Short Communication

CARIOSTATIC EFFECT OF PALATINOSE ON EXPERIMENTAL DENTAL CARIES IN RATS

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SUMMARY: Specific pathogen-free Sprague-Dawley rats infected with Streptococcus mutans MT8148R (serotype c) developed a severe dental caries when fed a diet containing 56% sucrose (diet #2000). Complete replacement of the dietary sucrose with palatinose, a structural isomer of sucrose, however, resulted in negligible caries induction and plaque accumulation. Replacement of half of the sucrose content of diet #2000 with palatinose induced dental caries; however, the caries score was significantly lower than that induced by diet #2000. No significant reduction in caries development was observed when a quarter of the dietary sucrose was replaced with palatinose.

In a preceding paper (Ooshima et al., 1983), we demonstrated that none of seven strains of Streptococcus mutans (serotype a–g) produced any significant amount of acid or insoluble glucan from palatinose (isomaltulose; α-D-glucopyranosyl-1,6-fructofuranose), a structural isomer of sucrose. It was also found that palatinose significantly inhibited synthesis of insoluble glucan from sucrose by the glucosyltransferase (GTase) of serotypes c, d and g S. mutans. Furthermore, in the animal experiment using S. mutans 6715 (g), the caries-inducing activity of palatinose was extremely low. Even when palatinose was substituted for half the sucrose content of a caries-inducing diet #2000 (Keyes and Jordan, 1964), the caries score decreased notably when compared with diet #2000 or diets replaced for half the sucrose by glucose, fructose or a mixture of glucose and fructose.

S. mutans strain 6715 failed to utilize palatinose even after subcultured 10 times in a broth medium containing 1% palatinose, while strains of serotypes c, e and f became to utilize it when subcultured in the medium (Ooshima et al., 1983; Ohta and Takazoe, 1983). It has been reported that serotype c is the most predominant type of S. mutans in Japanese, North-American and...
European populations (Hamada and Slade, 1980; Hamada, Masuda and Kotani, 1980). The purpose of the present investigation was to examine the caries-inducing or cariostatic activities of palatinose in specific pathogen-free Sprague-Dawley rats infected with a strain of serotype c S. mutans.

*S. mutans* MT8148R (serotype c), which had been isolated from a carious lesion of a Japanese child and made streptomycin resistant, was used in the present study. The time schedule of the experiment, the experimental procedures, and the maintenance of the experimental animals were described in the previous papers (Hamada et al., 1978; Ooshima et al., 1981, 1983). The basic experimental diet used in this study was cariogenic diet #2000 containing 56% sucrose (Keyes and Jordan, 1964). To examine various sugars including palatinose for the caries-inducing ability sucrose was replaced with different sugars to be tested (Table I). All sugars incorporated in the diets were finely pulverized and passed through a 100-mesh sieve. The diets were supplied *ad libitum* throughout the experiment.

Implantation or elimination of *S. mutans* MT8148R was confirmed according to Keyes and Fitzgerald (1962). No streptomycin-resistant streptococci were detected in any of uninfected rats throughout the experiment. *S. mutans* MT8148R was easily established and persisted at the highest level throughout the experiment in the oral cavity of the rats fed diets containing different sugars, irrespective of the presence of sucrose (Fig. 1). On the other hand, serotype g *S. mutans* 6715 has been found to be barely established when rats were fed diet containing palatinose or cornstarch (Fig. 1; Ooshima et al.,

### TABLE I

<table>
<thead>
<tr>
<th>Experimental group</th>
<th>Inoculation of <em>S. mutans</em></th>
<th>Sugars in diet</th>
<th>Plaque index 1</th>
<th>Caries score 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>(mean±S.E.)</td>
<td>(mean±S.E.)</td>
</tr>
<tr>
<td>1</td>
<td>–</td>
<td>Sucrose 56%</td>
<td>0.4±0.0***</td>
<td>8.1±0.4**</td>
</tr>
<tr>
<td>2</td>
<td>+</td>
<td>Sucreose 56%</td>
<td>1.1±0.1</td>
<td>43.9±5.2</td>
</tr>
<tr>
<td>3</td>
<td>+</td>
<td>Glucose 56%</td>
<td>0.6±0.1**</td>
<td>21.4±1.4**</td>
</tr>
<tr>
<td>4</td>
<td>+</td>
<td>Fructose 56%</td>
<td>0.7±0.0**</td>
<td>22.2±1.5**</td>
</tr>
<tr>
<td>5</td>
<td>+</td>
<td>α-Starch 56%</td>
<td>0.5±0.0**</td>
<td>8.0±0.6**</td>
</tr>
<tr>
<td>6</td>
<td>+</td>
<td>Palatinose 56%</td>
<td>0.4±0.0**</td>
<td>8.0±0.7**</td>
</tr>
<tr>
<td>7</td>
<td>+</td>
<td>Sucrose 28%+Glucose 28%</td>
<td>1.0±0.1</td>
<td>48.8±5.2</td>
</tr>
<tr>
<td>8</td>
<td>+</td>
<td>Sucrose 28%+Fructose 28%</td>
<td>0.9±0.1</td>
<td>36.2±5.5</td>
</tr>
<tr>
<td>9</td>
<td>+</td>
<td>Sucrose 28%+α-Starch 28%</td>
<td>0.7±0.1**</td>
<td>24.3±3.8*</td>
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<tr>
<td>10</td>
<td>+</td>
<td>Sucrose 28%+Palatinose 28%</td>
<td>0.8±0.1*</td>
<td>29.4±3.0*</td>
</tr>
<tr>
<td>11</td>
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<td>Sucrose 42%+Glucose 14%</td>
<td>1.2±0.1</td>
<td>59.4±6.8</td>
</tr>
<tr>
<td>12</td>
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<td>Sucrose 42%+Fructose 14%</td>
<td>1.3±0.1</td>
<td>60.0±7.5</td>
</tr>
<tr>
<td>13</td>
<td>+</td>
<td>Sucrose 42%+α-Starch 14%</td>
<td>0.9±0.1</td>
<td>42.4±3.7</td>
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<tr>
<td>14</td>
<td>+</td>
<td>Sucrose 42%+Palatinose 14%</td>
<td>0.8±0.1*</td>
<td>40.3±4.1</td>
</tr>
</tbody>
</table>

