

Environmental Impact on Aging and Life Span

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COMMENTARY

Aging related to breakdown of cellular and tissue function over time, which is related to increased prevalence of chronic diseases. Evidence in invertebrate model organisms and human studies support the idea that aging is regulated at the genetic level but also by non-genetic factors. Interestingly even the lifespan of isogenic individuals reveals large differences between the primary and last death in controlled environments, suggesting that even small environment variations may dramatically impact aging and lifespan. A number of environmental modulators of the aging process include dietary interventions, up regulated stress response, physical exercise and circadian rhythms.

After all, our body must work continuously day in the outing for dozens of years, which causes it wear and tear. The interesting thing is that this is often not really true. Aging isn't simply a results of inevitable wear and tear. Metabolism may be a collective term for all the processes within the body that allow the body to function: the beating of the guts, the contracting of muscles the breathing and the firing of nerve signals. Since mice and bats have a comparable, metabolism, one would expect that they also wear and age at an equivalent rate. However the average life span of a mouse is two years, where as a bat can live to be 30 years old or more.

The pervasiveness is age related alterations in the chromatin regulation across the cell types and species is now well documented exclusively focussed on studies of chromatin aging throughout organismal lifespan across model organisms. Though

it is clear that many epigenomic changes occur in the aging how these changes may ultimately impact the tissue and cell biology is less clear in the present circumstances. Because of the potential role of chromatin as a regulatory platform age related epigenomic may foster biological instability. First, changes to the chromatin landscape throughout life may cause decreased transcriptional precision and decreased cell and tissue function. The robustness and integrity of transcriptional network has been observed to decay during aging whether aging is also associated to increased cell to cell transcriptional noise another respect of transcription precision, remains an open question. Indeed whereas increased transcriptional noise has been observed for tested genes in cardiomyocytes with aging. Whether aging is additionally associated to increased cell to cell transcriptional noise, another aspect of transcription precision, is still an open question. Indeed, whereas increased transcriptional noise has been observed noise in hemopoietic stem cells from old mice for any of the six assayed genes. It is important to notice that, due to technical limitations, these pioneering studies were limited to few genes and cell types. Recent advances in single cell profiling techniques now allow high resolution genome wide analyses of single cell transcription across diverse cell types and will be key to understand the significance of transcriptional noise regulation during aging.

Age dependent changes in chromatin modification may impact the aspects of transcriptional precision. Recent studies support an important function for the promoting transcriptional process during aging.

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