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A Potential Treatment to Prevent Obesity-Related Liver Damage

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Editorial

One of the especially dangerous health risks of being extremely overweight occurs when an obese person begins to accumulate fat in their liver. This condition non-alcoholic disease disease (NAFLD) is the world's commonest chronic liver disease and is that the primary underlying cause for liver transplants in children and adults. Without such transplants, which are available to only a little percentage of patients, NAFLD over time are often fatal. In fact, (excluding alcohol-related liver damage) quite 30,000 people a year die from NAFLD.

For years, the first thanks to treat NAFLD has been through the utilization of varied weight control methods: diet programs, exercise regimens, medications of limited benefit, bariatric surgery, and more. But once people develop progressive NAFLD, simply losing weight isn't enough.

The research team reports that excessive fat deposition within the liver thanks to obesity can alter the microenvironment of the liver during a way that draws a highly specific population of immune T cells to the liver. These "inflammatory hepatic *CXCR3*+Th17 cells" or "ihTh17" cells continue to trigger excess inflammation and life-threatening liver damage.

By running a series of experiments using human tissues and cells and multiple lines of genetically modified mice, the team found that obesity itself triggers activity along a molecular "pathway" that starts with excess expression of the *CXCL10* and *CXCR3* genes.

This abnormal activity attracts more and more ihTh17 cells to the liver. The consequence being a scorched earth inflammatory feedback circuit that recruits additional immune cells and progressively damages liver function.

After tracing the ihTh17 cell liver recruitment pathway, the team began to seek out how to interrupt the unhealthy cycle of inflammation. They found success with mice bred to lack expression of the gene Pkm2 in their T cells, which appears to be crucial to continued activity along the *CXCR3* pathway.

When these modified mice got obesity-inducing diets, they still got fat. But they suffered notably less liver damage than non-modified mice. Next, the researchers tested human tissues collected from people with NAFLD. They confirmed that a lot of of the key genes and molecular activities occurring within the mice also might be detected within the human liver cells.

The results demonstrate for the primary time that ihTh17 cells represent a crucial component of the complex world of NAFLD pathogenesis. Learning more about the way to regulate ihTh17 cells' function and therefore the interaction with the liver cells and the system could lead on to new therapies to scale back the harm caused by NAFLD.

Human gene editing isn't likely to be a suitable option for this condition anytime soon. However, some drugs are known to be capable of blocking Pkm2 activity, Scientists says. Those drugs still require more in-depth laboratory evaluation. Ultimately, a promising compound also would wish to be tested in multi-year clinical trials. But now, for the primary time in years, the team features a promising cause explore.

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