Key gene found responsible for chronic inflammation, accelerated aging and cancer

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Researchers at NYU School of Medicine have, for the first time, identified a single gene that simultaneously controls inflammation, accelerated aging and cancer.

"This was certainly an unexpected finding," said principal investigator Robert J. Schneider, PhD, the Albert Sabin Professor of Molecular Pathogenesis, associate director for translational research and co-director of the Breast Cancer Program at NYU Langone Medical Center. "It is rather uncommon for one gene to have two very different and very significant functions that tie together control of aging and inflammation. The two, if not regulated properly, can eventually lead to cancer development. It's an exciting scientific find."

The study, funded by the National Institutes of Health, appears online ahead of print today in *Molecular Cell* and is scheduled for the July 13 print issue.

For decades, the scientific community has known that inflammation, accelerated aging and cancer are somehow intertwined, but the connection between them has remained largely a mystery, Dr. Schneider said. What was known, due in part to past studies by Schneider and his team, was that a gene called AUF1 controls inflammation by turning off the inflammatory response to stop the onset of septic shock. But this finding, while significant, did not explain a connection to accelerated aging and cancer.

When the researchers deleted the AUF1 gene, accelerated aging occurred, so they continued to focus their research efforts on the gene. Now, more than a decade in the making, the mystery surrounding the connection between inflammation, advanced aging and cancer is finally being unraveled.

The current study reveals that AUF1, a family of four related genes, not only controls the inflammatory response, but also maintains the integrity of chromosomes by activating the enzyme telomerase to repair the ends of chromosomes, thereby simultaneously reducing inflammation, preventing rapid aging and the development of cancer, Dr. Schneider explained.

"AUF1 is a medical and scientific trinity," Dr. Schneider said. "Nature has designed a way to simultaneously turn off harmful inflammation and repair our chromosomes, thereby suppressing aging at the cellular level and in the whole animal."

With this new information, Dr. Schneider and colleagues are examining human populations for specific types of genetic alterations in the AUF1 gene that are associated with the co-development of certain immune diseases, increased rates of aging and higher cancer incidence in individuals to determine exactly how the alterations manifest and present themselves clinically.

Provided by New York University School of Medicine