Correspondence

SIR,
Balldin and colleagues\(^1\) reported an increase of serum total amylase activity in cigarette smokers, but their results failed to be confirmed by Nasrallah et al.\(^2\).

We have evaluated serum total amylase activity, isoamylase fractions by electrophoretical method and salivary \(\alpha\)-amylase activity in 70 subjects, aged 30–65, divided as follows: group S consisted of 30 subjects who had smoked cigarettes for three to 20 years and at time of study smoked more than 20 cigarettes/day; group N consisted of 40 non-cigarette smokers who served as reference group.

No subject used had a history of alcohol abuse or of gastroenterological disease. The Table indicates that there is no difference in the fasting levels of total amylase and pancreatic isoamylase in the two groups considered.

<table>
<thead>
<tr>
<th></th>
<th>Group S (32)</th>
<th>Group N (40)</th>
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</thead>
<tbody>
<tr>
<td>Total amylase</td>
<td>236-69±11-18</td>
<td>293-40±18-19</td>
</tr>
<tr>
<td>Type P</td>
<td>111-56±9-02</td>
<td>120-84±8-66</td>
</tr>
<tr>
<td>Type S</td>
<td>125-13±6-02</td>
<td>172-56±12-73</td>
</tr>
<tr>
<td>Salivary amylase</td>
<td>274-3±16-05</td>
<td>437-5±32-62</td>
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</table>

All results are shown as \(M \pm \text{SEM (units/l)}\). Saliva diluted 1/1000.

It may be observed that cigarette smokers exhibit a reduction in serum type \(S\) isoamylase that correlates well with the significant reduction of salivary amylase activity.

Several studies reported that tobacco smoke contains agents with cytotoxic or carcinogenic effects on the exocrine pancreas,\(^3\)\(^4\) but little is known about the effect of tobacco smoke on salivary glands.

From our results it could be argued that chronic cigarette smoke may exert a toxic effect on salivary glands, by acting through a local effect or a neurovascular reflex. In any event, the real impact of findings linking cigarette smoking with abnormalities in salivary amylase activity should be further investigated.

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References


In vitro determination of small intestinal permeability

SIR.—We were interested to read the recent paper by Drs Bjarnason and Peters (Gut 1984; 25; 145–50) describing their \(in vitro\) technique for showing abnormalities of intestinal permeability in coeliac disease. While we agree with the authors that the results they present do not confirm a reduced intestinal permeability to small hydrophilic molecules, we believe this is a result of their selection of probe molecules, and does not provide evidence that such a reduction of permeability does not occur. The three molecules used in this work are all considerably larger than probe molecules such as mannitol, monosaccharides and low molecular weight polyethylene glycols, for which there is indisputable evidence of reduced absorption in coeliac disease.\(^1\)\(^4\) The inverse relationship between molecular weight and intestinal permeability to molecules with a molecular weight between 340 and 5200 daltons shown by Bjarnason and Peters confirms our own observations in an animal model,\(^5\) although we believe that molecular volume is a more appropriate parameter to relate to intestinal permeability than molecular weight. We have also found that there is a different inverse relationship for smaller molecules with a molecular volume between 130 and \(230\times10^{-3}\) nm,\(^3\)\(^5\) suggesting that such small molecules are absorbed \(via\) a different pathway from which EDTA, cyanocobalamin and inulin would be excluded by their physical size, and we believe it is this pathway which becomes less permeable in coeliac disease.

The major advantage of tests of intestinal permeability based on the simultaneous administration of two molecules is the contrasting changes in their absorption that occur in coeliac disease, reducing the influence of extraneous factors such as gastrointestinal transit time and renal function on the test result\(^6\) and increasing the sensitivity and specificity of the test.\(^7\) Thus we cannot agree that the