Back to the Future: Addressing the Early Life Determinants of Non-Communicable Diseases


Non-communicable diseases (NCDs) are the leading causes of death worldwide. Almost two-thirds of all global deaths are due to NCDs.¹ Although all countries are facing epidemics of NCDs, low- and middle-income countries (LMICs), and the poorest and most vulnerable populations within them, are the hardest hit. There is a global imperative to devise and implement effective prevention strategies, as the future costs of diagnosis and treatment are likely to be unaffordable.

The United Nations (UN) High-level Meeting on the Prevention and Control of Non-communicable Diseases, held in New York City in September 2011, has proposed the “four by four” strategy for NCD prevention. Prevention efforts for the priority NCDs addressed at the Meeting (Diabetes, Cardiovascular Disease, Cancer, and COPD) are focused on four predominantly adult risk factors: poor diet, physical inactivity, tobacco use, and alcohol consumption. Although Paragraphs 26 and 28 of the UN Political Declaration reference the roles of prenatal nutrition, maternal diseases, and household air pollution on NCD risk in later life, these paragraphs are only partial descriptions of the full scope of the problem and the opportunities for intervention. Emerging science on the role of both nutritional factors and exposures to environmental chemicals on the Developmental Origins of Health and Disease (DOHaD) suggests that far more attention is needed on early life interventions, optimizing nutrition, and reducing toxic exposures in order to stem the advancing prevalence of NCDs.

The current state of the science surrounding DOHaD and NCDs was discussed at the PPTOX III conference on Environmental Stressors in the Developmental Origins of Disease: Evidence and Mechanisms, held in Paris, France in May, 2012, and at a special symposium held just prior to the conference.²

Human studies have clearly shown that nutritional deprivation and maternal metabolic status (e.g. diabetes) in early intra-uterine life increases the risk of metabolic disorders and cardiovascular disease during adulthood.³⁴ These effects are seen not just in a setting of extreme deprivation, but throughout the range of “normal” population weights at birth and in early childhood.³ In recent years, associations between in utero exposures and childhood diseases, including Type II diabetes, also have been demonstrated.⁵ Environmental exposures in utero and early life, ranging from heavy metals to endocrine disrupting chemicals, have also been shown in a limited number of human and greater
number of animal studies to affect adult metabolism, immune system function, and reproductive function.2

Although causal relationships have yet to be established, the new science of epigenetics is providing insight into common mechanisms of early life predisposition to adult disease risk. During development, epigenetic marks such as DNA methylation, histone modifications, and non-coding RNA expression undergo dramatic alterations. These alterations target genes that are critical, not only for early life development, but also for later life physiological functions. Epigenetic modifications are stable through cell division can be transmitted transgenerationally.6 There is a growing body of evidence indicating 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 influence epigenetic changes, thus suggesting a mechanism for their effects on adult health.7,8 Advancing the science of DOHaD one step further, prenatal exposures to air pollutants have been linked with epigenetic changes and to subsequent effects on children’s respiratory health.9

Understanding that the risk of developing NCDs is affected by in utero and early childhood experiences affords an opportunity to effectively target interventions at the time when they will have the greatest impact. Because these exposures are not directly controlled by the individual, especially those that may have occurred to the individual’s parents or grandparents, early life interventions can reduce the perception of ‘blame’ that the individual’s own lifestyle caused his or her disease. This in turn has policy implications as the prevailing viewpoint often assumes that NCDs are largely a matter of individual responsibility, thus obviating societal and governmental responsibility. Substantial reductions of NCD risks could result from taking advantage of existing maternal–child health platforms to educate mothers on both nutritional and environmental exposures and integrate the health promotion and disease prevention agendas within social and economic development efforts. The Millennium Development Goals (MDGs), for example, address not only maternal and child health problems but also poverty and malnutrition, gender inequality, and lack of education, all of which are significant drivers of social disadvantage in LMICs and 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 Development, and the development of Sustainable Development Goals (SDGs) and appropriate environmental, nutritional and health indicators affords another opportunity to incorporate NCD prevention into broader, multi-sector programs. Tying NCD prevention to the attainment of the MDGs and SDGs could leverage major worldwide investments in health and development.

References:
2 Barouki R, Gluckman PD, Grandjean P, Hanson M, Heindel JJ. Developmental origins of non-communicable diseases: implications for research and public health. *Environ Health* 2012; 11:

Conflicts of Interest
None declared

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