In the present study dexamethasone showed marked reduction of both exudate volume and leucocyte numbers in all three experimental schedules. This is in agreement with the results of Miyasaka & Mikami (1982) but they did not determine its effect on differential cell counts. It has been postulated that anti-inflammatory corticosteroids exert their anti-inflammatory action through producing phospholipase A2 inhibitory protein in terms of macrocortin or lipomodulin (Flower et al 1984).

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Letters to the Editor

Chronic clenbuterol treatment modulates a 5-hydroxytryptaminergic system

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After prolonged or repeated treatment with antidepressants or β-adrenoceptor agonists, resistance to the drugs develops due to hyposensitivity of β_1- (Banerjee et al 1977) and β_2-receptors (Dooley & Hauser 1983). However, we have observed, that antagonism of reserpine-induced hypothermia in mice by clenbuterol (a liposoluble β-adrenoceptor agonist) is, on the contrary, facilitated by chronic treatment (Francès et al 1985), although under the same experimental conditions resistance develops to clenbuterol-induced hypomotility. This observation may be of importance since reserpine-induced hypothermia in mice is generally considered to be predictive of antidepressant activity in man, and β-adrenoceptor agonists have been successfully used to treat depression (Widlöcher et al 1978). Since 5-hydroxytryptamine (5-HT) is thought to play an important role in depression, we have sought to determine whether a 5-HT system is implicated in the facilitatory effect of chronic clenbuterol treatment on the antagonism of reserpine-induced hypothermia by the β-agonist. 5-HT neurons were lesioned in male, Swiss, NMRI mice (20–24 g) by intracerebroventricular injections of 5,7-dihydroxytryptamine (5,7-DHT) (0·2 mg per mouse, disolved in 0·01% ascorbic acid), 30 min after i.p. injection of the highly potent inhibitor of noradrenaline reuptake, nisoxetine (20 mg kg^{-1}).

* Correspondence.

One week after lesion, clenbuterol (0·25 mg kg^{-1}) was administered chronically, twice daily (0830–0930 h and 1700–1800 h), on days 1 to 5 and 8 to 11, and once on day 12 (total of 19 injections). Controls received water. The effect of acute treatment with clenbuterol on reserpine-induced hypothermia was tested 6 h after the last chronic treatment, according to the following protocol: reserpine (2·5 mg kg^{-1}) was administered i.p. 4 h before clenbuterol (0·5 mg kg^{-1}) or water. Rectal temperature was measured 1 h after clenbuterol administration with a thermoelectric rectal probe inserted to constant depth.

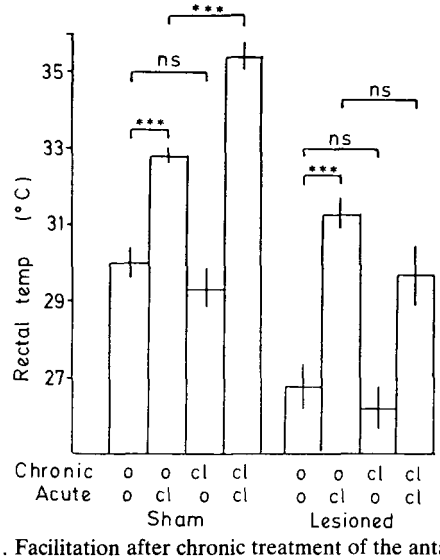

FIG. 1. Facilitation after chronic treatment of the antagonism of reserpine-induced hypothermia by acute clenbuterol: effect of 5,7-DHT lesions. All mice (6 per group) were treated with reserpine. O: water-treated controls. Cl: clenbuterol. Statistical significance was determined by analysis of variance and Student's *t*-test: ***$P < 0.001$; n.s., non-significant.

Results are presented in Fig. 1. Lesions with 5,7-DHT had no effect on body temperature (sham: 37·3 ± 0·3 °C, lesioned: 36·9 ± 0·1 °C). After reserpine treatment, the mean rectal temperature of the 5,7-DHT-lesioned mice was significantly lower than that of sham-lesioned animals. Acute clenbuterol significantly antagonized hypothermia in both sham and 5,7-DHT-lesioned animals; the effect was significantly greater in the latter. Chronic clenbuterol alone had no effect on the temperature of either group, but potentiated the antagonism of reserpine-induced hypothermia by acute clenbuterol in sham-lesioned animals. This effect was abolished by 5,7-DHT lesion.

Facilitation of the effect of acute clenbuterol by chronic treatment is probably not due to accumulation of clenbuterol, since the temperature attained under these conditions (35·4 °C) is never observed with acute clenbuterol treatment, no matter how high the dose (up to 64 mg kg^{-1}), and is absent in lesioned mice. This indicates that potentiation of the antagonism of reserpine-induced hypothermia by clenbuterol requires intact 5-HT innervation. This observation may be relevant to the well known latency period (1–2 weeks) required for the therapeutic efficacy of antidepressants including β-adrenoceptor agonists.

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Definitive IUPAC recommendations, 1985

The following IUPAC documents appeared in *Pure and Applied Chemistry* in definitive form during 1985.

Comments on these recommendations would be welcomed (addressed to the originating IUPAC Commission).

1. Names, symbols, definitions and units of quantities in optical spectroscopy (1985, 57: 105).
2. Nomenclature for regular single-strand and quasi single-strand inorganic and coordination polymers (1985, 57: 149).
3. Source-based nomenclature for copolymers (1985, 57: 1427).
4. Nomenclature, symbols, units, and their usage in spectrochemical analysis—V; Radiation sources (1985, 57: 1453).
5. Recommended terms, symbols, and definitions for electroanalytical chemistry (1985, 57: 1491).
6. Nomenclature for thermal analysis—IV (1985, 57: 1737).

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