of 10^{-6} M 5-HT. Incubations were terminated by filtration under reduced pressure through Whatman GF/C filter papers. The filtration procedure consisted of quenching the incubation with 4 ml of ice-cold buffer, rapid filtration, followed by two further washes with 4 ml of buffer. The complete washing procedure usually took less than 10 s. The residual radioactivity on the filter papers was counted at 31% efficiency in a PCS scintillant (Amersham–Searle). Protein was estimated by the method of Lowry *et al.*[18]. 'Nonspecific' binding, defined as the radioactivity left on the filter paper in the presence of 10^{-6} M 5-HT, was subtracted from total binding in each experiment to give 'specific' or receptor binding. Specific binding at 5 nM ^{3}H-5HT was in the range 55–65% of total binding.

In accord with Bennett and Snyder[4], we have shown that the specific portion of ^{3}H-5-HT binding is rapid, reversible, saturable and of high affinity ($K_D = 11.7 \pm 2.5 \times 10^{-9}$ M, average of three experiments, ± s.e.m.). The concentration at which specific ^{3}H-5-HT binding is inhibited by 50% (IC_{50}) by the two putative 5-HT blocking drugs, methysergide and cyproheptadine, are given in Table 1 and are close to the previously reported values[4]. Also shown in Table 1 are IC_{50} values for the 5-HT receptor of a number of β-adrenergic blocking drugs. Propranolol has a substantial affinity for the 5-HT binding site, indeed (−)-propranolol is equipotent with the 5-HT blocking drug, methysergide, in displacing ^{3}H-5-HT from its receptor. The interaction of propranolol with the 5-HT receptor is stereospecific as is evidenced by an approximately 58-fold lower affinity of the isomer (+)-propranolol, the racemate drug (±)-propranolol is about half as potent as the (−)-isomer alone. Other β-adrenergic blocking agents also have substantial affinity for the 5-HT receptor (Table) although β-blockers which are cardioselective *in vivo*, for example practolol and atenolol, have IC_{50}'s greater than 10^{-4} M.

Table 1 Effects of various drugs on ^{3}H-5-HT binding to crude synaptic membranes

Drug	Half maximal inhibition of specific ^{3}H-5-HT binding (M)
5-HT	$1.7 \pm 0.3 \times 10^{-8}$
Methysergide	$2.8 \pm 0.3 \times 10^{-7}$
Cyproheptadine	$4.8 \pm 1.1 \times 10^{-8}$
(−)—Propranolol	$2.1 \pm 0.4 \times 10^{-7}$
(+)—Propranolol	$1.2 \pm 0.2 \times 10^{-5}$
(±)—Propranolol	$6.0 \pm 1.2 \times 10^{-7}$
(±)—Alprenolol	$3.0 \pm 0.5 \times 10^{-7}$
(±)—Oxprenolol	$4.3 \pm 0.8 \times 10^{-7}$
(±)—Pindolol	$7.9 \pm 0.8 \times 10^{-7}$
(±)—Practolol	$> 10^{-4}$
(±)—Atenolol	$> 10^{-4}$

^{3}H-5-HT binding was assayed as described in the text. Results are means ± s.e.m. of triplicate determinations from at least three separate experiments using six different concentrations of the drugs indicated. The concentration of the drug giving half maximal inhibition of specific ^{3}H-5-HT binding was determined by log probit analysis.

Thus, we have demonstrated a stereospecific interaction of propranolol and other β-blockers with the 5-HT receptor, an action which was previously unknown for this class of drug. It seems that the affinity of β-blockers for the 5-HT receptor does not necessarily parallel their β-blocking potency in tissues outside the central nervous system since pindolol, an extremely potent β-blocker, is less potent in displacing ^{3}H-5-HT than (−)-propranolol. Green and Grahame-Smith[17] have suggested, on the basis of the blocking action of (−)-propranolol on a L-tryptophan induced hyperactivity syndrome in rats, that this drug inhibits central 5-HT function. The direct interaction of propranolol with 5-HT receptor reported here is in accord with these findings. The significance of these observations in relation to the effects of propranolol in the central nervous system of animals and man remains to be evaluated.

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Errata

In the article 'Possible detection of a radio event correlated with a γ-ray burst' by N. Mandolesi *et al.*, *Nature* **266**, 427–429, the legend to Fig. 2 should have read: Fig. 2*a*, Balloon horizon; *b*, Medicina horizon in equatorial coordinates . . .

In the article 'β-Endorphin *in vitro* inhibition of striatal doparine release' by Horace H. Loh *et al.*, *Nature* **264**, 567–568, the last sentence beginning in the second column of p.567 should have read: Met^5-enkephalin, on the other hand, did not produce a significant inhibition of tritium overflow in concentrations as high as 100 μM. Naloxone, at an equimolar concentration, produced a significant blockade of the inhibition by morphine and β-endorphin, the blockade being more complete in the presence of morphine than in the presence of β-endorphin.

Corrigenda

In the article 'Pigmentation of the ladybird beetle *Coccinella septempunctata* by carotenoids not of plant origin' by G. Britton, W. J. S. Lockley, G. Harriman & T. W. Goodwin, *Nature* **266**, 49–50 the authors stated that phytoene, ζ-carotene and β-zeacarotene had not been detected before in any insect. Although no details were given of their characterisation, the detection of these carotenoids in certain butterflies has, however, been reported previously[1–3].

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In the article 'Nucleosynthesis and anomalous xenon and krypton in carbonaceous chondrites' by J. B. Blake and D. N. Schramm, *Nature* **263**, 787, the references on the following sentences with proper reference numbers should have read: Perhaps the explanation of the anomalous light isotopes lies in either the p-process[9,10] or in fractionation[7]. If the light xenon component is due to the p-process then ^{130}Xe might be affected, which would change the normalisation[9].