Intestinal Calcium Absorption in Rats Is Stimulated by Dietary Lactulose and Other Resistant Sugars

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ABSTRACT Lactulose is a disaccharide analogue of lactose that is resistant to metabolism in the small intestine but not in the large intestine. The effects of lactulose and other sugars on intestinal Ca absorption were determined from the decrease in the $^{47}$Ca:$^{75}$Se ratio between diet and feces after feeding male rats diets containing these sugars during a single night. Dietary lactulose was more potent than lactose in stimulating Ca absorption and was effective between 5 and 38 wk of age. The component sugars of lactulose, galactose and fructose, did not influence Ca absorption when provided together at concentrations equimolar to that of lactulose. The stimulation of Ca absorption by dietary lactulose increased as dietary Ca concentration was raised and was not influenced by prior injections of calcitriol. Lactulose must be present in the same meal as Ca to stimulate Ca absorption, but this stimulation was lost if the rats were fed lactulose continuously for 2 or 7 d prior to the test diet. Other sugars thought to be poorly absorbed in the small intestine (xyitol, lactobionate, arabinose, raffinose, pyrogalulate, sorbitol, gluconate and raftlose) stimulated Ca absorption to an identical extent as lactulose. Cecectomy did not influence the enhancement of Ca absorption by lactulose. These results indicate that sugars resistant to metabolism and absorption in the small intestine but not the large intestine stimulate Ca absorption in the small intestine. J. Nutr. 123: 2186-2194, 1993.

INDEXING KEY WORDS:
- rats
- calcium
- sugars
- lactulose
- lactose

Lactose stimulates intestinal Ca absorption in laboratory rats when added to diets at concentrations of 10% or greater (Atkinson et al. 1957). This action of dietary lactose also occurs in vitamin D-deficient rats and, as a consequence of an elevation of Ca absorption, most of the skeletal defects associated with vitamin D deficiency can be corrected [Miller et al. 1988, Schaalma et al. 1988]. Dietary lactose also seems to be capable of increasing bone mass when fed chronically to normal, vitamin D-replete rats (Fournier et al. 1966, Gourvitch 1966).

When rats are fed diets containing lactose, part of this lactose is hydrolyzed to glucose and galactose in the small intestine (Dahlqvist and Thomson 1964, Kim et al. 1978), where these monosaccharides are readily absorbed. The remaining lactose reaches the large intestine (Dahlqvist and Thomson 1964, Kim et al. 1978) and is metabolized (Kim et al. 1979) to volatile fatty acids (acetate, propionate and butyrate acids), thereby decreasing the pH of the fluid within the cecum and colon and producing cecal hypertrophy (Pansu et al. 1978).

Given the presence of lactose and its metabolites throughout the intestine, several theories have been advanced to explain the stimulatory effects of dietary lactose on intestinal Ca absorption. First, the glucose and galactose produced from lactose in the small intestine might stimulate intestinal Ca transport as a consequence of an increase in water absorption (Birlouez-Aragon 1988, Schuette et al. 1989). Secondly, the unhydrolyzed lactose might promote Ca absorption in the jejunum and ileum, because the increased volume of fluid in these segments required to maintain isotonicity might increase the permeability of the intercellular junctions (Bronner 1987). Thirdly, the reduced pH resulting from lactose hydrolysis in the large intestine would be expected to increase the concentration of soluble Ca and thus allow greater rates of Ca absorption by the vitamin D-dependent Ca transport systems present in the cecum (Favus and Angeid-Backman 1985, Nellens and Goldsmith 1981) and colon (Karbach and Rummel 1986, Lee et al. 1980).

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Lactulose is an analogue of lactose that is not metabolized in the small intestine (Dahlqvist and Gryboski 1965, Saunders and Wiggins 1981) but hydrolyzed to the volatile fatty acids in the large intestine (Demigné et al. 1989, Saunders and Wiggins 1981). Thus, if the stimulatory effect of lactose on intestinal Ca absorption results from either the presence of unhydrolyzed lactose in the small intestine or its metabolites in the large intestine, lactulose and other such sugars that are resistant to hydrolysis in the small intestine should have a similar effect while being more potent. Such an action would explain the recently described stimulatory effects of dietary lactitol (Ammann et al. 1988) and xylitol (Hämäläinen et al. 1985) on intestinal Ca absorption.

