Fermentation and bulking capacity of indigestible carbohydrates: 
the case of inulin and oligofructose

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The bulking index (i.e. the increase in faecal fresh weight in gram per gram indigestible 
carbohydrate ingested) with oligofructose and inulin is similar to that produced with other 
easily fermented fibres such as pectins and gums. Most studies in man have been performed 
at a level of 15 g/d and more investigations on lower intakes are needed to appoint the least 
intake for an effect. Concerning short-chain fatty acids (SCFA) most studies have been using 
oligofructose and points at an increased butyric acid formation in the caecum of rats. In one 
study on rats with inulin high caecal proportions of propionic acid were obtained. As inulin 
has a higher molecular weight than oligofructose it might be speculated if this could be a 
reason to the different SCFA-profile formed. No effects on faecal concentrations of SCFA in 
humans have been revealed with inulin and oligofructose, which neither is expected as most 
of the SCFA formed during the fermentation already has been absorbed or utilized by the co-
lonic mucosa.

Bulking index: SCFA: Inulin: Oligofructose

Introduction

Carbohydrates, which are not digested nor utilized in the 
small intestine, reach the large intestine where they can 
be partly or totally degraded by the microflora. During 
this fermentation short-chain fatty acids (SCFA), mainly 
acetic-, propionic- and butyric acid, and gases are formed 
(Macfarlane & Cummings, 1991). Carbohydrates resistant 
to fermentation usually have a high bulking effect, thus 
reducing the risk for constipation and possibly also colonic cancer (Cummings et al. 1992). Easily fermentable carbo-
hydrates, on the other hand, may also be of nutritional 
interest as some of the short-chain fatty acids formed 
have been suggested to be of physiological importance. 
Butyric acid is the preferred energy substrate for the 
colonic mucosa and has been suggested to protect against 
colonic disease, e.g. ulcerative colitis and cancer (Scheppach et al. 1992; Lupton, 1995; Gamet et al. 1992). The proportion of propionic- and acetic acid, on the other hand, appears to affect glucose- and lipid metabolism beneficially and the higher the amount of pro-
pionic acid the more pronounced are the effects (Wolever et al. 1991). An increase in the SCFA-formation also 
leads to a decreased pH that may stimulate mineral 
absorption in the colon and increase the blood flow.

The most well-known source of indigestible carbo-
hydrates are the non-starch polysaccharides. Other 
examples are the oligosaccharides like the fructans found 
in, for example onion, chicory and artichoke and the 
α-galactosides found in legumes as well as some starch 
fractions that for various reasons may reach the colon 
undigested.

Bulking capacity

Carbohydrates resistant to bacterial fermentation increase 
the bulk passing through the colon. For some of the resist-
ant types of carbohydrates, the capacity to hold water may 
add to this effect. Different types of carbohydrates are fer-
mented to various degrees, and factors of importance are, 
for example the monomeric composition of the saccharides 
and type of glycosidic linkages present (Nyman, 1985). 
The degree of polymerization, the solubility and the struc-
tural arrangement of the carbohydrates may also be important 
for the degree of fermentation. Food processing may 
change some of these properties, and as a result the fermentability and the bulking effects will be altered (Svanberg et al. 1995).

Abbreviations: SCFA, short-chain fatty acids.

Note: For the definition of the terms inulin and oligofructose please refer to the introductory paper (p. S139) and its footnote.

