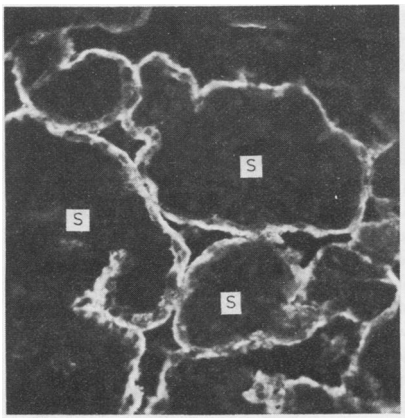

The distribution of transferrin receptor on human placenta. The syncytiotrophoblast of chorionic villi is positive with monoclonal antibody OKT-9. Note negativity of chorionic stroma (S). × 300.

tation of the polymorphism of human transferrin receptors. At present it would seem prudent to use a battery of techniques to confirm or refute the presence of transferrin receptors rather than using only monoclonal antibodies to the receptor.

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Dr Gatter replies as follows:

Dr Wells and colleagues report the interesting observation that a different monoclonal anti-transferrin receptor antibody to the four that we used in our study does not give staining of the basal layer of the epidermis in their laboratory. We too pointed out that there were differences between our antibodies, particularly between BK19·9 and the other three antibodies. The latter half of our discussion was concerned with the possible explanation for this and we raised the possibility that these discrepancies might reflect the fact that the transferrin receptor is not a single molecular entity but a family of molecules which are antigenically similar but not identical. Further studies, using a variety of different techniques, on the human transferrin receptor will be of great interest.

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Selective damage to type 2B muscle fibres in ethanol-fed rats

The recent report by Slavin *et al*[1] describes selective atrophy of the alkaline myofibrillar ATPase type 2B muscle fibres in chronic alcoholics. Slavin *et al* suggest that defective muscle anaerobic glycolysis may underlie the selective damage to type 2B fibres that are more heavily dependent upon glycolysis for energy production. The prototype disorder of defective skeletal muscle anaerobic metabolism is myophosphorylase deficiency (glycogenosis type V, McArdle's disease), although phosphofructokinase deficiency (glycogenosis type VII, Tarui's disease), phosphoglycerate mutase deficiency (DiMauro's disease), muscle lactate dehydrogenase deficiency (Kanno's disease) and muscle phosphoglycerate kinase deficiency (Dalakas' disease) have similar symptomatology.[2 3] These disorders are all characterised by painful, exercise-induced, electrically-silent muscle contracture followed by post-exercise rhabdomyolysis.[2 4] The usual diagnostic clinical test, the forearm ischaemic exercise test, is based upon the finding in these disorders of deficient lactate production during ischaemic exercise.[5] Radionuclide scanning with calcium tracers demonstrates marked uptake in contractured muscle.[6 7] Similar symptomatology including exercise-induced, painful muscle cramping, episodes of rhabdomyolysis, excessive muscle uptake of radionuclide calcium tracers, and defective lactate production during ischaemic exercise has been reported in chronic alcoholics.[8–11] In our histological studies of patients with myophosphorylase deficiency or phosphofructokinase deficiency, we found selective damage to the type 2B muscle fibres.[4 6]

We have developed an animal (rat) model for disorders with defective skeletal muscle glycolytic/glycogenolytic metabolism.[12–14] This model utilizes iodoacetate selectively to inhibit the second stage glycolytic enzyme D-glyceraldehyde-3-phosphate dehydrogenase. The animals develop muscle symptomatology completely analogous to that of the human patients, including the histological evidence of selective type 2B muscle fibre injury. We have also found a sexually dimorphic response in this model with male rats and ovariectomised female rats developing markedly more severe symptomatology than intact female rats.[15]

We have performed preliminary studies of the effect of ethanol in the animal model. For six to ten weeks a complete liquid diet (Ensure) to which was added 9·5% ethanol by volume was administered as the only nutrient to a group of Wistar Furth rats. Although these rats show no evidence of alcohol dependence, they do have persistent behavioural deficits.[16] After administration of less than half the usual