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ABSTRACTS OF MEMOIRS

RECORDING WORK DONE AT THE PLYMOUTH LABORATORY

BAKER, P. F., MEVES, H. & RIDGWAY, E. B., 1973. Effects of manganese and other agents on the calcium uptake that follows depolarization of squid axons. *Journal of Physiology, London*, **231**, 511–26.

The Ca-sensitive photoprotein aequorin was injected into squid axons and the light response to stimulation or depolarizing voltage clamp pulses recorded.

The effects of Mn^{2+}, Co^{2+}, Ni^{2+}, La^{3+} and of the organic Ca antagonists D-600 and iproveratril on the early tetrodotoxin-sensitive and late tetrodotoxin-insensitive components of the light response were studied.

The late tetrodotoxin-insensitive component can be blocked, reversibly, by concentrations of Mn, Co and Ni that reduce but do not block the tetrodotoxin-sensitive component. The late component can also be blocked by La^{3+} and the organic Ca antagonists D-600 and iproveratril.

Mn^{2+}, Co^{2+}, Ni^{2+} and the drug D-600 all reduce the Na currents, but have little effect on either outward or inward K currents. Tetraethyl-ammonium blocks the outward K current but has no appreciable effect on the tetrodotoxin-insensitive entry of Ca.

Concentrations of Mn between 5 and 50 mM substantially reduce the light output during a train of action potentials; they also slightly reduce the rate of rise of the action potential.

On pharmacological grounds it is concluded that the tetrodotoxin-insensitive component of Ca entry does not represent Ca ions passing through the K permeability channels. There must exist a potential-dependent late Ca channel that is distinct from the well-known Na and K channels of the action potential. A possible function for this late Ca channel in the coupling of excitation to secretion is discussed.

BAKER, P. F., MEVES, H. & RIDGWAY, E. B., 1973. Calcium entry in response to maintained depolarization of squid axons. *Journal of Physiology, London*, **231**, 527–48.

Intracellular aequorin was used to monitor changes in Ca entry in response to maintained depolarization either produced electrically or by exposure to K-rich solutions.

External K concentrations greater than 50 mM produce a phasic light response. The light rises to a peak in a few sec and then falls in 0·5–5 min to a new steady level that is always greater than the level in the absence of K.

The phasic light response does not result from depletion of available aequorin at the periphery of the axon, but rather seems to reflect a phasic entry of Ca in response to depolarization.

Similar phasic responses are produced by prolonged electrical depolarization. These results are consistent with depolarization serving both to activate and also to inactivate Ca entry.

Following inactivation and after return to normal sea water, there is an appreciable relative refractory period during which the response both to K-rich sea water and electrical depolarization is reduced in size. Complete recovery takes 10–15 min.

The response to 410 mM-KCl is dependent on the previous treatment of the preparation. Pre-treatment with 100 or 200 mM-KCl reduced the response to 410 mM-KCl. The potential for half inactivation was about -25 mV in 112 mM-Ca and -40 mV in 20 mM-Ca.

The rate of onset of inactivation is potential dependent and is faster for depolarizations to zero potential than for smaller ones.

The phasic Ca entry produced by K-rich solutions is insensitive to external tetrodotoxin and internal tetraethylammonium ions, but is blocked by external Mn^{2+}, Co^{2+} and Ni^{2+} ions and by the drugs D-600 and iproveratril. This suggests that the phasic Ca entry involves the late Ca channel.