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From *in vivo* studies on rats it can be concluded that the extent of fermentation varies for different types of non-starch polysaccharides and resistant starches. Cellulose, oat husk, ispaghula and sterculia are examples of non-starch polysaccharides that are highly resistant against microbial degradation, whereas β-glucans, pectins and gums are rapidly fermented (Nyman & Asp, 1985; Berggren et al. 1993). Similarly native resistant starch has been reported to be less fermented than retrograded starch (Schultz et al. 1993). Oligosaccharides, on the other hand, seem to be more or less completely fermented (Berggren et al. 1993). Nevertheless, easily fermentable types of carbohydrates have been reported to have a certain faecal bulking effect, due to an increased bacterial mass (Macfarlane & Cummings, 1991). Thus, bulking index (i.e. increase in fresh faecal weight in g/g of indigestible carbohydrates eaten) for pectin has been shown to be around one (Table 1), while that with wheat bran has been reported to be between 1.3 and 2.2 (Armstrong et al. 1993; Hansen et al. 1992; Nyman & Asp, 1985). The comparatively low bulking index of wheat bran is due to its low capacity to hold water. Bulking index for inulin and oligofructose when fed at a similar level as wheat bran and pectin has been reported to be around one (Roberfroid et al. 1993). However, the bulking index obtained in rats must be interpreted with care and cannot necessarily be expected to be indicative for man. It is difficult to determine faecal wet weight accurately in rats and human faeces are generally much smoother with higher water content than faeces of rats. A more relevant measure when extrapolating rat data to humans is probably faecal dry weight increment per gram of fibre taken. Both fibre fermentability and faecal dry weight increment per gram of fibre taken has been shown to be well correlated between humans and rats (Nyman et al. 1986).

### Human studies

Most studies in humans use six to twelve healthy volunteers, and the subjects have been given doses of 15 g indigestible carbohydrates per day for 2–5 weeks. Exclusion criteria for the studies have been use of antibiotics for at least the last 6 months and previous gut disorders.

When oligofructose was given to healthy volunteers (N = 8) the bulking index was reported to be between 1.0 and 1.2 (Gibson et al. 1995) but inulin, induced a higher bulking index of 2.1. However, as very few persons participated in the study (n = 4) it is difficult to draw any conclusion about the influence of the molecular weight. A very similar bulking index as with oligofructose (1.5) was also obtained when inulin with a higher chain length (average DP = 25) was given in doses of 5 g three times daily to slightly constipated volunteers (Den Hond et al. 2000), suggesting minor importance of the molecular weight on faecal bulking capacity. Neither a higher dose had any effect and such a high dose as 50 g gave a very similar...
Table 2. SCFA formed at fermentation of inulin and oligofructose compared to some other indigestible carbohydrates by human faecal bacteria in vitro, in the rat caecum or in human faeces

