Microbial Degradation of Whole-Grain Complex Carbohydrates and Impact on Short-Chain Fatty Acids and Health1–4

Knud Erik Bach Knudsen*
Department of Animal Science, Aarhus University, Tjele, Denmark

ABSTRACT

Whole-grain cereals have a complex dietary fiber (DF) composition consisting of oligosaccharides (mostly fructans), resistant starch, and nonstarch polysaccharides (NSPs); the most important are arabinoxylans, mixed-linkage β(1,3; 1,4)-D-glucan (β-glucan), and cellulose and the noncarbohydrate polyphenolic ether lignin. The highest concentration of NSPs and lignin is found in the outer cell layers of the grain, and refined flour will consequently be depleted of a large proportion of insoluble DF components. The flow and composition of carbohydrates to the large intestine are directly related to the intake of DF. The type and composition of cereal DF can consequently be used to modulate the microbial composition and activity as well as the production and molar ratios of short-chain fatty acids (SCFAs). When arabinoxylans were provided as a concentrate, the effect was only on total SCFA production. Increased SCFA production in the large intestine was shown by the concentration in the portal vein, whereas the impact on the concentration in peripheral blood was less because the majority of propionate and butyrate is cleared in the liver. Active microbial fermentation with increased SCFA production reduced the exposure of potentially toxic compounds to the epithelium, potentially stimulating anorectic hormones and acting as signaling molecules between the gut and the peripheral tissues. The latter can have implications for insulin sensitivity and glucose homeostasis.

Introduction

Several epidemiologic studies have indicated that the consumption of whole-grain cereals is protective against several chronic diseases such as cardiovascular diseases, type 2 diabetes, and some forms of cancer (1–4). Whole-grain cereals are a rich source of dietary fiber (DF)5 and bioactive compounds. The DF components, however, are unevenly distributed in the grain; the outer tissues (pericarp/testa, aleurone) contain the highest concentrations of DF (3, 5), and depleting the flour of the outer tissues may eliminate ~58% of DF, in particular its insoluble components (3).

DFs are the fraction of carbohydrates and lignin that are not degraded by endogenous enzymes in the small intestine (6). Although some degradation occurs in the small intestine, the majority portion passes to the large intestine, where it is fermented by the colonic microbiota (7). Fermentation of DF carbohydrates leads to the production of SCFAs, mainly acetate, propionate, and butyrate; gases (hydrogen and methane); and other metabolites (8). The ratio of SCFA production and absorption is dependent on fermentation substrate, the microbial composition, and colonic transit time (9). The substrate dependency has been used as a subject to manipulate the SCFA composition (10, 11). Most SCFAs are absorbed by the colonic epithelium or metabolized by other colonic bacteria, resulting in only 5–10% of SCFAs being excreted in feces (8). SCFAs play an important role in gut and potentially metabolic health (9, 12). The main purpose of the present review is to discuss current

Keywords: whole-grain cereals, complex carbohydrates, short-chain fatty acids, butyrate
knowledge concerning the microbial degradation of complex carbohydrates from cereals and influence on SCFA production and impact on health.

**Complex Carbohydrates in Whole-Grain Cereals and Refined Products**

The DF fraction of cereals consists of nonstarch polysaccharides (NSPs), resistant starch (RS), oligosaccharides (mostly fructans), and the noncarbohydrate polyphenolic ether lignin. The main NSPs in cereals are arabinoxylans, mixed-linkage β(1,3; 1,4)-D-glucan (β-glucan), and cellulose. These polymers are present in the cell walls along with lignin, lignans, phenolic acids, and minor amounts of protein. The cell walls of the different tissues have a complex structure with variable properties and composition (13). The cell walls from the outer part of the kernel primarily play a role in protection, and the cell walls in these tissues are consequently thick, hydrophobic, and consist of cellulose, xylans, and significant amounts of lignin. In endosperm tissues that include the aleurone layer, the cell walls are thin and hydrophilic and are primarily made up of 2 polymers: arabinoxylans and β-glucan. The cell wall polysaccharides of the aleurone, although part of the endosperm, are largely insoluble, in contrast to the cell wall polysaccharides of the remaining endosperm (14, 15). The compositions as well as the fine structure of cell walls of similar types of tissue differ: wheat and rye are more similar than are oats, barley, and corn (13–15). The cereal type and relative proportion of tissues in the flour fraction therefore have a great impact on NSPs and lignin concentrations as shown in Table 1. Dry milling is a convenient way to concentrate tissues with specific functional properties: for example, oat bran has a high content of β-glucan and aleurone from wheat has a high content of arabinoxylans (5, 16).

