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Poster Sessions

Monensin-induced Ca^{2+} overload suppresses mitochondrial ATP production in cardiac myocytes

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[Background] Monensin (Mo) has been reported to decrease the ATP content in various cells, however, its mechanisms are not fully understood in cardiac myocytes. In this study, the effects of Mo on ATP production was examined in guinea-pig ventricular myocytes. [Methods] The ATP production was evaluated from the time taken to open sarcolemmal K_{ATP} channel. Membrane potential and Ca^{2+} concentration of mitochondria (mito) was measured. [Results] When the extracellular solution containing 112 mM Na_o and the intracellular solution containing 10 mM Na_i and 0 mM ATP were used, Mo 10^{-5} M shortened time taken to open the K_{ATP} channel significantly. When 0 mM Na_o and 10 mM Na_i were used, Mo also shortened time. Next, when 0 mM Na_o and 0 mM Na_i were used, Mo shortened the time to a lesser extent. When EGTA 5 mM was replaced by BAPTA 40 mM in the patch pipette, the time taken to open the K_{ATP} channel was not shortened so much by Mo. Mo increased mito Ca^{2+} and depolarized the membrane potential in saponin-treated myocytes. [Conclusion] We conclude that the Mo-induced Na^+ influx into mito alters $\text{Na}^+/\text{Ca}^{2+}$ exchange function, and the mito membrane depolarization may cause Ca^{2+} influx to mito matrix, leading to suppression of ATP production.