<table>
<thead>
<tr>
<th>Carbohydrate</th>
<th>Level</th>
<th>Duration*†</th>
<th>SCFA concentration‡**††‡‡</th>
<th>SCFA-profile (%) C,&lt;sub&gt;2&lt;/sub&gt; C,&lt;sub&gt;3&lt;/sub&gt; C,&lt;sub&gt;4&lt;/sub&gt;</th>
<th>Model</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oligofructose‡</td>
<td>6</td>
<td>14</td>
<td>35.7</td>
<td>4.69 10.53 0.3 0.0</td>
<td>70.9:21</td>
<td>Rats, n = 10</td>
</tr>
<tr>
<td>Oligofructose (from sucrose) ‡</td>
<td>6</td>
<td>14</td>
<td>44.64</td>
<td>5.44 9.74 0.54</td>
<td>75.9:16</td>
<td></td>
</tr>
<tr>
<td>Xylooligosaccharides‡</td>
<td>6</td>
<td>14</td>
<td>35.34</td>
<td>4.90 5.95 1.30</td>
<td>76.11:13</td>
<td></td>
</tr>
<tr>
<td>Oligofructose§</td>
<td>10</td>
<td>56</td>
<td>31.42</td>
<td>8.65 14.83 1.65</td>
<td>57.16:27</td>
<td>Germ-free rats inoculated with human faecal flora, n = 6</td>
</tr>
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<td>Carrot§</td>
<td>10</td>
<td>56</td>
<td>32.16</td>
<td>8.80 3.66 2.63</td>
<td>72.20:8</td>
<td></td>
</tr>
<tr>
<td>Wheat bran§</td>
<td>10</td>
<td>56</td>
<td>28.40</td>
<td>9.42 4.14 1.63</td>
<td>68.22:10</td>
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<tr>
<td>Pea§</td>
<td>10</td>
<td>56</td>
<td>23.91</td>
<td>9.44 3.12 1.10</td>
<td>66.26:8</td>
<td></td>
</tr>
<tr>
<td>Oats§</td>
<td>10</td>
<td>56</td>
<td>23.27</td>
<td>9.15 3.20 2.01</td>
<td>65.26:9</td>
<td></td>
</tr>
<tr>
<td>Inulin†</td>
<td>5</td>
<td>21</td>
<td>54.6</td>
<td>19.8 10.3 0.0</td>
<td>65.23:12</td>
<td>Rats, n = 12</td>
</tr>
<tr>
<td>Inulin‡</td>
<td>10</td>
<td>21</td>
<td>66.2</td>
<td>58.4 31.0 19.3</td>
<td>42.38:20</td>
<td></td>
</tr>
<tr>
<td>Inulin†</td>
<td>20</td>
<td>21</td>
<td>42.6</td>
<td>35.9 28.1 30.7</td>
<td>40.34:26</td>
<td></td>
</tr>
<tr>
<td>Oligofructose (from sucrose)</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>– 3.66</td>
<td>62.13:25</td>
<td>In vitro, 6 h</td>
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<tr>
<td>Lactulose</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>– 3.66</td>
<td>74.7:19</td>
<td></td>
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<tr>
<td>Oligofructose**</td>
<td>15</td>
<td>15</td>
<td>76.8</td>
<td>23.0 18.5</td>
<td>65.22:13</td>
<td>In vitro, 6 h</td>
</tr>
<tr>
<td>Sucrose**</td>
<td>15</td>
<td>15</td>
<td>64.3</td>
<td>21.8 16.0</td>
<td>64.21:15</td>
<td>Humans, n = 8</td>
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<tr>
<td>Placebo††</td>
<td>–</td>
<td>7</td>
<td>22.8</td>
<td>3.6 2.3</td>
<td>80.12:8</td>
<td>Humans with ileal pouch-anal anastomosis, n = 16</td>
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<tr>
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<td>14.3</td>
<td>7</td>
<td>25.2</td>
<td>2.8 2.9</td>
<td>82.9:9</td>
<td></td>
</tr>
<tr>
<td>Resistant starch††</td>
<td>14.3</td>
<td>7</td>
<td>26.7</td>
<td>3.4 3.9</td>
<td>79.10:11</td>
<td></td>
</tr>
<tr>
<td>Inulin‡‡</td>
<td>20</td>
<td>7</td>
<td>137.5</td>
<td>60.5 33.1 6.0</td>
<td>60.26:14</td>
<td>Humans, n = 10</td>
</tr>
<tr>
<td>Inulin‡†</td>
<td>40</td>
<td>7</td>
<td>114.3</td>
<td>42.3 31.6 8.0</td>
<td>61.22:17</td>
<td>Humans, n = 15</td>
</tr>
<tr>
<td>Lactose‡†</td>
<td>20</td>
<td>7</td>
<td>137.6</td>
<td>45.4 49.0 5.7</td>
<td>59.20:21</td>
<td></td>
</tr>
<tr>
<td>Lactose‡‡</td>
<td>40</td>
<td>7</td>
<td>133.4</td>
<td>39.0 34.7 6.0</td>
<td>64.19:17</td>
<td></td>
</tr>
<tr>
<td>Rice‡‡</td>
<td>40-4</td>
<td>28</td>
<td>42.93</td>
<td>14.87 15.28 3.21</td>
<td>59.20:21</td>
<td>Humans n = 12</td>
</tr>
<tr>
<td>Inulin**</td>
<td>19-5</td>
<td>28</td>
<td>33.26</td>
<td>11.99 12.82 7.39</td>
<td>57.21:22</td>
<td></td>
</tr>
<tr>
<td>Oligofructose‡</td>
<td>40</td>
<td>3</td>
<td>73</td>
<td>18 19 2</td>
<td>67.16:17</td>
<td>Humans, n = 12</td>
</tr>
<tr>
<td>Oligofructose‡</td>
<td>80</td>
<td>3</td>
<td>87</td>
<td>14 19 28</td>
<td>72.12:16</td>
<td></td>
</tr>
<tr>
<td>Lactulose‡</td>
<td>40</td>
<td>3</td>
<td>72</td>
<td>14 17 9</td>
<td>70.14:16</td>
<td></td>
</tr>
<tr>
<td>Lactulose†</td>
<td>80</td>
<td>3</td>
<td>80</td>
<td>9 12 28</td>
<td>79.9:12</td>
<td></td>
</tr>
</tbody>
</table>

