Introduction

Mortality due to various kinds of non-communicable diseases (NCDs) has become an increasing focus of attention in recent years.\(^1\) With rapidly increasing globalization, lifestyles in low- and middle-income countries increasingly include high-fat diets and inadequate physical exercises are resulting in an increased worldwide burden of NCDs.\(^2\) A study by the International Diabetes Federation (IDF) showed that about 382 million people had diabetes in 2013, and this will rise to 592 million by 2035. The number of people with type 2 diabetes is increasing in every country, and 80% of people with diabetes live in low- and middle-income countries. The burden of NCDs and the prevalence of related risk factors such as overweight and diabetes have also increased in China over the past decades. In 2005, NCDs accounted for an estimated 80% of deaths and 70% of disability-adjusted life-years lost in China.\(^4\) In 2010, the leading causes of death among Chinese women were cardiovascular and cerebrovascular diseases and cancer, which together accounted for nearly 70% of all deaths.\(^5\) The incidence of hypertension increased from 5.1% to 33.5% between 1958 and 2010, while the prevalence of diabetes increased by 9.7% and the estimated prevalence of pre-diabetes was 15.5%, accounting for 92.4 million adults with diabetes and 148.2 million adults with prediabetes.\(^6,7\) The prevalence and burden of NCDs in China is creating a significant economic and social load on society, so epidemiological, demographic and socio-economic transitions has been made to prevent and control NCDs in China.

Theory of developmental origins of health and disease and NCDs

In the 1980s, retrospective cohort studies implemented by David Barker and colleagues indicated that
the incidence of certain adult diseases such as cardiovascular disease (CVD) and type 2 diabetes may be linked to the intrauterine environment. Based on Barker hypothesis, the theory of “Developmental Origins of Health and Disease (DOHaD)” and its conceptual framework has been established: stating that an offspring with susceptibility to chronic diseases in later life may be programmed in early life development. Initial studies on the DOHaD focused on the impact of maternal under-nutrition, and demonstrated that low birth weight was associated with an increased risk of obesity, CVD and metabolic disorders in later life. Small at birth and thin at two years of age and gaining weight rapidly thereafter is a pattern of growth during childhood associated with insulin resistance and coronary problems in later life. Children born from preeclamptic pregnancies were more prone to hypertension, insulin resistance and diabetes mellitus, neurological problems, stroke, and mental disorders throughout life. Recent evidence on the effect of over-nutrition showed that fetal exposure to a diabetic environment in utero is associated with an increased incidence of hypertension and glucose and lipid metabolism disorders in adulthood. A case–control study by identification and prevention of dietary- and lifestyle-induced health effects in children and infants (IDEFICS) showed that parental body mass index (BMI) and gestational weight gain were the key risk factors of childhood obesity at the age of nine years. Moreover, maternal obesity (BMI > 30 kg/m²) and high-fat diets were associated with an increased risk of premature death and adiposity in an adult offspring by a record linkage cohort analysis. Recently, it has become widely recognized that NCDs are the long-term outcome of physiological adaptations to the environment, and the complicated process referred to as “programming”. “Programming” refers to the process where by a stimulus at a critical window of development has long-term effects. Epigenetics is the study of chemical changes in DNA and histones that affects how genes are expressed without alterations of DNA sequences. More evidence from epidemiological work based on large cohort studies and animal models that explored epigenetic mechanisms confirmed that malnutrition or over-nutrition during the fetal period alters the epigenetic expression status of metabolic genes in the fetus and that this altered expression can persist and have a profound impact on the development of NCDs in adulthood. Epigenetic mechanisms have also determined the transgenerational disease transmission.

Pregnancy as a stress test for future metabolic syndrome

A number of studies have reported women with gestational diabetes mellitus (GDM) have a substantially increased risk for type 2 diabetes in later life. A study conducted showed that women with GDM have a sevenfold increased risk of developing type 2 diabetes in their lifetime. Approximately half of these women develop diabetes in the 1st 5–10 years after the index pregnancy. The ADA in 2015 recommended follow up and screening for women with GDM to detect persistent diabetes at 6–12 weeks postpartum, and women with a history of GDM should have lifelong screening for the development of diabetes or pre-diabetes at least every three years. Women with a history of GDM found to have pre-diabetes should receive lifestyle interventions. In addition, coexisting obesity and progressive weight gain are additive factors for progression to type 2 diabetes. A study by O’Sullivan showed that the incidence of diabetes in participants who had previous transient gestational glucose intolerance was significantly higher for overweight subjects (46.7%) than for those of normal weight (25.6%). He stated that among persons at high risk, like GDM, excess weight also predicted the severity of the subsequent diabetic condition. What is more, women treated for GDM had lower rates of hypertensive disorders of pregnancy, cesarean deliveries, and less weight gain.

