TREATING FIBROSIS

California Researchers Find Treatment for Fibrosis

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Scleroderma, autoimmune liver diseases, pretibial myxedema, thyroid and other conditions are often complicated by fibrosis. Learn the latest treatment news.

Fibrosis in Autoimmune Disease

Fibrosis is a thickening or hardening of tissue causing changes similar to those seen in scarring. Fibrosis is an important feature of many autoimmune diseases.

For instance, fibrosis of the liver in patients with autoimmune hepatitis can lead to cirrhosis. And fibrosis of thyroid tissue in patients with Hashimoto’s thyroiditis leads to the destruction of normal thyroid cells and exacerbates hypothyroidism. In both sarcoidosis and systemic sclerosis, fibrosis of the lungs (pulmonary fibrosis) is a common complication. In both pretibial myxedema, which can occur in patients with Graves’ disease and in conditions of nephrogenic fibrosing dermopathy, the upper and lower extremities may be affected by a dermal fibrosis, which causes a tightening and hardening of the skin. In patients with thyroid eye disease, fibrosis can restrict eye muscle movement.

Stopping and Reversing Liver Fibrosis

Researchers at the University of California San Diego (UCSD) have shown in animal studies that fibrosis of the liver can be stopped and reversed. This study shows the possibility of treating and curing conditions characterized by excessive tissue scarring, such as scleroderma, pulmonary fibrosis, hepatitis, fatty liver disease, and primary biliary cirrhosis.

Six years ago, the UC San Diego School of Medicine research team discovered that excess collagen production related to oxidative stress causes the excess fibrous tissue growth that leads to liver fibrosis and cirrhosis. Specifically, oxidative stress causes cell injury and this activates a protein called RSK. RSK, in turn, activates stellate liver cells, and initiates the process of fibrosis. In these earlier studies, UCSD researchers developed a way to block excess scar tissue in mice.

Current Research

While early studies showed the mechanism in which fibrosis occurs, the latest research, published in December 2007, describes the method in which the researchers stopped and reversed liver fibrosis in mice. The researchers developed an RSK inhibitory peptide that effectively blocked the activation of RSK. Furthermore, blocking the excessive collagen
production via RSK inhibition doesn’t harm the liver. When mice with severe liver fibrosis were given the RSK-inhibitory peptide, the peptide stopped the hepatic stellate cell proliferation. In addition, this compound directly activated the caspase or ‘executioner’ protein, which killed the cells responsible for liver cirrhosis while sparing the normal liver cells.

The researchers also found that human liver cells have a similar pathway in which the activation of RSK protein leads to cirrhosis. This pathway is not seen in normal control livers. Biopsy samples taken from patients with liver cirrhosis also showed activated RSK. This implies that RSK inhibitor peptides may reverse liver fibrosis in humans. Encouraged by their results, the researchers report that the ability to reverse fibrosis would lead to treatments for fibrosis in other autoimmune disorders that are characterized by fibrosis.

**Resources:**


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