* Level of indigestible carbohydrate in the diet given to rats is represented in g/100 g.
† Level of daily intake in humans is represented in g.
‡ SCFA-concentration in caecum of rats in mmol/l.
§ SCFA-concentration in caecum of rats in mmol/g.
** SCFA-concentration in mmol/kg wet faeces.
†† SCFA concentration in faeces in mmol/d.
‡‡ SCFA-concentration in μmol/g dry faeces.
bulking index as the lower doses used (Castiglia-Delavaud et al. 1998).

Due to a high production of gases during the fermentation of easily fermentable carbohydrates high levels of oligosaccharides can lead to intestinal discomfort. Therefore it may be interesting to know the minimum dose to obtain an effect. Not many studies have been performed at levels lower than 15 g/d, but Menne et al. (2000) concluded a tendency to confirmation of the bulking effect previously reported, with only 8 g oligofructose per day. They also reported a change in stool frequency and appearance (softer) of the stool. Further, an increased stool weight and stool frequency has been reported with oligofructose (from sucrose) at levels as low as 3 g/d for subjects having a low stool frequency (Tominaga et al. 1999).

The bulking index obtained with inulin and oligofructose thus seems to be in the similar range as reported for easily-fermented types of dietary fibre such as pectin (1–2), guar gum (1–2) and sugar-beet fibre (1·5) (Cummings, 1984; Spiller, 1993; Castiglia-Delavaud et al. 1998), but lower than that of wheat bran (2·5–5), one frequently consumed resistant type of fibre (Cummings, 1984; Spiller, 1993; Spiller et al. 1986). The laxative effect of inulin has also been reported to be better, as judged by the higher stool frequency, than with lactose in constipated elderly, when given in doses from 20 to 40 g (Kleeßen et al. 1997). In contrast, other studies have reported minor effects with oligofructose and inulin (Alles et al. 1996; van Dokkum et al. 1999). Thus, faecal weight was not affected in healthy men when added to the ordinary diet at a level of 5 and 15 g/d, which could be due to the fact that these patients already had a high intake of non-starch polysaccharides (40 g/d) (Alles et al. 1996).

In conclusion, inulin and oligofructose seem to have a similar bulking effect as gums and pectins. However, more large-scale studies are needed. Further, most studies have been performed on inulin and oligofructose at a level of 15 g/d or more. Further studies on lower intakes are therefore needed.

**Short-chain fatty acids**

The pattern of short-chain fatty acids (SCFA) formed upon fermentation of easily fermentable carbohydrates is another factor of nutritional importance. Butyric acid appears to be essential in the maintenance of a healthy colonic mucosa, whereas propionic acid is increasingly connected with beneficial effects on carbohydrate and lipid metabolism. SCFA may also influence colonic motility. Of special interest is that various indigestible carbohydrates give rise to different SCFA patterns (Berggren et al. 1993). This might be due to the fact that the substrate favours growth of colonic bacteria, producing a specific short-chain fatty acid pattern.

The formation of SCFA is not that easy to measure and different methodologies are used to analyse the formation and pattern of short-chain fatty acids. Studies in humans focus on the SCFA found in faeces, and not at the actual site for fermentation. To enable evaluation of SCFA production from various substrates, relevant experimental models are therefore needed. Examples of such models are in vitro incubations with human or animal faeces, or animal studies. Studies in rats mostly focus on short-chain fatty acid pattern in the caecum.

**Animal studies**

Particularly high amounts of propionic acid have been obtained with guar gum, and nearly 30 % of the SCFA has been shown to be propionic acid, whereas β-glucans, raffinose and oligofructose (from sucrose) promoted a high production of butyric acid (15–22 %) (Berggren et al. 1993). Interestingly, dietary fibre rich in uronic acids (pectin and linseed fibre) gave very low levels of butyric acid, only 7 or 4 %, respectively. This is noteworthy, in light of the fact that uronic acid containing fibres, like pectin but also fibre isolated from carrots, appear to stimulate chemically induced cancer in rats (Bauer et al. 1981).

