significantly inhibit low affinity uptake. High affinity uptake of [^{35}S]-L-homocysteate (0.1 μM) was not significantly inhibited by 0.1 mM D- or L-glutamate, D- or L-aspartate or D-homocysteate, but was inhibited 70% by 0.1 mM *p*-chloromercuriphenylsulphonate. At 0°C, low affinity uptake as reduced by more than 90% and high affinity uptake was abolished. No metabolism of [^{35}S]-L-homocysteate (0.1 mM) was detected (37°C/10 min).

These results provide a possible explanation for the different characteristics of neuronal excitation produced by D- and L-homocysteate. The functional significance of the energy-dependent uptake systems for L-homocysteate, the high affinity component of which appears to possess a high degree of stereospecificity, remains to be investigated.

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The response of the rat anococcygeus muscle to stimulation of the individual extrinsic nerves and its modification by drugs

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Two extrinsic nerves supply the rat anococcygeus (Gillespie & Lullman-Rauch, 1974). These nerves subdivide and supply a nerve plexus comprising ganglia and interconnecting tracts. In this study the response of the muscle to stimulation of each of the two extrinsic nerves has been compared with that to stimulation of the combined extrinsic nerve supply and that to field stimulation (Gillespie, 1972).

Each nerve (one a branch of the external spermatic division of the genito-femoral nerve and the other a branch of the posterior scrotal division of the perineal nerve) was stimulated individually (1–100 Hz for 5 s, 0.3 ms, supramaximal) with suction electrodes positioned 0.5–1 cm from the muscle which was perfused with a modified saline solution (Creed, Gillespie & Muir, 1975).

A contraction (latency 0.5–2.5 s) was produced on stimulation of either nerve (optimal frequency about 50 Hz); the contraction following stimulation of the external spermatic branch was usually the larger. No contraction was produced on stimulation of either nerve with a single stimulus or indeed below 2 Hz. The excitatory response was abolished by tetrodotoxin (TTx, 1 μg/ml) or by phentolamine mesylate (Ph) (10 μg/ml) confirming earlier suggestions (Gillespie, 1972) that it was mediated via an adrenergic nerve pathway. The excitatory response was reduced by the ganglion blocking agents, tubocurarine chloride (Tc, 0.1 mg/ml) or hexamethonium bromide (C_6, 0.5 mg/ml).

When the tone of the muscle was raised (e.g. following carbachol chloride (2 μg/ml) with or without subsequent Ph (10 μg/ml), or following guanethidine sulphate (9 μg/ml) or in a few instances in the absence of any drug) stimulation produced an inhibitory response in about half the experiments. The most obvious explanation for the absence of inhibitory responses in some muscles is that the fibres were missed or damaged in the dissection. When present, the inhibitory responses resulted from stimulation of either or both extrinsic nerves. The inhibitory response which had a variable latency (1.5–5 s) was abolished by TTx (1 μg/ml). The inhibitory response (optimal at about 10 Hz) was not reduced and indeed in some cases was increased in amplitude and/or prolonged by either C_6, or Tc (up to 1 mg/ml) or atropine sulphate (up to 1 μg/ml).

The present evidence, in contrast to previous views (Gillespie, 1972) suggests that synaptic interruption occurs in the excitatory pathway—probably in the ganglionic plexus lying close to the muscle. Two puzzling features of the inhibitory response require explanation—its long latency and the effects of acetylcholine antagonists. The organization of the inhibitory pathway remains obscure.

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