

A coordinated response to cardiac stress

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Myocardial hypertrophy, a thickening of the heart muscle, is an adaptation that occurs with increased stress on the heart, such as high blood pressure. As the heart muscle expands, it also requires greater blood flow to maintain access to oxygen and nutrients, necessitating an expansion of the cardiac vasculature.

In this issue of the <u>Journal of Clinical Investigation</u>, Daniela Tirziu and researchers at Yale University identified a molecular mechanism by which the growth of new blood vessels (angiogenesis) and <u>heart muscle</u> growth are coordinated.

Using a mouse model of myocardial hypertrophy, Tirziu and colleagues determined that nitric oxide triggers the destruction of a protein known as RGS4.

Nitric oxide typically drives physiological changes associated with the relaxation of blood vessels, while RGS4 attenuates the activity of a cellular signaling pathway that promotes cardiac growth.

These findings reveal how increases in heart muscle and <u>blood vessel</u> <u>growth</u> are coordinated, linking changes in vasculature to changes in heart size.

More information: NO triggers RGS4 degradation to coordinate angiogenesis and cardiomyocyte growth, <u>doi:10.1172/JCI65112</u>



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