



## Editorial

# Are we nearing the extinction of Gerontology?

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If you had to choose how much you wanted to live? What would you choose? 80 yrs, 100 yrs, 120 yrs or more!!!! and on reaching the desired age would we still like to stay at that age or change our minds?? Going back in time around 50,000yrs ago human beings had a very short life span; as humans evolved, their lifestyles, dietary habits, and living conditions also improved which resulted in prolonged survival and longer life spans. Today human beings are living longer and healthier lives than ever before however there are unforeseen consequences to this prolonged survival, that is aging or reaching the geriatric age group. The aging results in more disease and sickness in the geriatric age group from processes like decreased oxygen consumption, negative metabolism, solar radiation exposure and many more which hit the body, bringing it down to a worn downstate, ultimately there is loss of the special senses, bones and muscles degenerate, skin wrinkles, immune system becomes weaker, memory and cognition decline.<sup>1</sup>

If diseases and damage are the highlights of the geriatric years, is aging acceptable? So, then the question arises, Is it good to age? can we slow or prevent aging? If so, to what age should we get old?? Current day medical science is trying is to prolong life and decrease suffering. Getting old means getting more sick and less disease-free. We are currently trying methods to prevent aging by some

ineffective ways like organ transplants, using chemotherapy to eliminate cancers, utilizing vaccines, using antibiotics to prevent diseases. In a jist aging is caused by physical wear and tear making our bodies much more fragile with prolonged aging, until we die as one of our system fails.

Scientists need to work on optimising health span rather than life span. Present day research is being focused on senescent cells and their expiration dates. A senescent cell ages due to chromosomal aging within a cell which is regulated by telomeres. The telomeres shorten as the cell ages ultimately a senescent cell is formed. These cells are harmful for the near by cells and tissues as they release toxins and are linked to diseases like diabetes and renal failure. Scientists have created genetically engineered rats which can remove their senescent cells, such rats have shown to live healthy lifespans and were much more active than normal rats. Target apoptotic therapy is also being tried to remove these senescent cells from rats there by increasing their overall life spans also increased by 30%. Other age reversing studies going on focus on biochemical compound called NAD<sup>+</sup>, which is a coenzyme vital for better functioning of all cells in body, older individuals have half the amount of NAD<sup>+</sup> inside the body as compared to younger individuals. Higher levels of NAD<sup>+</sup> are associated with dramatic improvement in cell function and activity as seen by experiments in rats. Lastly the studies are also aiming on stem cell therapy.<sup>2</sup> Stem cells are key for improvement of health life span. Stem cells decrease in

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aged individuals. Experiments carried out in rats have shown that transplant of stem cells into older rats tends to boost the functioning of the neighbouring tissue cells by improving cellular machinery, biochemical response, cell signalling and cell metabolism, making the rat more active and healthier than before in comparison to their counterparts. All this recent updates suggest that there are multiple pathways to regulate aging and lots has to be understood before actual human trials can begin.

The present-day scientific world boasts its discovery of the fountain of youth that is telomerase which holds the key and secret to stop, modulate, slow or delay aging. Longevity researchers have evolved over time, from many theories of aging, putting forth the one which is most conclusive and universally accepted based on seven hallmarks of aging. The present theory presents three important molecular mechanisms that play an important role in cellular senescence and can hold the key to modulate aging. The First mechanism the “Initiator trigger mechanism” includes two subsets – a. the Genome flux which is based on the functioning of the gatekeeper and caretaker genes inclusive of the putative DNA repair genes and the mismatch, nucleotide excision and repair genes. The cell pathways help prevent the entry of mutated genes within the cell cycle, thereby preventing genomic instability and replication of mutated cells and b. the telomere wear and tear, it basically includes the activity of the telomerase enzyme responsible for modulating the telomere length located at the chromosome ends thereby regulating cell division and cellular senescence.<sup>3</sup> The second molecular mechanism is known as the “Reactionary shield mechanism” which includes three subsets, i.e. a. Malfunctioning nutrient cell signals, b. Mitochondrial breakdown, and c. cellular senescence. This molecular mechanism promotes aging by the Insulin/insulin growth factor 1 (IIS Pathway) through modulating the DAF pathway (Drosophila gene encoding insulin like growth factor 1) which is responsible for controlling antioxidants and heat shock proteins and also regulate the caloric restriction which controls Sirtuins, (FOXO) The Fork head box O pathway, and inhibition of TOR (The mammalian target of rapamycin) pathways, all of them regulate cellular aging and improved mitochondrial functioning.<sup>4</sup> The third molecular mechanism can be dubbed as the culprits of the phenotype changes, and this includes two subsets -a. Stem cell fatigue and b. Fl awed intercellular communication. This mechanism holds the key to stem cell quiescence and long term functionality of stem cells. Identifying these hallmarks of aging will shed light on the intricate matrix of molecular pathways that regulate aging and will pave the

way for future research.<sup>5</sup>

Irrespective of the battles of youth and old the question which shakes the ethos of nature and geriatric medicine of the present day is that, Is it ethical to stop or modulate aging?. Are we ready to accept immortality?? Our present-day world, the society, the family a re set in a biological clock- a natural order that starts with birth to childhood progressing to adolescence and youth, with middle-age years and finally the golden years. The society is programmed to see, hear, live in their relationships. Altering aging means to alter the fabric and ethos of nature, which revolves around the one eternal truth that every organism lives to perish one day. The dilemma and debate regard aging has persisted through history and mythology, and the quest for immortality is nothing new for the present-day world and will continue on the theatrical ground of gerontology, hence the irony of life – If we live longer, Will we decrease our stress? or make our surrounding a better place? Will we improve our social interactions? will we realize the importance of nature around us? Can humanity uplift for the betterment of mankind itself in the prolonged years of its longevity is a matter to ponder!!

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None.

## 2. Conflict of Interest

None.

## References

1. Bass SA. Gerontological theory: the search for the holygrail. *Gerontologist*. 2006;46:139–144.
2. Magni G, Amici A, Emanuelli M, Orsomando G, Raffaelli N, et al. Enzymology of NAD+ homeostasis in man. *Cell Mol Life Sci*. 2004;61:19–34.
3. Hoeijmakers JH. DNA damage, aging, and cancer. *N Engl J Med*. 2009;361:1475–1485.
4. Barzilai N, Huffman DM, Muzumdar RH, Bartke A. The critical role of metabolic pathways in aging. *Diabetes*. 2012;61:1315–1322.
5. Rera M, Bahadorani S, Cho J, Koehler CL, Ulgherait M, et al. Modulation of longevity and tissue homeostasis by the Drosophila PGC-1 homolog. *Cell Metab*. 2011;14:623–634.

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