The purpose of this study was to further characterize the stimulatory action of dietary lactulose (Brommage et al. 1991) and other nonmetabolizable (resistant) sugars on intestinal Ca absorption and determine the potential role of the cecum in this process. These studies were performed using a modification of the in vivo $^{47}$Ca:$^{47}$Sc isotope ratio assay described by McCredie et al. (1984).

**MATERIALS AND METHODS**

Lactulose syrup was purchased from ICN Biochemicals (Cleveland, OH), raftilose “P95” from Raffinerie Tirlemontoise (Tirlemont, Belgium) and the other sugars (including lactulose powder) from Fluka (Buchs, Switzerland); $^{47}$Ca with its daughter isotope $^{47}$Sc was purchased from either Amersham (Little Chalfont, England), Medgenex Diagnostics (Brussels, Belgium) or Riso Isotolaboratoriet (Roskilde, Denmark).

Male rats of the Fischer 344 strain were purchased from IFFA-CREDO (Les Orcins, France), housed in individual cages, and fed a modified AIN-76A purified diet (AIN 1977 and 1980) containing by weight 0.5% Ca as CaCO$_3$, 0.5% P, 20% casein, 7% corn oil, 15% sucrose, 50% cornstarch and 3% cellulose. Additions of lactulose and other sugars were made by omitting an equivalent amount of cornstarch from the test diets. Cecectomy was performed under Nembutal (Abbott Laboratories, North Chicago, IL) anesthesia according to the surgical procedure previously described (Bruckner-Kardoss and Wostmann 1967). In one experiment, rats were injected intraperitoneally under Enflurane (Anaquest, Liberty Corner, NJ) anesthesia with 2 nmol of calcitriol in a volume of 50 µL of ethanol during the morning of the experiment.

Intestinal Ca absorption was determined from the decrease in the ratio of $^{47}$Ca to $^{47}$Sc in feces relative to the diet, using the method of McCredie et al. (1984) modified to permit inclusion of $^{47}$Ca and $^{47}$Sc in the diet rather than giving these isotopes by stomach gavage. The $\gamma$-emitting $^{47}$Ca ($t_{1/2} = 4.53$ d; energies of 78, 130 and 207 fJ) decays to the $\gamma$-emitting $^{47}$Sc ($t_{1/2} = 3.43$ d, energy of 26 fJ). Both isotopes were counted simultaneously by discrimination of their gamma energies. Because $^{47}$Sc is not absorbed by the intestine, it serves as a nonabsorbable marker, and thus only representative samples of diet and feces need to be counted.

The test diets were identical to the standard diet except for the specific changes indicated for each experiment and the inclusion of $^{47}$Ca, $^{47}$Sc, 0.01% Sc as ScCl$_3$ (a carrier for the $^{47}$Sc) and 0.02% of the food dye Fast Green FCF. This dye permitted easy identification of the radioactive feces. The test diets were thoroughly mixed using a kitchen-type blender, and water was added to produce a thick paste from which pellets were made by hand. The formation of these pellets was facilitated by using cornstarch as the major carbohydrate source in the diet. In some experiments, these pellets were dried for 24 h at 60–70°C before being fed to the rats.

On the day of each experiment, the standard diet was removed during the morning and replaced with the radiolabeled test diet in the evening just before darkness. The radiolabeled diet was removed the following morning and replaced with the standard diet. Feces were collected after 3 d, with care taken to count only the green-colored, radioactive fecal pellets. Throughout the experiment the rats remained in their individual plastic cages with wood chips as bedding material and had free access to demineralized water. Thus, an advantage of this assay is that rats consume the test diets under normal feeding conditions.

An Auto-Gamma counter (Packard Instruments, Downers Grove, IL) was employed with the pre-set window for $^{57}$Co/$^{75}$Se used to detect $^{47}$Sc and an energy range of 58 to 250 fJ for $^{47}$Ca. Diet and fecal samples were counted in plastic tubes (11 mm internal diameter and 75 mm height), with care taken to fill each tube to a height of 56 mm to maintain constant counting efficiencies. The sample depth control was set to 3 cm. Appropriate corrections were made for the 21% spillover of $^{47}$Ca counts into the $^{47}$Sc channel. The $^{47}$Ca contents of the test diets were $\sim$0.8 MBq/kg.

