ta Group, which theoretically at least, should have a much smaller impact worldwide.

What is the purpose of the oral glucose tolerance test? Clinically it is a minor diagnostic tool (except in pregnancy). In my own clinic last year, we performed less than ten glucose tolerance tests for diagnostic purposes. The main purpose of the oral glucose tolerance test is the identification of at-risk groups in population surveys. One major group contains those individuals at risk of developing macrovascular disease, and the recent survey by Fuller et al. [3] would suggest that the impaired glucose tolerance criteria fulfil this need, although 'normality' may have been set too high rather than too low, as suggested by Massari et al. The second group are those at risk of developing diabetic microangiopathy and neuropathy. Only time, and prospective studies, will tell whether the new criteria for diabetes accurately identify these individuals. Certainly based on information available today the new criteria are a considerable improvement on the old.

Finally, Massari et al. criticise the rounding up of numbers in the WHO criteria – most of us would agree with their criticism, not because one set of values is wrong and one is right, but more because it detracts from efforts to obtain uniformity of approach.

In summary, Massari et al. have emphasized the differences between the old and the two new sets of criteria. Sadly they have unwarrantedly included the new WHO criteria in their accusations of imprecision when this criticism can only really be levelled at the National Diabetes Data Group criteria, where 33% of subjects were unclassifiable. I would suggest that all future studies be based on the new WHO criteria and that studies to test the predictive accuracy of these new criteria should be set in train now.

Yours sincerely,
K. G. M. M. Alberti

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HLA-DR Antigens and Diabetic Nephropathy

Dear Sir,

In a recent preliminary communication, Christy et al. claimed an association between the HLA-DR3/DR4 phenotype and diabetic nephropathy in patients with Type 1 (insulin-dependent) diabetes [1]. In their series of 26 patients with Type 1 diabetes and nephropathy, the phenotype DR3/DR4 was significantly less common than in a matched group of patients with Type 1 diabetes without nephropathy.

A former study at our clinic [2] showed no evidence for association between HLA-B locus antigens and Type 1 diabetic patients with nephropathy, although associations of HLA-B15 [3] and DR4 [4, 5] with diabetic proliferative retinopathy were suggested.

We have studied HLA-DR antigen frequencies in 61 Type 1 diabetic patients with nephropathy and in 61 Type 1 diabetic patients free of renal disease (no proteinuria) matched as closely as possible for duration of disease and age at onset (mean age at onset: 10.9 versus 10.8 years; mean duration of disease: 24.97 versus 25.11 years, respectively).

Table 1. HLA-DR antigen frequencies in Type 1 diabetic patients with and without nephropathy

HLA-DR	Type 1 diabetic patients			
	With nephropathy ($n=61$)	(%)	Without nephropathy ($n=61$)	(%)
3,*	15	24.59	11	18.03
4,*	21	34.43	24	39.34
3, 4	22	36.06	22	36.04
,	3	4.92	4	6.56

* HLA antigen other than DR3 or DR4

Out of the 61 diabetic patients with nephropathy, 35 have received a renal allograft. Table 1 shows that we do not confirm the suggested association between DR3/DR4 and nephropathy, even though the number of patients in our study was larger than in the Danish study.

We conclude that there is no evidence for an association between HLA and diabetic nephropathy.

Yours sincerely,
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Free Inositol, Sorbitol and Fructose Levels in Sciatic Nerve

Dear Sir,

In a recent paper by Mayhew et al. [1], free inositol, sorbitol and fructose levels in sciatic nerve, obtained post-mortem from diabetic and control subjects, were compared with data we obtained on sural nerve biopsy [2]. Mayhew et al. found inositol levels to be lower in diabetic patients ($n=23$) than in control subjects ($n=15$; $p=0.016$). This is the expected direction of change based on studies in experimental

diabetes [3–5]. In their comparison with our work, Mayhew et al. point out that most of our diabetic patients ($n = 17$) had inositol levels insignificantly different from our control group ($n = 13$) and that these values were similar to the control values obtained by themselves. A separate group of *three* diabetic patients (without evidence of neuropathy) that we studied had inositol levels about twice the control/other-diabetic levels ($p = 0.037$). This small group was singled out by Mayhew et al. for comment as follows: "Dyck et al. quote inositol concentrations more than double the normal for nerves from diabetic patients. Such high values suggest an error in their analytical methods". Since our control group results are not significantly different from those of Mayhew et al. and since we used the same analytical method, it is difficult to see how an error in the method could explain the divergent results from three patients. To us it seems more likely to be a Type 2 statistical error resulting from the small sample number. Whether the results obtained post-mortem by Mayhew et al. truly support the view that there is a decrease in inositol in human diabetic nerve must be confirmed by further research. Considering the effect on nerve level of dietary inositol supplementation [6, 7] and deficit [7], it is clear that patient status and diet is probably a large factor, even without considering biopsy *versus* post-mortem sampling. In our hands, the significance of the data was obscured by large inter-patient variation: the relative standard deviation of control *myo*-inositol values was 43%, of diabetics 76% (compared with 62% control and 70% for the data of Mayhew et al.). Just how much scientific significance can be drawn from such data is certainly open to question.

Yours sincerely
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