1, 2) Dental plaque index and caries score were evaluated according to Regolati and Hotz (1972) and Keyes (1944) as modified by Ooshima et al. (1981, 1983).

3) Statistical analyses were carried out between group 2 and the other groups. (* *p* < 0.005, ** *p* < 0.01, t-Test)
In the rats fed glucose, fructose, or a mixture of glucose and fructose, the recovery of strain 6715 was very low at the time of the first bacteriological examination after infection. The level of recovery increased gradually and reached the highest level at the end of the experiment. In the rats fed sucrose-containing diet, serotype g S. mutans was established promptly after infection.

The difference in the implantation of inoculated S. mutans may suggest that the dependency of serotype g S. mutans on sucrose is higher than that of serotype c S. mutans. These findings are in agreement with the results of in vivo experiments by Van Houte et al. (1976) and Tanzer (1979). Furthermore, the results of in vitro experiments using human enamel or nichrome wire (Tinanoff, Tanzer and Freedman, 1978; Dummer and Green, 1981) demonstrated that serotype d S. mutans could attach to these solid surfaces only in the presence of sucrose. On the other hand, serotype c S. mutans could attach, although less tenaciously, to tooth enamel and nichrome wire, even when grown in glucose-containing medium. In a recent investigation in which simultaneous infection of S. mutans serotype c and d occurred (Huis in't Veld, Drost and Havenaar, 1982), serotype d S. mutans could only be established on the rat tooth surfaces when sucrose-containing diet was supplied, whereas serotype c strain was always present in high proportions. Using gnotobiotic rats simultaneously infected with strains of S. mutans serotype a-e, Thomson et al. (1979) showed that serotype d S. mutans required sucrose to colonize and remain on the tooth surface.

![Graph](image)

Fig. 1. Recovery of inoculated S. mutans MT8148R (serotype c, ▢) from oral swabs of rats fed on diet containing sucrose (S), palatinose (P), glucose (G), fructose (F), or a mixture of sucrose and palatinose (SP). For comparison, the recovery of S. mutans 6715 (g) was shown by (□), which was reported in our previous paper (Ooshima et al., 1983).
Rats were sacrificed in 55 days after inoculation of *S. mutans*. Plaque index and caries score were evaluated according to Regolati and Hotz (1972), and Keyes (1944) as modified by Ooshima et al. (1981) (Table I). The rats fed palatinose (rat group 6) or α-starch (group 5)-containing diet showed baseline levels of plaque accumulation and caries incidence, whereas those fed sucrose (group 2) resulted in notable plaque formation and caries induction. The caries-inducing ability of glucose and that of fructose (groups 3 and 4) were similar and approximately half of that of sucrose. This result is in agreement with that of our previous experiment using serotype g *S. mutans* 6715 (Ooshima et al., 1983). Replacement of half of the sucrose content by palatinose (group 10) resulted in a marked reduction in caries incidence, when compared to the caries score of rats fed diet #2000. Rats fed a diet containing 42% sucrose plus 14% palatinose or α-starch induced similar levels of dental caries to that of those fed diet #2000. However, replacement by α-starch (group 9) showed a similar result with that of group 10, which suggest that the decrease in caries score may have been due not to the cariostatic effect of palatinose but to a simple decrease in the sucrose content of the diet.

Huxley (1977) demonstrated that an almost linear response was found between the sucrose content (0, 15, 30 and 56%) of diet #2000 and the caries incidence in Sprague-Dawley rats. Hefti and Schmid (1979) also showed similar results in Osborne-Mendel rats fed diet #2000. Therefore, it appears that the decrease in the sucrose content from 56% to 28% may have a significantly reduced carious lesions in the present investigation. Further studies should be required to elucidate the minimum sucrose concentration to induce maximum dental caries in our experimental caries system.

Of interest is that addition of glucose or fructose to sucrose diet (rat groups 11 and 12 in Table I) increased the caries incidence. Sucrose can contribute to the formation of a heavy dental plaque, and *S. mutans* produces significant amount of lactic acid from glucose or fructose as well as many other sugars. Thus, the presence of sucrose and either glucose or fructose should enhance the cariogenicity of *S. mutans*, as shown in this study. In view of the findings shown in this and previous experiments, the incidence of caries development would significantly be reduced if palatinose is substituted for sucrose in the diet.

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**REFERENCES**


