concentration may be important.[6] Surprisingly, Maran and colleagues do not discuss this possibility.

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EDITOR,—After being transferred to human insulin a number of patients have complained that they do not recognise hypoglycaemia as clearly as they used to with animal insulin. Scientific debate about this has developed into a veritable battle of faith. Having been responsible for recruiting patients to one of the landmark studies in this field,[1] which Alberto Maran and colleagues reference,[2] I can testify to what can happen in the heat of combat.[3] In the report of this study 14 of 28 episodes of severe hypoglycaemia which occurred in my patients were attributed to the wrong insulin and patients were subsequently excluded selectively. This became obvious because different figures had been published earlier.[4]

The design of Maran and colleagues' study would seem to exclude such trouble. Important inconsistencies emerge, however, from a reading of their earlier abstract.[5] The threshold for psychomotor deterioration was higher with human insulin in the abstract (3·1 *v* 2·8 mmol/l); in the paper the thresholds are different but nearly identical (2·8 *v* 2·7 mmol/l).

But why bother? The pharmaceutical industry is willing to keep animal insulins available for patients who need them, although it will not fund a definitive large study assessing severe hypoglycaemia. Animal insulins continue to be needed both from patients' and from doctors' points of view.

Because of the doubtful reanalysis of the data Maran and colleagues' findings should be interpreted with caution. Remarkably, their table I shows that the median symptom scores for episodes of hypoglycaemia during the two months' treatment are higher with porcine than human insulin for seven of the nine items and identical only for the remaining two; the scores at the onset of attacks show the greatest difference. The authors' conclusion that no causal relation exists between human insulin and reduced hypoglycaemia awareness may well seem disdainful to some patients—for example, those who refused to participate in the study because of the problems experienced with human insulin. I suggest that doctors should end the battle of faith and stand together to assure continued supply of animal insulin for those who need or want it.

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AUTHORS' REPLY,—We acknowledge the numerical discrepancies between the data published in abstract form for the American Diabetes Association and those in the final version of the paper in the *BMJ*. These differences are small and in no way alter the conclusions. They are due to differences in the data available for the preliminary analysis (the abstract was written in December 1991, when data collection and analysis were incomplete) and to the limited space available in the abstract (200 words). We could not, for example, explain how we had analysed the frequency of clinical episodes of hypoglycaemia during treatment with the two insulins. In the abstract we used only the numbers from the last six weeks of each treatment period to avoid a changeover effect. This is explained in detail in the paper, and the analysis in the paper (and, indeed, the analysis presented at the time of the summer meeting for which the abstract was drafted) is more sophisticated and based on a complete dataset.

With regard to Matthias Egger and colleagues' other points, the adrenaline concentrations were not significantly different between the two studies (p for the peak value was 0·23), nor (which these authors do not mention) was the earlier response with human insulin significant. As regards the size of the study, the power calculations are clearly described in the text. The meta-analyses referred to show that it is often different components of the counterregulatory responses that vary between studies. All the evidence available suggests that the rate of fall of the glucose concentration does not affect the response to hypoglycaemia in a clamp.[1]

Ernest von Kriegstein's suggestion that our reanalysis is dubious is unworthy. It is by no means uncommon for preliminary results in abstracts to differ from those achieved finally—indeed, we were surprised that these discrepancies were thought worthy of remark. The analysis is fully explained in the paper. The values in table I are medians, and the quoted statistics clearly show the lack of significant difference. We had no preconception of the results and analysed our data without prejudice. We deny that we have been disdainful of patients: we were investigating a concern expressed by them in the most objective way we could. We ourselves suggested that, notwithstanding our data, any patient who believes that he or she has a problem with human insulin should resume animal insulin, which we certainly would not wish to see withdrawn. The subject is certainly emotive, but we believe that our study has helped to move the "battle of faith" into the arena of science.

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Prevalence of toxoplasma IgG

EDITOR,—The prevalence study of Gilbert and colleagues,[1] aimed at identifying the possible connection between toxoplasma seropositivity and ethnic group and place of birth of an antenatal population from west London, was of considerable value as it is the first study of its kind to be published. However, the conclusions drawn from the data are of some concern.

There was no indication whether individuals within the subgroups had travelled to either their country of origin, if appropriate, or—if born in one geographical location—had travelled to other areas where the incidence of environmental toxoplasma exposed them to increased risk of this infection. In this context, it is of note that the prevalence of toxoplasma infection can vary in different geographical areas within a single country.[2]

The authors gave considerable emphasis to the role of ingestion of raw or undercooked meat as a source of toxoplasma infection. Meat must be uniformly heated to 66°C (150°F) to kill intracystic toxoplasma. Such food is undoubtedly involved in acquiring toxoplasma infection. This has been shown by lower prevalence of antibodies to toxoplasma in immigrants to Paris than in women of French origin,[3] a factor suggested to be the consequence of a cultural preference for poorly cooked meat.

It is important, however, not to exclude from the equation the ingestion of food contaminated by infective oocysts from cat faeces. This route of toxoplasma infection to humans has been extensively investigated and recorded—for example, in a series of related studies from Costa Rica.[4]

Two of the longitudinal studies cited showed a temporal decline in toxoplasma seroprevalence; this was suggested to be due to improved standards of food storage. However, these studies started in 1969, which coincides with the first report of the role of the domestic cat in the life cycle of toxoplasma.[5] Could it be that the awareness of this factor has contributed, at least in part, to the observed decrease in human toxoplasma infection, at least in some geographical areas?

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Prescribing folic acid

EDITOR,—Anna Livingstone's statement that 400 μg folic acid tablets (classed as a dietary supplement) cannot be prescribed on FP10 is incorrect.[1] The factor that decides whether a product can be prescribed is whether the product appears on the blacklist rather than whether it has a product licence. A great many FP10s are written