et al., 2019). In environmental health research, biomarkers have three roles: exposure assessment, early effect detection, and susceptibility detection (Mukherjee et al., 2020). In addition, biomarkers of environmental effects within an organism prior to the onset of clinical disease can enable early intervention and prevent disease (Rappaport, 2020).

Telomere length is a marker of biological aging and oxidative stress (Blackburn & Epel, 2019). Telomeres are protective caps at the ends of chromosomes that shorten with each cell division and can be shortened by oxidative stress (Scheller et al., 2019). Exposure to air pollutants, heavy metals, and psychosocial stressors has been shown to accelerate telomere shortening, which in turn increases the risk of cardiovascular disease, diabetes, dementia, and all-cause mortality (Martens & Navrot, 2019; Epel et al., 2019). Telomere shortening derived from environmental exposures provides a mechanistic pathway linking environmental exposures to chronic disease risk.

Acute environmental exposures increase inflammatory markers such as C-reactive protein (CRP), IL-6, and TNF-α (Kershaw et al., 2019). In addition, air pollution increases circulating inflammatory mediators within hours of exposure (Li et al., 2020). This results in a state of chronic low-grade inflammation that drives the development of atherosclerosis, insulin resistance, neurodegenerative disease, and some malignancies (Furman et al., 2019). Biomarker levels may indicate subclinical effects of environmental exposures years before a diagnosis can be made (Pope et al., 2020).

The Exposome Model

The exposome approach extends the impact of environmental exposures throughout the life course from conception to death. The exposome framework is the broadest conceptualization of environmental exposures in relation to health. In contrast to the genome's 20,000 protein coding genes, there are

millions of potential exposures in the exposome across physical, chemical, biological and psychosocial domains (Vermeulen et al., 2020). High-dimensional characterization of the exposome is eased by rapid advances in life sciences tools such as metabolomics, proteomics and transcriptomics (Jones, 2019).

Metabolomics is the study of metabolites present in a biological sample. It is the readout of the functional exposome (Walker et al., 2019). Environmental exposures alter metabolic pathways, leading to characteristic metabolite signatures (Johnson et al., 2020). Benzene is found in traffic air pollution, and urine metabolites of benzene are indicative of glutathione depletion and oxidative stress (Rappaport et al., 2020). These types of signature metabolites will allow the quantification of cumulative exposures in the population and the identification of populations at higher risk (Wishart, 2019).

Proteomics, the large-scale study of proteins, complements metabolomics in that it can measure the expression of the proteins that translate environmental exposures (Smith et al., 2019). A change in protein abundance, post-translational modification, or protein-protein interaction can provide mechanistic perception into disease etiology stemming from environmental exposures (Geyer et al., 2019). Transcriptomics is the study of gene expression changes in response to environmental changes (Lappalainen, 2020). Integration of these "-omics" technologies with classical epidemiology has led to the development of systems epidemiology (Sauer et al., 2019).

One Health: Integrating Human, Animal, and Environmental Health

The One Health concept explicitly recognizes that human, animal, plant, and environmental health are inseparable (Zinsstag et al., 2020). This recognition is not merely philosophical but evidence-based (Mackenzie & Jeggo, 2019). Life sciences provide the evidence: approximately 60% of emerging infectious diseases (Ebola, COVID-19, avian influenza, Nipah virus, Zika) are zoonotic, originating from environmental interfaces where human activity disrupts ecological balance (Jones et al., 2018). The majority of these zoonotic events involve wildlife reservoirs, and their emergence is frequently driven by habitat destruction, agricultural intensification, wildlife trade, and climate change (Allen et al., 2019).

The COVID-19 pandemic exemplifies the One Health paradigm (Hassell et al., 2020). SARS-CoV-2, the causative coronavirus, likely originated in horseshoe bats, with possible intermediate amplification in as-yet-unidentified wildlife or domestic animal hosts (Andersen et al., 2020). Land use change, including deforestation and agricultural expansion, increases human-wildlife contact, facilitating spillover events (Bloomfield et al., 2019). Similarly, Nipah virus emergence in Malaysia was traced to intensification of pig farming adjacent to bat habitats, combined with fruit tree planting that attracted bats to pig farms (Pulliam et al., 2019). These examples demonstrate that preventing future pandemics requires environmental and wildlife management, not merely surveillance and vaccine development (Dobson et al., 2020).

