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.

MICHAEL WELLS* CHANG-JING G YEHS BEA-LI HSII W PAGE FAULK
*Department of Pathology, University of Leeds, Leeds LS2 9JT Inserm U210, Laboratoire d’Immunologie, Faculté de Médecine, Avenue de Vallombrose, 06034 Nice-Cédex, France

References


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.

KC GATTER
Nuffield Department of Pathology, John Radcliffe Hospital, Oxford OX3 9DU

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

The recent report by Slavin et al1 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 contraction followed by post-exercise rhabdomyolysis.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 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.5,7 In our histological studies of patients with myophosphorylase deficiency or phosphofructokinase deficiency, we found selective damage to the type 2B muscle fibres.6

We have developed an animal (rat) model for disorders with defective skeletal muscle glycolytic/glycogenolytic metabolism.11-15 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 ovarietomised 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
minimum iodoacetate dosage and after only one-third the usual minimal exercise (compared to model animals not given ethanol), the ethanol-fed rats developed the muscle symptomatology. In addition, both male and intact female rats developed the more severe male-pattern of injury. Histological study revealed damage exclusively to the type 2B muscle fibres.

Our preliminary findings in the ethanol-fed rats would tend to support the contention by Slavin et al. that selective vulnerability of type 2B muscle fibres in chronic alcoholism is the result of alcohol-induced alteration of anaerobic glycolysis. It is possible that in chronic alcoholics the syndromes of acute rhabdomyolysis and chronic muscle atrophy are opposite ends of a spectrum of type 2B muscle fibre response to varying degrees of alcohol-induced interference with energy metabolism.

ROGER A BRUMBACK
University of Rochester Medical Center, Rochester, New York 14642, USA

References

Elution of antibodies to Mallory's hyaline from kidneys of patients with alcoholic liver disease and mesangial IgA deposits

We were most interested to read the paper by Burns et al. reporting a Mallory body antigen (JMB2) in the mesangium of three patients with alcoholic liver disease. Data from our elution experiments with such kidneys help to confirm the significance of this report.

Kidney were obtained at post-mortem from nine subjects with alcoholic liver disease and mesangial IgA deposits. Washed glomerular suspensions were eluted with citrate buffer pH 3-2 and the concentrated eluates were tested for IgA, IgG and IgM class antibodies to Mallory bodies by indirect immunofluorescent fluorescence using frozen sections of liver from a patient with alcoholic liver disease and abundant Mallory's hyaline. IgA anti-Mallory body staining was seen in seven of the eluates. The same pattern of staining was seen with serum from a patient with acute alcoholic hepatitis and with two titre smooth muscle antibody (SMA) sera. No staining was seen with an eluate from a normal kidney or with normal serum or serum from a patient with IgA myeloma. No staining was found with reagents for IgG or IgM and there was no reactivity with normal liver sections.

These findings suggest that IgA anti-Mallory body/Mallory body complexes contribute to the mesangial deposits seen in some patients with alcoholic liver disease. However, other immune mechanisms are also operative in alcoholic liver disease, notably increased gut permeability to antigens and impaired hepatic sequestration of antigens, immune complexes and IgA polymers. The participation of IgA in each of the above mechanisms explains the predominance of IgA in the associated hyperglobulinaemia, serum immune complexes and mesangial immune deposits.

JANE D LOMAX-SMITH ANDREW J WOODROFFE
Renal Unit and Department of Pathology, Royal Adelaide Hospital, North Terrace Adelaide SA 5000 Australia

Letters to the Editor

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<th>Total Ig (µg IgG equiv/ml)</th>
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R A Brumback

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