Crohn's, eczema, and psoriasis have the same genetic cause

Diseases like Crohn's, psoriasis, and eczema are all caused by defects in the same genes, reveals new study.

An international team of scientists have identified ten new genetic variants that cause eczema.

For the first time, they have identified which biological signals--communication pathways between cells--these genes affect. They have also examined what effect these biological signals have on other diseases.

The new research shows that many of the genetic defects that lead to development of eczema are also present in people who develop autoimmune diseases such as psoriasis, and the inflammatory bowel disease Chron’s.

According to the scientists behind the new study we are now one step closer to understanding the cause of these diseases, and this knowledge could lead to new types of treatment.

“This study gives us an understanding of some of the common mechanisms that come into play in the development of many different diseases,” says co-author Johannes Waage from COPSAC (Copenhagen Prospective Studies on Asthma in Childhood), Danish Paediatric Asthma Centre.

“It’s important that we understand these mechanisms if we are to have any hope of being able to prevent or treat them one day,” he says. “It’s not enough to know which genes are affected--we also need to know what role the genes play, and the interactions between different biological mechanisms that result in the disease.”

The new research is published in the scientific journal Nature Genetics.

Colleague: Results can lead to better treatments

Lone Skov is a professor at University of Copenhagen, Denmark, and consultant at the Skin and Allergy Department at Herlev and Gentofte Hospital, Copenhagen.

She was not involved in the new study but has read it and thinks that the results are interesting.

Skov says that the study could bring us a step closer to better treatments for eczema.

“It’s important that we find out which genes play a role in the disease and what changes occur in the immune system when it goes wrong, in order to get closer to new treatments for those patients that are hit the hardest,” says Skov.

“This study identifies new genes, which may eventually help us to find new targets for treatments,” says Skov.
Largest genetic study of eczema

The new study is the largest of its kind and involved 21,000 people with eczema and 95,000 control subjects. The data was collected from many different research institutions.

The genomes of both the control group and the group with eczema were mapped and analysed, which allowed the researchers to identify the genetic differences between the two groups.

Waage and colleagues discovered 31 locations on the genome where some of the DNA building blocks had been substituted, and in which the genetic differences could be associated to eczema. Scientists call these genetic variants.

21 variants were already known, but 10 of them were brand new.

"When we conduct such a big study it gives us an opportunity to find even more genetic variants. In addition to the 31 genetic variants that were strongly associated with eczema, we also found hundreds of other genetic variants, which are only weakly associated with the disease," says Waage.

Overlap between diseases

Waage and colleagues examined the biological signalling pathways in the genes and identified other diseases that could be linked to the same pathways.

They discovered that many of the genes affected pathways that have a strong impact on the development of various autoimmune diseases.

For example, they discovered that some of the genetic variants affected certain signalling pathways that specifically has to do with the skin’s immune system tolerance.

In an autoimmune disease like psoriasis, the skin can become hypersensitive, and react to either bacteria or viruses that are naturally present in the skin.

This is due to a signalling pathway that prevents the immune system from functioning as it should. In the new study, this very same signalling pathway was affected by the genetic variants identified in patients with eczema.

"We can find lots of these coincidence of various diseases in which the same signalling pathways are defective in some way. Scientists around the world are currently working on this large puzzle, trying to link the various genes, diseases and signal paths together to form a greater understanding of the cause of all diseases," says Waage.

If such an overview is achieved, it may provide an opportunity to intervene and prevent the diseases in the first place or to develop new treatments, he says.

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