RS is the fraction of starch that is not digested in the small intestine. Starch may be nondigestible because it is trapped within whole plant cell matrices (RS₁), the starch granules are resistant (RS₂), the starch is retrograded (RS₃), or if it is chemically modified (RS₄) (17). The amounts of RS are usually low in common cereal starches, with an amylose content of 20–30%, whereas high-amylose starch types may contain higher amounts (18). RS can also be formed in connection with the cooling process of a starch gel after heating (18).

Fructans consist of 1 glucose unit linked to a varying number of fructose units (3–20) present predominantly in the endosperm and germ tissues (19). Both branched and unbranched chains occur.

**Regulation of the Flow of Substrate for Fermentation in the Large Intestine**

Individuals who have undergone an ileostomy have been used as a model to study and quantify the amount of nutrients that pass from the small to the large intestine and thereby potentially can be available for fermentation (Figure 1). In studies with cereal products (soft and crisp breads and breakfast cereals), there is a strong relation between the intake of DF (NSPs + RS) and the flow of dry solids and carbohydrates to the large intestine (20–24). Carbohydrates account for 30–43% of the dry solids. The structure of the starch granule in cereals (20–30% amylose content) makes starch relatively easily accessible for amylolysis in the small intestine when provided as breads or breakfast cereals. The luminal viscosity created by the soluble β-glucan and arabinoxylans may delay the digestion and absorption processes and thereby impede starch hydrolysis (25, 26). However, most studies showed that, quantitatively, starch digestion was only marginally affected, by 1.3% and 1.9%, after consumption of low- and high-DF diets, respectively (21, 23). This is consistent with studies in ileal-cannulated pigs fed wheat or rye breads (27, 28); although intact kernels may reduce the digestibility of starch, the reduction was <1% (29).

No endogenous carbohydrases capable of cleaving the NSP bonds are secreted to the small intestine in humans. Nevertheless, degradation of NSPs occurs during passage in the small intestine because of microbial carbohydrates. The molecular weight reduction in linear β-glucans is substantial in humans and pigs in contrast to the negligible degradation of the more structurally complex arabinoxylans from wheat,

---

**Table 1.** Starch and NSP content of whole grain, refined flour, and bran from wheat, rye, and oats

<table>
<thead>
<tr>
<th></th>
<th>Wheat</th>
<th>Rye</th>
<th>Oats</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Flour</td>
<td>Bran</td>
<td>Flour</td>
</tr>
<tr>
<td>Extraction rate, %</td>
<td>100</td>
<td>~60</td>
<td>100</td>
</tr>
<tr>
<td>Starch, g/kg DM</td>
<td>696</td>
<td>832</td>
<td>55</td>
</tr>
<tr>
<td>NSP, g/kg DM</td>
<td>9</td>
<td>6</td>
<td>17</td>
</tr>
<tr>
<td>Fructans</td>
<td>19</td>
<td>2</td>
<td>114</td>
</tr>
<tr>
<td>Cellulose</td>
<td>6</td>
<td>2</td>
<td>24</td>
</tr>
<tr>
<td>β-Glucan</td>
<td>71</td>
<td>21</td>
<td>337</td>
</tr>
<tr>
<td>Arabinoxylans</td>
<td>9</td>
<td>19</td>
<td>58</td>
</tr>
<tr>
<td>Others*</td>
<td>124</td>
<td>40</td>
<td>550</td>
</tr>
<tr>
<td>Total NSP, g/kg DM</td>
<td>124</td>
<td>40</td>
<td>550</td>
</tr>
<tr>
<td>Lignin, g/kg DM</td>
<td>15</td>
<td>0</td>
<td>72</td>
</tr>
<tr>
<td>Dietary fiber, g/kg DM</td>
<td>139</td>
<td>40</td>
<td>622</td>
</tr>
</tbody>
</table>

*DM, dry matter; NSP, nonstarch polysaccharide.

