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Cardiac SERCA2a/b: Overview of the Medication Targets

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Abstract

Calcium (Ca2+) recycling during excitation-contraction coupling is a key mechanism for effective relaxation of the contracted cardiac muscle. Failure of recycling calcium through the sarcoplasmic reticulum (SR) results in severe impairment of myocardial relaxation and consequently contraction on a beat-to-beat basis. The Sarco(Endo)plasmic reticulum Ca+2 ATPase (SERCA) plays an instrumental role in claiming the cytosolic calcium (70%) into the lumen of the SR. To date, three types of SERCA proteins were characterized SERCA1, SERCA2, and SERCA3. SERCA2 is known to have 3 isoforms identified as 2a, 2b, and 2c. While SERCA2b is found ubiquitously expressed, SERCA2a is expressed on slow-switch skeletal muscles and on the cardiac myocytes. SERCA2c has been found recently to be localized in human hearts. The expression and function of SERCA2a/b were altered in heart diseases (i.e. myocardial infarction and hypertrophy). Restoring this alteration is considered to be one of the potential targets in management of cardiac dysfunction. Over the last 12 years, many drugs have shown their effect on both the expression and function of SERCA2a/b. In this review we shed the light on the effect of several drugs adopted in clinical practice that affect the function of SERCA2a/b. They can either modulate the expression or the activity of the pump. This raises an awareness for drugs that antagonize cardiac function.

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