The voltage-current relationship of this current resembled that of T-type Ca^{2+} current but not R-type Ca^{2+} current. However, the rapid inactivation, one of the properties of T-type Ca^{2+} current, was not observed in the present experiment.

6. This residual current, insensitive to Ca^{2+} channel blockers, was inhibited by low concentrations of Ni^{2+} (0.01–0.1 mM), and was not suppressed by ATP (500 μM).

7. Catecholamine release induced by 60 mM KCl was significantly inhibited by nifedipine (3 μM), ω-conotoxin MVIIC (3 μM) and mixture of four blockers, but neither ω-conotoxin GVIA (1 μM) nor ω-agatoxin IVA (0.1 μM).

8. These results suggest that guinea pig adrenal chromaffin cells possess L- and N- and P- and Q-type Ca^{2+} channels and other subtypes of Ca^{2+} channel insensitive to these blockers, and that L- and Q-type channels and the unidentified Ca^{2+} channels mainly contribute to the catecholamine release by stimulation with high K^+. Some properties of unidentified Ca^{2+} channel currents were different from those of both T-type and R-type Ca^{2+} channel currents. As we can not identify the subtype of this Ca^{2+} channel, further studies are required to identify these Ca^{2+} channels.

MECHANISMS OF CATECHOLAMINE SECRETION BY Ca^{2+}-REINTRODUCTION AND Na^+ REMOVAL IN ADRENAL CHROMAFFIN CELLS OF THE GUINEA-PIG

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1. We investigated the mechanisms of increases in catecholamine (CA) secretion evoked by reintroduction of Ca^{2+} after removal of extracellular Ca^{2+} (Ca^{2+}-reintroduction) and removal of extracellular Na^+ in chromaffin cells of the guinea-pig.

2. CA secretion was increased with increasing the concentration of Ca^{2+} (0.1–10 mM) reintroduced. This secretory response was not affected by 1 μM atropine, 100 μM hexamethonium and 1 μM tetrodotoxin.

3. In the presence of 1 mM $CoCl_2$ or 1 mM $MgCl_2$, Ca^{2+}-reintroduction failed to increase CA secretion. The secretory response to Ca^{2+}-reintroduction was significantly inhibited by 10 μM methoxyverapamil (D600) and 1 μM nifedipine, but not by 1 μM ω-conotoxin GVIA and 0.1 μM ω-agatoxin IVA, and was greatly potentiated by 1 μM Bay K 8644.

4. Ca^{2+}-reintroduction caused an increase in the intracellular free Ca^{2+} concentration ($[Ca^{2+}]_i$) which was inhibited by D600 in isolated chromaffin cells loaded with fura-2.

5. CA secretion evoked by high K^+ (56 mM) which directly depolarizes the cell membranes was also inhibited only by nifedipine, and was greatly potentiated by Bay K 8644. However, the rate of inhibition in CA secretion evoked by high K^+ was much smaller than that by Ca^{2+}-reintroduction.

6. CA secretion was evoked by the removal of extracellular Na^+ in the absence of Ca^{2+} but not in its presence. This secretory responce was also significantly inhibited by D600, and was

greatly potentiated by Bay K 8644.

7. These results suggest that CA secretion evoked by Ca^{2+}-reintroduction, high K^+ and the removal of extracellular Na^+ and Ca^{2+} is mainly mediated by Ca^{2+} entered through L-type voltage dependent Ca^{2+} channels in adrenal chromaffin cells.

EFFECTS OF TYROSINE KINASE INHIBITORS AND NON-SELECTIVE CATION CHANNEL BLOCKERS ON CAPACITATIVE Ca^{2+} ENTRY IN RAT ILEAL SMOOTH MUSCLES

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1. The present experiment was performed to examine the involvement of tyrosine kinase and non-selective cation channels in capacitative Ca^{2+} entry (CCE) in the rat ileal smooth muscles. The effects of tyrosine kinase inhibitors (genistein and tyrphostin 47), an inactive analogue of genistein (daidzein), non-selective cation channel blocker (SK & F96365) and Ca^{2+} entry blocker (tetrandrine) were examined in the presence of methoxyverapamil.

2. In the presence of external Ca^{2+}, carbachol-induced sustained contractions were dose-dependently inhibited by genistein, daidzein, tyrphostin 47, SK & F96365 and tetrandrine.

3. Under Ca^{2+}-free conditions, after the depletion of stored Ca^{2+} by carbachol or caffeine, the application of Ca^{2+} evoked transient contractions due to CCE. These contractions were inhibited by genistein, daidzein, tyrphostin 47, SK & F96365 and tetrandrine. The inhibitory potency of genistein was greater than that of daidzein. The application of Ca^{2+} evoked sustained contractions due to CCE after the depletion of stored Ca^{2+} with the treatment of thapsigargin. These five drugs also inhibited the contraction, but the potency of daidzein was greater than that of genistein.

4. SK & F96365 produced no inhibitory effects on the carbachol- and caffeine-induced contractions due to Ca^{2+} released from Ca^{2+} store. Genistein, daidzein and tyrphostin 47 inhibited these contractions. However, these drugs were less effective in inhibiting the contraction evoked by Ca^{2+} release than that by CCE. Tetrandrine inhibited contraction induced by carbachol but not caffeine.

5. Genistein slightly suppressed Ca^{2+}-induced contractions in β-escine treated skinned fibers.

6. These results suggest that CCE induced by carbachol and caffeine may be mediated by tyrosine kinase and this pathway is sensitive to SK & F96365 and tetrandrine. However, the inhibitory effects on CCE were produced by not only genistein but also daidzein, indicating that further studies are nesessary to evaluate this hypothesis.