

Compounds Used in Treatment of Autoimmune Disorders

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EDITORIAL NOTE

The immune system is programmed to rid the body of biological bad guys like viruses and dangerous bacteria but its precision isn't guaranteed. During the tens of millions of Americans suffering from autoimmune diseases, the system mistakes normal cells for malicious invaders, provoking the body to take part in self-destructive conduct. This diverse class of conditions, which includes Type I diabetes, lupus, and multiple sclerosis, can be very difficult to treat. In a previous study the researchers describe their advancement of small molecules that restrain one of the main enzymes involved in misguided immune responses. That could lead to new treatments for people with certain autoimmune disorders and, all the more extensively, shed light on the causes of autoimmunity.

In eukaryotes, including humans, DNA commonly resides in a cell's nucleus, or in other sequestered organelles such as mitochondria. So in case DNA is found outside of these compartments in the cell's cytosol the immune system goes into high alert, assuming the genetic material was leaked by an attacking bacterium or virus. In 2013, researchers discovered a protein called cyclic GMP-AMP Synthase (cGAS) that detects and binds to cytosolic DNA to initiate a chain reaction a cascade of cellular signaling events that leads to immune activation and usually ends with the destruction of the DNA-shedding pathogen.

However, cytosolic DNA isn't always a sign of infection. Sometimes it's created by the body's own cells and cGAS does not discriminate among infectious and innocuous DNA. The protein will tie to totally harmless genetic material, prompting an immune response even in the absence of an intruder. There is no specificity. So as well as sensing unfamiliar microbial DNA, cGAS will also sense aberrant cytosolic DNA made by the host. Furthermore, this absence of self-versus non-self-specificity could be driving autoimmune reactions.

Since the discovery of cGAS, researchers in the Tuschl lab have sought to understand its likely clinical significance. Assuming autoimmune disorders are the result of an erroneously activated immune system, perhaps, they believe, a cGAS inhibitor could be used to treat these conditions.

Up to this point, no powerful and specific small-molecule compound existed to obstruct cGAS in human cells, however the researchers previously distinguished one that can do the job in mouse cells. Through their screen, the researchers distinguished two molecules that showed some movement against cGAS however this result was just the start of a long process towards developing an inhibitor that may be used in a clinical setting. The hits from library compounds were an extraordinary starting point; however they were not strong enough. So we used them as molecular scaffolds on which to make improvements, adjusting their structures in ways that would increase strength and also reduce toxicity.

Working with the Tri-Institutional Therapeutics Discovery Institute, the researchers changed one of their unique scaffolds to create three compounds that blocked cGAS action in human cells making them the first molecules with this capability. The compounds are presently being additionally upgraded for likely use in patients, with an underlying focus on treatment of the uncommon hereditary disease Aicardi-Goutières syndrome. People with this condition accumulate abnormal cytosolic DNA that activates cGAS, prompting serious neurological problems. A drug that blocks the enzyme would therefore be of tremendous therapeutic value to those with the disease, who as of now have not many treatment options. This class of drug might actually also be used to treat more common diseases, such as systemic lupus erythematosus, and possibly neurodegenerative diseases that incorporate inflammatory contributions, such as Parkinson's disease.

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