Networking to advance progress in children's environmental health

The 2010 Global Burden of Disease (GBD) estimates show a marked shift in the past 20 years away from deaths from acute communicable diseases to chronic non-communicable diseases (NCDs).[1] Early childhood deaths have reduced and years lived with disability (YLD) have increased.[1] The YLD attributable to communicable, maternal, neonatal, and nutritional diseases decreased by 19·5% between 1990 and 2010, whereas the YLD attributable to chronic disease increased: deaths from cardiovascular diseases by 17·7%; from chronic respiratory disease by 8·5%; from neurological disorders by 12·2%; from diabetes by 30·0%; and from mental and behavioural disorders by 5·0%.[2]

These estimates and assessments of the hazards posed by the diseases' underlying risk factors are based on a rigidly conservative method that requires a very high (sufficient) level of proof before a risk factor can be included in GBD estimates.[3] This approach creates a structural bias because it necessarily restricts the inclusion of newly recognised and emerging risk factors that have not yet achieved the full weight of evidence required for consideration.

Adverse environmental exposures in early life are among the risk factors systematically excluded from consideration in the present method. Yet understanding is increasing that many NCDs are initiated by early-life exposures to toxic chemicals, nutritional imbalances, and psychosocial stress.[4] For example, exposure to toxic or endocrine-disrupting chemicals in early life can affect metabolism that changes brain growth[5] or promotes obesity[6] and increases later risk of cardiovascular disease, diabetes, and cancer. Evidence is increasing that adverse environmental exposures could play a substantial part in progression of NCDs, including for respiratory diseases such as asthma, chronic obstructive pulmonary disorder, and lung cancer; neurobehavioural disorders, including attention deficit hyperactivity disorder, depression, and other mental disorders; mild mental disability; obesity and type 2 diabetes; and childhood cancer.[7]

The main reason that new environmental exposures have been systematically excluded from GBD analyses is an absence of full evidence of causation, which can be due to factors including the long time required to link low-dose exposures in early life with disease outcomes many years later; ubiquitous population exposures confounding traditional epidemiological methods; non-monotonic dose-response curves confounding traditional toxicology methods; and age-dependent and species-dependent susceptibility and metabolism-confounding translation of results of animal studies to human beings and from adults to infants. The result is that environmental exposures have not been accorded due consideration in public health planning and resource allocation.

To overcome these problems and to improve children's health by reducing the effect of adverse environmental exposures, WHO has been actively engaged in efforts to assess the environmental contribution to GBD. In the past decade, several WHO collaborating centres, affiliated with WHO's Public Health and Environment section, have been designated with terms of reference and work plans that advance the WHO agenda in children's environmental health. The recognition that children are increasingly at risk of living with chronic disease of environmental origin shows the importance of research and advocacy in this specialty.[7–9]

To further this agenda, the Public Health and Environment section, its WHO collaborating centres, and other partners have formed a collaborative network. This network aims to provide a coordinated approach to address priority areas of children's environmental health, including early recognition of new and emerging threats to children's health such as exposure to environmental toxicants from improper electronic waste management practices;[9,10] low-dose exposures in early life that increase risks of chronic NCDs in today's children and tomorrow's adults; exposure to environmental pharmaceutical pollutants; exposures to children working in and exposed to extractive industries; exposure to emerging chemicals; and the effects of global environmental change. This work is premised on the concept that effective intervention can be achieved before full achievement of so-called sufficient evidence of causality. Additionally, network activities will include action on the traditional environmental threats to children including indoor air pollution; water and

sanitation; ionising radiation; environmental chemicals and metals; pesticides; occupational exposures and child labour; and injuries, disasters, and conflict.

The overall objective of the collaborative network is to reduce morbidity and mortality of children through the identification and control of environmental risks. The network plans to achieve this objective through activities intended to set a research agenda aimed at building evidence and research capacities in children's environmental health; collaborative research coordination and analysis of evidence; education, communication, and awareness raising in children's environmental health; and development of interventions aimed at capacity building, reducing exposure, and preventing or decreasing the burden of disease for children. Two specific initiatives are the WHO e-waste project that was officially launched in September, 2013, and the 4th International Conference on Children's Environmental Health, which is being planned for 2015.

For more on **WHO e-waste declaration** see https://www.qcmri.uq.edu.au/chep/e-waste-network/e-waste-declaration.aspx]

The aim of this Comment is to alert the global health community to the activities of the network and to invite participation from interested groups. At present, the network is being coordinated by the National Institute of Environmental Health Sciences, an institute of the US National Institutes of Health.

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