*The sum of noncellulosic residues galactose, mannose, uronic acids, and glucose not accounted for as β-glucan or cellulose.
rye, and oats when provided as bread or breakfast cereals (20, 21, 23, 24). RS present as RS2 or RS3 is also more or less quantitatively recovered in ileal effluent (20–24). Currently, it is not known to what extent cereal fructans are degraded in the small intestine of humans, but animal studies (19) and studies with inulin from Jerusalem artichokes suggest that fructans that survive the baking process will be recovered in the ileal effluent (30).

**Microflora of the Large Intestine**

The environment in the large intestine has all of the conditions for prolific bacterial growth: it is warm, moist, anaerobic, and filled with feed residues that flow at a relatively low speed. The numbers of bacterial cells can reach $10^{11}$–$10^{12}$/g, 10-fold greater than the number of cells in the human body (7, 31, 32). Colonic bacteria have a far larger repertoire of degrading enzymes and metabolic capabilities than their host, and dietary changes may affect or interact with the microbiota and the metabolic outcome through several interrelated mechanisms. First, the metabolism is regulated within each individual species of gut bacterium where alternative substrates can give rise to different products as a result of fermentation via different metabolic routes; second, the same substrate can be processed via different routes depending on the rate of supply or the physiology and environment of the bacterial cell (31, 32).

*Firmicutes, Bacteroidetes,* and *Actinobacteria* are the dominant bacterial phyla in healthy humans, with *Proteobacteria* and *Verrucomicrobia* present in lower numbers (33, 34). Some of the species are commonly detected in high numbers in most adult fecal samples. Tap et al. (35) reported 66 particularly abundant phylotypes among 17 healthy individuals, and Walker et al. (36) found 50 dominant phylotypes, which each represented > 0.5% of total 16S ribosomal RNA sequenced across 6 obese male individuals. Five of the top 10 species (*Bacteroides vulgatus, Eubacterium rectale, Faecalibacterium prausnitzii, Clostridium eubacterium,* and *Ruminococcus bromii*) corresponded to the top 5 most abundant bacteria detected in an entirely culture-dependent study.

**Impact of Carbohydrates on Microbial Composition in the Large Intestine**

The degradation by the microbial community of carbohydrates in the large intestine occurs in a hierarchic fashion: sugar residues $>$ oligosaccharides $>$ starch residues $>$ soluble NSPs $>$ insoluble NSPs. In pigs, almost all sugars, fructans, residual starch, and the soluble DF components ($\beta$-glucan and soluble arabinoxylans) are degraded in the cecum and proximal colon where the microbial activity is high (28, 37, 38), whereas insoluble arabinoxylans and cellulose are degraded at more distal locations (28, 37). By linkage composition analyses (39), it was also shown that the aleurone arabinoxylans resemble those of the ileal sample more than the fecal sample, whereas endosperm arabinoxylans had the fecal xylose substitution pattern already in the proximal colon. For pericarp/testa arabinoxylans, the substitution pattern was similar in the ileum, proximal colon, and feces (39).

The fecal microbiota profile in healthy adults is generally quite stable over time, but the consumption of selected prebiotics (i.e., nondigestible oligosaccharides and RS) were shown to change the microbial community (40, 41). The fecal microbiota profiles of obese male subjects given diets differing in type and content of DF (RS3, NSPs from wheat bran, and a weight-loss diet) for 3-wk periods tended to group by individuals more than by diets (36). However, marked changes were found in the relative abundance of several dominant phylotypes in response to intake of RS, whereas there was no effect of NSPs from wheat bran (36). The changes occurred within a few days, and they were reversed equally quickly by a subsequent dietary shift. The lack of effect of the NSPs from wheat bran is presumably a consequence of the diverse NSP composition of wheat bran, with no single NSP being dominant (Table 1). Similar results were found by Costabile et al. (42). Feeding whole-grain wheat, however, increased *Bifidobacterium* spp. and resulted in a higher lactobacilli:enterococci ratio. Another intervention study with whole grain vs. refined wheat documented significantly higher ($P = 0.04$) numbers of *Bifidobacterium* spp. (43). Thus, whole grains have a potential to influence microbial composition, although the effect is less pronounced than when using prebiotics with a dominating polysaccharide (RS, inulin) or oligosaccharide (41, 44).

