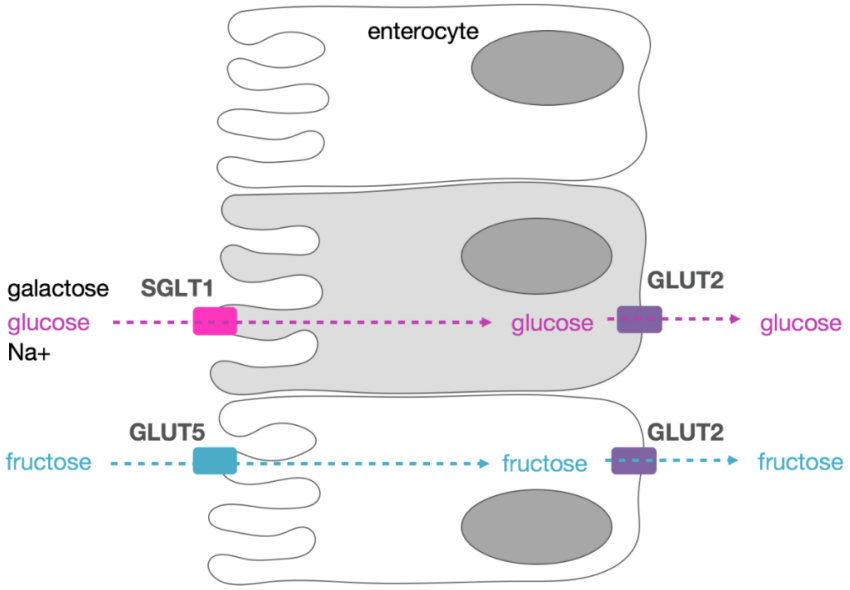
1.2 Theory into practice

1.2.1 Carbohydrate absorption

> Once emptied from the stomach, most fluid and sugar absorption will take place in the duodenum and jejunum. Glucose and galactose are transported across the luminal membrane of enterocytes by the sodium dependent glucose transporter SGLT1.
>
> Absorption of glucose (and galactose) is coupled with sodium transport and the associated electrochemical gradient. A Na/K^+ ATP-ase, located at the basolateral membrane, is responsible for maintaining the electrochemical gradient. (Jeukendrup, 2017, https://bit.ly/2soS2oQ)

In most mammalian studies, SGLT1 has been shown to be expressed on the brush border of enterocytes (Batchelor et al., 2011; Dyer et al., 2009; Margolskee et al., 2007; Moran, Al-Rammahi, Arora, Batchelor, Coulter, Daly, et al., 2010; Takata, Kasahara, Kasahara, Ezaki, & Hirano, 1992) (Figure 4). Expression levels are usually highest in the jejunum followed by the duodenum and ileum (Balen et al., 2008). This is in line with our understanding that most absorption takes place in the jejunum. SGLT1 is not expressed in the large intestine (Balen et al., 2008).

Figure 4: Absorption of glucose and fructose. Glucose and fructose are absorbed via different pathways involving SGLT1 and GLUT5 respectively. (SGLT1=sodium dependent glucose transporter 1, GLUT5 = glucose transporter 5 (fructose transporter), GLUT2 = glucose transporter 2)



Source: Jeukendrup, 2017, https://bit.ly/2soS2oQ

Fructose uses a different transporter (GLUT5) to glucose, that is not sodium dependent and is highly specific to fructose (Figure 4). The regulation of GLUT5 is more rapid than the regulation of SGLT1. Changes in fructose transport are typically paralleled by similar changes in GLUT5 mRNA and protein abundance. In rats, GLUT5 mRNA doubles within 3h after intestinal perfusion with a fructose solution (Kishi, Takase, & Goda, 1999). It must be noted that these effects have only been demonstrated at unnatural high fructose intakes (at least 30% of energy in the diet coming from fructose, a typical intake in a Western diet is around 9%).

From the enterocyte to the systemic circulation, the sugars need to pass the basolateral membrane. All three monosaccharides use the bidirectional transporter GLUT2 which is also sodium independent. The capacity of GLUT2 to transport glucose across a concentration gradient is believed to be very large (Kellett, 2001; Kellett, Brot-Laroche, Mace, & Leturque, 2008).

