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.

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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 (JM2B) in the mesangium of three patients with alcoholic liver disease. Data from our elution experiments with such kidney help to confirm the significance of this report.

Kidneys 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 immunofluorescence using frozen sections of liver from a patient with alcoholic liver disease and abundant Mallory’s hyaline. IgA anti-Mallory body staining was seen with seven of the eluates. The same pattern of staining was seen with serum from a patient with acute alcoholic hepatitis and with two high 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 hypergoblinemia, serum immune complexes and mesangial immune deposits.

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<table>
<thead>
<tr>
<th>Total Ig</th>
<th>Indirect immunofluorescence staining of Mallory bodies (Graded 0 – ++++)</th>
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<tbody>
<tr>
<td></td>
<td>IgA</td>
</tr>
<tr>
<td>Eluates</td>
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<tr>
<td>1</td>
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</tr>
<tr>
<td>2</td>
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<td>3</td>
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<td>8</td>
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<td>9</td>
<td>186</td>
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<tr>
<td>Normal kidney</td>
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<table>
<thead>
<tr>
<th>Sera</th>
<th>Total Ig (µg IgG equiv/ml)</th>
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</thead>
<tbody>
<tr>
<td>NHS</td>
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</tr>
<tr>
<td>ALD</td>
<td>1/10</td>
</tr>
<tr>
<td>SMAA</td>
<td>1/10</td>
</tr>
<tr>
<td>SMAd</td>
<td>1/10</td>
</tr>
<tr>
<td>IgA myeloma</td>
<td>1/20</td>
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</tbody>
</table>
The Howie code and the price of safety

I endorse the need for caution expressed by Dr Whale¹ about interpreting the tuberculosis figures in the last survey carried out for the ACP with help from the IMLS and others.² This adds to the importance of the further data to be collected in the next survey covering 1982–83, and continued co-operation is hoped for in this important surveillance project. Attention will be paid to classification of groups other than "medical, science, MLSO" since the breakdown which served adequately for the original problem of hepatitis is less satisfactory for tuberculosis. Dr Whale's caution about the high attack rates calculated from only two cases in "porters, assistants" should be related to the even higher rates calculated for those "technicians and attendants" in mortuary and post-mortem work in 1979–81.³ Hepatitis B surprised many of us when inapparent parenteral contagion emerged as an important mode of infection in laboratories, and in the light of the study by Newsom and others⁴ it will be interesting to discover whether routes other than airborne prove to be important in the spread of tuberculosis to laboratory staff.

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References


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J D Lomax-Smith and A J Woodroffe

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