We hypothesised that phosphoinositide 3-kinase p110α (PI3K) is critical for exercise-induced protection, as PI3K is activated in cardiomyocytes during exercise and is critical for heart growth following long-term training (athlete's heart).

Methods: Cardiac-specific transgenic mice with elevated or reduced PI3K activity (caPI3K and dnPI3K mice, respectively) and non-transgenic controls underwent four weeks of swim training followed by pressure-overload (ascending aortic banding, $n = 5$–9 per group). Systolic function was assessed by echocardiography one week post-surgery and heart tissue collected for histological and molecular analyses. Aortic banding was also performed in dnPI3K mice overexpressing heat shock protein 70 (Hsp70) to assess the relative importance of PI3K versus Hsp70 in mediating cardioprotection.

Results: Exercise training protected non-transgenic mice from developing heart failure following banding [improved systolic function ($P < 0.001$) and less cardiac hypertrophy ($P < 0.05$), lung congestion ($P < 0.001$) and fibrosis ($P < 0.05$) compared with untrained controls]. Banded caPI3K mice were protected from developing heart failure regardless of whether or not mice were exercised prior to banding (fractional shortening (FS) = 58%, no significant difference vs sham-operated caPI3K controls). In contrast, exercise had no protective effect in banded dnPI3K mice (FS = 30–35%; depressed vs non-transgenic, $P < 0.05$). Transgenic overexpression of Hsp70 did not improve the cardiac phenotype of banded dnPI3K mice.

Conclusion: PI3K is critical for mediating the protective effects of long-term exercise training in a mouse model of pressure-overload. Future studies will use a gene therapy approach to investigate the therapeutic potential of PI3K in heart failure.

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Vascular/Hypertension

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2011 Draft Position Statement on the Use of Ambulatory Blood Pressure Monitoring in Australia: National Heart Foundation (NHF) & High Blood Pressure Research Council (HBPRCA)

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A Novel Framework to Assess Neural and Peripheral Characteristics in Hypertension using Baroreflex Equilibrium Diagram

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Background: Sympathetic nervous hyperactivity as well as the cardiovascular abnormality (arterial atherosclerosis and cardiac hypertrophy) has long been suspected to contribute to the pathogenesis of hypertension. However, quantitative, simultaneous and individual assessment of the neural and the peripheral characteristics responsible for increased arterial pressure has been difficult.

Methods: We investigated the open-loop characteristics of baroreflex function in anesthetised spontaneously hypertensive rats (SHR, $n = 7$) and normotensive Wistar Kyoto rats (WKY, $n = 6$) by isolating the carotid sinus baroreceptor. Carotid sinus pressure (CSP) was controlled at various pressures and the sympathetic nervous activity (SNA) and arterial pressure (AP) responses were recorded simultaneously. The transfer function from CSP to SNA [neural arc], and from SNA to AP [peripheral arc] were described in a baroreflex equilibrium diagram. Peripheral characteristics were additionally estimated by measuring plasma norepinephrine concentrations (NE) before and after autonomic blockade, and by examining AP response to phenylephrine administration.