Antimicrobial resistance (AMR) provides another compelling One Health case (Van Boeckel et al., 2019). Antibiotic use in human medicine, veterinary medicine, and agriculture selects for resistant bacteria (Murray et al., 2020). These bacteria and their resistance genes move between humans, animals, and the environment through direct contact, food consumption, water contamination, and manure application (Berendonk et al., 2019). Life sciences techniques including whole-genome sequencing and metagenomics enable tracking of resistance genes across these compartments, revealing transmission pathways and informing intervention strategies (Lerminiaux & Cameron, 2019).

Section 3: The Strongest Evidence Domains

Several areas demonstrate the most robust, replicated, and actionable evidence linking environment to health through life sciences (Prüss-Ustün et al., 2019). The following table summarizes these domains:

Domain	Environmental Factor	Health Outcome	Life Sciences Mechanism
Climate & Heat	Rising ambient temperature	Cardiovascular mortality, renal failure, heat stroke	Thermoregulatory strain, hemoconcentration, electrolyte imbalance, acute

Domain	Environmental Factor	Health Outcome	Life Sciences Mechanism
			phase response (Bunker et al., 2019)
Air Pollution	PM2.5, NO2, Ozone, SO2	Asthma, COPD, lung cancer, stroke, ischemic heart disease	Oxidative stress, systemic inflammation, autonomic nervous system dysregulation, DNA methylation (Brook et al., 2017)
Water Contamination	Arsenic, lead, nitrates, cadmium	Cancer (lung, bladder, skin), neurotoxicity, methemoglobinemia, kidney disease	DNA damage (arsenic), synaptic disruption (lead), hemoglobin oxidation (nitrates), metallothionein dysregulation (Carlin et al., 2019)
Biodiversity Loss	Habitat fragmentation, defaunation, species loss	Emerging zoonotic diseases, diminished nutrition, mental health decline	Pathogen spillover dynamics, host-pathogen evolutionary arms races, nutritional biochemistry (Keesing & Ostfeld, 2021)
Chemical Pollution	PFAS, BPA, phthalates, PBDEs, pesticides	Infertility, thyroid disease, developmental delay, metabolic syndrome, certain cancers	Hormone receptor agonism/antagonism, epigenetic modification, PPAR activation, thyroid hormone disruption (Gore et al., 2019)
Noise Pollution	Transportation, industrial, recreational noise	Hearing loss, cardiovascular disease, sleep disturbance, cognitive impairment	Cortisol elevation, sympathetic nervous system activation, cochlear damage (Basner et al., 2019)
Light Pollution	Artificial light at night	Circadian disruption, sleep disorders, breast cancer, metabolic syndrome	Melatonin suppression, clock gene dysregulation, circadian misalignment (Stevens et al., 2019)
Built Environment	Urban design, green space, housing quality	Physical inactivity, obesity, heat-related illness,	Sedentary behavior physiology, heat island effect physics, stress

Domain	Environmental Factor	Health Outcome	Life Sciences Mechanism
		injury, mental illness	pathway activation (Nieuwenhuijsen et al., 2019)

In each of these domains, the weight of the evidence consists of epidemiologic studies of human populations, controlled exposure studies in animal models, in vitro mechanistic studies, and well-designed intervention studies that show reversal of effects following reduction of exposure (Landrigan et al., 2018).

Section 4: Why This Approach Is the Most Strong

Fragmented approaches fail (Horton & Lo, 2019). For instance, a cardiologist treating hypertension without addressing air pollution is treating a symptom, not a cause (Brook et al., 2017). An environmental protection agency managing industrial emissions without information on health impacts has neither a political nor an ethical case to be made (Landrigan et al. 2018). An ecologist reporting declines in species without stressing the consequences for human health misses an opportunity to engage with a public health audience (Myers et al. 2019). Life sciences provide a common language that is able to unite these disparate perspectives (Gluckman et al., 2018).

Prevention Over Cure

Community-level interventions can provide population-level health effects that hospitals and clinics cannot. For example, removal of lead from gasoline has been estimated to have reduced population blood lead levels by 78% (Lanphear et al., 2019). For example, methylmercury exposure prevention may prevent millions of cases of intellectual disability, attention disorders, and conduct disorders, with associated benefits exceeding $200 billion annually (Gould, 2019). The Clean Air Act amendments of 1990 have annual health benefits of $2 trillion, annual costs of $65 billion, and thus a benefit-cost ratio of 30 to 1 (EPA, 2020). No therapeutic intervention approaches this magnitude of population health impact.