Fractional intestinal Ca absorption was calculated from the $^{47}$Ca:$^{47}$Sc ratios in diet [at the time of diet administration and also the time of fecal counting] and feces as described by McCredie et al. (1984), using Lotus 1-2-3 software. Statistical analyses (indicating in the legends to the table and figures) were performed using Student’s $t$ test and a one-factor ANOVA (both with @Library—Statistical Application software as an “add-in” to Lotus 1-2-3) and a two-factor ANOVA (with Statgraphics software). Probability values of <0.001 were regarded as statistically significant.
RESULTS

The stimulation of intestinal Ca absorption by lactose and lactulose at various concentrations in the diet is shown in Figure 1. Lactose increased Ca absorption in an approximately linear fashion but was less potent than lactulose, because similar absorption values were found with diets containing 5% lactulose and 15% lactose. The effect of lactulose was not further increased by rising dietary concentrations from 10 to 15%. As shown in Figure 2, the stimulatory action of lactulose was found when this sugar was provided as either a powder or syrup but was not observed when its two component sugars, galactose and fructose, were added together to the diet at amounts equimolar to that of lactulose. The data presented in Figure 3 demonstrate that dietary lactulose stimulated intestinal Ca absorption in rats between 5 and 38 wk of age. As expected, basal Ca absorption declined continuously during this period.

The influences of dietary Ca level and an injection of calcitriol on the stimulation of intestinal Ca absorption by dietary lactulose are shown in Figure 4. In the absence of lactulose in the diet, fractional Ca absorption was elevated following calcitriol injection and by lowering the dietary Ca concentration. Compared with the 0.5% Ca diet normally employed, the enhancement of Ca absorption by lactulose was eliminated by feeding a 0.2% Ca diet but was further increased by feeding a 1.2% Ca diet. The combined stimulatory actions of lactulose and calcitriol were additive.

Figure 5 presents the results of experiments in which lactulose was fed either before or after the test meal containing $^{47}$Ca. Lactulose must be present in the meal in order to observe its stimulatory effect, because giving lactulose during either of the 2 d after the experiment did not influence the absorption of
the \(^{47}\text{Ca}\) previously consumed. Feeding lactulose continuously for either 2 or 7 d prior to the test meal containing lactulose eliminated the stimulation of intestinal Ca absorption by lactulose.

The lack of specificity of the stimulation of intestinal Ca absorption provoked by lactose and lactulose is presented in Figure 6 and Table 1. Compared with lactulose, virtually identical increases in Ca absorption were produced by similar dietary concentrations of xylitol, lactobionic acid, arabinose, raffinose, pyroglutamic acid, sorbitol, gluconate and raftilose.

Data obtained in rats that were cecectomized at 10 wk of age and fed the test meals with or without lactulose 16 to 18 d later are presented in Figure 7. Cecectomy did not influence intestinal Ca absorption in either the presence or absence of dietary lactulose.

**DISCUSSION**

We employed a simple \(^{47}\text{Ca}^{47}\text{Sc}\) ratio assay to confirm that dietary lactose stimulates intestinal Ca absorption and extended this observation by showing that lactulose and other "resistant" sugars have a similar, but more potent, effect as lactose. An advantage of this assay is that rats consume a pelleted test diet during one night under normal physiological conditions without time for adaptation to occur. In additional experiments employing this assay, we have shown that Ca absorption varies during the normal rat estrous cycle (Brommage et al. 1993) and that Ca absorption is inhibited by dietary oxalate (unpublished data).

Our observation that all sugars that are presumably not metabolized and absorbed in the small intestine

**FIGURE 3** Influence of age on the stimulation of intestinal Ca absorption by dietary lactulose. Rats between the ages of 5 and 38 wk were fed the test diet containing either 0 or 5% lactulose. Values are means ± SEM for eight or nine rats. The effect of lactulose was significantly different from the control group for every age at \(P < 0.001\).