Inulin and oligofructose, appear to be highly fermented both in vitro and in vivo, yielding high levels of short-chain fatty acids. Most studies have been performed on oligofructose (Table 2). Thus, in a study on rats, oligofructose at a level of 6 g/100 g gave a higher caecal concentration of butyrate (10·53 and 9·74 mmol/l) compared with the same amount of xylo-oligosaccharides (5·95 mmol/l) (Campbell et al. 1997). No significant differences were obtained in the concentration of acetate, propionate or lactate. Similarly, in a study on germ-free rats, inoculated with human faecal flora, oligofructose (10 g/100 g) gave a four-fold increase in caecal concentration of butyrate (14·83 mmol/g) compared to 3·66, 4·14, 3·12 or 3·20 mmol/l for dietary fibre from carrots, wheat bran, peas and oats, respectively (Roland et al. 1995). However, all these food items, except carrots, are relatively resistant against fermentation that may explain the low caecal concentrations of SCFA. The amount of propionate was not affected by diet in this study either, while the yield of acetate was lower with peas and oats than in the other materials. A significantly higher caecal concentration of butyrate with oligofructose has also been obtained by Poulsen & Molck (2002). They found that there was a significant increase in butyrate concentration when rats were given oligofructose (15 g/100 g) for 5 or 10 weeks, independently if the rats were administered with dimethylhydrazine or not, compared with the other groups studied (inulin 5 g/100 g and 15 g/100 g or oligofructose 5 g/100 g).

Some studies have also been performed on inulin. There was a significant dose-dependent increase in the total amount of caecal SCFA and of lactic acid when rats received inulin at the level of 0, 5, 10 or 20 g/100 g (Levrat et al. 1991). The proportion of propionic acid formed was very high (34–38 %) compared with studies on oligofructose, and it may be speculated that the higher molecular weight of inulin is a reason for this. The high level of lactic acid formed at the level of 20 g/100 g with inulin led to very acidic conditions (pH 5·65) and as proposed by the authors to an accelerated absorption of SCFA.

**In vitro studies**

Studies in vitro, with human faecal inoculum, have also
shown that different substrates give various SCFA patterns. Starch has been shown to yield high proportions of butyric acid, and arabinogalactans high proportions of propionic acid (Englyst et al. 1987). In vitro fermentation of oligofructose (from sucrose) resulted in higher molar ratios of propionate and butyrate (13 and 25%, respectively) than with lactulose (7 and 19%, respectively) (Luo et al. 1996), but lower than many types of starches (Ferguson & Jones, 2000). However, Clausen et al. (1998) found very similar proportions of acetate, propionate and butyrate with both lactulose and oligofructose.

**Human studies**

Generally no effect on faecal concentration or of molar proportions of acetate, propionate and butyrate has been seen with inulin or oligofructose in humans (Gibson et al. 1995; Alles et al. 1996; Alles et al. 1997; Kleessen et al. 1997; Clausen et al. 1998; Brighenti et al. 1999; Kruse et al. 1999). However, an increased concentration of lactic acid at higher doses (80 g/d) has been reported (Clausen et al. 1998). Further, in the study by Kleessen et al. (1997) the proportion of butyrate tended to be higher at the higher doses (40 g v. 20 g) with inulin, but these data are a bit confusing.

In conclusion, most studies on SCFA have used oligofructose and points at an increased amount of butyric acid in the caecum of rats. In humans no effects on faecal SCFA can be seen. In one study on rats using inulin very high proportions of propionic acid were obtained, and more studies on inulin are therefore needed. Although inulin is completely fermented the degradation may be slower than with oligofructose, which might give rise to another SCFA pattern. Changes in SCFA pattern may also be reflected in faeces with a slowly fermentable carbohydrate. The place of fermentation is important to evaluate, as most colonic diseases appear in the distal end. It is important to study the SCFA pattern of fructo-oligosaccharides in combinations with other food items.

**References**


