

# Aging Biomarkers-Disorders Characterized by Premature Aging

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Citation: Gatta DM, Botcha SS (2021) Aging Biomarkers-Disorders Characterized by Premature Aging. Biomark J Vol.7 No.1: 81.

Received date: January 21, 2021; Accepted date: February 04, 2021; Published date: February 11, 2021

## Abstract

Aging is a normal and multi-factorial phenomenon characterized by the accumulation of degenerative processes that are in turn underpinned within molecular pathways by multiple alterations and damage, ultimately the alterations and damage compromise cell and tissue functions. As such, for almost all non-communicable diseases, including cardiovascular diseases, cancer, diabetes and neurological diseases, aging is the most profound risk factor. DNA injury, changes in gene and non-coding RNA expression, genotoxicity, oxidative stress, and the occurrence of shorter telomeres are the proposed mechanisms that lead to the aging process and the development of these chronic, age-associated diseases.

**Keywords:** Biomarker; Biogerontologists; Aging

## Description

The nature of biomarker research is the products of these physiological, biochemical, and/or molecular-genetic processes and the predictive associations between these products as they are linked to age.

Aging biomarkers are biomarkers that may better predict functional ability than chronological age at some later age. In other words, aging biomarkers will provide the actual "biological age," which could be different from the chronological age [1,2]. Validated aging biomarkers would allow lifespan to be extended by testing therapies, because changes in biomarkers would be measurable over the organism's lifespan. While maximum lifespan would be a means of validating aging biomarkers, it would not be a realistic means for long-lived organisms such as humans because longitudinal studies would take too long to validate aging biomarkers.

Although hair graying increases with age, it is not possible to call hair graying a biomarker of ageing. Similarly, skin wrinkles and other typical improvements seen with aging are no better markers than chronological age for potential functionality [3]. Biogerontologists have continued their attempts to identify and confirm aging biomarkers, but progress has been limited so far.

## Diseases

Rare human genetic disorders characterized by premature aging phenotypes with a reduced life span are Hutchinson-Gilford Progeria Syndrome (HGPS) and Werner syndrome. To a certain extent, this category of diseases resembles physiological ageing, serving as excellent models to gain insight into the biology of human aging. These diseases are due to either a mutation in genes encoding the machinery for DNA repair or the A-type lamina, leading to disorganized structures of chromatin.

### Hutchinson-Gilford progeria syndrome

Is a genetic condition, the drastic, rapid emergence of aging starting in childhood characterizes the genetic disorder. Usually, affected children appear fine at birth and in early childhood, but then develop slower than other children and do not gain weight at the predicted rate (failure to thrive). Including prominent eyes, a thin nose with a beaked tip, thin lips, a small chin and protruding ears, they grow a distinctive facial appearance. Alopecia, aged-looking skin, joint defects, and fat loss under the skin are also caused by Hutchinson-Gilford progeria syndrome (subcutaneous fat). Extreme artery hardening (arteriosclerosis) occurs in people with Hutchinson-Gilford progeria syndrome starting in childhood. This disorder dramatically raises the risk of a heart attack or stroke at a young age.

**Caused by:** Due to the development of an irregular lamin A protein, mutations in the LMNA gene cause Hutchinson-Gilford progeria syndrome. The illness is inherited by people; only one copy of the LMNA gene is necessary to induce the illness since it is an autosomal dominant gene.

### Werner syndrome

The dramatic, rapid appearance of features associated with normal aging is characterized by Werner syndrome. Typically, individuals with this condition develop and grow normally before they reach puberty. Typically, affected adolescents do not have a growth spurt, resulting in small stature.

Usually, when they are in their twenties, the distinctive aged appearance of individuals with Werner syndrome starts to evolve and involves graying and loss of hair; a hoarse voice; and thin, hardened skin. They may resemble with a facial appearance called "bird-like." Due to irregular fat deposition, many people with Werner syndrome have thin arms and legs and a thick trunk.

**Caused by:** WRN gene mutations cause Werner syndrome. The WRN gene provides guidance on the development of the Werner protein, which is believed to perform many tasks related to DNA maintenance and repair.

## Aging Biomarkers Applications

DNA methylation, loss of histones, and histone alteration are the key pathways known as possible biomarkers of aging [4]. The uses of aging biomarkers are ubiquitous and the discovery of a biological aging physical parameter will enable humans to assess our true age, mortality, and morbidity.

## Conclusion

The physical biomarker change should be proportional to the age change of the species. Thus, human beings will be able to

delve into research on extending life spans and finding timelines for the advent of possible genetic disorders after developing a biomarker of aging.

## References

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