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Swiprosin-how cardiomyocytes can reorganize their sarcomeres and couple remodeling to β-adrenoceptor desensitization

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Cardiomyocytes are terminally differentiated cells in a term that they lose their ability for cell division shortly after birth. However, cardiac remodeling requires intensive reconstruction of contractile units that is still possible in these cells. Cultivation of adult rat ventricular cardiomyocytes (ARVC), terminally differentiated cells, on culture dishes requires remodeling of cells in order to adapt cell shapes to the two-dimensional surface. It is known that ARVC are able to degrade their contractile units (sarcomeres) and reform new sarcomers alongside stress fibres. However, it is not known which molecules trigger this process and whether this process is comparable to remodeling processes *in vivo*. We recognized that swiprosin, a calcium-dependent protein that stabilizes actin filaments and thereby stabilizes stress fibres, is required for the reformation of sarcomeres in ARVC. When swiprosin activation is blocked by verapamil or when swiprosin is downregulated by administration of siRNA directed against swiprosin, ARVC were unable to rebuilt sarcomeres. Moreover, in vivo expression of swiprosin was induced in post-infarct hearts during a phase of intensive cardiac remodeling. Swiprosin expression strongly correlated with GRK2 expression that represses β-adrenoceptor coupling. *In vitro*, silencing of swiprosin reduced GRK2 expression and this improved β-adrenoceptor coupling. A role for increased diastolic calcium levels in cardiac calls has well been described in cardiac remodeling. However, these effects were linked to calcineurin-dependent transcription factor activation and could exlplain only part of the remodeling process. Here we describe another link between diastolic calcium levels and remodeling requiring activation (dimerization) of swiprosin in cardiomyocytes.

Biography

Klaus-Dieter Schlüter, born 09.07.1959, studied biology at the Westfälische-Wilhelms-University, Münster, Germany. He made his PhD at Gesellschaft für Biotechnologische Forschung, Braunschweig, Germany, and spent his Post-Doc education at Heinrich-Heine-University Düsseldorf, Germany. In 2002 he became professor for physiology at Justus-Liebig-University in Giessen, Germany. Research areas are biology of cardiomyocytes, hypertensive heart disease, and ischemia/reperfusion. He published over 100 original articles and several review articles, editorials and book chapters. Current activities include editing of a book entitled "Cardiomyocytes – Active players in cardiac disease". He is member of the German Society of Cardiology and of the German Physiological Society.

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