**FIGURE 4** Influence of dietary Ca level and a previous injection of calcitriol on the stimulation of intestinal Ca absorption by dietary lactulose. In both experiments, 13-wk-old rats were studied, and values are means ± SEM for eight or nine rats. In the first experiment [top], the test diet contained various levels of Ca in the presence and absence of 5% lactulose. *Indicates statistically significant differences between lactulose-fed and control rats at \(P < 0.001\). In the second experiment [bottom], 2 nmol of calcitriol was injected intraperitoneally into one half of the rats 7 h before providing the test diet with or without 10% lactulose. The effects of lactulose and calcitriol were both significant at \(P < 0.001\), and these effects were additive but not synergistic.
stimulate intestinal Ca absorption confirms the extensive work on this topic performed almost 40 years ago by Fournier and colleagues. These investigators employed balance techniques in rats to show that, in addition to lactose, mannose (Fournier et al. 1955a), arabinose (Fournier et al. 1955b), xylose (Fournier et al. 1955b) and glucosamine (Fournier 1956), among other sugars, all increased intestinal Ca absorption. Analogous to the term “resistant starches” used to describe starches that are not completely metabolized in the small intestine, we have employed the adjective “resistant” to describe sugars that escape metabolism and absorption in the small intestine. The resistant sugars examined in the present study are all thought to be metabolized by the bacterial flora in the large intestine and the metabolites generated absorbed in this segment. Thus, lactose is a resistant sugar when given to lactase-deficient human subjects or at high dietary levels to rats. Lactose is less potent than lactulose in stimulating intestinal Ca absorption in rats because it is partially hydrolyzed by lactase in the small intestine. Dietary Ca can be provided as salts of gluconate, lactobionate and pyroglutamate, and these Ca salts would be expected to produce higher values of Ca absorption than CaCO\(_3\) as a result of the presence of a “resistant” sugar as an anion.

The ability of dietary lactulose to stimulate intestinal Ca absorption throughout the life of the rats agrees with similar data obtained when 20% dietary lactose was fed chronically (Dupuis and Fournier

**FIGURE 5** Influence of feeding 5% dietary lactulose before and after the test diet. In the first experiment (top), 9-wk-old rats consumed the lactulose diet only on the day of the study (DAY 0) or continuously for either 2 d (DAYS -2 to 0) or 7 d (DAYS -7 to 0) before the test meal containing \(^{47}\text{Ca}\). In the second experiment (bottom), 38-wk-old rats consumed the lactulose diet only on the day of the study (DAY 0) or only on the first day (DAY 1) or only the second day (DAY 2) after the test meal containing \(^{47}\text{Ca}\). In both experiments, a control group of rats (NEVER) consumed only the lactulose-free diet. Values are means ± SEM for eight rats. *Indicates significant differences from the control group at \(P < 0.001\).

**FIGURE 6** Comparison of dietary lactulose with other resistant sugars on the stimulation of the intestinal absorption of Ca. Rats at 17 wk of age were examined. Sugars were added to the test diet at a concentration of 5% (wt/wt). Values are means ± SEM for eight rats. All sugars gave similar values of Ca absorption, but each sugar was significantly different from the control group at \(P < 0.001\).
LACTULOSE AND RESISTANT SUGARS STIMULATE Ca ABSORPTION

TABLE 1

<table>
<thead>
<tr>
<th>Experiment</th>
<th>Age [wk]</th>
<th>Diet</th>
<th>Fractional Ca absorption %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>5</td>
<td>Control</td>
<td>65.0 ± 1.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5% Lactulose</td>
<td>81.3 ± 1.6*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5% Sorbitol</td>
<td>84.8 ± 1.8*</td>
</tr>
<tr>
<td>2</td>
<td>6</td>
<td>Control</td>
<td>60.6 ± 1.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5% Lactulose</td>
<td>76.9 ± 2.6*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4.9% Gluconate</td>
<td>78.8 ± 2.3*</td>
</tr>
<tr>
<td>3</td>
<td>38</td>
<td>Control</td>
<td>12.9 ± 0.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5% Lactulose</td>
<td>22.2 ± 1.0*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5% Raftilose</td>
<td>11.6 ± 1.6*</td>
</tr>
</tbody>
</table>

1Values are means ± sem for eight or nine rats. Gluconate was provided as an equimolar mixture of D-gluconic acid and sodium D-gluconate at a concentration (4.9%) calculated to be equimolar to the amount of carbonate present in a diet containing 0.5% Ca as CaCO₃. *P < 0.001 compared with control group using Student’s t test.