Some epidemiologists have commented that preeclampsia is not only a pregnancy disease but also a risk factor for developing diseases later in life. Women who have had preeclampsia seem to be at higher risk of premature death, mortality from ischemic heart disease, and CVDs including ischemic heart disease and hypertension. A case control study showed that patients with hypertensive pregnancies showed an abnormal activation of the endothelium which persists after pregnancy, which may represent an explanation of the increased risk of CVD later in life. Pregnant women with adverse perinatal complications can also risk NCDs in later life.

Early intervention to NCDs

Over the past decade, China has strengthened its primary healthcare system and increased investment in public health interventions. Although China has made good progress in developing and implementing these strategies and policies for NCDs prevention and control, many challenges still remain. Such as, there is insufficient public funding for NCDs care and
management, and NCDs patients are economically burdened due to limited benefit packages covering NCDs treatment offered by health insurance schemes. A life-course approach to reduction of risk of NCDs suggests that early-life interventions may be more effective and improve functional capacity. The major force, which is responsible for the emergence of NCDs, is the rapid increase in high-risk lifestyle behavior like tobacco use, the harmful use of alcohol, physical inactivity, and an unhealthy diet, which are causes of overweight and obesity, raised blood pressure, raised blood glucose and dyslipidemia. Early life development (1000 days, including gestation, and after two years of age) is not only the critical time for development of tissues, organs, and the nervous system, but also increasingly recognized as period of peak susceptibility to nutritional insults. It is pivotal to teach the importance of healthy behavior before and during pregnancy, in early infancy and in childhood, for future well-being. Nutritional guidelines in pregnancy, based on evidence to promote healthy fetal development, should be well established and widely disseminated. Research on the association between early nutrition and adult outcomes showed that a focus on improvements in nutrition in pregnancy and linear growth in infancy and childhood, which are optimal periods of peak susceptibility to nutritional insults, could share the achievements of research in our country at the congress of DOHaD. Making knowledge available to develop an understanding of the DOHaD presumption that controlling weight and obesity during pre-pregnancy and pregnancy, reducing the incidence of GDM, preeclampsia, and even more effective for reducing the mortality and morbidity of the fetus.

So it is highlighted that intervention in early life may be able to alter trajectories of later disease risk more cost-efficiently than traditional lifestyle modifications or treatment in later life.

Conclusions

The global burden of NCDs is rising dramatically worldwide and is causing a double poor-health burden in China. Early life influences play an important role in the prevalence of NCDs because maternal lifestyle and nutritional conditions will affect the risk of metabolic disorders in the next generation. Although there is an absence of high-quality cohort data for the long-term outcome of maternal and infant intervention to prevent related NCDs, the current recommendations based on the theory of DOHaD presume that controlling weight and obesity during pre-pregnancy and pregnancy, reducing the incidence of GDM, preeclampsia in pregnancy, promotion of a well-balanced dietary pattern, and promotion of health literacy in the public would bring potential benefits to reduce the potential risk of NCDs. The DOHaD Society had established affiliated societies in China in 2009, and Chinese doctors could share the achievements of research in our country at the congress of DOHaD. Making knowledge available to develop an understanding of the DOHaD offers the potential to encourage informed diet and lifestyle choices supporting reduction of NCDs risk in current and future generations.

References

1. World Health Organization. Global Status Report on Non-communicable Diseases 2010. Geneva: WHO; 2011.

2. Jones AC, Geneau R. Assessing research activity on priority interventions for non-communicable disease prevention in low- and
middle-income countries: a bibliometric analysis. *Glob Health Action*. 2012;5:1–13.