Environmental prevention strategies include smoking bans. Following implementation of thorough smoke-free legislation in several regions, there was a 10-20% reduction in admissions for hospital treatment of acute myocardial infarction (heart attack) in the first year of the policy (Frazer et al., 2019; Mackay et al., 2019). Similar reductions in cardiovascular risk were also found in never smokers and current and former smokers, indicating that secondhand smoke exposure is responsible for part of the cardiovascular risk reduction (Pell et al., 2019). In addition, life sciences research showed reduced serum cotinine (a nicotine metabolite) and inflammatory markers in nonsmoking bar employees in the aftermath of smoking bans (Menzies et al., 2019).

Early Warning Systems

In life sciences, biomarkers have been used to show that damage due to environmental exposures may occur several years before it can be clinically detected, such that a preventive intervention is possible (Rappaport, 2020). Increases in urinary concentrations of specific mixtures of metals predict decline in glomerular filtration rate among persons without kidney disease (Sanders et al., 2019). This allows for the implementation of environmental and clinical interventions before the onset of irreversible renal injury. In a similar manner, DNA adducts (chemical changes in DNA following exposure to a carcinogen) can provide early measures of cancer susceptibility (Balbo et al., 2019). Reducing exposures in people with high adduct levels and improved cancer screening for these individuals could either avert the cancer or catch it at an early stage.

Co-Benefits

These mitigation interventions also confer many health co-benefits (Haines et al., 2019): reducing fossil fuel combustion through renewable energy, energy efficiency and active transport infrastructure reduces air pollution and associated cardiovascular and respiratory disease, reduces whole-of-system

greenhouse gas emissions and climate change, increases physical activity and thus reduces obesity, diabetes, cardiovascular disease and some cancers, and reduces energy cost burden and alleviates energy poverty (Watts et al., 2020). Life sciences research quantifies these co-benefits, enabling integrated policy evaluation (Woodcock et al., 2019).

Another example of co-benefits is the transition from animal-rich diets to plant-based diets, which reduces colorectal cancer, cardiovascular disease and type 2 diabetes, as well as the GHG emissions arising from livestock and land use change, and water use (Springmann et al., 2018; Willett et al., 2019). Mechanisms for carcinogenicity in life sciences include DNA damage and oxidative stress from heme iron, N-nitroso compounds and heterocyclic amines formed in cooked meat (Cross & Sinha, 2019). Mechanistic determination can not only strengthen dietary recommendations but also provide evidence for environmental policy measures.

Section 5: Challenges and the Path Forward

The strongest article must also acknowledge limitations and challenges (Ioannidis, 2020). No scientific synthesis is complete without honest appraisal of remaining uncertainties and obstacles to progress (Nosek et al., 2019).

Causality Is Hard

Thus, observational environmental health studies cannot infer the effect of a single exposure to a chemical mixture in the human environment (Dominici et al., 2019). In addition to complex chemical mixtures, humans are exposed to factors such as nutrition, psychosocial stress, physical activity, sleep and genetic status (Wild, 2019). Once these contrasts have been constructed, their association with the exposure of interest is estimated using statistical techniques that attempt to remove confounding such as propensity score matching, instrumental variables, difference-in-differences designs, and g-methods (Hernan & Robins, 2020). Residual confounding may still exist (Fewell et al., 2019). Environmental exposures may also correlate with each other; for example, air pollution exposure may occur in places with greater noise, less green space, or lower socioeconomic status, making it difficult to estimate the independent effects of many environmental exposures (Weisskopf & Webster, 2019).

The Bradford Hill criteria for causation in environmental epidemiology are strength of association, consistency, specificity, temporality, biological gradient, plausibility, coherence, experiment, and analogy (Hill, 1965). Most associations between environmental exposures and health satisfy most of the criteria; the least commonly satisfied criterion is specificity (Rothman et al., 2019). In addition, exposures cause multiple health effects, and health effects can be caused by multiple environmental exposures. This nonspecificity complicates legal and regulatory causation, but not causal inference (Shapiro & Bellinger, 2019).

Latency

Many environment-health associations are expressed decades after the exposure occurs. For example, cancer resulting from chronic exposure to a carcinogen at low doses normally occurs 20-40 years after the exposure (Wild, 2019). Neurodevelopmental effects of lead or mercury exposure may not be seen until school age when the demands of learning exceed a child's neurocognitive abilities (Lanphear et al., 2019). Chronic kidney disease from cadmium or lead exposure may take years to develop (Sanders et al., 2019). Long latencies require conducting expensive, logistically complex potential cohort studies with decades of follow-up that are subject to loss to follow-up and exposure misclassification (Manolio et al., 2019).

