produced apomorphine-like (contraversive) circling behaviour in only 40–50% of animals tested, and then at the very high drug levels of 1 and 1.5 mg kg^{-1}. In the turning mouse model of von Voigtlander and Moore[2], where 1 mg kg^{-1} apomorphine induced a mean rate of turning of 5 min^{-1}, no satisfactory data for LSD could be obtained. In the dose range 0.025–0.2 mg kg^{-1}, LSD induced neither turning behaviour or postural asymmetries nor significantly modified apomorphine or amphetamine-induced circling, as may be predicted were it a potent dopamine agonist. Where rotation was induced following administration of 1.5 mg kg^{-1} LSD the duration was of the order of 35–45 min; not 2 h as Pieri *et al.* suggest. We agree with them that rotation was prevented by previous treatment with haloperidol (0.5 mg kg^{-1}), and not after α-methyl-*p*-tyrosine (250 mg kg^{-1}).

In another experimental animal model, namely, audiogenic seizures in inbred strains of mice, dopamine agonists have been shown to diminish the severity of the seizure response[3,4]. In our experiments, apomorphine was at least 10 times more potent than LSD in blocking the clonic phase of the seizure in 50% of animals.

The pharmacology of LSD is very confused, but has previously been associated with central 5-HT neurones[5–7]. Pieri *et al.*, however, have claimed a direct and potent action of LSD on the dopaminergic receptor *in vivo*. Our data do not confirm LSD as being a potent dopamine agonist, although it seems to be capable of stimulating the dopamine receptor in very high doses.

CHRIS PYCOCK
GILL ANLEZARK
Department of Neurology,
Institute of Psychiatry,
De Crespigny Park,
London SE5 8AF, UK

1 Pieri, L., Pieri, M., and Haefely, W., *Nature*, **252**, 586-588 (1974).
2 Von Voigtlander, P. F., and Moore, K. E., *Neuropharmacology*, **12**, 451-462 (1973).
3 Lehmann, A., in *Physiological Effects of Noise* (edit. by Welch, B. L., and Welch, A. S.), 227-257 (Plenum, New York, 1970).
4 Anlezark, G. M., and Meldrum, B. S., *Br. J. Pharmac.*, **53**, 419-421 (1975).
5 Andén, N. E., Corrodi, H., Fuxe, K., and Hökfelt, T., *Br. J. Pharmac.*, **34**, 1-7 (1968).
6 Chase, T. N., Breese, G. R., and Kopin, I. J., *Science*, **157**, 1461-1463 (1967).
7 Boakes, R. J., Bradley, P. B., Briggs, I., and Dray, A., *Br. J. Pharmac.*, **40**, 202-218 (1970).

PIERI ET AL. REPLY—The results reported[1] correspond to a well defined model, namely rats unilaterally lesioned in the medial forebrain bundle with 5,6-HT or 6-OHDA. These two types of lesion have been extensively investigated with biochemical[2,3] and histofluorescence methods (H. P. Lorez, unpublished), and result in a marked and long-lasting depletion of dopamine (DA), possibly leading to the subsequent development of denervation supersensitivity, (1 mg kg^{-1} apomorphine being able to induce more than 15 turns min^{-1} for 30 min). Lesioned animals were challenged with apomorphine and only those responding with a clear circling (about 50–60%) were subsequently used for the study of the effect of other agents, including LSD (80% of responses). The data obtained with several hundreds of rats have been described in part previously[4] and a more extensive paper is in the press[5]. The fact that Pycock and Anlezark[6] seem to have used a different animal species and a different lesion, clearly yielding less denervation supersensitivity (1 mg kg^{-1} apomorphine inducing a mean rate of turning of 5 min^{-1}), precludes any reasonable comparison.

Concerning the relative potencies of apomorphine and LSD as striatal DA receptor agonists, it seems from our data that LSD is slightly more potent than apomorphine. By no means, however, do we claim that this applies to completely different models such as that reported by Pycock and Anlezark[6] (namely, audiogenic seizures in mice).

