Further proof awaits the isolation of carrier molecules from these mucosae.

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Aspirin inhibition of glucose absorption from the human mouth

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Aspirin inhibited glucose absorption from the gastrointestinal tract of the human in vivo (Arvanitakis et al., 1977) and the rat in vitro (Cooke et al., 1979). Glucose was absorbed from the human mouth apparently by a Na⁺-dependent carrier-mediated process (Manning & Evered, 1976). The aim of the present study was to investigate whether aspirin also inhibited glucose transport across buccal mucosa.

Absorption of D-glucose from the human mouth in vivo was studied in the absence and presence of aspirin (acetylsalicylic acid) at pH 6 by a published method (Manning & Evered, 1976). Equimolar citric acid was substituted for sodium citrate in the buffer solution and 3.5 mM-CaCl₂ was added. D-Glucose was measured enzymically (Dahlqvist, 1964). Absorption, measured as a loss from the buccal cavity, was at an initial concentration measured enzymically (Dahlqvist, 1964). Absorption, measured as a loss from the buccal cavity, was at an initial concentration of 10 mM-glucose. This value was on the upper linear portion of absorption rate in relation to glucose concentration (Manning & Evered, 1976).

The results given in Table 1 show that aspirin inhibited glucose absorption from the buccal cavity of a male human adult aged 22 years. By comparison, aspirin at 10 and 20 mM inhibited transport of 40 mM-glucose across everted sacs of rat small intestine by 62 to 70% (Cooke et al., 1979). It is probable that buccal mucosa is permeable to aspirin, presumably in its un-ionized form. Salicylic acid and salicylamide were absorbed from the human mouth at pH 6 in vivo (Sprake, 1977).

There are two likely hypotheses to account for aspirin inhibition of glucose absorption across gastrointestinal mucosa. Firstly, aspirin uncouples oxidative phosphorylation (Brodie, 1956). Buccal absorption of glucose was inhibited by salicylate (Evered et al., 1977), which also uncouples oxidative phosphorylation (Brodie, 1956). Moreover, phenobarbitone inhibited buccal absorption of glucose (Evered et al., 1977), and barbiturates too inhibited cellular respiration (Jalling et al., 1955).

Secondly, aspirin decreased significantly the net flux of Na⁺ ions across the epithelium of rat small intestine (Cooke et al., 1979). Ca²⁺ ions stimulated glucose transfer across the mucosa of the human mouth (McMullan et al., 1977) and rat small intestine (Manning et al., 1978). This effect of Ca²⁺ ions was thought to be due to a stimulation of Na⁺ fluxes. There was no such effect of Ca²⁺ with fructose transport (Manning et al., 1978), which is independent of Na⁺ ions. Significantly, aspirin did not inhibit absorption of fructose across intestinal epithelium (Cooke et al., 1979).

The present experiments do not discriminate between these rival theories. However, the results provide further support for the presence of a Na⁺-dependent carrier-mediated transport process for D-glucose in human buccal mucosa, as in mammalian small intestine.

Table 1. Effect of aspirin on the absorption of 10 mM-D-glucose across human buccal mucosa

Mean values are shown, ± S.E.M. for six experiments. The subject was a 22-year-old male Caucasian, and informed consent was first obtained. Statistical significance was determined by Student's t test.

<table>
<thead>
<tr>
<th>Aspirin concn. (mM)</th>
<th>Glucose absorbed (μmol/5min)</th>
<th>Statistical significance</th>
<th>Inhibition (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 (control)</td>
<td>68.1 ± 0.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>48.0 ± 1.1</td>
<td>P &lt; 0.005</td>
<td>30</td>
</tr>
<tr>
<td>0 (control)</td>
<td>68.5 ± 0.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>56.1 ± 0.6</td>
<td>P &lt; 0.005</td>
<td>18</td>
</tr>
</tbody>
</table>