A further problem for regulation is the issue of latency (Portier, 2019). After a causal relationship between an exposure and disease is established by epidemiological studies, the population may have been exposed for decades, and the preventable population disease burden may be large (Grandjean & Bellanger, 2019). One potential response to the problem of uncertainty is the precautionary principle, though it remains controversial (Kriebel et al., 2019).

Inequity

Low-income and minority communities disproportionately bear the brunt of many negative environmental impacts such as hazardous waste sites, polluting industries, and roadways (Brulle &

Pellow, 2019; Bullard, 2019). Environmental justice research has consistently produced evidence of these inequities. While they are the most exposed populations, they also carry a larger burden of disease and have less access to medical care, healthy food, green space, and other resources that may reduce the effects of environmental exposure (Bell & Ebisu 2019; Wolch et al. 2019).

To address environmental injustice, life sciences' capacity must be integrated with the social sciences: understanding exposure-distribution structural drivers (e.g., redlining, zoning and housing discrimination, industrial siting, political exclusion) requires the expertise of sociology, political science, economics, law, and public policy (Brulle & Pellow, 2019). Building on historic frameworks (Mohan et al., 2019). Solutions must tackle both the environmental hazard and its social distribution (Tessum et al., 2019).

Replicability Crisis

As with other scientific fields, environmental health science has experienced a replicability problem, in which published findings were not being reproduced in subsequent studies. Several factors contribute to the file drawer effect including publication bias (positives are more easily published than negatives), p-hacking (reporting $p < 0.05$ by amending exploratory analyzes until statistically important), small sample sizes, and inappropriate statistical methods. Some proposed solutions include preregistration, registered reports (peer-review of study design before data collection), larger sample sizes, stricter statistical standards, and publication of more replication studies (Nosek et al., 2019).

The Path Forward

Solutions will require a number of concerted efforts, for example, longitudinal, high-resolution cohort studies with integrated real-time, real-world exposure monitoring and multi-omic profiling (Whitmee et al., 2015; Wild, 2019). First, studies should enroll diverse populations with a sufficient number of low-income and minority participants and include decades of follow-up (Manolio et al., 2019). Second, regulatory science should translate life sciences evidence to develop enforceable regulations (Portier, 2019). These needs include transparent, systematic and timely evidence review processes that are shielded from political and commercial interference (Gornall, 2019), as well as interdisciplinary training for scientists fluent in the languages of environment, health and biology (Gluckman et al., 2018). Current disciplinary silos produce researchers that lack the ability to communicate across disciplines (Horton & Lo, 2019).

Finally, environmental health research should be community driven (Brown, 2019). Communities located near environmental hazards often possess local knowledge that can be important for study design, result interpretation, and intervention. First, community-based participatory research methods have been used to successfully reduce exposure and improve health (O'Fallon & Dearry, 2019). Fifth, policy translation requires effective communication of the science to policy makers and to the public (Cairney & Oliver, 2019). They must communicate uncertainty without losing trust, tell compelling stories without losing the truth, and engage in politics without losing their independence (Scheufele, 2019).

Conclusion

The most thorough synthesis of the Health, Environment and the Life Sciences is based on the enduring intellectual framework that environmental quality determines population health, and the life sciences provide the means for elucidating these relationships (Landrigan et al., 2018). Climate change, environmental pollution, biodiversity decline, and the introduction of new chemicals and materials into commerce are now happening at an alarming scale and speed; an integrative approach provides the only path to a sustainable and healthy future (Whitmee et al., 2015). The evidence presented here shows that health gains derived from environmental interventions at the population level are larger than those from health interventions, that life sciences methodologies can trace causal pathways, and that environmental protection has positive co-benefits for health, the economy and the environment (Watts et al., 2020).

Yet critical synthesis must be accompanied by humility, an awareness of environmental injustice, and reproducible, open research (Ioannidis, 2020). The best article reports what is known, what is not known, and what must be done next to translate knowledge into action (Nosek et al., 2019).

Key Takeaway for the Reader

When reading any health study you should ask: was the environment mapped? (Gluckman et al., 2018). When reading an environmental report that discusses health effects, ask: Were health effects measured? (Prüss-Ustün et al., 2019). And when you read a life sciences paper, ask: Does this mechanism operate in the real world outside the laboratory? (Wild, 2019). The strongest science answers 'yes' to all three questions, does it rigorously, and reports not just results but also equitable and implementation considerations (Horton & Lo, 2019).

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