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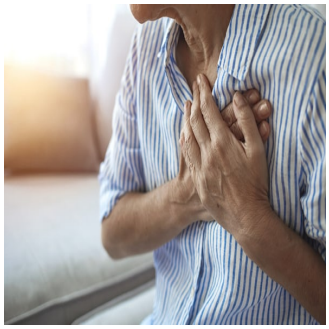
Heart Failure and Multimorbidity Due to Innate Immune Memory



Recent research indicates that heart failure induces a lasting stress memory in the body, potentially contributing to recurrent heart failure and other health issues. This memory involves changes to the DNA modifications of haematopoietic stem cells, which are crucial for producing immune cells like macrophages that protect heart health. During heart failure, a key signalling pathway, transforming growth factor beta (TGF-β), becomes suppressed in these stem cells, impairing macrophage production. Restoring TGF-β levels could offer a promising approach to treating recurrent heart failure, while early detection of stress memory could serve as a pre-emptive warning system.

On a global scale, efforts towards achieving the United Nations' Sustainable Development Goals aim for healthier lives and increased well-being. Recent studies suggest that global life expectancy may rise by approximately 4.5 years by 2050, largely due to advancements in public health and improved survival rates from conditions like cardiovascular diseases. However, heart disease remains the leading cause of death worldwide, affecting an estimated 26 million people through conditions such as heart failure, which often recurs alongside complications in other organs like the kidneys and muscles.

Researchers investigating heart failure recurrence in Japan sought to understand its underlying causes and potential preventive measures. They hypothesised that the stress endured during heart failure might accumulate within haematopoietic stem cells, affecting their function. This stress imprinting was observed in mice models, where epigenetic changes were noted in the DNA of haematopoietic stem cells. Specifically, the suppression of the TGF-β pathway was identified, leading to the production of dysfunctional immune cells, a factor linked to organ damage and recurring heart failure.

This stress memory persisted even after bone marrow transplantation from affected mice to healthy counterparts, demonstrating a prolonged impact on cellular function. Coined as stress memory, this phenomenon underscores the lasting systemic effects of heart failure stress. The discovery opens new avenues for potential therapies aimed at preventing stress memory accumulation during heart failure treatment. Experimental approaches, such as supplementing active TGF-β in animal models, show promise in mitigating these effects and potentially correcting the epigenetic changes in haematopoietic stem cells.

Researchers aim to develop diagnostic tools capable of detecting and preventing stress memory accumulation in humans. This long-term goal not only targets the prevention of recurrent heart failure but also seeks to intervene before the condition progresses. This holistic approach could revolutionise treatment strategies by addressing the underlying mechanisms that perpetuate heart failure and related health complications.

Source: University of Tokyo
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