Healthy Ageing and Longevity

Volume 9

Series Editor

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Rapidly changing demographics worldwide towards increased proportion of the elderly in the population and increased life-expectancy have brought the issues, such as "why we grow old", "how we grow old", "how long can we live", "how to maintain health", "how to prevent and treat diseases in old age", "what are the future perspectives for healthy ageing and longevity" and so on, in the centre stage of scientific, social, political, and economic arena. Although the descriptive aspects of ageing are now well established at the level of species, populations, individuals, and within an individual at the tissue, cell and molecular levels, the implications of such detailed understanding with respect to the aim of achieving healthy ageing and longevity are ever-changing and challenging issues. This continuing success of gerontology, and especially of biogerontology, is attracting the attention of both the well established academicians and the younger generation of students and researchers in biology, medicine, bioinformatics, bioeconomy, sports science, and nutritional sciences, along with sociologists, psychologists, politicians, public health experts, and health-care industry including cosmeceutical-, food-, and lifestyle-industry. Books in this series will cover the topics related to the issues of healthy ageing and longevity. This series will provide not only the exhaustive reviews of the established body of knowledge, but also will give a critical evaluation of the ongoing research and development with respect to theoretical and evidence-based practical and ethical aspects of interventions towards maintaining, recovering and enhancing health and longevity.

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Early Life Origins of Ageing and Longevity

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Preface

Traditionally, gerontological research is focused on later stages of life cycle. Accumulating evidence, however, indicates that the rate of aging-associated functional decline (senescence) as well as the risk for chronic pathological conditions can originate early in life. It is already well established in numerous studies that all living things are highly plastic during their early development. Through particular developmental stages ("critical windows"), the organism is extremely sensitive to various environmental cues with the outcomes impacting subsequent stages of life cycle and later-life health status. Such kind of developmental plasticity has adaptive value because it attempts to match an individual's responses to particular environmental conditions that are likely to prevail in subsequent life; inadequate prediction, however, leads to an increase of the risk for chronic disease in later life. These processes are commonly referred to as "developmental programming." The mechanistic basis for the link between early and late stages of the life cycle is not entirely clear, but modulation of epigenetic regulation of gene expression likely plays a critical role in linking the adverse environmental conditions early in life to the risk of various aging-related pathological conditions in adulthood. Currently, these mechanisms are the subject of active debate and investigation.

Initially, these ideas were articulated by David Barker in the "fetal origins of adult disease" hypothesis on the basis of his early observation that small size at birth is linked to an elevated risk of hypertension, abnormalities in lipid metabolism, insulin resistance, and cardiovascular disease in adulthood. Subsequently, Barker's hypothesis has been confirmed by numerous observations and is currently accepted by most of the scientific community. Over the last few decades, it has evolved into the Developmental Origins of Adult Health and Disease (DOHaD) hypothesis that postulates that adverse environmental exposures during critical *in-utero* and early postnatal stages of development may permanently change physiological responses and cause functional impairments and disorders in adult life. Among the determining factors affecting these processes, developmental malnutrition seems to be the most important, although other factors such as stress or hypoxia may also be crucially involved. Collectively, these factors may to a great

extent determine not only the risk for subsequent adult-onset disorders but also the aging rate *per se* and, ultimately, longevity.

The possibility of developmental origins of aging-associated processes draws attention to factors contributing to developmental programming phenomenon that largely affect modern human societies. Among them, there are factors related to present-day lifestyle trends, such as Westernized dietary habits, low physical activity, high level of social stress, drug and substance abuse, and exposure to xenobiotics. All these factors may affect pregnant and lactating women, and they were shown to be able to adversely modulate epigenetic and other pathways that contribute to developmental programming of aging-related pathological conditions. It can be certainly assumed that these factors will substantially influence future trends in incidence of aging-associated disorders and in life expectancy. Therefore, further translational research is required to improve the understanding of early etiological mechanisms of age-related pathology. One of the most promising outcomes of such translational studies is development of epigenetic approaches aimed to predict which early life exposures would put exposed subjects at risk for a particular disease and which individuals will be more susceptible to develop particular pathological conditions later in life.

The elaboration of interventional strategies targeted at restoration of developmentally disrupted epigenetic patterns is increasingly considered as a promising treatment option to alleviate adverse effects of early life malprogramming and to reduce the risk of adverse age-related conditions in later life. Indeed, since developmentally induced epigenetic disturbances are potentially reversible, they can likely be corrected by specific epigenome-targeted pharmacological and/or nutritional interventions. Further development of such approaches will, of course, require the use of highly sensitive methods to detect potentially disadvantageous epigenetic alterations long before the clinical diagnosis of disease. After appropriate clinical trials, the implementation of such treatment strategies in clinical practice may hold great promise in preventing and treating a wide variety of chronic diseases and human healthspan extension.

This book brings together current research evidence and knowledge on the early life origin of aging and longevity in chapters written by leading experts in this area from around the world. It can likely be a relevant and useful resource not only for professional scientists and clinicians but also for scientifically interested amateurs wishing to know more about the current research in this rapidly evolving field.

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Contents

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