saline control injections as well as with (+)-amphetamine (0.25 mg/kg) which does not increase checking. Control and 0.25 mg/kg treated animals spent about 25/50 s at windows when they were uncovered but less than 5/50 s when covered. But 10 min after injection with (+)-amphetamine (4 mg/kg), the time spent at uncovered windows fell to less than 10/50 s ($P < 0.01$, 2-tailed matched pair t test, compared with controls). This indicates a decrease in the influence of the external environment during amphetamine induced checking.

In contrast, the time spent at uncovered windows was not decreased by apomorphine (0.25 mg/kg) which does stimulate checking, when compared with 0.063 mg/kg apomorphine, which does not, or to controls. However, the time spent at covered windows was increased slightly by apomorphine ($P < 0.05$, 0.063 mg/kg; $P < 0.01$, 0.25 mg/kg). This probably reflects increased locomotion around the perimeter of the drum.

Thus it would appear that the persistent checking induced by amphetamine is not visually determined and is, in fact, incompatible with visually determined behaviour, whereas the moderate checking and the increase in locomotion induced by apomorphine does not prevent the external visual control of behaviour.

Inter-relationships between behavioural and neurochemical indices of supersensitivity in dopaminergic neurones

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There is considerable biochemical and behavioural evidence to suggest that supersensitivity of dopamine (DA) receptors develops in denervated rat striata following lesions of the nigrostriatal pathway (Ungerstedt, 1971). However, such lesions have not resulted in systematic changes in the activity of DA stimulated adenylate cyclase in striatal homogenates (Iversen, 1977). We have attempted therefore to inter-relate the rotational response to apomorphine with DA depletion, receptor sensitivity and DA stimulated adenylate cyclase activity in rat striatal homogenates after unilateral 6-hydroxydopamine lesions of the nigrostriatal pathway.

Rats were lesioned unilaterally in the medial forebrain bundle with a range of 6-hydroxydopamine concentrations (1–9 μg in 4 μl saline containing ascorbate, 1 mg/ml) and tested 14 days later in an automated rotameter for rotational response to 0.05 mg/kg of subcutaneously administered apomorphine (Waddington & Crow, 1978). Mean contralateral rotation was 298 turns/hour. Twenty-one days post-lesion rats were stunned and decapitated and striata stored at −40°C for subsequent assays. Mean DA depletion (assayed by the method of Coyle & Henry, 1973) was 83% ($n = 19$) in denervated striata. There was a significant correlation ($r = 0.68$; $P < 0.01$) between the degree of DA depletion and apomorphine-induced turning with a strong suggestion that DA depletions below 60% did not result in a rotational response. DA receptor sensitivity, assessed by [^{3}H]-spiperone binding (Reisine *et al.*, 1977) was elevated by 42% (range −28% to 186%; $P < 0.01$) in striatal preparations after nigrostriatal lesions with a significant correlation ($r = 0.58$; $P < 0.05$) between increases in spiperone binding and apomorphine induced turning with the regression line intersecting the axes near the origin. After 6-hydroxydopamine lesions adenylate cyclase activity was significantly increased ($P < 0.05$) with stimulation by DA (100 μM) but was unchanged in the presence of DA (10 μM) or under basal conditions. There was no significant correlation between adenylate cyclase activity and DA depletion, spiperone binding or rotational response to apomorphine.

These results suggest that in the rat behavioural manifestations of DA supersensitivity may not be exhibited below 60% DA depletion in denervated striata. However, as the regression line relating spiperone binding to rotational behaviour passes through the origin, the binding index of DA receptor supersensitivity may be directly proportional to the behavioural index of supersensitivity.

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