Discovery Links Proteins Necessary to Repair Membranes

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(PhysOrg.com) -- Researchers at UMDNJ-Robert Wood Johnson Medical School are a step closer to treating, and perhaps preventing, muscle damage caused by disease and aging. In their study, published in the June issue of *Journal of Biological Chemistry*, the scientists have linked the newly discovered protein MG53 to a pathway that repairs human muscle tissue along with the proteins caveolin-3 (Cav3) and dysferlin.

Prior to this study, the underlying interactions that inhibited membrane repair in muscle tissue were unknown. Linking these proteins creates a mechanism that allows damaged membranes to be repaired, which may transform treatment for patients who suffer from severe complications of diseases such as muscular dystrophy, as well as cardiovascular disorders and conditions related to advancing age.

The study was led by Jianjie Ma, PhD, professor of physiology and biophysics at UMDNJ-Robert Wood Johnson Medical School, in collaboration with Professor Hiroshi Takeshima at Kyoto University, Japan.

According to Dr. Ma, human cells are continuously injured and naturally repaired through the life span. For instance, micro tears can occur as muscles contract within the body during normal everyday activities. However, diseases such as diabetes, cardiovascular disorders and muscular dystrophy, and even aging, compromise the method in which the body repairs its own tissues, resulting in severe damage. His research

team announced in December 2008 that it had discovered MG53 as a key initiator of membrane repair in damaged tissue, making it the first group to specifically pinpoint a protein responsible for promoting cell repair.

In the new study, the team's research has revealed that MG53 acts first as the initial sensor of damaged tissue during the repair process. Then, through its interaction with Cav3, MG53 recruits intracellular vesicles to the injury site in the membrane, acting as a trafficking agent in the repair process. The vesicles interact with dysferlin to fuse with the membrane, thereby creating a repair patch and allowing for normal membrane function.

"Dysferlin has previously been linked to muscle repair, but our findings show that it can not complete the process when MG53 is absent," said Dr. Ma. "The discovery of MG53 as a necessary element in the repair mechanism provides a foundation in which to study the broader implications of how MG53 fits into the next generation of therapeutic treatments for patients with muscle and cardiovascular disease. We are also looking at its potential to prevent damage from ever occurring."

Source: UMDNJ-Robert Wood Johnson Medical School

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