There is little evidence for other carbohydrate transporters in addition to SGLT1 and GLUT5 transporters at the luminal

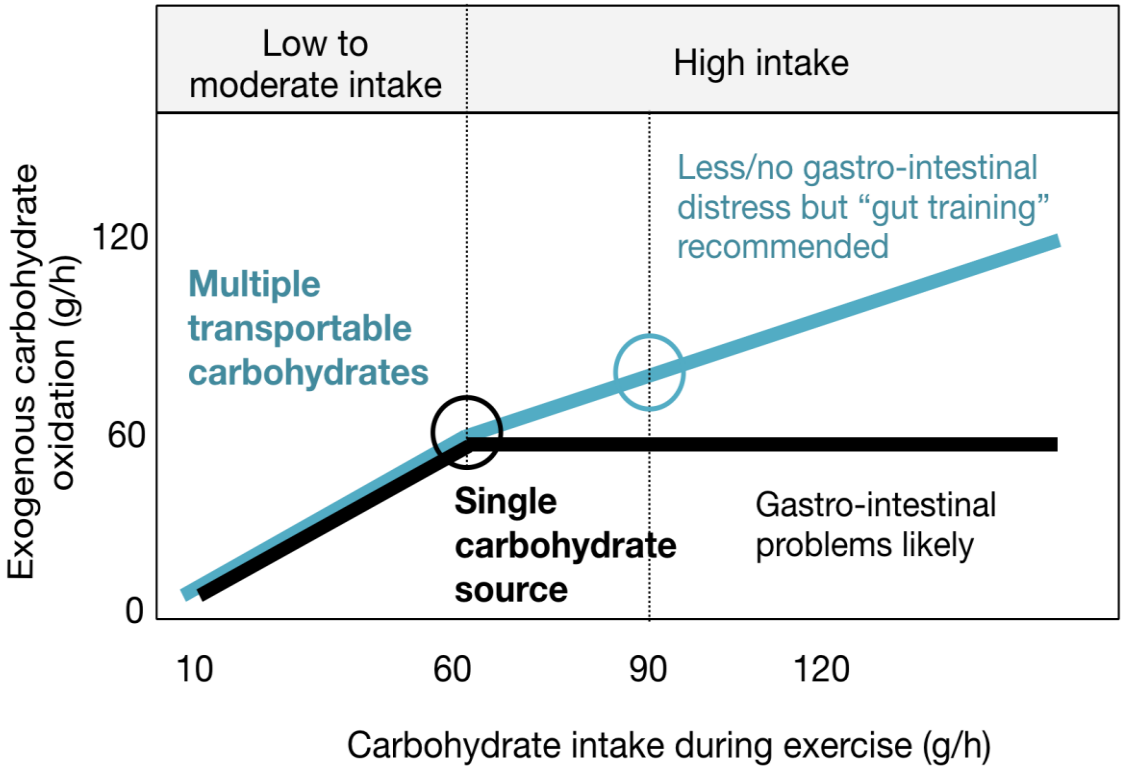
membrane and GLUT2 at the basolateral membrane. There have been suggestions of other transporters, but it seems that if they exist, they will be relatively unimportant for transport of carbohydrates from a quantitative point of view. Since GLUT2 does not seem to be limiting, here we will focus mostly on SGLT1 and GLUT5.

The regulation of carbohydrate transport proteins is essential for the provision of glucose to the body in resting conditions. Also, during exercise, when exogenous delivery of carbohydrate may be important for performance, the transporters will be responsible for glucose delivery to the working muscle. Exercise studies have provided indirect, but strong evidence that the delivery of carbohydrate is limited by the transport capacity of SGLT1 (for reviews see Jeukendrup, 2011a, 2011b, 2013, 2014). A recent review based mostly on more direct measurements in animals also concluded that the intestine has the capacity to absorb glucose via basal levels of SGLT1, but that this capacity becomes limiting when dietary carbohydrate exceeds a certain level (Shirazi-Beechey, 2011). (Jeukendrup, 2017, https://bit.ly/2soS2oQ)

This may have relevance in football because studies suggest that >60 g carbohydrate should be ingested before and during a match and even a single dose of carbohydrates can result in saturation of carbohydrate transporters (Adopo, Peronnet, Massicotte, Brisson, & Hillaire-Marcel, 1994; Jeukendrup, 2010).

At ingestion rates over 60-70 grams of carbohydrate per hour (glucose, sucrose, maltose, maltodextrin, starch), exogenous carbohydrate oxidation peaks around 60 grams per hour (**Figure 5**) (Jeukendrup, 2011a, 2011b, 2013, 2014).

