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Research uncovers key difference between our bodies' fight against viruses and bacteria

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Scientists at The University of Nottingham have discovered a key difference in the biological mechanisms by which the immune system responds to viral and bacterial pathogens.

The study, published in the journal *Nature Immunology* and led by Professor Uwe Vinkemeier in the University's School of Life Sciences, centred on STAT1, a protein that can bind DNA and hence plays a vital role in regulating genes in the body.

STAT-1 responds to interferon signals, hormone-like molecules which control communication between cells to trigger defensive action by the body's immune system when pathogens such as bacteria, viruses, or parasites are detected. These powerful defensive actions are also part of the body's ability to control the growth of malignant tumours that can ultimately achieve their complete elimination.

It was previously thought that all interferons used single STAT1-containing units rather than STAT1 chains to regulate the activity of genes. However, using mice bred specially to express a mutated form of STAT1 which is limited to forming single STAT1 units, the Nottingham team has demonstrated that this abolishes the function of some interferons while leaving others largely unaffected.

They found that when the assembly of STAT1 chains was inhibited, type I interferons responsible for protecting against viruses such as vesicular stomatitis virus were unaffected, whereas type II interferons, which

protect against bacterial infections such as listeria, no longer functioned effectively.

Professor Vinkemeier said: "The core of these findings is that we are revising a central aspect of what we thought we knew about how these proteins worked. The molecular mechanisms underlying type I and type II interferon functioning are actually more distinct than we previously imagined. This in turn offers new options for rational pharmacological intervention."

For example, type I interferons, involved in the anti-viral response also play a role in stopping cells from growing and replicating—and therefore inhibiting the spread of the virus throughout the body. These interferons are already in clinical use against Hepatitis virus and several cancers and in the treatment of auto-immune diseases like multiple sclerosis. Type-II interferon, in contrast, has been shown to be detrimental in some of these conditions, namely multiple sclerosis and melanoma, an aggressive type of skin cancer.

"In situations like these our finding offers a new target for making current treatments more effective. There is good reason to assume that an inhibitor of STAT1 chain formation could potentially block detrimental type-II interferon responses while keeping type I activities, including anti-viral protection, intact. This would avoid an important shortcoming of current STAT1 inhibitors."

More information: Paper: dx.doi.org/10.1038/ni.2794

Provided by University of Nottingham

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