3. Vellakkal S, Millett C, Basu S, et al. Are estimates of socioeconomic inequalities in chronic disease artefactually narrowed by self-reported measures of prevalence in low-income and middle-income countries? Findings from the WHO-SAGE survey. *J Epidemiol Community Health*. 2015;69:218–225.

4. Wang L, Kong L, Wu F, Bai Y, Burton R. Preventing chronic diseases in China. *Lancet*. 2005;366:1821–1824.

5. Yu W. National Disease Surveillance Points System—Death Cause Surveillance 2010. Beijing: Military Medical Science Press; 2012.

6. Li YC, Wang LM, Jiang Y, Li XY, Zhang M, Hu N. Prevalence of hypertension among Chinese adults in 2010. *Chin J Prev Med*. 2012;46:409–413.

7. Yang W, Lu J, Weng J, et al. Prevalence of diabetes among men and women in China. *N Engl J Med*. 2010;362:1090–1101.

8. Barker DJ, Osmond C. Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. *Lancet*. 1986;1:1077–1081.

9. Barker DJ, Osmond C, Golding J, Kuh D, Wadsworth ME. Growth in utero, blood pressure in childhood and adult life, and mortality from cardiovascular disease. *BMJ*. 1989;298:564–567.

10. Fleming TP, Velazquez MA, Eckert JJ. Embryos, DOHaD and David Barker. *J Dev Orig Health Dis*. 2015;20:1–7.

11. Hanson M. The birth and future health of DOHaD. *J Dev Orig Health Dis*. 2015;1:1–4.

12. Barker DJ, Osmond C, Forsén TJ, Kajantie E, Eriksson JG. Trajectories of growth among children who have coronary events as adults. *N Engl J Med*. 2005;353:1802–1809.

13. Intapad S, Alexander BT. Pregnancy complications and later development of hypertension. *Curr Cardiovasc Risk Rep*. 2013;7:183–189.

14. Yan J, Li X, Su R, Zhang K, Yang H. Long-term effects of maternal diabetes on blood pressure and renal function in rat male offspring. *PLoS One*. 2014;9:e88269.

15. Su RN, Zhang K, Yang HX. Long-term effects of mild intrauterine hyperglycemia on glucose and lipid metabolism in intergenerational rat offspring. *Chin J Perinatal Med*. 2015;18:455–461.

16. Bammann K, Pepjes J, De Henauw S, et al. Early life course risk factors for childhood obesity: the IDEFICS case—control study. *PLoS One*. 2014;9:e86914.

17. Reynolds RM, Allan KM, Raja EA, et al. Maternal obesity during pregnancy and premature mortality from cardiovascular event in adult offspring: follow-up of 1 323 275 person years. *BMJ*. 2013;347:f4539.

18. McMillen IC, Robinson JS. Developmental origins of the metabolic syndrome: prediction, plasticity, and programming. *Physiol Rev*. 2005;85:571–633.

19. Yan I, Yang H. Gestational diabetes mellitus, programing and epigenetics. *J Matern Fetal Neonatal Med*. 2014;27:1266–1269.

20. Bellamy L, Casas JP, Hingorani AD, Williams D. Type 2 diabetes mellitus after gestational diabetes: a systematic review and meta-analysis. *Lancet*. 2009;373:1773–1779.

21. American Diabetes Association. Standards of medical care in diabetes-2015 abridged for primary care providers. *Clin Diabetes*. 2015;33:97–111.

22. O’Sullivan JB. Body weight and subsequent diabetes mellitus. *JAMA*. 1982;248:949–952.

23. Lazzarin N, Desideri G, Ferri C, et al. Hypertension in pregnancy and endothelial activation: an emerging risk factor for cardiovascular disease. *Pregnancy Hypertens*. 2012;2:393–397.

24. Bhutta ZA. Early nutrition and adult outcomes: pieces of the puzzle. *Lancet*. 2013;382:486–487.

25. Yang HX, Zhang MH, Su SP, et al. *Diabetes Beyond Pregnancy and Practical Manual*. People’s Medical Publishing House; 2012.

26. Marques RdeFdaSV, Taddei JAdeAC, Konstantyner T, et al. Anthropometric indices and exclusive breastfeeding in the first six months of life: a comparison with reference standards NCHS, 1977 and WHO, 2006. *Int Breastfeed J*. 2015;10:20.

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