

More Lack in the World

The Complex Connection between Undernutrition and Climate Change

Anthropogenic climate change is projected to reduce cereal yields and food security and therefore to undermine future efforts to reduce child undernutrition. But models are needed to better measure the potential impacts of climate change on population health. Now researchers have developed a model to estimate future undernutrition attributable to climate change as a function of its impact on crop productivity [*EHP* 119(12):1811–1816; Lloyd et al.].

Undernutrition is measured using criteria such as stunting (smaller-than-average height-for-age) and underweight (smaller-than-average weight-for-age). The researchers developed and validated the model using previously published data about past food availability, the prevalence of stunting, and gross domestic product. Then they used projections of future calorie availability under two climate change scenarios and a reference scenario of no climate change to estimate undernutrition among children under age 5 years in five regions of South Asia and sub-Saharan Africa in 2050.

The model estimates that climate change will lead to an average relative increase in moderate stunting (height that is more than two standard deviations



A Ugandan child scrapes out extra food from a cooking pot. Climate change is expected to undermine efforts against child undernutrition by reducing cereal yields and food security.

below the expected height-for-age) of 1–29%, depending on region, compared with a future without climate change. Climate change will have a greater impact on rates of severe stunting (height more than three standard deviations below the expected mean), which the authors estimate will increase by an average of 31–62%, depending on region.

Climate change is likely to affect undernutrition through a variety of means in addition to crop production, including impacts on infectious diseases in humans, plant pests and diseases, labor productivity, and water availability. One limitation of the current study is the difficulty in quantifying the impact of climate change in the face of uncertainty about how countries will develop and manage their food systems. The authors state that their current study illustrates the importance of the outcome used to predict impacts—undernourishment (lack of food) versus stunting, for instance, or moderate versus severe stunting have different implications for decision making and for population health.

The study adds to the evidence suggesting that climate change is likely to increase future hunger and undernutrition even under optimistic assumptions of future emissions and economic growth. The study results suggest that to reduce and prevent future undernutrition, it is necessary to not only reduce emissions of greenhouse gases but also increase food access and improve socioeconomic conditions.

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Hormone Impact

BPA Linked to Altered Gene Expression in Humans

Urinary metabolites of bisphenol A (BPA), a widely used component of polycarbonate plastics and epoxy resins, serve as biomarkers for exposure to the chemical and are detectable in more than 90% of individuals tested in the United States and Europe. Studies to date suggest positive associations between BPA and cardiovascular disease, diabetes, and reproductive and developmental abnormalities in humans, although further research is needed to confirm these findings. Recent studies have shown links between BPA and changes in total testosterone concentrations and altered estradiol:testosterone ratio in men, but evidence for a mechanism behind such links has been lacking. A new study now links BPA exposure with altered expression of estrogen- and androgen-responsive genes in humans [*EHP* 119(12):1788–1793; Melzer et al.].

The InCHIANTI study—a prospective study of mid- and late-life morbidity risk factors among 1,453 participants in Chianti, Italy—provided the data for the current study. A subset of 96 men provided same-day blood and urine samples in 2008–2009. Urine samples were analyzed for concentrations of BPA, and blood leukocytes were used for transcript analysis of six estrogen- and androgen-responsive genes: *ESR1*, *ESR2*, *ESRRA*, *ESRRB*, *ESRRG*, and *AR*. These genes code nuclear hormone receptors involved in the control of developmental and physiological pathways shown to be activated by BPA in laboratory studies.

Urinary BPA concentrations ranged from 0.73 to 56.94 ng/mL and were positively associated with increased expression of *ESR2* and *ESRRA* based on models adjusted for potential confounding factors. Transcripts for other genes were either not detected (*ESRRG*) or were not associated with BPA concentrations (*ESR1*, *ESRRB*, and *AR*). Mean expression of *ESR2* and *ESRRA* increased by 65% and 38%, respectively, in the highest versus lowest BPA exposure tertile.

The implications of altered gene expression in blood leukocytes are unknown, and this measure has not been validated as a surrogate measure of effects on hormone-responsive gene expression. However, the results suggest that BPA is bioactive in humans, and the authors argue that the potential link between exposure, hormone signaling, and related disorders is biologically plausible. For example, estrogen receptor β , coded by *ESR2*, plays a significant role in maintaining the structure and function of tissues in the cardiovascular and central nervous systems.

The cross-sectional design, lack of distinction between free and conjugated BPA in urine samples, and possible unidentified confounding factors are limitations of the study. Additional research is needed to confirm the findings and further investigate gene expression changes and effects of BPA exposure in other estrogen-regulated target tissues.

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