Figure 5: Schematic of exogenous carbohydrate oxidation from a single carbohydrate (orange) and multiple transportable carbohydrates (blue), based on data presented elsewhere



Source: Jeukendrup, 2014. https://bit.ly/2QINjYO

It is clear that higher oxidation rates can be achieved with multiple transportable carbohydrates, especially at high intakes. At intakes up to 60 g/h there is no difference between single and multiple transportable carbohydrates but when intake increases above 60 g/h and the Sodium Dependent Glucose Transporter 1 (SGLT1) becomes saturated, added fructose will result in higher exogenous carbohydrate oxidation rates. The recommended intake for single and multiple transportable carbohydrates are indicated with a circle. If single carbohydrate sources are ingested at higher rates than 60 g/h, gastro-intestinal problems are likely. With multiple transportable carbohydrates fewer symptoms have been observed but "training the gut" (and getting used to high intakes) is recommended.

Even ingestion at 144 g/h (Jentjens & Jeukendrup, 2005) or 180 g/h (Jeukendrup, 1999) did not increase exogenous

carbohydrate oxidation rates much above 60 g/h. Because this limitation was not caused by gastric emptying, muscle glucose uptake or liver glycogen storage, it was deduced that absorption had to be limiting (Jeukendrup, 2010). When fructose was ingested in addition to larger amounts of glucose, carbohydrate oxidation rates were elevated above 60 g/h (Jentjens, Venables, & Jeukendrup, 2004). These studies strongly suggested that glucose transport across the epithelial cell was the limiting factor and that SGLT1's maximal transport capacity was reached (Pfeiffer, Stellingwerff, Zaltas, & Jeukendrup, 2010). There appears to be a dose response relationship between carbohydrate intake and performance (Smith et al., 2013; Smith et al., 2010; Vandenbogaerde & Hopkins, 2011) and football studies also seem to find greater benefits with higher intakes. A reduced capacity of the intestine during exercise in combination with a higher carbohydrate intake may also result in gastro-intestinal distress (de Oliveira et al., 2014). This means that we must find ways to improve the capacity to absorb carbohydrate.

1.2.2 Training the intestine

Training the gut has been proposed as a way to increase SGLT1 transporter number and/or activity, but evidence in humans thus far is limited (Jeukendrup, 2013).

Using a segmental perfusion technique, Shi et al. (1995) reported a close relationship between water absorption and solute absorption in the duodenojejunum, especially when multiple transportable substrates are present (i.e., glucose, sucrose, glycine, Na+). We confirmed this in humans during exercise: multiple transportable carbohydrates increased carbohydrate absorption and oxidation and this was associated with an increased fluid absorption (Jeukendrup, 2010). Therefore, one other benefit of increasing the transport capacity for carbohydrate is that fluid intake is likely to be improved too (for a given carbohydrate intake). Improved fluid absorption can help towards preventing dehydration (and help prevent dehydration induced reductions in performance), but more complete absorption may also reduce the chances of GI discomfort (de Oliveira et al., 2014).

In order to develop practical recommendations, it is important to understand the regulation of intestinal glucose transport. Below, we will therefore discuss the regulation in more detail, before providing suggestions of practical implication.

Regulation of glucose absorption has been shown to be directly linked to the expression of SGLT1 protein. Bob Crane proposed the existence of a Na^+/glucose co-transport in 1960 at the Symposium on Membrane Transport and Metabolism in Prague (Kleinzeller, 1961) but the actual transporter was not identified until the 1980s (Hosang, 1981) . Studies in the 1960s also observed that dietary carbohydrate intake can influence the capacity to absorb glucose (Ginsburg & Heggeness, 1968). In 1983 it was demonstrated that intestinal transporters were upregulated and downregulated depending on dietary composition (Karasov, Pond, Solberg, & Diamond, 1983). At least in rats, it appears that dietary changes do not have to be extreme in order to observe effects on absorption and these effects have been seen not only for sugars but also for amino acids (Karasov et al., 1983). Increases in absorption have been observed in as little as 0.5 days in rats (Karasov et al., 1983). It was also observed very early on that digestive enzymes were upregulated in response to dietary composition. For example, Deren et al (1967) demonstrated in 1967 that rats who were fasted for 3 days displayed 4 fold increases in sucrase and maltase activity in response to a sucrose diet compared with a casein diet. This was correlated with increases in sucrose hydrolysis and in fructose absorption.