**SCFA Production and Absorption**

SCFAs are present at concentrations of $\sim 100$ mmol/kg colonic material but with a decreasing concentration from the cecum/proximal colon to the distal colon in humans (45) and animals (38). Cecum and proximal colon are the most active site for fermentation and with the lowest pH (5.4–6.4) depending on the rate of fermentation. Acetate accounts for approximately two-thirds of the produced SCFAs and is formed by many of the bacteria groups that inhabit the colon (32) (Figure 2), whereas the number of bacteria...
groups that form propionate and butyrate is more restricted (32). There is also a substantial utilization of acetate during butyrate formation (46). As discussed in the previous section, the potential for changing the microbial composition by whole-grain cereals and cereal fractions is lower than by prebiotics with dominating oligo- or polysaccharides. However, several studies showed that β-glucan and arabinoxylans, when present in the whole-grain matrix, can be regulatory factors for SCFA production and profile in the large intestine. An arabinoxylan-rich cereal-based diet was found to stimulate the proliferation of butyrate-producing microorganisms (i.e., *F. pausnitzii*, *Roseburia intestinalis*) (46), butyrate production in the large intestine (47), and the net portal absorption of butyrate (48) to a larger extent than a diet with equal amounts of DF in the form of RS (Table 2). A Western-style diet high in refined carbohydrates from sugar and refined wheat flour was used as a reference diet (47, 48). In contrast to these findings, studies in rats and pigs fed an arabinoxylan-rich fraction from wheat did not specifically show enhanced butyrate but only total SCFA production (49, 50). The reasons for this difference in butyrate response between these 2 sets of experiments cannot be determined by any certainty but may be related to the way the arabinoxylans was provided, as a concentrate from wheat (49, 50) or as part of a whole-grain matrix (27, 47, 48). The fermentation of the arabinoxylan concentrate can be expected to be rapid, as indicated by the results in references 49 and 50, and with a decrease in pH mainly in the cecum (48), whereas the fermentation of arabinoxylans from a whole-grain matrix will be slower, with a decrease in pH not only in the cecum but also in the proximal colon (28). pH has been found to play a crucial role for the balance between acetate and butyrate production in the gut (51), and it cannot be excluded that this is a key factor why arabinoxylans fermentation under some conditions leads to enhanced butyrate production. In support of an impact of the whole-grain matrix on butyrate production is a human study in which the subjects consumed wheat and rye breads (WR) that were prepared without (WR−) and with (WR+) xylanases and compared with refined wheat (Table 3) (52). It was found that the concentration of total SCFAs and butyrate in feces was increased after the intervention with the WR diet, but it was only for the WR+ treatment that the effect was significant. Likewise, the concentrations of acetate, propionate, and butyrate in feces all increased when human subjects consumed either wheat arabinoxylan- or inulin-enriched bread, but only the butyrate concentration changed significantly with wheat arabinoxylans (53). A range of other in vivo studies (27, 37, 54–58) also pointed toward arabinoxylans as a fermentative substrate that stimulates butyrate

![FIGURE 2](https://academic.oup.com/advances/article-abstract/6/2/206/4558057)

A schematic of the gut microbiota involved in the metabolism of SCFAs. Acetate and lactate are shown as intermediates. Representative species and phyla are indicated based on information from cultured microorganisms. CoA, coenzyme A. Reproduced from reference 32 with permission.
formation, whereas the outcome of in vitro fermentation studies is more variable, with some finding stimulating effects (59, 60) but others not (61).

β-Glucan also seems to have the potential to influence SCFA production. In patients with ulcerative colitis who daily consumed 60 g oat bran rich in β-glucan, it was found that the fecal butyrate concentration increased significantly, whereas the remaining fecal SCFA concentrations were unchanged (62). The data from the ulcerative colitis study corroborated studies in rats fed barley β-glucan (63, 64), pigs fed oat bran (37, 55), and portal vein–catheterized pigs fed an oat bran and an oat β-glucan–rich concentrate (65, 66).

