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Early-life prevention of non-communicable diseases

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Non-communicable diseases (NCDs) are major causes of death worldwide and underlie almost two-thirds of all global deaths.¹ Although all countries face epidemics of these diseases, low-income and middle-income countries, and the poorest and most vulnerable populations within them, are affected the most. There is a global imperative to create and implement effective prevention strategies, because the future costs of diagnosis and treatment are likely to be unaffordable.

At the UN High-Level Meeting on the Prevention and Control of Non-Communicable Diseases, held in New York, USA, in September, 2011, the so-called four by four strategy for NCD prevention was proposed. Prevention efforts for the priority NCDs discussed at the meeting (diabetes, cardiovascular disease, cancer, and chronic obstructive pulmonary disease) focus on four, mainly adult, risk factors: poor diet, physical inactivity, tobacco use, and alcohol consumption. Although paragraphs 26 and 28 of the UN Political Declaration refer to the roles of prenatal nutrition, maternal diseases, and household air pollution on NCD risk in later life, these paragraphs only partially describe the full scope of the problem and opportunities for intervention. As scientific knowledge emerges on the role of both nutritional factors and exposures to environmental chemicals in the developmental origins of health and disease, evidence suggests that much more attention is needed on early-life interventions, optimisation of nutrition, and reduction of toxic exposures to curtail the increasing prevalence of NCDs.

The present state of the science on the developmental origins of health and disease and NCDs was discussed at the Prenatal Programming and Toxicity III conference, Environmental Stressors in the Developmental Origins of Disease: Evidence and Mechanisms, held in Paris, France in May, 2012, and at a symposium just before the

conference.² Studies in human beings have shown that nutritional deprivation and maternal metabolic status (eg, diabetes) in early intrauterine life increase the risk of metabolic disorders and cardiovascular disease in adulthood.^{3,4} These effects occur not only in settings of extreme deprivation, but also throughout the normal range of population weights at birth and in early childhood.³ Investigators have also reported associations between in-utero exposures and childhood diseases, including type 2 diabetes.⁵ In-utero and early-life exposures to environmental toxicants, ranging from heavy metals to endocrine-disrupting chemicals, affect adult metabolism, immune system function, neurodevelopment, and reproductive function.²

Although causal relations have not yet been established, the new science of epigenetics offers insight into mechanisms of early life predisposition to adult disease risk. During development, epigenetic marks, such as DNA methylation, histone modifications, and noncoding RNA expression, undergo substantial changes. These changes affect genes that are essential for both early life development and later life physiological functions. Epigenetic modifications are stable during cell division and can be transmitted transgenerationally.⁶ An increasing amount of evidence suggests that developmental exposure to nutritional imbalance or environmental contaminants—including metals, pesticides, persistent organic pollutants, and chemicals in drinking water, such as triethyltin, chloroform, and trihalomethanes—can affect epigenetic changes, thus suggesting a mechanism for their effects on adult health.^{7,8} Similarly, prenatal exposure to air pollutants has been associated with epigenetic changes and subsequent effects on children's respiratory health.⁹

Knowledge that in-utero and early childhood experiences affect the risk of NCD development provides an opportunity to target interventions at the time when they have the greatest effect. Because these exposures are not controlled directly by the individual, especially when the exposures might have occurred to the individual's parents or grandparents, early-life interventions can reduce the perception of blame that the individual's own lifestyle has caused his or her disease. This notion has policy implications, because the prevailing viewpoint often assumes that NCDs are mainly a matter of individual responsibility, thus obviating societal and governmental responsibility. Substantial reductions of NCD risks could be achieved through the use of existing maternal-child health platforms to educate mothers about both nutritional and environmental exposures and to integrate the health promotion and disease prevention agendas within social and economic development efforts. For example, the Millennium Development Goals (MDGs) address not only maternal and child health problems, but also poverty and malnutrition, sex inequality, and lack of education, all of which are notable drivers of social disadvantage in low-income and middle-income countries and are underlying causes of NCDs.^{10,11} Poverty alleviation, sustainable food production, and reductions in exposures to toxic chemicals are all key themes emerging from the Rio+20 UN Conference on Sustainable Development¹² held in Rio de Janeiro, Brazil, in June, 2012, and the development of Sustainable Development Goals (SDGs) and appropriate environmental, nutritional, and health indicators provides another opportunity to incorporate NCD prevention into broader, multisector programmes. The integration of NCD prevention with the attainment of the MDGs and SDGs could leverage major worldwide investments in health and development.

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