When sugar transporters were identified in the gut in the 1980s, studies started to measure changes in SGLT1 content and activity in response to diet. Both the activity and abundance of SGLT1 have been shown to be regulated by dietary carbohydrate intake in a number of rodent models (Dyer et al., 2009; Ferraris, Villenas, Hirayama, & Diamond, 1992). It is clear that SGLT1 protein responds to glucose concentrations in the lumen. However, when membrane impermeable glucose analogues were used, SGLT1 was stimulated to the same degree (Dyer, Vayro, King, & Shirazi-Beechey, 2003). This suggested that a glucose sensor detects glucose or its analogues, initiating the upregulation of the SGLT1 transporters.

Specialized cells (L-cells and K-cells) in the intestinal luminal membrane have been shown to express taste receptor cells. In particular, it has been demonstrated that T1R2 and T1R3 receptors detect sweetness. The T1R2 and T1R3 cells are coupled through a G-protein (alha-gustducin) to a cascade of downstream cellular events that ultimately lead to upregulation of SGLT1. A more detailed discussion of the potential pathways involved will be provided in the following sections.

SGLT1 is not only upregulated in response to dietary carbohydrate but also in response to sweeteners. Margolskee et al. (2007) confirmed earlier findings by reporting that wild-type mice whose diet was supplemented with carbohydrate, almost doubled their SGLT1 protein expression compared with mice on a low carbohydrate diet. However, when the low carbohydrate diet was supplemented with the sweeteners sucralose, acesulfame K or saccharine, but not aspartame, SGLT1 expression also doubled. The observation that aspartame had no effect is not surprising because it is known that mice do not experience aspartame as sweet.

A number of dietary constituents have been implicated in the regulation of glucose transport. Sodium chloride consumption appears to modulate intestinal glucose transport. Studies suggest that chronically elevated luminal concentrations of glucose and sodium will lead to increased expression of the SGLT1 protein (Bindslev, Hirayama, & Wright, 1997). There are still many questions about the mechanisms and if the effects of sodium and glucose are additive (Ferraris, 2001).

Dietary fibre is another constituent with potential effects, but studies have been inconclusive with some studies showing a decrease, some no change and some even an increase in intestinal glucose transport with increasing dietary fibre intake (Ferraris, 2001). Fibre is a broad term to describe vastly different characteristics and fibre can have effects on gastric emptying, motility and also the composition and structure of the intestinal tract. It may therefore not be surprising that results of studies have been inconclusive.

To the best of my knowledge no human studies have investigated the effects of dietary constituents on intestinal

glucose absorption and therefore it is premature to develop firm guidelines in the absence of these findings.

SGLT1 protein is upregulated in response to a number of stimuli including, but not limited to glucose and galactose: 3-O-methylglucose (non-metabolizable substrate of SGLT1) and fructose (not a substrate of SGLT1). Upregulation of the SGLT1 protein is dependent on availability of these sugars but metabolism of these sugars is not necessary. The fact that SGLT1 expression responds to glucose analogues and sugars not transported by SGLT1 suggests that there is a separate receptor that detects these glucose analogues.

