Gil Atzmon

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Biography

Gil Atzmon is Professor of Human Biology at Haifa University in Israel, where he runs the Laboratory of Genetics and Epigenetics of Aging and Longevity, and at Albert Einstein College of Medicine in New York. The foremost focus of Prof Gil Atzmon's entire research career has been the understanding of the association of the whole genome to disease, performance, health and longevity. Since 2001, he has focused on human genome and its impact on aging and longevity.

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Note:

DETECTION OF EPIGENOMIC VARIATION ASSOCIATED WITH LONGEVITY AMONG MULTI ETHNIC CENTENARIAN POPULATION IN ISRAEL

Genetic, epigenetic and environmental factors play a crucial role in determining life span. Epigenetics has emerged as an important factor in the control of gene expression and therefore effect disease risk. Specifically, methylation changes at specific gene regions have been associated with cancer risk and autoimmune disorders. Studies have shown that age related epigenetic changes could serve as a marker for chronological age. We hypothesize that *i) aging is associated with epigenetic changes in humans, and ii) centenarians have distinct pattern of methylation that protects them from age-related diseases, and therefore affects healthy lifespan*. We propose that epigenetic changes are one of the central mechanisms by which aging predisposes to many age-related diseases, and therefore influence disease risk and lifespan. We test this hypothesis using a unique population of individuals with prolonged life span (i.e. centenarian) who represent a very small segment of human population. We systematically assess the contribution of genomic methylation changes in three major sub-groups, of centenarians:

1. **Survivors:** Those who survive after onset of major age associated disease like diabetes, CVD or metabolic syndrome at an age comparable to general population, i.e at the age of 60+/-5 years (thus long life span but short healthy lifespan),
2. **Delayers:** Develop age related diseases mentioned above much later that control population i.e at the age of 80+/-5 years (therefore have a longer healthy life span)
3. **Dodgers:** Fail to develop age-related illnesses naturally at the age of 100+/-5 years. We hypothesize that subjects within the three groups will exhibit differential methylation at sites distinct from each other as well as appropriate age matched controls (healthy cohort subjects available for 60+/-5 and 80+/-5 yr old). Furthermore, we found that the survivors and delayers exhibit different gene expression pattern as they approach their chronic life time condition. We test our hypothesis, by employing novel high-throughput technology (genome-wide methylation assay- Infinium MethylationEPIC) to probe into the epigenomic methylation hallmark for healthy life span in a unique population (i.e. Israeli multi ethnic centenarian study cohort) of subjects between ages 55-110. In addition, we did a combination of large-scale epigenomic studies to identify the most distinctive epigenetic loci (i.e. those with the greatest differential methylation). We then perform Multi-locus validation for methylation status using MassARRAY (Sequenome). We incorporated validation of candidate epi-loci in extended original population, to define the role of epigenetics on specific mechanisms related to age related diseases and healthy lifespan (such as mitochondrial mutations, oxidative stress).