# Sirtuins in the development of cardiac hypertrophy 

Ravi Sundaresan<br>Department of Microbiology and Cell Biology, Indian Institute of Science, Bangalore, India

Correspondence: Ravi Sundaresan, Department of Microbiology and Cell Biology, Indian Institute of Science, Bangalore 5600I2, India, Email: rsundaresan@mcbl.iisc.ernet.in

Received: November 10, 2017 | Published: November 14, 2017
Copyright© 2017 Sundaresan. This is an open access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Lysine acetylation is one of the reversible post-translational modifications linked to the pathogenesis of cardiovascular diseases. Emerging evidence indicates that the activity of cellular histone and non-histone proteins, from prokaryotes to humans, is controlled by lysine acetylation. The acetylation sites are evolutionary conserved. The reaction is catalyzed by enzymes lysine acetyltransferases (KATs) and deacetylases (KDACs). Sirtuins are class III KDACs, which
are homologs of the yeast silencing information regulator 2 (Sir2) that require NAD+ as cofactor. In mammals, seven sirtuin isoforms (SIRT1-7) having a common catalytic core domain, but structurally different N - and C-terminal extensions have been characterized. The role of Sirtuins in the development of cardiac hypertrophy will be discussed further in the talk.