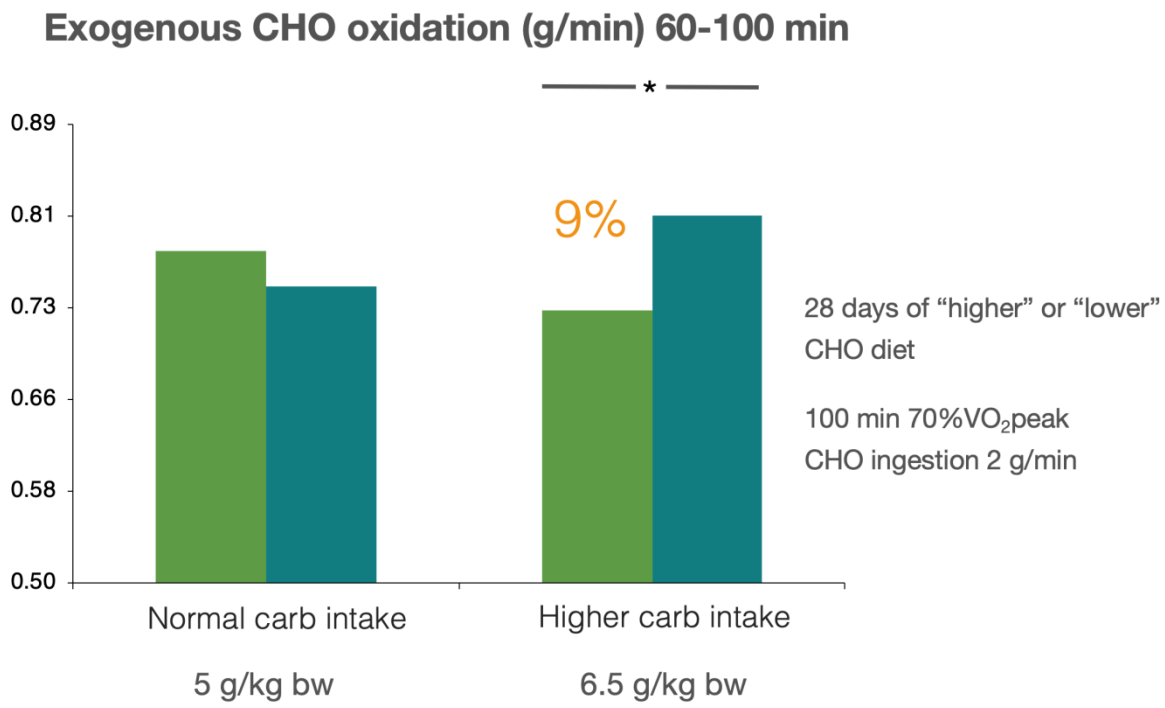
In mice, intestinal SGLT1 protein in brush-border membrane vesicles in the mid small intestine increased 1.9 fold after 2 weeks of a high carbohydrate diet (Margolskee et al., 2007). In a study of horses, which are believed to be slow adapters to an increase in carbohydrate, SGLT1 protein expression from intestinal biopsies was increased after just 1 week of high carbohydrate feeding and the abundance increased further after 1 months and 2 months on the diet. Piglets who received a higher carbohydrate diet for 3 days showed increases of SGLT1 protein as well as glucose absorption (Moran, Al-Rammahi, Arora, Batchelor, Coulter, Ionescu, et al., 2010).

Although no direct human studies exist, a large number of animal studies suggest that the time course of changes in SGLT1 expression is relatively rapid. Several studies have observed significant changes after only a few days of dietary change (Margolskee et al., 2007). It seems therefore reasonable to suggest that several days of a high carbohydrate intake can increase SGLT1 content and the capacity to absorb glucose, but more prolonged exposure to the diet could result in greater adaptations.

An elegant study by Cox et al. (2010) gives us the most important clues today that diet manipulation can result in improved delivery of carbohydrate during exercise. In this study 16 endurance-trained cyclists were divided into a high carbohydrate and a control group. For 28 days both groups trained (16 h/week) and their performance improved as a result of this training. Both groups received a diet with a moderate carbohydrate content (5 g/kg/day). The high carbohydrate group were supplemented with an additional 1.5 g/kg per hour of exercise performed daily.

The carbohydrate supplement was provided mainly in the form of a glucose drink. In addition, they received carbohydrate rich foods to meet the hourly demands of exercise. The control group also received a nutritional supplement but this was composed of fat and protein rich foods with limited carbohydrate content. Subjects in the high carbohydrate groups consumed the supplements before and during exercise as well as immediately after exercise. The cyclists in the control group consumed their supplement after exercise. On average the carbohydrate supplemented group had a high daily carbohydrate intake of 8.5 g/kg whereas the control groups consumed 5.3 g/kg/day. (Jeukendrup, 2017, https://bit.ly/2soS2oQ)

Figure 6: A human study that shows that adaptation in the gut can take place after increasing carbohydrate intake



Source: Jeukendrup A, 2018, mysportscience, adapted from Cox, 2010

Before and after the 28-day training period all subjects performed an exercise trial in which they received a 10% carbohydrate solution. Isotopic tracers were used to measure the oxidation of the exogenous carbohydrate. It was observed that exogenous carbohydrate oxidation was improved after the carbohydrate supplemented diet. The most likely explanation is an increase in the ability to

absorb carbohydrate as a result of an upregulation of SGLT1 transporters. It was concluded that for athletes who compete in endurance events where exogenous carbohydrate is an important energy source, and there is ample opportunity to ingest carbohydrate, this higher carbohydrate intake approach may be beneficial (Cox et al., 2010; Jeukendrup, 2013, 2014).

