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Relationship between oxidative stress and energy metabolism in hypertension induced cardiac remodeling

Renuka R. Nair
Sree Chitra Tirunal Institute for Medical Sciences and Technology, India

Hypertension induced left ventricular hypertrophy is an independent risk factor for cardiac failure. Hence, strategies that promote reverse remodeling forms an important component in the treatment of hypertension. Cardiomyocytes incubated with human serum samples showed a direct correlation of oxidative stress with cellular hypertrophy. *In vitro* experiments using cardiac myocytes and fibroblasts; and studies in spontaneously hypertensive rat (SHR) have established the role of oxidative stress in the initiation and progression of hypertrophy. Hypertrophy is accompanied by a shift in energy substrate preference from fatty acids to glucose. Though initially adaptive, chronic dependence on glucose can be maladaptive. Stimulation of fatty acid oxidation in SHR using the peroxisome proliferator activated receptor- alpha agonist fenofibrate showed an age dependent paradoxical effect. Association between oxidative stress and hypertrophy was apparent, as young SHR (2-month-old) showed regression of hypertrophy and oxidative stress; whereas the converse was observed in older rats (6-month-old). The fatty acid transporter CD36 is defective in hypertensives. Medium chain triglycerides (MCT) bypass CD36. Significant reduction of oxidative stress and hypertrophy was observed in older SHR on supplementation of MCT (5%) in the diet. Blood pressure was maintained, thereby delinking blood pressure reduction from cardiac remodeling. In SHR, an inverse relation was observed between NFκB and MCAD establishing the relation between oxidative stress and metabolic shift and is possibly intimately linked with regulation by PGC-1α. The concept that hypertrophy is an essential compensation for increased wall stress needs reconsideration; and treatment should be directed at prevention of transition from adaptive cardiac remodeling to cardiac failure.

Biography

Renuka R. Nair received her Ph.D. from Osmania University (India) in 1979 and joined as a Postdoctoral Fellow at the Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum, India. She was appointed as a faculty at the Institute in 1984, and is currently the Head of the Division of Cellular and Molecular Cardiology. Her research interest is focused on prevention of adverse cardiac remodeling in chronic pressure overload. She is also working on the role of cardiac stem cells in myocardial regeneration. She is a fellow of the National Academy of Medical Sciences, India.

renuka@sctimst.ac.in