Characterization of Contractile Activity and Intracellular Ca^{2+} Signalling in Mouse Myometrium

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OBJECTIVE: *To characterize the contractile responses of mouse myometrium, the associated calcium (Ca^{2+}) changes and the role of the sarcoplasmic reticulum (SR), and to better understand excitation contraction coupling in this tissue.*

METHODS: *Strips of longitudinal myometrium were used, and Ca^{2+} was measured after loading with Indo-1.*

RESULTS: *Intracellular Ca^{2+} transients, produced by Ca^{2+} entry, preceded phasic spontaneous contractions. Depolarization with high potassium concentration significantly increased the amplitude of the contractions and transformed the pattern of activity from phasic to tonic, with accompanying changes in intracellular Ca^{2+} concentration ($[Ca^{2+}]_i$). Oxytocin significantly stimulated contractile activity and $[Ca^{2+}]_i$ above the level occurring spontaneously. Thus all forms of contractile activity were closely correlated with Ca^{2+}. When the SR was emptied using a blocker of the SR calcium-adenosinetriphosphatase, cyclopiazonic acid, spontaneous Ca^{2+} and force transients increased greatly in frequency and amplitude. Ryanodine, a blocker of Ca^{2+}-induced Ca^{2+} release (CICR), did not impair activity. In the absence of external Ca^{2+}, oxytocin was able to release Ca^{2+} from the SR through IP_3 but produced only a small increase in force, demonstrating a requirement for Ca^{2+} entry as part of the mechanism of agonist action.*

CONCLUSION: *Mouse myometrium, (1) produces contractile activity reflecting changes in $[Ca^{2+}]_i$ irrespective of the stimulus, (2) has a significant SR Ca^{2+} content releasable by agonists but not CICR, (3) has an SR acting to inhibit spontaneous activity, and (4) behaves qualitatively similarly to human and rat myometrium in major aspects of excitation contraction coupling and is therefore a useful model tissue.*
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KEY WORDS: Smooth muscle, SR Ca^{2+} release, agonists, sarcoplasmic reticulum.