ally from direct or indirect stimulation of indoleamine receptors (Jacobs, 1976), were each rated, on a blind basis, with a score of 0, 1, or 2.

The rated response to fenfluramine was significantly ($P<0.01$) increased in the lithium pretreated rats. By contrast, the response to 5-HTP was only significantly ($P<0.05$) greater than that of controls in LL rats (Figure 1). Although the enhanced response to 5MeODMT in LL rats was not significant, a further study in groups of 9–10 rats, with doses of 0.75–2.00 mg/kg, demonstrated a significantly ($P<0.05$) increased overall response to 5MeODMT in LL rats (Kolmogorov-Smirnov two-sample test, 2-tailed, $D=0.29$, n_1, $n_2=46$).

The greater behavioural response to fenfluramine in SL and LL rats may reflect an increase in the amount of extra-granular 5-HT released by the drug, due to lithium impaired intra-neuronal storage. The enhanced behavioural response to 5-HTP and 5MeODMT in LL, but not SL rats, supports the suggestion that indoleamine receptors become supersensitive as a result of continued reduction of normal 5-HT release by lithium.

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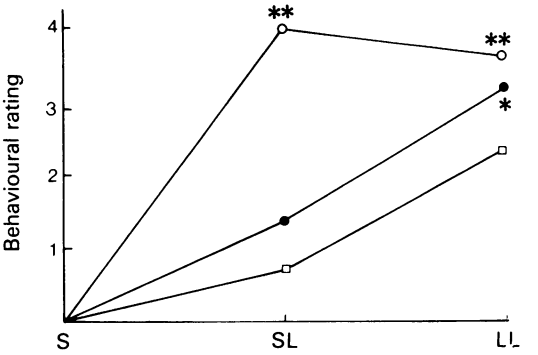

Figure 1 Ratings for postural tremor, fore-paw treading, hind-limb splay, Straub tail, head-weaving, and hypersalivation and/or ejaculation, were summed for each rat. Mean ratings of SL and LL rats are expressed as differences from mean ratings of S rats. There were significant differences between ratings after (○) fenfluramine and (●) 5-HTP, (Kruskall-Wallis one-way analysis of variance, $P<0.01$ and $P<0.05$ respectively). LL rats had higher scores than controls after fenfluramine and 5-HTP, as did SL rats after fenfluramine, (Mann-Whitney U test, 2-tailed, $*P<0.05$; $**P<0.01$). Lithium pretreatment did not significantly affect scores after (□) 5MeODMT, although the trend was similar to that after 5-HTP.

Exploratory behaviour and aversive thresholds in rats following microinjection of morphine into central and medial nuclei of the amygdala

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The amygdaloid complex, an area rich in opiate receptors (Atweh & Kuhar, 1977) and opioid peptides (Watson, Akil, Sullivan & Barchas, 1977), has recently been implicated in morphine-induced alterations in aversive thresholds and open field behaviour (Rodgers, 1977, 1978). In this experiment we extend investigations to the effects of intra-amygdaloid opiate injections on exploratory behaviour.

Male hooded rats were anaesthetized with Equithesin and bilaterally implanted with guide cannulae aimed at the central (A/P: + 6.0, L: ± 3.9, V: 8.5) or medial (A/P: + 5.2, L: ± 3.5, V: 9.5) nucleus of the amygdala. Two weeks post-operative recovery was allowed before testing commenced. Rats were randomly assigned to flinch-jump and holeboard tests and then randomly allocated to vehicle (sterile water), morphine sulphate (10 μg), naloxone hydrochloride (1 μg) or morphine plus naloxone (10 μg + 1 μg) groups. In all cases injections were made bilaterally in a volume of 0.5 μl over 20 s. Cannula placements were verified histologically after trypan blue injection and data from animals with placement errors were excluded.

In the flinch-jump test, electric shock was applied through the grid bars of the test chamber (18 × 15 × 13 cm) as previously described (Rodgers, 1978). Mean jump thresholds were determined for each rat before and after injection. At both sites,

morphine significantly elevated response thresholds ($P<0.01$ for central; $P<0.001$ for medial, related t-tests). In medial, but not central, sites naloxone antagonized the morphine effect; naloxone alone had no significant effect at either site.

Exploration was measured in a holeboard (4 equally spaced holes in a floor 65 x 65 cm). Infrared photocells connected to counters provided automated measures of exploration (the number of head dips and the time spent head-dipping) and locomotor activity (File & Wardill, 1975). Rats received a single 10 min trial and were tested in randomized order between 0800–1200 h. When injected into the medial amygdaloid nucleus, morphine significantly reduced duration of head-dipping ($F(1,38) = 5.9$, $P<0.02$), an effect not antagonized by naloxone. However, in the central nucleus, morphine produced naloxone-reversible decreases in both the number of head-dips and time spent head-dipping ($F(1,42) = 6.1$ and 5.1, $P<0.02$ and 0.001, respectively) and in locomotor activity ($F(1,42) = 6.1$, $P<0.02$).

In conclusion, our results suggest that the presence or absence of specific morphine receptors cannot be deduced on the basis of a single behavioural test. We have shown that in the central amygdala morphine acts on specific receptors to reduce exploration and on non-specific receptors to raise electric shock thresholds. The converse appears to apply in the case of the medial amygdala.

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Behavioural effects of chronic amphetamine and their reversal by haloperidol in the marmoset

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Acute administration of amphetamine to the marmoset results in an increase in small head movements (checking), a decrease in activities and social contact, but no change in the amount of locomotion (Ridley, Baker & Crow, 1979; Scraggs & Ridley, 1978a). The increase in checking is blocked by haloperidol although activities and social contact cannot be reinstated by further drug treatment (Scraggs & Ridley, 1978b).

Chronic administration of amphetamine in rodents results in a progressive augmentation of stereotyped behaviour (Segal & Mandell, 1974). In primates, chronic methamphetamine treatment produces a progressive deterioration in purposeful and co-ordinated behaviour (Ellinwood & Kilbey, 1975).

In this study, (+)-amphetamine was administered via the drinking water to an established group of six marmosets in increasing doses of 1–4 mg/kg over 27 days (phase I). Haloperidol (0.01 mg/kg) was then given in addition to amphetamine (4 mg/kg) for 51 days (phase II) followed by 33 days of amphetamine (4 mg/kg) alone (phase III). Finally the animals were observed without drugs for 33 days (phase IV).

Behaviour was observed each day on alternate 3 day periods except when drug treatment changed, when behaviour was observed on the 3 days immediately before and after the change. Means of behaviour over 3 day periods were calculated and compared with pre-drug control readings (phase 0). On each observation day, each animal was observed for 100 s. Behaviour was classified each second into 5 mutually exclusive categories:

1. Checking: movement of the head only.
2. Activities: movement of only part of the body excluding head only. This category included activities such as eating, drinking and self-grooming.
3. Social contact: physical contact between 2 animals including grooming, fighting, play and sexual activity.
4. Locomotion: displacement of the whole body.
5. Inactivity: no discernible movement.

It was found that during phase I checking increased initially ($P<.001$) but then declined to normal levels: locomotion was initially unchanged but then declined ($P<.05$). Social contact was reduced ($P<.05$) while inactivity was increased ($P<.01$) throughout phase I. Purposeful activities were initially slightly decreased, but not significantly. However, during phase I a syn-