Beat: Miscellaneous

Immunosuppression drug may reverse age-related heart diseases, study finds

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USPA News - Elderly mice suffering from age-related heart disease saw a significant improvement in cardiac function after being treated with an immunosuppression drug during a three-month-long study, indicating that humans with age-related heart dysfunction may be able to improve their condition with appropriate drug treatment. The research, led by a team of scientists at the Buck Institute for Research on Aging in California, showed how the drug rapamycin impacts mammalian tissues, providing functional insights and possible benefits for a drug that has been shown to extend the lifespan of mice by as much as 14 percent.

Rapamycin is an immunosuppressant drug which can be used to help prevent organ rejection after transplantation, but it is also included in treatment regimes for some cancers. In the study, rapamycin was added to the diets of mice that were 24 months old - the human equivalent of 70 to 75 years of age. Similar to humans, the aged mice in the study exhibited enlarged hearts, a general thickening of the heart wall and a reduced efficiency in the heart`s ability to pump blood. The mice were examined with ultrasound echocardiography before and after the three-month treatment period, using metrics closely paralleling those used in humans. Buck Institute faculty Simon Melov, PhD, the senior author of the study, said age-related cardiac dysfunction was either slowed or reversed in the treated mice. "When we measured the efficiency of how the heart pumps blood, the treated mice showed a remarkable improvement from where they started," he said. "In contrast, the untreated mice saw a general decline in pumping efficiency at the end of the same three month period." Adding that the treated mice saw a reduction in heart size, reduced stress signaling in heart tissues and a reduction in inflammation, Melov said the study provides the first evidence that age-related heart dysfunction can be improved even in late life via appropriate drug treatment. Buck researchers, utilizing genome analysis tools, uncovered suites of related genes which rapamycin modulates in the heart. "Rapamycin affected the expression of genes involved in calcium regulation, mitochondrial metabolism, hypertrophy and inflammation," said Melov. "We also carried out behavioral assessments which showed the treated mice spent more time on running wheels than the mice who aged without intervention." The findings are significant for human health as heart disease is the leading cause of death in the United States, claiming nearly 600,.000 lives per year. "Little has been known about the functional ramifications of rapamycin in mammalian tissues," explained Buck Institute President and CEO Brian Kennedy, co-author of the paper. "These findings are significant because we have no interest in simply extending lifespan without an accompanying improvement in the health and quality of life," Kennedy added. "It is particularly encouraging that, in this case, an already-approved drug that extends lifespan also improved function late in life." However, chronic treatment with rapamycin has been problematic in both humans and mice as the drug has the potential to cause deleterious metabolic side effects, including weight gain and glucose insensitivity. Melov said the drug had only mild transient metabolic effects during the study. Researchers at the Mayo Clinic are currently recruiting seniors with cardiac artery disease for a clinical trial involving low dose treatment with rapamycin. Future studies will focus on better understanding the molecular targets that drive age-related heart dysfunction, and why rapamycin treatment is so beneficial to the aging hearts.

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