Friday Favorites Epigenetic Clocks for Testing Your Biological Age Transcript

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Epigenetics and Cell Identity

My book How Not To Age, is all about the mechanisms of aging and how we can affect them through diet and lifestyle. Can looking at epigenetics, modifiers of gene expression help predict our health span and lifespan?

Watch to find out. "Epigenetic Clocks for Testing Your Biological Age" Epigenetics-the differential expression of genes- both establishes the character and function of a cell and maintains that identity over time through round after round of cell division.

So, a heart cell stays a heart cell and divides to make more heart cells, instead of skin cells or kidney cells, even though all our main cells have the same entire complement of DNA to potentially be anything.

This is accomplished by methylation, chemical markers that silence inappropriate genes in a particular cell. The fidelity of that maintenance of methylation is good- 97 percent to 99.9 percent every division-but not perfect.

Twin Drift and Age-Related Methylation Changes

Over time, those tiny errors may add up and may help explain why the methylation patterns of identical twins drift apart as they age. The epigenetic markers of young identical twins are essentially indistinguishable but then diverge over time.

Identical twins have the same DNA, the same genes, but the difference in gene expression among older identical twin pairs were found to be about four times greater than those observed in young pairs.

This may result in them each getting different diseases. An age-related disease like Alzheimer's only has an identical twin concordance rate of about 50 percent, meaning if one twin gets it, there's only about a coin flip chance that the other will too, despite identical DNA.

Or, even if they do both get it, the disease may manifest decades apart. Any epigenetic differences that may contribute to differential disease rates may arise from having different diets and lifestyles or may be a result of random epigenetic drift.

However, there are certain DNA sites on our chromosomes that predictably methylate or demethylate as we age. So predictable, in fact, it's like clockwork.

Epigenetic Clocks and Biological Age

One of the earliest attempts to study aging and the epigenome (which is only like a dozen years ago) found that the DNA from a 103-year-old appeared to be less methylated overall than the DNA of a newborn infant, suggesting, perhaps, that aging involves the general loss of epigenetic markings.

We now know it's more complicated than that. Of the methylation sites that reliably change as we age, about 60 percent go from methylated to unmethylated, and the other 40 percent become more methylated over time.

Some so reliably change with age that they've been considered a "molecular crystal ball for human aging." In a remarkable triumph of Big Data, out of the millions of methylation sites in our DNA, a tiny subset so dependably shift over time that you can predict someone's age within a few years just by strategically measuring the methylation pattern in a few hundred or even just a few dozen sites in someone's three-billion-letter genome.

Over just the last few years, these "epigenetic clocks" have become established as robust measures of chronological age, surpassing telomere length as the best age predictor.

Who cares, though? I mean, why invent some costly Rube Goldberg approach to divining someone's age when you can simply ask them? Well, you can imagine forensic applications, the determination of an unidentified victim's age with a blood sample, but that's just scratching the surface.

The kicker is that epigenetic clocks don't just track your chronological age but appear to measure your true biological age. In other words, your epigenetic age can better predict your remaining life expectancy than your calendar age.

It's like science fiction. Feed a drop of blood into some futuristic machine that scans the placement of chemical markers on a strand of DNA, and it spits out your true age, reflecting a lifetime of lifestyle choices.

Healthspan Prediction and Anti-Aging Tracking

If the machine calculated that you have the DNA methylation pattern of a 60-year-old, but you've only had 50 birthdays, that would be an example of "epigenetic age acceleration," when your epigenetic clock age is older than your actual chronological age.

That would be an indication that you're aging too fast. As a 50-year-old, you'd think you have another, mm, 30 years on this Earth, but because the epigenetic clock shows that you're aging at such an accelerated pace, it's more like you only have another 20 years left.

Every five years of epigenetic age acceleration is associated with an 8 to 15 percent increased risk of mortality. In addition to predicting time-to-death, epigenetic clocks also appear to foretell healthspan indicators, such as cognitive decline, frailty, arthritis, and the progression of diseases like Alzheimer's and Parkinson's.

As you can imagine, the insurance agency* has jumped on this, and your premiums may soon be determined by your epigenetic age. But it's not some Gypsy fortune teller curse set in stone.

You can change the rate at which you age and may soon be able to use epigenetic clocks to track your progress, potentially presenting a radically faster and cheaper way to test anti-aging interventions.