ERRATA

Br. J. Pharmac. (1980), **68**, 22–24

'Enhancement of rat brain metabolism of a tryptophan load by chronic ethanol administration' by A.A.-B. Badawy, M. Evans & N.F. Punjani.

Part of the last sentence on page 22 of this short communication was omitted. The complete sentence should read: 'Ethanol, however, decreases the load-induced rise in brain 5-HT and this may be partly explained by the finding (Gál, Young & Sherman, 1978) that, although brain tryptophan concentration rises in proportion to the dose of tryptophan administered (in the range of 0 to 100 mg/kg), those of 5-HT and 5-HIAA decline with doses above 25 mg/kg.

One of the abstracts was misprinted in the January 1980 issue of the journal. The correct version is given below.

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β-Adrenoceptor antagonists in L-tryptophan and L-DOPA induced behavioural syndromes

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Evidence that propranolol and certain other β-adrenoceptor antagonists may also possess significant antagonist activity at 5-HT receptors is growing and comes from studies *in vitro* (Schechter & Weinstock, 1974; Middlemiss, Blakeborough & Leather, 1977) and from inhibition of behavioural syndromes thought to result from raised brain 5-HT activity. Propranolol and related antagonists have been shown to block head-twitch responses in mice (Weinstock, Weiss & Gitter, 1977) and the characteristic hyperactivity syndrome in rats which follows monoamine oxidase inhibition and L-tryptophan (Green & Grahame-Smith, 1976). Although the effects of propranolol in the latter are stereospecific they are, however, only reported at doses well in excess of peripheral β-adrenoceptor blocking doses.

This hyperactivity syndrome is complex and may depend as much on dopamine (DA) as 5-HT stimulation in the CNS. Thus a closely related syndrome is induced when L-DOPA is substituted for L-tryptophan and 5-HT and DA antagonists may have similar effects on both syndromes (Jacobs, 1974; Deakin & Green, 1978). Costain & Green (1978), however, found no effect of propranolol on the L-DOPA syndrome.

We have considered the possibility that high precursor doses used in previous hyperactivity studies may preclude a high sensitivity to antagonist drugs and increase the risk of non-specific neurotransmitter effects. We have, therefore, re-examined the effects of propranolol's isomers on L-tryptophan and L-DOPA induced hyperactivity using precursor doses, 3–6 and 10–20 times lower, respectively, than were commonly used in the above studies.

Table 1 Effect of antagonists on rat hyperactivity. Receptor antagonists or saline were administered to groups of three male rats (Alderley Park strain, 180–200 g) simultaneously with tranylcypromine (TCP) (20 mg/kg) followed thirty minutes later by L-tryptophan (L-TRYP) (15 mg/kg) or L-DOPA (5 mg/kg). All injections were i.p. Hyperactivity was measured on 'Columbus' activity meters, 40–60 min after precursor load

	Rat Hyperactivity			
	TCP + L-TRYP		*TCP + L-DOPA*	
	M.E.D. (mg/kg)	*Effect*	*M.E.D. (mg/kg)*	*Effect*
(−)-Propranolol	0.8	Inhibition	0.8	Inhibition
(+)-Propranolol	25.0	None	25.0	None
Haloperidol	0.2	Inhibition	0.3	Inhibition
Methysergide	15.0	Potentiation	2.5	Potentiation

Results expressed as the minimum effective dose (M.E.D.) of antagonist to give $P < 0.05$ by Student's '*t*' test relative to controls, $n \geq 6$.