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8 Pomorska R, Lech B, Droszcz W, Purska-Rowińska E. The effect of long-tern triamcinolone acetonide therapy on the function of the neuromuscular system. *Pneum Pol* 1981;**49**:627.

***This letter was sent to the authors, who reply below.

Sir,—Professor Droszcz and Dr Piotrowska make some extremely important points in their letter. They are correct in the belief that our study was performed on the assumption that there was no difference between the potencies of triamcinolone and triamcinolone acetonide. The investigation was stimulated by the publication of the report in the *British Journal of Disease of the Chest* in 1979 entitled "Triamcinolone in corticosteroid-resistant asthma."[1] The authors of that study, like ourselves, used doses of Kenalog (triamcinolone acetonide) within the range recommended by the manufacturer in the ABPI *Data Sheet Compendium*[2] which does not indicate that there is any difference in potency between triamcinolone and triamcinolone acetonide. In experimental animal models it is apparent that triamcinolone acetonide is very much more potent than triamcinolone, but no data from studies in man appear to be available. We therefore have to concede that Professor Droszcz and Dr Piotrowska are perhaps correct in their criticism of the way in which we discussed our data. We think it possible, however, that the information provided by ER Squibb and Sons Ltd about their product Kenalog may have misled the majority, if not all, of the physicians who use this corticosteroid preparation. Although we accept that it is very difficult to assess the relative potencies of corticosteroids, especially when they are administered by different routes, this controversy about the potency of triamcinolone and triamcinolone acetonide highlights the great need for companies to be obliged to state the potency of their products. Perhaps hydrocortisone could be the standard drug with an assumed potency of 1 and the activity of all other corticosteroid preparations for oral, intramuscular, or intravenous compared with it.

If the argument put forward by Professor Droszcz and his colleague about the potency of triamcinolone acetonide is accepted, and so far as we are aware there are no data to refute it, it remains difficult to explain why it causes less suppression of the hypothalamopituitary axis (HPA) than daily oral prednisolone in a dose of at least 10 mg. One explanation could be that a large dose of corticosteroid is available very soon after injection of Kenalog, but is not maintained for a full period of four weeks, towards the end of which serum and tissue levels may fall below physiological requirements, with consequent stimulation of the HPA axis. If this is the case treatment with Kenalog could be dangerous when given to patients who have HPA suppression, such as those patients in our study who had been taking large doses of oral prednisolone for a considerable time.

We concluded that we would not normally recommend triamcinolone (meaning triamcinolone acetonide) in preference to prednisolone because of side effects. If triamcinolone acetonide is indeed 10 times more potent than triamcinolone it could never be justified in preference to oral prednisolone in the long term management of bronchial asthma in the doses recommended by the manufacturers. Unfortunately, the data about the relative potencies of triamcinolone acetonide, prednisolone, and hydrocortisone are not published and are only available from ER Squibb and Sons Ltd as confidential information for clinical investigators.

Since publication of our paper we have learned from the manufacturers of Kenalog that it is not an intramuscular depot preparation and the reason for its prolonged but unpredictable duration of action is unknown. We have therefore to admit that even the title of our paper is incorrect.

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1 Peake MD, Cayton RM, Howard P. Triamcinolone in corticosteroid-resistant asthma. *Br J Dis Chest* 1979;**73**:39–44.
2 Squibb ER and Sons LTd. In: *ABPI data Sheet compendium 1983–84*. London: Datapharm Publications, 1983.

Corrections

Peak flow rate records in surveys: reproducibility of observers' reports

In the paper by Dr KM Venables and colleagues (November 1984;**39**:828–32) we regret that there are errors in the first paragraph of the methods section, in which it is stated that recordings from 61 men were studied. Of the 23 persons employed in the electronics factory, 18 were in fact women. The beginning of the last paragraph of page 828 should read: "Recordings from 61 subjects formed the basis of the study. Thirty eight subjects (all male) were currently employed in a steel coating plant. . . and 23 (18 female) were employed in an electronics factory. . . ." Elsewhere in the paragraph the word men should be taken to indicate subjects.

Bronchial reactivity to inhaled histamine and annual rate of decline in FEV_1 in male smokers and ex-smokers

Smoking, allergy, and the differential white blood cell count

In the two papers by Dr RG Taylor and others (January 1985) we regret that page numbers are missing from two of the references. In ref 10 on p 16 the pages are 17–22 and in ref 24 on p 21 they are 9–16.