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Aging: Facts and theories, L. Robert, T. Fulop, editors (Karger, Basel, Switzerland) 2014. 216 pages. Price: USD 231.00 / CHF 196.00 / EUR 163.00 ISBN 978-3-318-02652-8

Due to demographic transition and its impact on economics and quality of life, ageing has assumed center stage for research in the scientific community. In the last five decades there has been an extensive conceptualization and experimentation to unravel the mysteries of ageing. This book is a part of an ongoing series, "Interdisciplinary Topics in Gerontology" and an attempt to critically appraise various mechanisms proposed as cause of ageing in light of scientific evidence and reasoning. The book has 12 chapters dealing with

ageing process, authored by different experts in the subject. Each chapter dwells upon theoretical aspects in the particular field and experimental or observational facts supporting or refuting the theories or concepts.

The book sojourns through oxidative stress and mitochondrial theory of ageing, commitment of cells to senescence, evolutionary theories of ageing, control of cell replication during ageing, cell senescence, ageing of cell communication, immunological theories of ageing, ageing as alteration, and longevity and its regulation. The chapter on the theories of ageing underlines the limitations of the popular Hayflick limit theory. It draws attention to the commitment theory of cellular ageing to fill in those lacunae in the experimental system due to which we are unable to explain the variability of different somatic cells (with division potential) with life span variability. Also, the limitation of extrapolation from experimental results could be countered.

Evolutionary theory of ageing puts forth that ageing is a result of an accumulation of mutations: a negative effect of certain alleles in old age, originally selected for a positive effect at a younger age or a trade-off between reproduction and maintenance. Evolutionary theories explain some but not all aspects of ageing.

The chapter on control of cell replication during ageing underlines the fallacious lead taken by researchers resulting in contradictions and paradoxes which need to be resolved to re-establish logic and understanding of cell replication control during ageing and its implications. Telomere shortening has shown variable results in different cell lines. The relationship between cell senescence and cancer has also not been justified. Experimental evidences for different theories of control of cell ageing, senescent phenotype, relation of senescence with cancer, etc. have been dealt with. Ageing of cell membrane and its biochemical consequences; the relationship between membrane hypothesis of ageing and genetic changes have also been explained along with anti-ageing experiments with centrophenoxine and growth hormone.

The molecular basis of ageing mitochondrial dysfunction and role of oxidative stress have been well covered. A new perspective of reactive oxygen species (ROS) as signaling molecules in ageing has been mentioned in the chapter, "Oxidative stress, Mitochondrial Dysfunction and the Mitochondrial Theory of Ageing". The concept put forward by the authors is that ROS may not be detrimental forces in

ageing but simply the body's way of coping with ageing related cellular damage. However, a lot of work is still being done on ROS mediated cell damage in ageing. Antioxidants have been proven to be protective against such damage in animal models. A more balanced opinion comparing the two contrasting viewpoints could have been given instead of a casual mention of new concepts.

Age dependent modifications in connective tissues in cellular and extracellular compartments are elaborated upon. Changes with ageing in macromolecular components of extracellular matrix, decline in receptor-mediated cell matrix interactions and implications of these changes in cardiovascular diseases and longevity are well covered.

Changes in immunity with ageing predisposes to chronic diseases associated with inflammation affecting longevity. Age associated immune dysfunction partly explains ageing. Immune risk profile (IRP) as a predictor of longevity has been suggested but not substantiated. The relative fitness of the immune system in centenarians points to the role of the immune system in longevity; but there is increase in pro-inflammatory interleukin - 6 (IL-6) in centenarians rebuking this.

The chapter on ageing of the brain and dementias discusses pathogenetic aspects of dementia syndromes. However, too much emphasis has been given on certain pathogenetic mechanisms such as protein misfolding. Oxidative stress, epigenetic mechanisms, age related gene up/ downregulation have not been accorded just place. *Drosophila* has been taken up as a model of human neurodegeneration. Other models and comparative benefits of each could also have been mentioned.

The chapter, "Aging as Alteration" could have been clarified further for the non-specialist reader as certain concepts and sentences are confusing.

Overall, this book is a good attempt to highlight new insights in this explosive area of research. In such an ever changing field, it is difficult to touch upon every aspect but barring a few shortcomings mentioned above, the authors have produced a comprehensive piece of work.

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