1964) and also with observations made using the ligated intestinal loop technique (Armbrecht 1987). Observations that dietary lactose increases Ca absorption in vitamin D-deficient rats and that the actions of lactulose and calcitriol are additive both strongly suggest that resistant sugars stimulate the passive rather than the vitamin D-dependent, active component of Ca absorption.

The hypothesis that the stimulation of intestinal Ca absorption by dietary lactulose and other resistant sugars occurs in the cecum is attractive because the cecum can readily absorb Ca both in vitro (Favus and Angeil-Backman 1985, Nellens and Goldsmith 1981) and when perfused in vivo (Ammann et al. 1986, Petith et al. 1979), and feeding lactulose is known to increase the concentration of Ca in the cecal fluid (Demigné et al. 1989). However, three separate lines of evidence argue against this hypothesis. First, cecctomized rats absorbed Ca to the same extent as the sham-operated rats in both the presence and absence of dietary lactulose. Cecctomy has previously been reported to be without effect on the enhanced intestinal Ca absorption produced by dietary lactose (Fournier et al. 1960) and vitamin D repletion (Innes and Nicolaysen 1937). Secondly, lactose stimulates Ca absorption in both germ-free and conventional rats (Andrieux et al. 1982, Andrieux and Sacquet 1983), indicating that the low pH and high Ca concentrations resulting from the metabolism of resistant sugars in the cecum are not related to Ca absorption. Thirdly, Andrieux and Sacquet (1983) measured the ratio of Ca to the nonabsorbable marker TiO₂ within various intestinal segments and calculated that the stimulatory action of lactose was manifested in the small intestine rather than the large intestine in conventional rats. These authors did observe that in germ-free rats Ca was absorbed in the cecum and part of the stimulation provoked by lactose could be explained by enhanced cecal Ca absorption. Nonetheless, the best available evidence indicates that under normal conditions the small intestine, and not the cecum, is the site of the stimulation of Ca absorption by resistant sugars.

Because the stimulation of intestinal Ca absorption produced by resistant sugars does not involve the metabolism and absorption of these sugars in the small intestine, the increased water absorption resulting from lactose metabolism in the perfused intestine (Birlouez-Aragon 1988, Schuette et al. 1989) does not seem to be related to Ca absorption following a meal. This conclusion is not surprising, because in diets or meals lactose usually replaces an absorbable sugar having a similar influence on water absorption.

The presence of osmotically active resistant sugars in the small intestine must increase the amount of fluid within the lumen to maintain isotonicity. As postulated by Bronner (1987), this additional fluid might increase the distention and permeability of the intracellular junctions between enterocytes, thereby increasing the passive absorption of Ca and other elements. Such a mechanism of action for the stimulation of intestinal Ca absorption by resistant sugars

![FIGURE 7 Effect of cecectomy on the stimulation of intestinal Ca absorption by dietary lactulose. Rats were either sham-operated (sham) or cecctomized (CCX) at 10 wk of age, and intestinal Ca absorption measured 2.5 wk later. Lactulose was added at a concentration of 5% (wt/wt) to the test diet. Values are means ± SEM for seven to eight rats. Lactulose (P < 0.001) but not cecectomy (P > 0.15) had a significant effect on Ca absorption.](https://academic.oup.com/jn/article-abstract/123/12/2186/4723330)
is entirely consistent with our data and those of others. These data include the greater potency of lactulose than lactose, the independence of age and vitamin D status, the noninvolvement of the cecum, the effect of increasing dietary Ca concentration, and the lack of specificity of the various resistant sugars. In further support of this hypothesis, Pansu et al. (1976) demonstrated that increasing the fluid content of jejunal loops in situ by filling them with hypertonic solutions stimulated Ca transport.

