Research finds new gene is key to immune response

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Credit: Stuart Hay, ANU

Researchers have identified a new gene which is crucial for the immune system to respond to infection and vaccination.

The finding could help scientists better understand immunodeficiency and why some people are more susceptible to infections.

One of the lead researchers, Dr Ian Parish from The Australian National University (ANU), said removal of the Etaa1 gene in a mouse model resulted in the animal not being able to mount an immune response to vaccination or infection.

"This finding may help researchers better understand certain patients who have an immunodeficiency with an unknown cause and are not able to control infections or respond to vaccination," said Dr Parish, from The John Curtin School of Medical Research at ANU.

This is the first time the gene has been removed in an animal, with scientists until now only trialling the removal of the gene from cells in a dish.

"Based on the biology of this gene, we would have predicted that it would be impossible for any of the body's cells to survive without the Etaa1 gene. However, deleting this gene only affects immune cells for reasons that we still don't fully understand," Dr Parish said.

Dr Parish said although researchers know that a patient's genetics can contribute to immunodeficiency, the genetic changes that cause disease are not known in a large proportion of immunodeficiency patients.

"Given how specific the Etaa1 gene is in controlling the immune response to infection, we are now interested to see whether damage to the Etaa1 gene could explain some patient cases of immunodeficiency," Dr Parish said.

He said researchers would now look to find whether certain people with immunodeficiency also have defects in the Etaa1 gene.

More information: Lisa A. Miosge et al. Systems-guided forward genetic screen reveals a critical role of the replication stress response

protein ETAA1 in T cell clonal expansion, *Proceedings of the National Academy of Sciences* (2017). DOI: 10.1073/pnas.1705795114