Healthy Aging Research

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Commentary

The Concept of DNA Damage and Age-Related Growth

Egill Rostrup*

Department of Clinical Medicine, University of Copenhagen, Copenhagen, Denmark

ABOUT THE STUDY

According to the DNA damage theory of aging, the buildup of naturally occurring DNA damage that is not repaired leads to aging. In this context, damage is defined as a DNA modification with an aberrant structure. Nuclear DNA is the main focus of this investigation, even though both mitochondrial and nuclear DNA damage can speed up aging. Aging can be caused directly or indirectly by nuclear DNA damage.

Because DNA damage happens often in mammals, including humans, DNA repair mechanisms have developed to counteract this. According to estimates for mice, DNA lesions occur in each cell on average 25 to 115 times every minute, or 36,000 to 160,000 times per cell per day. Even after repair processes have been active, some DNA damage may still be present in any cell. Certain cell types, especially non-reproducing or slowly replicating cells like brain, skeletal, and cardiac muscle cells, are more likely to accumulate unrepaired DNA damage.

Age-associated accumulation of DNA damage

DNA damage can build up over time in tissues made up of cells that seldom or never replicate and can eventually result in cell death or loss of gene expression in surviving cells. It is common practice to evaluate cumulative DNA damage directly. This type of research has shown that oxidative DNA damage is particularly significant. Both at the mRNA level and the protein level, it is possible to identify the loss of expression of particular genes.

Increased transcriptional variability, which was first identified in a chosen panel of genes in heart cells and, more recently, across the entire transcriptomes of immune cells and human pancreatic cells, is another type of age-related alteration in gene expression.

The breakdown of gene to gene coordination based on the transcriptional interactions between genes is another intricate form of transcriptional alterations in aging tissues.

Brain: The majority of the neurons in the adult brain are non-dividing, terminally differentiated cells. Numerous glaring signs of aging are caused by a loss in neural function.

This resulted in the discovery of a group of genes whose expression changed after the age of 40. These genes are essential for mitochondrial function, vesicular transport, and synaptic plasticity. Promoters of genes with decreased expression have significantly higher DNA damage in the brain. These gene promoters are specifically harmed by oxidative stress in cultured human neurons.

Muscle: In humans and other species, muscle strength and the capacity for continuous physical exertion decrease with age. Multinucleated myofibers, which are made up of mononucleated myoblasts fused together, make up the majority of the tissue that makes up skeletal muscle.

In skeletal and cardiac muscle, protein synthesis and breakdown reduce with age, as would be expected given that DNA damage prevents gene transcription. Piec discovered numerous alterations in protein expression in aging rat skeletal muscle, including decreased levels of several myosin and actin-related proteins. Myosin thick filaments and actin thin filaments interact to produce force in striated muscle.

Liver: Hepatocytes in the liver don't normally divide and seem to be terminally differentiated, yet they nevertheless have the capacity to multiply when damaged. With advancing years, the liver loses mass, blood flow is diminished, metabolism is compromised, and microcirculation changes.

Correspondence to: Egill Rostrup, Department of Clinical Medicine, University of Copenhagen, Copenhagen, Denmark, E-mail: RostrupE123@gmail.com

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