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LETTER TO THE EDITOR

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The role of muscle and glial cells in potassium homeostasis

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There has not to date been any clearly assigned role for the β -adrenoceptors of the plasma membrane of skeletal muscles. It is known that adrenoceptors stimulate Na^+ , K^+ -ATPase in skeletal muscle (Cheng et al 1977). Recent experiments have shown that stimulation of the Na^+ - K^+ transport system (Na^+ -pumping) across muscle-cell membranes by a β -adrenoceptor-mediated mechanism contributes to the regulation of membrane potential and the distribution of Na-K across the sarcolemma (Clausen & Flatman 1977; Edstrom & Phillis 1981). β -adrenoceptor blockade can both potentiate the potassium rise in plasma during exercise and delay the reversion of plasma potassium to normal values (Carlsson et al. 1978) and we would like to suggest that muscle tissue plays a major role in the control of plasma potassium concentrations under normal physiological conditions. Catecholamines released by sympathetic nerves and the adrenal medulla would stimulate the muscle membrane sodium-potassium pump and thus accelerate the return of plasma potassium concentrations to normal following periods of muscular activity. Consistent with this suggestion is the observation that there is prolonged decrease in plasma potassium (hypokalaemia) after intravenous administration of adrenaline, which has been attributed to an increased uptake of potassium by muscle (Ellis 1956).

There is an adrenoceptor-mediated activation of Na^+ - K^+ transport systems in glial cells of the central nervous system (c.n.s.) (Narumi et al 1979). It is known

that glial cells are important for the maintenance of the homeostasis of extracellular electrolytes in the c.n.s. (Hertz 1977). By controlling extracellular potassium concentrations in the brain, glial cells would also influence the excitability of central neurons (Phillis 1979), since any decrease in extracellular potassium levels would lead to a subsequent hyperpolarization and thus inhibition of adjacent neurons. In this context it is interesting to note that adrenaline causes a decrease in extracellular levels of potassium in the brain (Pelligrino & Siesjo 1980) presumably as a result of enhanced sodium-potassium transport.

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