Surprising find may yield new avenue of treatment for painful herniated discs

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An immune cell known to cause chronic inflammation in autoimmune disorders has been identified as a possible culprit in low back pain associated with herniated discs, according to doctors at Duke University Medical Center.

The finding implicates the cytokine molecule interleukin-17, and supports the burgeoning theory that an immune response plays a significant role in disc disease, says William J. Richardson, MD, an orthopedic surgeon at Duke. It may also open the door for new, therapeutic approaches that target a specific immune response in hopes of halting disc destruction, and possibly reversing the disease process.

"By identifying the specific subpopulation of lymphocytes (immune cells that are excited into action by the cytokine), it may soon be possible to arrest the body's inflammatory response to disc cells," says Richardson, senior author of the research published online this week in the July issue of *Arthritis and* Rheumatism. Doing so could reduce the painful inflammation associated with degenerative disc disease, and halt the evolution of arthritis. It may also reduce the need for back surgery.

"Mechanical forces may initiate the degenerative process, but biochemical inflammatory changes certainly play a role in disc pathology," says the study's first author, Mohammed Shamji, MD, PhD, senior neurosurgery resident at The Ottawa Hospital, Ontario, Canada, who participated in the research while at Duke. Decreasing the inflammation may arrest or reverse the patient's disease process and

perhaps reduce the need for surgery. "Now we are learning which pathways we have to block."

Low back pain is one of the most common reasons people seek medical care, and both degenerative and herniated discs -- also referred to as slipped discs or ruptured discs -- are common causes of that pain. The economic impact of medical care for herniated discs in the U.S. is estimated to be as high as $200 billion per year.

Herniated discs occur when the tough outer layer of cartilage cracks, allowing pieces of the softer inner material to protrude into the spinal canal. Until recently, it was thought that pain occurs when the material touches a nerve. Now doctors believe the pain is the result of an immune response caused by the presence of inflammatory cells.

"The center of the disc is immune-privileged since it has never been exposed to the immune system," says Shamji. When a disc is injured or degenerates, the body reacts against the invading inner material as it would against any virus or foreign body, and launches a response targeted at destruction. The nerve root, which is present near the protruding disc material, becomes painfully inflamed, swollen and damaged during that cascade of events.

In recent years, several anti-immune therapies, including steroids, have been injected into the space between the disc and the nerve, but with limited success, doctors say, because they don't target a specific immune response, and because low doses are used to minimize potentially serious side effects that include a higher predisposition to infection, activation of tuberculosis and a six-fold increase in lymphoma incidence.

The identification of IL-17 in the cascade of events is significant, Shamji says. "It's a product of a specific subgroup of immune cells that are involved in auto immune phenomena like rheumatoid arthritis and

asthma, but not in the body's response against infection or tumor. If you target this specific lymphocyte, you may avoid compromising the body's ability to protect itself against infection or tumor."

Researchers say they're still several steps away from human studies of IL-17 blockers currently in development.

Provided by Duke University Medical Center