Published online 2014 May 25.

Editorial

The Early Origins of Adult-Onset Diseases and the Role of the Pediatricians in Primary Prevention of Chronic Diseases

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Received: December 21, 2013; Accepted: May 18, 2014

Keywords:Cost of Illness; Chronic Disease; Prevention

Noncommunicable diseases, which are the leading cause of the burden of disease and mortality worldwide, originate in early living. Epigenetic elements are proposed as the possible etiologic element of the primary-life scheduling of adult-onset diseases (1). Based on the developmental origins of health and disease (DOHaD) theory, intrauterine or suitability following birth to the surroundings gives rise to the morphologic, physiologic, or metabolic alterations that affect the health afterward.

The DOHaD hypothesis proposes a correlation between low weight at birth, a complex clinical indicator of unsuitable intrauterine growth, and risk of chronic disorders such as hypertension, obesity, diabetes, coronary heart diseases, etc. in adult life. Nonetheless, developmental scheduling also presents a new method to avoid cardiovascular and related diseases during ostensible "Rescheduling": performance of suitable elements or prevention of harmful factors around the birth or genetic patterns improved adverse growth outline of these investigations, suggests that similar to what has been already recognized in scheduling, numerous and completely various rescheduling interventions all have analogous protective influences (2). The "Economical Phenotype Theory" expresses that once the fetal surroundings is poor in a necessary element or contains a detrimental element, the fetus suits by favoring development of vital organs; e.g. the brain at the cost of other organs, which have "keep" ability and are considered less significant for continued existence under these undesirable environmental circumstances. The suitability may give rise to the structural, physiological, and metabolic alterations that affect health afterward throughout the life. This event is recognized as the "Developmental Flexibility" or "Scheduling", because the

genetic schedule suits to the obtainable environmental circumstances leading to the diverse phenotypes (3).

Moreover, it should be considered that lifestyle elements as well as the major risk factors of noncommunicable diseases are established from the childhood. Pediatric investigations on elements, which may affect the risk of adult-onset diseases, have examined several biomarkers. The tracking of risk factors from the childhood through adulthood is well documented. Vascular endothelial malfunction commences in the primary life and is an essential pathological condition in the primary development of atherosclerosis. Likewise, early-life risk factors, particularly the childhood obesity, are significantly associated with the endothelial malfunction and the expansion of the atherosclerosis and subsequent diseases.

There are considerable evidences showing that the risk factors in the childhood are associated with the advanced or in-between vascular diseases in the adulthood, e.g. diabetes, hypertension, and increased carotid intima-media thickness. Moreover, accumulation of risk factors happens in the childhood and poses an even bigger risk than the total of the single risk factor to an adult health (4).

Investigations have lately suggested that epigenetic alterations in the metabolic pathways may not be confined to the prenatal malnutrition. Stunting refers to poor growth in height owing to malnutrition and infection, with height z-marks of more than two standard deviations under normal height curve for the respective age. Mounting evidences suggest that being overweight in the first two years of life and later stunting in children predisposes to a worse metabolic outcome in adulthood. These associations between the primary stunting and subsequent disease are important, presuming that stunting influences around 25% of children in the underdeveloped regions while the obesity epidemic arrives in developing regions worldwide.

Implication for health policy/practice/research/medical education:

All investigations have underscored the importance of developing adult-onset diseases from early life. This justifies the crucial role of pediatricians in primary prevention of chronic diseases of adulthood.

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With regard to this elevated outbreak of stunting, any association between stunting and the risk of subsequent metabolic diseases would be of considerable significance.

The distinct initiation of prompt weight gain is called "adiposity rebound" and is defined as the time when the physiologic elements of body mass index converses and commences to boost with age. Possible explanations are genetic characteristics or, otherwise, epigenetic alterations endowed with an augmented propensity for the prompt overweightness (5). All these evidences underscore the importance of adult-onset diseases from early life. This justifies the crucial role of pediatricians in primordial and primary prevention of chronic diseases of adulthood.

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