Researchers link aging to cellular interactions that occur across generations

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(Phys.org) —The evidence for what causes aging has typically been limited to the study of a single organism's lifespan; our cells divide many times throughout our lives and eventually cause organs and our bodies to age and break down. But new research from the UNC School of Medicine suggests that how we age might depend on cellular interactions that we inherit from ancestors throughout many generations.

By studying the reproductive cells of nematodes – tiny worms found in soil and compost bins – Shawn Ahmed, PhD, an associate professor of genetics, identified the Piwi/piRNA genome silencing pathway, the loss of which results in infertility after many generations. He also found a signaling pathway – a series of molecular interactions inside cells – that he could tweak to overcome infertility while also causing the worms to live longer adult lives.

The research, in collaboration with researchers at the University of Cambridge and described in a paper published in the journal *Cell Reports*, suggests that it's possible to manipulate the aging process of progeny before they're even born.

The finding gives scientists a deeper understanding of what may govern aging and age-related diseases, such as some cancers and neurodegenerative conditions.

Typically, nematodes produce about 30 generations in a matter of months and remain fertile indefinitely. Ahmed and colleagues found that

a mutation in the Piwi/piRNA cellular pathway of germ cells gradually decreased the worms' ability to reproduce as the mutation was passed down through the generations and eventually caused complete sterility. But when Ahmed's team manipulated a different protein – DAF-16/FOXO – the nematodes overcame the loss of the Piwi pathway. The worms did not become sterile; generations of worms reproduced indefinitely, achieving a sort of generational immortality. Moreover, it has been well established that DAF-16/FOXO plays a role in nematodes living longer.

Achieving longer life suggests that there's an effect on the aging of somatic cells – the cells that make up the body and organs of an organism.

"That's the really interesting thing about this," said Ahmed, a member of the UNC Lineberger Comprehensive Cancer Center. "What we've found implies that there's some sort of relationship between somatic cell aging and this germ line immortality process we've been studying."

What that relationship is, precisely, remains unknown. But so does the exact mechanism by which human somatic cells age as they divide throughout our lives. That is, exactly how we age – at the cellular level – is still not entirely understood.

"The field is fairly open in terms of what might cause aging of somatic cells," Ahmed said. "What makes our study unique is that we've found something that could be transmitted over many generations that could affect aging but is not necessarily a genetic mutation. Instead, whatever is being transmitted likely affects how a segment of the genome is silenced, and that genome segment can be modulated by a genetic mutation."

Think of it like this: when you were born, there could have been

something in the reproductive cells of your parents that triggered how the somatic cells of your liver or kidneys would age after you were born.

"This inheritable factor could be dictating the rate at which some of your organs are aging," Ahmed said, "and this may have been set during embryogenesis."

More information: "Reduced Insulin/IGF-1 Signaling Restores Germ Cell Immortality to Caenorhabditis elegans Piwi Mutants." Matt Simon, et al. *Cell Reports*. DOI: dx.doi.org/10.1016/j.celrep.2014.03.056

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