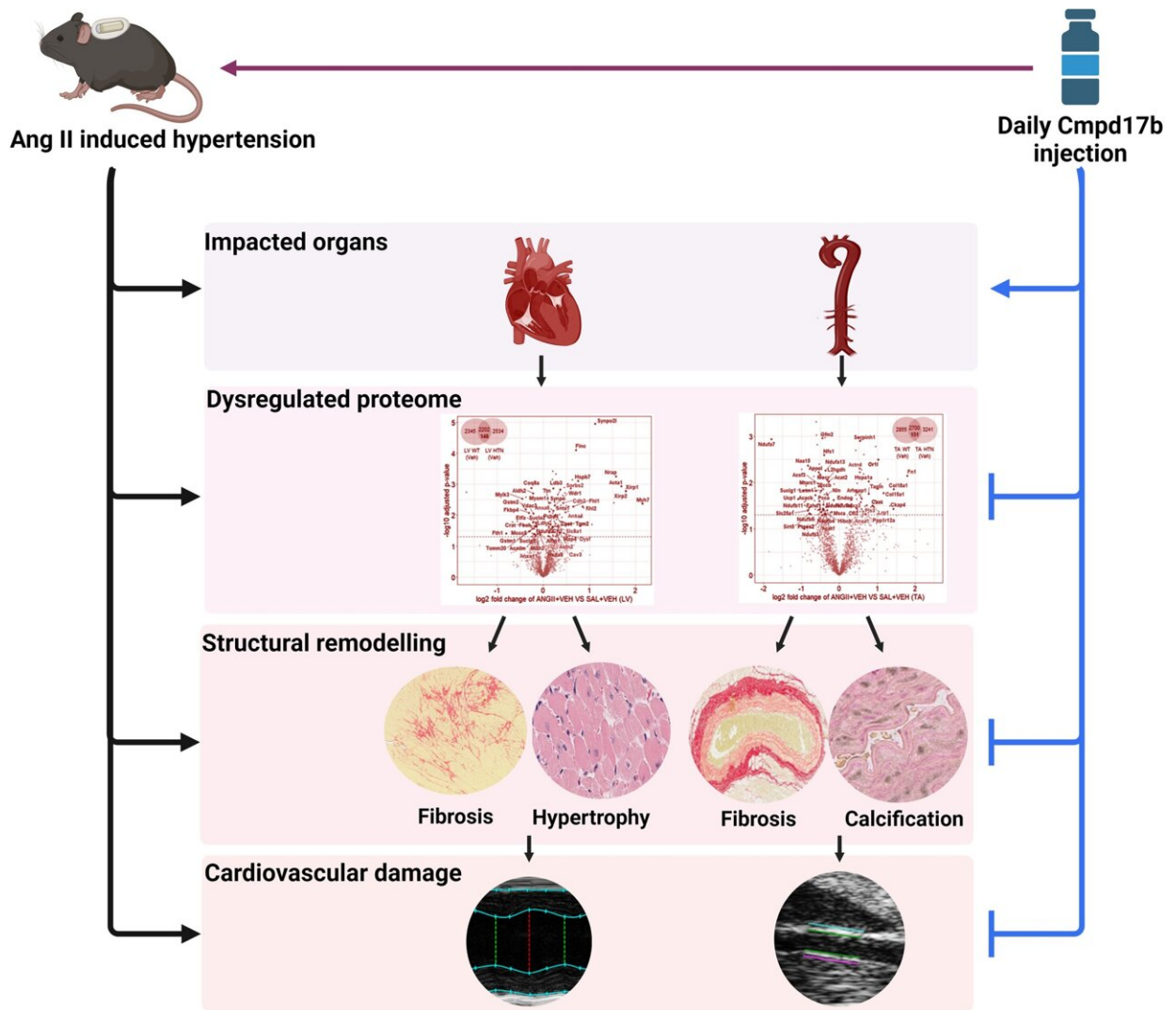


New therapy shows promise in treating hypertension-induced organ damage

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Graphical abstract. Credit: *Cardiovascular Research* (2024). DOI: 10.1093/cvr/cvae103

A team of researchers led by Monash University and the Baker Heart and Diabetes Institute have [published](#) the first compelling evidence of the therapeutic potential of a new therapy to treat hypertension-induced organ damage. The paper appears in the journal *Cardiovascular Research*.

Hypertension, commonly referred to as high blood [pressure](#), may lead to heart, kidney and [blood vessel damage](#). Current treatments often fall short, leaving those affected vulnerable to complications like enlarged hearts and weakened blood vessels. Addressing underlying inflammation is crucial to improving outcomes and reducing associated risks.

Consequently, the team from the Monash Institute of Pharmaceutical Sciences (MIPS) and The Baker Heart Institute set out to investigate how a novel small-molecule pro-resolving activator called 'compound17b' (Cmpd17b)—which MIPS researchers have previously shown to protect against [heart attack](#)—might also protect against hypertension-induced end-organ damage.

Through a comprehensive investigation involving both animal and human studies, the research team has unveiled the potential of Cmpd17b in mitigating the detrimental effects of hypertension-induced organ damage. By activating the formyl peptide receptor (FPR) family, known for its pivotal role in regulating inflammation, Cmpd17b emerges as a potent therapeutic agent capable of safeguarding against the ravages of high blood pressure on vital organs.

Co-first author on the study, and MIPS Ph.D. candidate Jaideep Singh, said the discovery of Cmpd17b as a potential treatment for hypertension-induced end-organ damage has been both encouraging and exciting for the team behind the research.

"Organ damage is a pathological feature of hypertension, responsible for significant morbidity and mortality, however current drugs to treat hypertension are limited when it comes to treating hypertension-induced end-organ damage, so there is absolutely a need to resolve this," said Mr. Singh.

"Our team has shown, for the first time, that not only does Cmpd17b normalize structure and function of heart and blood vessels in hypertensive mice, there is also a clear correlation with human hypertension suggesting Cmpd17b might also be effective in clinical settings."

Professor Geoff Head AM, a senior author on the study and Head of the Neuropharmacology Laboratory at the Baker Institute, said organ impairment as a result of hypertension is prevalent and remains a significant contributor to poor outcomes.

"FPRs are like bodyguards that control inflammation, a big problem in high blood pressure. As a team it's exciting to report that Cmpd17b, which activates these FPRs, could be a promising way to prevent and treat the damage high blood pressure does to our organs in the long run," said Professor Head.

Dr. Chengxue Helena Qin, corresponding author on the study, MIPS lab head and National Heart Foundation Future Fellow said the study revealed significant changes in the proteins and pathways in the hearts and blood vessels of mice with high blood pressure.

"We found that Cmpd17b, a new type of medication, can reverse some of these changes and improve heart and blood vessel health. This suggests that similar treatments might work in people with high blood pressure too," said Dr. Qin.

"Using medications like Cmpd17b could be a promising new approach to treating complications of [high blood pressure](#), potentially reversing damage to organs like the heart and blood vessels. Combining Cmpd17b with existing treatments could provide even better results in managing cardiovascular problems related to hypertension."

More information: Jaideep Singh et al, Novel formylpeptide receptor 1/2 agonist limits hypertension-induced cardiovascular damage, *Cardiovascular Research* (2024). [DOI: 10.1093/cvr/cvae103](https://doi.org/10.1093/cvr/cvae103)

Provided by Monash University

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