Columbia Replaces Master's Degree in Physical Therapy with Three-Year Doctorate

By Matthew Doucet

F or more than 20 years, the master of science degree in physical therapy has been the top rung of the ladder for Columbia physical therapy students. But that’s about to change, possibly as early as this fall.

To remain competitive in physical therapy education, Columbia plans to offer a three-year doctor of physical therapy degree, known as a DPT, for the first time. The DPT will replace the two-year master’s degree.

“Some other universities either offer or are about to offer the DPT that Columbia needed to act now,” says Risa Granick, associate professor of clinical physical therapy at the College of Physicians and Surgeons (P&S) and director of the program in physical therapy. The program in physical therapy is part of the Department of Rehabilitation Medicine at P&S.

In his previous work on cardiac physiology, Granick expects to admit an inaugural DPT class of 30 students. Class size will increase to 36 in both 2004 and 2005 and level off at 40 in 2006 and beyond.

The program will use a three-semester calendar—fall, spring, and summer—so the eight-semester sequence can be finished in three years. The program includes a total of 38 weeks of clinical education. During the clinical component, students will have the option of training at New York Presbyterian Hospital or at any of the 350 clinical sites across the country that have contracts with the Columbia physical therapy program.

Students also must complete a capstone research project that meets peer review publication standards. Working with a faculty mentor, each student will select a project related to basic research, a clinical case study, administration or education. Current faculty research efforts include studies related to low back pain in pre- and postpartum women, integration of arm and hand movements in Parkinson’s patients, recovery and quality of life in stroke survivors, and the impact of arthritis on motor skills, physical function, and quality of life in children.

Researchers Reveal that Skeletal Muscle Defect Explains Heart Patient Fatigue

By Susan Conover

P atients with heart failure often don’t have the energy to perform everyday tasks such as getting out of bed, cooking and shopping. But it’s not just the heart’s inability to pump enough adequate amounts of blood and oxygen that causes such severe fatigue. Researchers have suspected that changes in skeletal muscle also contribute to the tiredness and shortness of breath.

Until now, however, no molecular change had been found to explain why skeletal muscle, including the diaphragm, fatigues more quickly in heart failure patients. College of Physicians and Surgeons (P&S) cardiologists have found that a key calcium channel in skeletal muscle contributes to these symptoms.

“For the first time we have a tangible mechanism for skeletal muscle fatigue that new drugs could target,” says the study’s senior author, Dr. Andrew Marks, Wu Professor of Molecular Cardiology, director of the Center for Molecular Cardiology, and chairman of the Department of Physiology and Cellular Biophysics.

The research was published in the March 17 Journal of Cell Biology. One of the intriguing paradoxes of heart failure is that heart function does not always correlate with the severity of fatigue. Some patients whose hearts pump little blood can walk for miles, while other patients whose hearts pump fairly well are unable to walk around the block. The lack of a correlation suggested other mechanisms were at work.

In his previous work on cardiac muscle, Dr. Marks found that during heart failure, changes in a calcium channel, RyR2, cause the heart to weaken. The changes are caused by the chronic stimulation of the sympathetic, or “fight or flight,” nervous system that is characteristic of heart failure. Because a similar channel, RyR1, exists in skeletal muscle, the researchers wanted to see if the changes in the cardiac channel also would weaken skeletal muscle.

The process, they found, is analogous to what occurs in cardiac muscle. Normally RyR1 must be phosphorylated by a neighboring protein to open up channels in times of stress. Phosphorylation causes another protein, FKBP12, to move away from the channel.

Three authors of a paper on heart failure published in the Journal of Cell Biology, from left: Steven Reiken, Ethan Lehnart, and Andrew Marks.

The dissociation of FKBP12 opens the calcium channel, and calcium flows from the sarcoplasmic reticulum into the cell to help contract the muscle. The more calcium that enters, the stronger the contraction.

In heart failure, the calcium channel, which has four phosphorylation sites, becomes covered in phosphates, with three to four per channel instead of the normal zero to one. The resulting hyperphosphorylation causes the calcium channel to leak, so that calcium builds up inside the muscle cell. In the heart, calcium build-up eventually leads to a weaker heart and sometimes sets up a fatal arrhythmia.

But the hyperphosphorylated channel also causes skeletal muscle fatigue? The researchers found the answer is yes. Muscle from rats with heart failure fatigued faster than normal muscle, and the degree of channel phosphorylation correlated with how quickly the muscle fatigued.

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