**SCFA Metabolism**

DF amount and composition had a profound influence on the net portal absorption of SCFAs, including butyrate (27, 48, 65, 67) (Table 2). Within each experiment, butyrate absorption varied by 2 to 3.6 times between the lowest and highest concentrations due to differences in DF content (48, 65) and composition (27, 67). However, not only did DF content and composition influence total SCFA and butyrate absorption, but there was also a strong relation between net butyrate absorption and the butyrate concentration in the portal vein; for the data presented in Table 2, the correlation was 0.962. This is supported by pig studies in which pigs were deprived of feed for 24 h and then fed a pulse dose of either a barley-based diet rich in β-glucan (68) or a rye-based diet rich in arabinoxylans (27). In these studies, the concentration of butyrate in the portal vein started to increase 4 h postdosing (i.e., the time when the bulk of digesta reaches the large intestine) (27, 68). These data therefore seriously questioned the general view that butyrate is mainly consumed by the colonic epithelium (69–71). Rather, the liver is the main site for the clearance, with clearance rates of propionate and butyrate of ~94% and ~82%, respectively, and a clearance rate of ~41% for acetate (48). Augmenting DF intake therefore not only increased peripheral acetate but also propionate and butyrate, as shown in studies in pigs (Table 2) and humans (72–75).

**Physiologic and Health Effects of SCFAs**

An unhealthy dietary pattern (i.e., consumption of diets high in fat and refined carbohydrates and low in DF) is regarded to be a significant health risk factor, limiting the production of SCFAs and giving rise to high luminal concentrations of putative risk factors (secondary bile acids, protein degradation products) (28, 49, 76). Diets with a higher DF content stimulate SCFA production and reduce the exposure of colonic epithelium to potentially toxic compounds (28, 49), which have been shown to oppose the induction of DNA stand breaks due to high protein concentrations. Although the study by Belobrajdic et al. (49) did not result in a highest relative proportion of SCFAs, the higher pool size indicates an enhanced total butyrate production, which is of interest because butyrate is believed to play an important role in maintaining colonic health and function. In addition to being an energy source for colonic epithelial cells (70, 71), butyrate can induce changes in gene expression and to upregulate PPARγ expression (77). Oxidative stress

---

**TABLE 2** Net portal absorption of total SCFAs and butyrate concentrations in the portal vein and mesenteric artery as estimated in conscious catheterized pigs in vivo fed cereal-based diets varying in dietary fiber content and composition 1

<table>
<thead>
<tr>
<th>Diet</th>
<th>Content, g/kg DM</th>
<th>Absorption, mmol/d</th>
<th>Butyrate, μmol/L</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NDO</td>
<td>RS</td>
<td>Cellulose</td>
</tr>
<tr>
<td>HF wheat bran</td>
<td>28</td>
<td>5</td>
<td>58</td>
</tr>
<tr>
<td>HF oat bran</td>
<td>22</td>
<td>40</td>
<td>44</td>
</tr>
<tr>
<td>WSD</td>
<td>2</td>
<td>6</td>
<td>29</td>
</tr>
<tr>
<td>RSD</td>
<td>133</td>
<td>34</td>
<td>1</td>
</tr>
<tr>
<td>AXD</td>
<td>29</td>
<td>8</td>
<td>37</td>
</tr>
<tr>
<td>WFRF</td>
<td>4</td>
<td>131</td>
<td>12</td>
</tr>
<tr>
<td>WGRBB</td>
<td>18</td>
<td>11</td>
<td>20</td>
</tr>
</tbody>
</table>

1Values are medians (IQRs), n = 27. *Different from pre-WR+ period, P < 0.05. Adapted from reference 51 with permission. W+, wheat flour bread without in situ–produced arabinoxylan oligosaccharides; WR+, wheat/rye bread without in situ–produced arabinoxylan oligosaccharides; WR−, wheat/rye bread with in situ–produced arabinoxylan oligosaccharides.

---

**TABLE 3** Fecal SCFA excretions after 3-wk consumption of wheat and rye breads that were prepared without and with xylanases 1