It should also be stressed that low doses of LSD elicit a significant slowing of DA turnover in rat striatum (0.2 mg kg^{-1} intraperitoneally) and retina (0.5 mg kg^{-1} intraperitoneally). In addition, the striatal adenylate cyclase is activated by concentrations of LSD as low as 10^{-6} M (ref. 7). Thus, there is also biochemical evidence for DA receptor stimulating properties of LSD.

We did not intend to suggest that the DA receptor stimulant effect of LSD observed in our experimental conditions should be the final answer to the confused pharmacology of the compound. The DA-like component of LSD action in the central nervous system is, in our opinion, additive and not alternative to the stimulant action of the compound on 5-HT receptors. In circling behaviour, however, DA receptor stimulation seems to be important, at least in our model[5].

L. PIERI
M. PIERI
W. HAEFELY
F. Hoffmann-La Roche & Co. Ltd,
4002 Basel, Switzerland

1 Pieri, L., Pieri, M., and Haefely, W., *Nature*, **252**, 586 (1974).
2 Saner, A. Pieri, L., Da Prada, M., and Pletscher, A., *Experientia*, **30**, 697 (1974).
3 Saner, A., Pieri, L., Moran, M., Da Prada, M., and Pletscher, A., *Brain Res.*, **76**, 109 (1974).
4 Pieri, L., and Pieri, M., *Experientia*, **30**, 696 (1974); *J. Pharmac. (Paris), Suppl.* **2**, 5, 77 (1974).
5 Pieri, M., Pieri, L., Saner, A., Da Prada, M., and Haefely, W., *Archs int. Pharmacodyn.* (in the press).
6 Pycock, C., and Anlezark, G., *Nature*, **257**, 69-70 (1975).
7 Da Prada, M., Saner, A., Burkard, W. P., Bartholini, G., and Pletscher, A., *Brain Res.* (in the press).

Mimetic talking by parrots

THE paper by Gregory and Hopkins[1] has prompted me to speculate on the question; What is the biological significance (or survival value) of 'talking' by parrots? Of course, parrots do not actually talk, they only imitate speech. In fact, they imitate, not only speech, but many sorts of discernible sound patterns that are likely to be produced repeatedly in their environment. Indeed, the articulated sounds, I suggest, signify a kind of 'functional' mimicry, whereby an animal is camouflaged by becoming one more source of its natural environmental noise. Yet, the animal does not presumably utter the mimetic sound patterns at random times and spaces, but rather it may utter them as responses to specific outer signals. This behaviour implies the existence of integrated neuronal circuits; and as a dominant role of the visual system is known in birds[2], the pupil constriction observed, suggests that certain (learned) visual patterns may act as signals (stimuli) that trigger talking behaviour. Thus, pupil constriction may serve to obtain clearer, optimal images of such visual patterns. Guzmán-Flores (personal communication) has found that parrots fail to learn recurrent, tape-recorded utterances, in which the correlative significant visual stimuli are missing.

F. ALONSO DE FLORIDA
Facultad de Medicina,
Departamento de Fisiología,
Apartado Postal 70-199,
Ciudad Universitaria,
México DF, Mexico

1 Gregory, R., and Hopkins, P., *Nature*, **252**, 637–638 (1974).
2 Polyak, S., *The Vertebrate Visual System* (University of Chicago Press, 1957).

Hereditary persistence of foetal haemoglobin

ALTHOUGH the interpretation of Martinez and Colombo[1] may be correct, their conclusions are based on uncertain evidence and an alternative explanation that fits within the classical scheme is equally plausible. Their argument rests entirely on the statement that "foetal haemoglobin (HbF) levels show intrafamilial segregation in β^{thal}". From this statement, they conclude that III_2 cannot simply be a β^{thal} heterozygote like I_2. Our experience shows that this is not invariably so: parents, offspring, and siblings who are β^{thal} heterozygotes may differ in level of HbF. Consequently, we believe that III_2 is only a β^{thal} heterozygote and that his slightly elevated HbF is the result of his particular expression of the condition. The inheritance pattern then follows normally. I_1, II_1, and III_1 all have a chromosome with S and hereditary persistence of foetal Hb (HPFH); II_1 also has β^{thal}; III_1 and III_2 have a normal chromosome from II_2.