OPEN ACCESS Freely available online

ISSN: 2329-8847
Journal of
Aging Science

Perspective

The Construction and Consequences of the Longevity Network in Aged Persons

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DESCRIPTION

Human ageing is a very heterogeneous phenotype that can be compared to a complex mosaic created by the interaction of several environmental, stochastic, and genetic-epigenetic factors. As a result, each elderly person must be seen as a singleton, making it impossible to define the "ageing phenotype." We cover the centenarian phenotype, which is the best illustration of successful ageing, as well as other models used to research human ageing and longevity, including twins and cohorts of unrelated patients.

High diversity, maternal inheritance, and the lack of recombination are characteristics of Mitochondrial DNA (MtDNA). Research on human populations has uncovered ancestral linked polymorphisms, whose combination creates MtDNA haplogroups, which are now employed to retrace the lineages of human evolution. The MtDNA population pools of a sample of people chosen for successful ageing and longevity (212 subjects older than 100 years and in good clinical condition) and a sample of 275 younger people (median age 38 years), carefully matched as to gender and place of origin, were compared using such inherited MtDNA markers.

The average lifespan of people has significantly increased over the past 200 years, yet there are still significant regional differences. The advancements in nutrition, hygiene, and health care are primarily to blame for this increase in lifespan. However, the average lifespan has not increased at the same rate, and in Europe, men and women respectively spend an average of 20.5% and 25.4% of their lives dealing with disabilities brought on by illness or trauma. Although the majority of prevalent diseases that cause disability have age as their primary risk factor, being older does not always imply a higher level of age-related disability. This is demonstrated by the existence of old people from families with outstanding longevity who can live to old ages without suffering from significant problems. Also, compared to controls of a similar age, their offspring, who are referred to as "decelerated" or "healthy agers," have a decreased prevalence of age-related illnesses such cancer, cardiovascular disease, hypertension, and type 2 diabetes. They exhibit favourable or "youthful" profiles for numerous metabolic and immune-related metrics concurrently.

The overwhelming majority of studies on ageing have concentrated on specific genes or proteins without sufficiently exploring the potential significance of their interconnections. This research is the initial effort to build a "longevity network" by examining human Protein-Protein Interactions (PPIs). They identified the human orthologs of the longevity genes, produced a comprehensive list of established longevity genes from many species, including those that most likely affect human longevity, and tested whether the encoded proteins could be arranged into a network. The longevity gene-encoded proteins and the proteins that interact with them make up a continuous network that satisfies the requirements for a scale-free network with a significant hub contribution to network connectivity.

Most of them have never had annotations related to longevity before. Surprisingly, it was found that nearly every hub of the "longevity network" was associated with at least one Age-Related Disease (ARD), and many were associated with multiple ARDs. This could be one of the ways that proteins with numerous interactions affect how long organisms live. The hubs have the potential to be the main targets of therapies aimed at extending lifespan.

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Received: 02-Jan-2023, Manuscript No. JASC-23-20302; **Editor assigned:** 05-Jan-2023, Pre QC No. JASC-23-20302 (PQ); **Reviewed:** 20-Jan-2023, QC No JASC-23-20302; **Revised:** 26-Jan-2023, Manuscript No. JASC-23-20302 (R); **Published:** 03-Feb-2023, DOI: 10.35248/2329-8847.23.11.304

Citation: Jun M (2023) Effects of Nutrition and Exercise on Ageing People. J Aging Sci. 11:304.