<table>
<thead>
<tr>
<th>Metabolite</th>
<th>Treatment period, μmol/g wet feces</th>
<th>Pre-WR+ (W− treatment)</th>
<th>WR+ (W− treatment)</th>
<th>Pre-WR− (W+ treatment)</th>
<th>WR− (W+ treatment)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetic acid</td>
<td>26.4 (22.0–39.5)</td>
<td>36.6 (24.0–46.7)</td>
<td>29.0 (18.0–38.4)</td>
<td>33.8 (26.6–403)</td>
<td></td>
</tr>
<tr>
<td>Propionic acid</td>
<td>7.9 (6.1–10.7)</td>
<td>11.1 (7.4–14.2)</td>
<td>8.5 (5.7–11.0)</td>
<td>9.8 (7.8–11.2)</td>
<td></td>
</tr>
<tr>
<td>Butyric acid</td>
<td>10.7 (6.0–19.8)</td>
<td>18.3 (12.4–28.0)*</td>
<td>11.4 (5.5–18.6)</td>
<td>13.7 (9.4–21.7)</td>
<td></td>
</tr>
<tr>
<td>Total SCFAs</td>
<td>45.5 (32.3–70.0)</td>
<td>70.8 (46.8–84.0)*</td>
<td>48.9 (32.0–67.0)</td>
<td>58.5 (40.3–75.1)</td>
<td></td>
</tr>
</tbody>
</table>

1Values are medians (IQRs), n = 27. *Different from pre-WR+ period, P < 0.05. Adapted from reference 51 with permission. W−, wheat flour bread without in situ–produced arabinoxylan oligosaccharides; W+, wheat flour bread without in situ–produced arabinoxylan oligosaccharides; WR+, wheat/rye bread without in situ–produced arabinoxylan oligosaccharides; WR−, wheat/rye bread with in situ–produced arabinoxylan oligosaccharides.
is involved in both inflammation and the initiation and progression of carcinogenesis. During oxidative stress, there is an imbalance between the generation of reactive oxygen species and the antioxidant defense mechanisms, leading to a cascade of reactions in which lipids, proteins, and/or DNA may be damaged (78). Furthermore, butyrate was shown to have a potent effect on the expression of genes involved in regulating cell proliferation, apoptosis, differentiation, and metastasis in a human colonic epithelial cell line (79).

It has also been recognized that the binding of SCFAs to free fatty acid receptor (FFAR) 2 and FFAR3 located on colonic L-cells may be involved in controlling anorectic hormones including peptide YY and glucagon-like peptide 1 (GLP-1) (80). Oat β-glucan increased net SCFA absorption in portal vein–catheterized pigs, leading to a decreased apparent insulin production, which was associated with GLP-1 mediation (66); and supplementing a diet with 10% β-glucan increased total SCFA content in the cecum, suppressed central neuronal activity in the hypothalamic appetite centers, and decreased food intake (64). A recent study by Ingerslev et al. (48) in catheterized pigs, however, did not confirm a link between net portal SCFA absorption and the net portal GLP-1 flux but identified a lower insulin flux at higher net portal SCFA absorption.

SCFAs are also considered to be signaling molecules between the gut and the peripheral tissues, with implications for insulin sensitivity and glucose homeostasis (12, 74). The mechanisms appear to involve adipocyte cell differentiation, regulation, and metabolism (12, 75). In response to an increased DF intake, there is an increased absorption of SCFAs; and although the major part of propionate and butyrate is cleared in the liver, micromolar concentrations of all 3 SCFAs will reach the peripheral tissues (10) (Table 2). Here, the SCFAs can act as ligands on adipocytes (12, 81) and thereby influence the balance of adipokines released and, in turn, increase adipogenesis and differentiation of fat cells and thus reduce their average size. SCFAs further inhibit lipolysis within adipose tissue as indicated by the reduced plasma concentrations of nonesterified FAs after the intake of fermentable carbohydrates (82–84) and the lower fasting concentrations of nonesterified FAs with higher SCFA production (48). The consequence is reduced availability of FAs for uptake into ectopic fat depots (e.g., liver and pancreas) (12). Overall, the above-mentioned conditions have been linked to improved insulin sensitivity and glucose homeostasis (12, 74, 75, 85, 86).

Conclusions

Whole-grain cereals have a diverse DF composition that can be used to produce diets with the potential to influence microbial composition, the production and molar proportions of SCFAs, and gut and metabolic health.

Acknowledgments

The sole author had responsibility for all parts of the manuscript.

References


11. van Munster IP, Tangerman A, Nagengast FM. Effect of resistant starch on colonic fermentation, bile acid metabolism, and mucosal prolifera

12. Robertson DM. Metabolic cross talk between the colon and the periph


15. Saulnier L, Guillon F, Sado P-E, Rouau X. Plant cell wall polysacch


17. Englyst HN, Kingman SM, Cummings JH. Classification and measure


