УДК 61:615.1(062)(476-25)
ББК 52я73
А 43
ISBN 978-985-21-1008-2

Senanayake V., Borham A. K

PATHOPHYSIOLOGY AND MECHANISMS INVOLVED IN AGING

Tutor PhD, Associate Professor Zhadan S.A.
Department of Pathological Physiology
Belarusian State Medical University, Minsk

Aging has been the talk of centuries of people, scientist, philosophers. But what aging is yet to be understood as it is a vast topic of interest. Dating back up until now scientists are at a path to find a cure for aging. Aging is an irreversible pathological process that cause functional degradation of cells and tissues leading to problems in metabolic processes of the body. These changes lead to a risk of chronic diseases of the body in time. There are different markers of aging discovered by science over the years.

The main tasks and goals of this review is to discusses in detail the different molecular mechanisms which could cause and exacerbate ageing. For instance, Genome instability is considered one of the hallmarks of aging, it is caused by accumulation of damaged genetic material inside cells that would cause disturbances in the normal homeostasis of the cell various endogenous and exogenous agents cause DNA damage. Telomere Attrition is caused due to the shortening of telomeres by telomerase inactivation, its discovered that with each subsequent cell division a part of telomeres are lost and most mammalian somatic cells do not express telomerase. Epigenetic mechanisms are now discovered to be key affecters to the alterations of genome structure and function that affect aging there are three methods by which this occurs they are DNA methylation, histone modifications, and noncoding RNA species. Proteostasis or protein hemostasis is the regulation, stabilization and preservation of the functions of proteomes in the organism, the breakdown of protein homeostasis is linked to the onset of aging and the majority of aging-related illnesses. Mitochondrial dysfunction is heavily correlated with the ageing process, increasing age in mammals correlates with accumulation of somatic mitochondrial DNA mutations and deterioration in respiratory chain function subsequently accumulation of mitochondrial DNA mutations and increased reactive oxygen species (ROS) production causes oxidative damage to cellular macromolecules. Cellular senescence is the irreversible cessation of cell proliferation due to an exogenous or endogenous stimuli, the principal factors which cause senescence are the senescence-related secreted phenotype (SASP) and DNA damage. The gradual decline in functioning of a stem cell that generally accompanies aging may result from many of the aforementioned mechanisms of aging, alone or in combination, metabolic research has also shown that, as stem cells age they experience important changes in the balance between glycolysis, oxidative phosphorylation, and response to oxidative stress. Several metabolic interventions can increase longevity, including global caloric restriction (CR), the selective limitation of specific nutrients like methionine, physical exercise, and the administration of agents such as ‘‘CR mimetics’’.

Studying and further researching the molecular pathways involved in this age-dependent deterioration of mechanism will be critical for developing new therapies for diseases of aging that target the specific causes of age-related functional decline.