The failure of lactulose to continue to enhance total intestinal Ca absorption when fed chronically presumably resulted from a decrease in active Ca transport that counterbalanced the lactulose-induced increase in passive Ca absorption. This adaptive response is presumably mediated by decreased serum concentrations of calcitriol. Although changes in serum calcitriol concentrations in response to dietary lactose or resistant sugars have not been studied, Pansu et al. (1978 and 1979) have shown that lactose-fed rats have lower active Ca transport and Ca-binding protein levels in the duodenum. These two changes are well-known indicators of reduced serum calcitriol concentrations. Our finding of an adaptation to lactulose feeding was observed with 5% lactulose in the diet. Undoubtedly, the capacity for this adaptation is limited, and providing higher levels of lactulose would presumably have resulted in continued elevated intestinal Ca absorption. This hypothesis is consistent with previous studies in which Ca absorption and bone mass were increased in rats fed diets containing 35% lactose (Fournier et al. 1966, Gourvitch 1966) or 16% sorbitol (Fournier et al. 1967) for several months.

The results of studies examining the effects of lactose and resistant sugars on intestinal Ca absorption in humans are conflicting. Ziegler and Fomon (1983) employed a 3-d balance technique in infants to show that a formula containing lactose as the sole carbohydrate increased Ca absorption from 33 to 48% compared with a similar formula containing cornstarch hydrolysate and sucrose as carbohydrates. However, Wirth et al. (1990), also using a 3-d balance technique, found no difference in Ca absorption in premature infants fed formulas containing either lactose as the sole carbohydrate or a 50:50 mixture of lactose and polymers of glucose.

In adult humans, all of the present evidence indicates that removing the lactose normally present in milk (or reconstituted milk powder) by lactase treatment does not influence intestinal Ca absorption. This lack of an effect of lactose has been observed in both lactase-deficient (Griessen et al. 1989a, Kocián et al. 1973, Tremaine et al. 1986) and lactase-sufficient (Griessen et al. 1989a, Kocián et al. 1973, Schuette et al. 1991, Tremaine et al. 1986) subjects. The amount of lactose present in these studies varied from 12 to 75 g and the Ca intake from 200 to 970 mg. Interestingly, the rate of appearance of milk Ca in serum was increased by lactose in normal subjects but delayed in lactase-deficient subjects (Kocián et al. 1973).

Adding lactose to a test meal did stimulate intestinal Ca absorption in two studies involving lactase-sufficient subjects. Schuette et al. (1991) added 12 g of lactose to an artificial milk containing 217 mg of Ca and observed a 25% increase in Ca absorption. Likewise, a 59% stimulation of Ca absorption was found by Cochet et al. (1983) when their subjects drank a solution containing 500 mg of Ca as CaCl₂ to which 50 g of lactose was added.

Three studies indicate that resistant sugars can reduce intestinal Ca absorption. Cochet et al. (1983) observed that 50 g of lactose, added to a solution of 500 mg of Ca as CaCl₂, decreased Ca absorption by 18% in lactase-deficient subjects. This same group also observed that 15 g of lactitol, again added to a drink containing 500 mg of Ca as CaCl₂, decreased Ca absorption by 15% in normal subjects (Griessen et al. 1989b). The resistant sugar sorbitol, at a dose of 10 g added to CaCl₂ solutions containing either 20 mg or 500 mg of Ca, inhibited Ca absorption when compared with glucose (Francis et al. 1986).

All of these studies in adult humans have employed established assays involving tracer methodologies to quantify intestinal Ca absorption. Opposite results have been found by Pansu and colleagues when intestinal Ca absorption was monitored by the less rigorous technique of feeding subjects 500 mg of Ca as calcium gluconate in the presence of various sugars and then measuring acute increases in serum Ca concentrations. With this assay, 100 g of lactose was effective in raising serum Ca concentrations in lactose-deficient subjects but not in normal subjects (Pansu et al. 1969). Similarly, xylose and sorbitol were more hypercalcemic than glucose in normal subjects (Pansu and Chapuy 1970).

The experimental data in adults can be summarized by stating that the lactose present in milk does not influence intestinal Ca absorption but, under certain conditions in lactase-sufficient subjects, lactose can improve Ca absorption. The resistant sugars lactitol and sorbitol, and lactose in lactase-deficient subjects, decrease Ca absorption when added to a drink containing only CaCl₂. Resistant sugars have not yet been examined as part of a regular meal composed of protein, carbohydrate and fat. This issue is particularly important because additional nutrients will certainly influence the transit time of resistant sugars through the small intestine.

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