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Mini Review

Modulation of Nutrient-Responsive Pathways in Lifespan and Health

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ABSTRACT

Studies in bio gerontology are driven by cellular, tiny invertebrate and vertebrate models. Our understanding of the connection between nutrition, nutrient-response signalling pathways, and lifespan control has greatly expanded because to the use of many models, including yeasts, suitable tissue culture cells, Drosophila, the worm Caenorhabditis elegant, and the mouse. In recent years, high-throughput screens, mutagenesis, combinatorial pharmacological therapies, and multi-omics methods have all contributed to the discovery of previously unheard-of understandings of cellular metabolism, differentiation, and ageing. Hence, in order to find potential therapies that might promote healthy ageing and the amelioration of age-related disorders in humans, scientists are working to characterise the intricate architecture and interactions of growth and stress networks. In this concise review, we skim the most recent research on nutrient-response pathways.

Keywords: Gerontological nursing; Health; Nursing labour force

INTRODUCTION

Aging is a multifaceted, complicated process and essentially universal phenomena in life. It is the progressive accumulation of changes over time that raises the likelihood of mortality. Notably, lifespan regulation and the pace of ageing are dependent on both genetic and non-genetic variables as well as environmental influences. The environment has a major impact on lifespan regulation, with nutrition and stress being the main determinants of survival at the cellular, tissue, and organismal levels, according to a considerable body of research. Through nutrient-responsive pathways that are hard-wired to fundamental metabolic functions like gene transcription, protein translation, proteostasis and protein degradation rates, mitochondrial function, such as detoxification and respiration, as well as autophagy, cells perceive nutrients, such as amino acids and sugars.

DISCUSSION

Nutrient-responsive pathways, including the Insulin Growth Factor and the mechanistic Target of Rapamycin that regulate longevity and health span, as well as an update on recent developments in this scientific field. We briefly mention the underlying ideas behind these paths. In conclusion, whereas early in life activation of the associated signalling regulated by IGF and motor supports growth and development, it appears deleterious to longevity and health span. While the basic molecular actors in these pathways are satisfactorily delineated in smaller species like yeast, D. melanogaster, and C. elegant, further research is required to comprehend the functional connections on a genome-wide scale. Moreover, single or combined medication therapies [1].

In order to investigate impacts on longevity and health span, such as the amelioration of pathological conditions that might mimic age-related illnesses or syndromes, nutrient-responsive and other signalling pathways that regulate growth have been used. Nonetheless, research on human genetics, including the genetics of ageing and the impact of nutrition on regulating human longevity, is ongoing. To reduce this gap, the field is employing stem cell technology, patient samples, and organisation. It has reached a point of maturity that allows it to go forward with major investigations and clinical trials using canine breeds that are closely related to humans. Yet, cross-species analyses show that fundamental processes like proteostasis and protein half-life patterns, which can influence ageing processes and differentiation programmes, also occur at varying rates across various species [2].

These studies demonstrate that the exact, quantitative results in model organisms may not be the same as those in the human body or even in cohorts of humans. Therefore, interdisciplinary studies combining genetics, biomarker analyses, diet and drug surveys, and interventions in human populations are now needed within the field of bio gerontology, despite the fact that the contribution of model organisms in bio gerontology studies is prolific in understanding underlying molecular mechanisms [3].

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According to the hyper function theory of ageing, a developmental programme acts early in life to promote growth and fertility, which ensures survival during the reproductive peak, continues during later stages of life when it becomes "hyper-functional," causing cell senescence, the development of geriatric diseases, and ageing. However, its pleiotropic activity later in life seems to be harmful to lifespan and some aspects of health span. Increases lifespan and fitness, hence bolstering the hyper function theory of ageing. Although targeting lowered worm fertility in the early stages of development, its downregulation in adulthood boosted the organism's lifetime. On the basis of this discovery, it has also been demonstrated that adulthood insufficiency has a favourable effect on late-life reproductive potential [4,5].

CONCLUSION

But how late might such an intervention be done before it would no longer have a positive impact on longevity? Surprisingly, auxin-induced deterioration of the organism at higher ages nevertheless resulted in a doubling of its longevity. Provided further mechanistic information by revealing decreased of a number of proteins, one of which being a particular RNA-binding protein. Creation of a mutant version of car-1 that cannot have its lifetime increased, which codes for the protein, is expressed less as a result of knockdown, and this is followed by fewer neuronal cells. In turn, this appears to lessen the build-up of harmful proteins and increase lifetime. The increase of nonsense-mediated mRNA degradation, one prominent trait identified for long-lived.

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CONFLICT OF INTEREST

None.

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