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Author(s)	NAKAMURA, Minako
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EFFECTS OF GAMMA AMINOBUTYRIC ACID
ON SPONTANEOUS Ca^{2+} OSCILLATIONS
IN ISOLATED PREPARATIONS OF RAT CHROMAFFIN CELLS

Minako NAKAMURA

*Department of Physiology,
Faculty of Veterinary Medicine,
Hokkaido University, Sapporo 060, Japan*

1. Spontaneous $[\text{Ca}^{2+}]_c$ oscillations have been reported in the majority of chromaffin cells and were reconfirmed in the present study by a microfluorometric method with the Ca^{2+} -sensitive dye, fura-2, in about 60% of the rat adrenal chromaffin cells. The oscillations were promptly and reversibly arrested by application of gamma aminobutyric acid (GABA; 50 μM -1mM) and, in the majority of the preparations used, the arrest was preceded by a transient and rapid rise in $[\text{Ca}^{2+}]_c$.

2. The oscillations were also arrested rapidly and reversibly by the GABA_A agonist muscimol, but not affected by the GABA_B agonist baclofen. These results indicate that the inhibitory effect of GABA may be mediated by GABA_A receptors. The inhibitory effect of GABA could be due to a depolarization-induced activation and potential-dependent-inactivation of Ca^{2+} channels, as shown by the following results.

3. Oscillations were not observed in about 40% of the cells examined. In these quiescent cells, $[\text{Ca}^{2+}]_c$ remained stable at around 100nM, and a large spike-like rise in $[\text{Ca}^{2+}]_c$ was evoked by application of GABA (100 μM). The GABA-induced $[\text{Ca}^{2+}]_c$ rise was enhanced when $[\text{Cl}^-]_o$ was lowered, suggesting that GABA caused depolarization as a consequence of an increase in Cl^- efflux.

4. The oscillations were rapidly arrested when extracellular Ca^{2+} was chelated by EDTA or EGTA, and when Ni^{2+} , a non-selective Ca^{2+} channel blocker, was applied, indicating that extracellular Ca^{2+} entry is importantly involved in the oscillatory $[\text{Ca}^{2+}]_c$ rises.

5. Taking the present results and reported results into consideration, we now propose the following mechanism regulating the spontaneous $[\text{Ca}^{2+}]_c$ oscillations in rat chromaffin cells; 1) the oscillations may correlate with spontaneous fluctuations in membrane potential, and depolarizing phases of the fluctuations may coincide with oscillatory Ca^{2+} entry through voltage-dependent Ca^{2+} channels, although the possible involvement of an intracellular mechanism of Ca^{2+} release cannot be excluded, and 2) GABA may open the Cl^- channels to cause depolarization followed by a plateau phase, which may be responsible for a transient and rapid rise followed by gradual decay in $[\text{Ca}^{2+}]_c$.