Specific stimulation of [^{3}H]-dopamine release from dendrites of rat substantia nigra by glycine

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It has been suggested that glycine may be a transmitter in the substantia nigra. Unilateral intra-nigral injection of glycine produces turning behaviour in rats (Mendez, Finn & Dahl, 1976), and inhibits dopamine release from the ipsilateral caudate nucleus in cats (Cheramy, Nieullon & Glowinski, 1978). To study possible presynaptic actions of glycine in the nigra, we have studied the effect of this amino acid on the efflux of [^{3}H]-dopamine ([^{3}H]-DA), [^{3}H]-5-hydroxytryptamine ([^{3}H]-5HT) and [^{3}H]-γ-aminobutyric acid ([^{3}H]-GABA) from prelabelled, superfused slices of rat substantia nigra. The methods used have been described in detail elsewhere (Srnivisan, Neal & Mitchell, 1969). Amino-oxyacetic acid (10 μM) and pargyline (50 μM) were present to inhibit labelled transmitter metabolism where appropriate.

A depolarizing stimulus (50 mM KCl) increased the rate of efflux of [^{3}H]-5HT, [^{3}H]-DA and [^{3}H]-GABA. In all cases this effect of K^+ was markedly reduced in a low calcium, high magnesium medium. Glycine (50 & 100 μM) stimulated the efflux of [^{3}H]-DA and this effect was abolished when strychnine hydrochloride (10 μM) was included in the superfusate. The efflux of [^{3}H]-5HT and [^{3}H]-GABA was unaffected by glycine (100 μM). Furthermore, taurine, GABA and β-alanine (100 μM) had no effect on the efflux of [^{3}H]-DA. Stimulated [^{3}H]-DA release from substantia nigra is presumed to be dendritic (Geffen Jessell, Cuello & Iversen, 1976) and the observed reduction of striatal dopamine release seen by Cheramy *et al* (1978) after intra-nigral glycine may therefore, be a result of stimulation of presynaptic dopamine receptors, reducing activity in the ascending nigrostriatal dopaminergic pathway (Maggi, Bruno, Cattabeni, Grobetti, Parenti & Racagni, 1978).

In additional studies, slices of rat substantia nigra were incubated for 5 min with [^{3}H]-glycine (0.2–2.0 μM) and radioactivity was accumulated with an apparent Km of 2.44 μM, indicating a transmitter-specific high affinity uptake (Balcar & Johnston, 1973). However, 50 mM K^+ had no effect on the efflux of [^{3}H]-glycine from prelabelled substantia nigra. This was in contrast to 3 day old rat spinal cord where K^+ stimulated the release of [^{3}H]-glycine in a calcium-dependent fashion. This suggests that glycine may be released only from a very small number of terminals in the nigra, whereas more diffuse elements such as glia in this region may take part in its inactivation.

RK is an MRC student.

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