Decoding The Hallmarks Of Aging Transcript

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Longevity Hallmarks Overview

The next, circle, after life energy is the longevity circle, which is really looking at the hallmarks of aging. So there were nine published in 2013. Another five were added in 2022.

Now we're up to about 16. Some people think there's 22. Some people think there's 30. The point is, these are basically processes that occur both as a expression of aging.

Aging Drivers and Metabolic Balance

In other words, telomeres get short. That's an expression of aging. But then it also becomes a driver of aging. It actually accelerates the aging process.

And so when you actually understand that these are really what's responsible for the exponential acceleration in the aging process. It makes you start to understand that they need to have an exponential response to reverse them.

And so these hallmarks of aging, whether it's shortening telomeres, mitochondrial dysfunction, lack of protein manufacturing, folding and disposal, crosslinking of proteins with endogenous sugars that happen or exogenous sugars from what we eat.

The risk of cancer, a lack of DNA repair, alterations in the gut microbiome as we age, epigenetic changes that accelerate our aging. This balance between mTOR and Ampk, mTOR is really what, builds us and makes us strong.

But if we have too much more, we have more cancer and we die sooner. Ampk is what rejuvenates us. And and yet, if we have too much Ampk, we're weak and frail.

Inflammation, Biomarkers, and Cellular Damage

And so it's a teeter totter. This is part of the symphony of longevity balancing being on mTOR part of the time. And then being on a Ampk side of the equation part of the time.

And there are more elements here. Inflammation. Inflammation is such a big driver of aging, and it's because it has so much oxidative stress associated with it.

Looking at NAD levels, looking at transcriptomics and proteomics, this is basically the expression of your genes both in the RNA and the proteins that actually tell us what genes are turned on and what genes are turned off.

This becomes a clue to actually what's happening with us in the aging process. And then there are things related to cellular macromolecular damage where the cells are damaged.

You don't have a great way to measure this currently. And altered intracellular communication is also will accelerate inflammation. And we can measure it that way.

And then so mechanical properties we don't have a great way to look at cell mechanical properties at the moment. But there's some really fascinating work being done around micro tubules in the cell, that actually relate to consciousness.

Stem Cells, Senescence, and RNA Splicing

And they're also sort of pathways and highways, if you will, for cells to move things around in the cells and actually for cells to divide. So that's a very fascinating area of research.

And then stem cells, we know how important stem cells are to rejuvenate ourselves. And we may have a limited supply of those. And how do we actually keep from depleting that supply.

We'll talk about that in this conference as well. And then the senescent cell burn. Senescent cells are essentially old cells that no longer work. They still take up space and they can become sources of secretions of inflammatory cytokines.

So in other words, they're fanning the flames of, of, aging by increasing inflammation. In doing so, not only do they take up space, but when they start to excrete these inflammatory cytokines, they're actually recruiting normal cells that also become senescent.

And this becomes part of the exponential acceleration in aging also. And then messenger RNA splicing dysregulation. So when a protein is coded for on a gene, RNA will come in and code along that DNA and then come off the DNA.

Now that RNA this segment over here is part of the protein. This segment here is part of the protein. This segment over here is part of the.

Immune Aging and Applied Longevity

Part of the protein. And put it back all together. That's how you actually get it. So when there's splicing dysregulation when you're splicing these together it can lead to faulty protein production.

And this is now seen to be a hallmark of aging. And then the immune system. This is a massive one when the immune system becomes senescent. Short telomeres will do this increase inflammation.

And senescent cells will do this. But when the immune system is no longer useful, it can no longer fight off, a virus like Covid, and it can no longer detect a cancer cell and, kill it off, and it can no longer prune senescent cells from the body, all of which are incredibly important for staying useful.

So these are all needs to be looked at. And these are things that are applied longevity. We deconstruct everything around us that we possibly can to understand not only where somebody is in the aging process, but then also how do we actually take them forward towards this youthful 30 year old body?