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Is the Body's Sodium Channels the New Route to Preserve Joint Structure in OA?

WASHINGTON, D.C. – Research at ACR Convergence 2024, the American College of Rheumatology's annual meeting, found that certain channels in cartilage cells play a crucial role in osteoarthritis (OA). This discovery paves the way for the development of treatments that may slow or prevent joint damage and pain caused by OA.

OA is the most common form of arthritis, affecting 32 million people in the U.S. and millions more worldwide. It occurs most often in older adults. Once considered a disease where cartilage wears away over time, it is now known that OA involves all parts of the joint, including the joint lining, ligaments and bone. Weight loss and exercise can help reduce pain and inflammation but cannot prevent or reverse joint damage.

Yale researchers have discovered that a specific type of sodium channel, called Nav1.7, might be key to treating osteoarthritis. Chuan-Ju Liu, PhD, a professor of orthopaedics and vice chair of research for the department of orthopaedics and rehabilitation at Yale University School of Medicine co-led the research with Stephen Waxman, MD, a professor of neurology, neurobiology and pharmacology at Yale University.

Professor Liu explains that sodium channels are specialized proteins that open and close to allow sodium to pass through a cell's membrane. They are usually found in cells of the brain and muscle, where they play a leading role in generating electrical signals.

Nav1.7 is a type of sodium channel normally found in brain cells (neurons), where it plays a big role in how pain is felt– a discovery of Dr. Waxman's. About a decade ago, Liu and colleagues were surprised to find the same channel in cells of the cartilage (chondrocytes) in patients with osteoarthritis.

"When we inactivated this channel, we observed significant protection against cartilage breakdown and joint degeneration, along with a reduction in joint pain," Liu explains. "This discovery suggests that Nav1.7 could serve as a treatment target for both reducing pain and preventing joint deterioration."

"Additionally, we observed that blocking Nav1.7 channels reduced the progression of osteoarthritis," he continued.

Most of Liu's recent research has been performed in animal models, but he points out that sodium channels function the same way in almost all species.

The next step, he says, is to find cost effective and feasible ways to inhibit Nav1.7. A drug originally developed to treat pain by inhibiting Nav1.7, is currently being tested in clinical trials.

“Our discovery of the sodium channel nav1.7 represents a significant stride [forward],” he says. “Our findings suggest that modifying the activity of these channels could offer a novel approach to managing OA symptoms and, more importantly, disease progression.”

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About ACR Convergence

ACR Convergence, the annual meeting of the American College of Rheumatology, is where rheumatology meets to collaborate, celebrate, congregate, and learn. With hundreds of sessions and thousands of abstracts, it offers a superior combination of basic science, clinical science, business education and interactive discussions to improve patient care. For more information about the meeting, visit the ACR Convergence page, or join the conversation on X by following the official hashtag (#ACR24).

About the American College of Rheumatology

Founded in 1934, the American College of Rheumatology (ACR) is a not-for-profit, professional association committed to advancing the specialty of rheumatology that serves nearly 9,600 physicians, health professionals, researchers and scientists worldwide. In doing so, the ACR offers education, research, advocacy and practice management support to help its members continue their innovative work and provide quality patient care. Rheumatology professionals are experts in the diagnosis, management and treatment of more than 100 different types of arthritis and rheumatic diseases. For more information, visit rheumatology.org.