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EFFECTS OF K^+ ON Ca^{2+} INFLUX AFTER DEPLETION OF INTRACELLULAR Ca^{2+} STORES IN ILEAL SMOOTH MUSCLE OF THE RAT

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1. The present experiment was performed to examine the effects of K^+ on a rise in intracellular Ca^{2+} level ($[Ca^{2+}]_i$) and a contraction induced by application of Ca^{2+} when intracellular Ca^{2+} stores were transiently or continuously depleted in fura-2-loaded longitudinal smooth muscle strips of the rat ileum.

2. After transient depletion of Ca^{2+} stores with carbachol (0.1 mM), application of Ca^{2+} caused a rise in $[Ca^{2+}]_i$ and a contraction, both of which were increased with increasing K^+ concentrations ($[K^+]_0$) applied simultaneously. The effect of K^+ was inhibited by methoxyverapamil (10 μM) and potentiated by Li^+ (10 mM).

3. After application of Ca^{2+} together with various concentrations of K^+ under Ca^{2+}-free conditions, carbachol caused a rise in $[Ca^{2+}]_i$ and a contraction, both of which increased ($[K^+]_0 < 40$ mM) or decreased (>40 mM) depending on the K^+ concentration used. The increasing effect of K^+ was inhibited by methoxyverapamil.

4. The intracellular Ca^{2+} stores were continuously depleted by pretreatment with thapsigargin or ryanodine. Under these conditions, the rise in $[Ca^{2+}]_i$ and contraction induced by application of Ca^{2+} were dose-dependently and markedly increased even in the presence of methoxyverapamil or nifedipine.

5. The rise in $[Ca^{2+}]_i$ and contraction induced by Ca^{2+} after treatment with thapsigargin or ryanodine were inhibited by the replacement of Na^+ with K^+. The inhibition by K^+ was attenuated by increasing the extracellular Ca^{2+} concentration.

6. The influx of Mn^{2+} was much greater in tissues pretreated with thapsigargin or ryanodine than in intact tissues. The enhanced Mn^{2+} influx was inhibited by the replacement of Na^+ with K^+.

7. These results suggest that the Ca^{2+} are replenished by Ca^{2+} stores passing through a voltage-dependent Ca^{2+} channel and another unknown pathway, when the intracellular Ca^{2+} stores are transiently depleted by an agonist in ileal smooth muscle of the rat. On the other hand, Ca^{2+} entry through another unknown pathway is markedly increased by continuous depletion of Ca^{2+} stores with thapsigargin or ryanodine, which is inhibited by increasing $[K^+]_0$. The inhibitory effect of K^+ on Ca^{2+} entry might be due to the reduction of the electrochemical gradient for Ca^{2+} across the plasma membrane.