Researchers reveal protein's role in preventing growth of heart muscle leading to heart failure

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(Medical Xpress)—Cardiovascular disease remains the number one cause of death in the Western world, with heart failure representing the fastest-growing subclass over the past decade. The stage that precedes heart failure in a significant number of cardiovascular diseases is pathological hypertrophy—the growth of the heart muscle in an attempt to increase its output. Not all hypertrophy is pathological; for example, during pregnancy or high physical exertion, the muscle of the heart grows but myocardial function remains normal. But when hypertrophy is excessive, prolonged and unbalanced, it becomes pathological, leading to heart failure and arrhythmias.

Now, for the first time, researchers at the Hebrew University of Jerusalem's Faculty of Medicine have revealed how a protein called Erbin acts as a brake against this excessive and pathological growth of heart muscle. They also demonstrated that damage to this protein leads to excess growth of heart muscle, a decrease in function, and severe pathological growth of heart muscle.

The research was conducted by Ms. Inbal Rachmin as part of her doctoral thesis, under the supervision of Prof. Ehud Razin and Dr. Sagi Tshori at the Institute for Medical Research Israel–Canada in the Faculty of Medicine at the Hebrew University of Jerusalem. The study, "Erbin is a negative modulator of cardiac hypertrophy," was published in the *Proceedings of the National Academy of Sciences* (*PNAS*).

Ms. Rachmin detected a significant decrease in the expression of the protein Erbin in the heart tissue of patients suffering from heart failure. Moreover, the induction of hypertrophy in mice lacking Erbin led to the early death all of these mice, compared to only about 30 percent mortality observed in the control group. Histological examination showed that heart failure was the main reason for this.

This important research also has further implications in the area of breast cancer treatment. Erbin interacts with the receptor Her2/ErBb2, which is overexpressed in approximately 30% of breast cancers. The standard treatment in these cases is the use of Herceptin, an antibody to this receptor. Studies have shown that 5-10 percent of breast cancer patients who received this treatment together with chemotherapy have a significant decrease in heart function. The researchers describe a cardioprotective role for Erbin, which suggests it is a potential target for cardiac gene therapy.

More information: Paper: www.pnas.org/content/111/16/5902.abstract

Provided by Hebrew University of Jerusalem

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