It has become clear that an increase in dietary carbohydrate intake can increase the abundance and activity of intestinal SGLT1 transporters and that this results in an improved capacity to absorb carbohydrate. The reverse may be true as well. With carbohydrate restriction through reducing carbohydrate intake, high fat or even ketogenic diets, or by reducing total energy intake, the daily carbohydrate intake can become very low. Studies in lambs have demonstrated that as the diet changes from milk to grass, so the rumen, where dietary carbohydrates are fermented into volatile fatty acids, develops. Rumen formation effectively prevents the delivery of monosaccharides to the intestine. As a result, there is a marked decrease in both the SGLT1 protein content of the intestine as well as the capacity of the small intestine to absorb carbohydrate (Shirazi-Beechey, 1991; Dyer, 1997). (Jeukendrup, 2017, https://bit.ly/2soS2oQ)

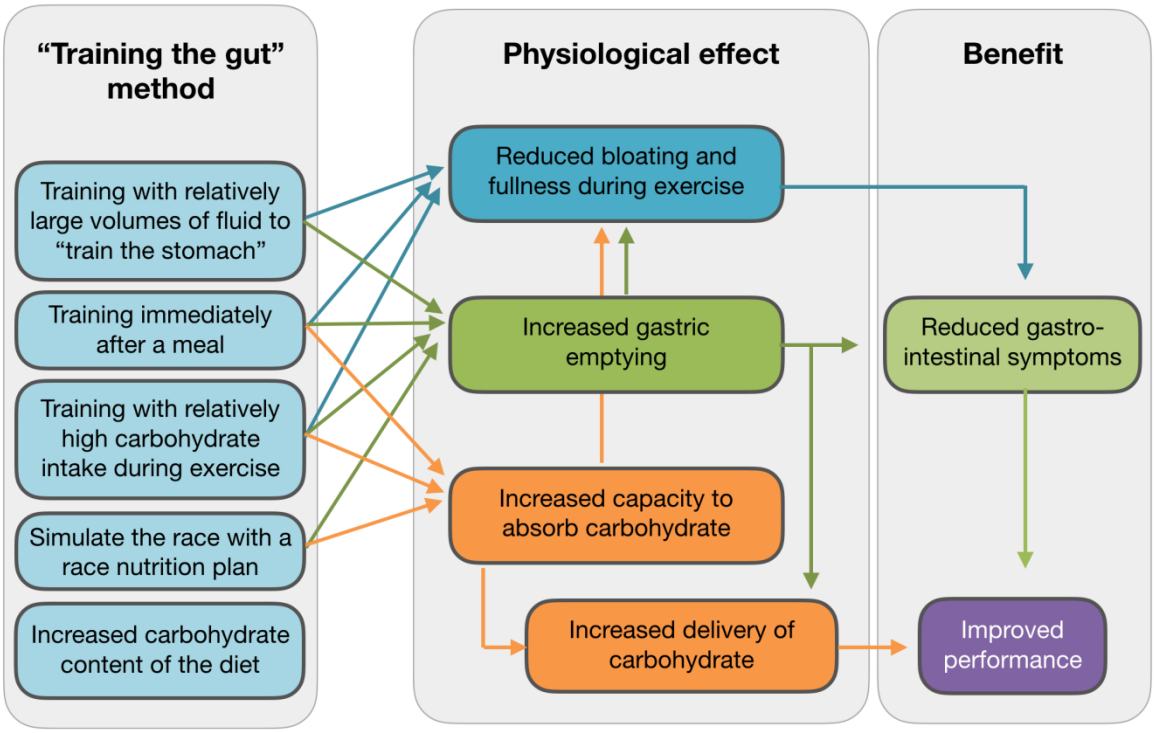
1.2.3 How to train the gut?

In the previous sections, we have made the case that training the gut may be a good idea, also for football players. There are numerous ways to do this (Figure 7). Especially those players that struggle to eat anything close to the match or are afraid that this will cause gastrointestinal problems, can benefit from it. While some extrapolations from animal studies are required, it is likely that adaptations in the human stomach and intestine are as rapid as those seen in other mammals. This means that several days and certainly 2 weeks of a high carbohydrate diet would result in significant increases in the SGLT1 content of the intestinal lumen and just a few days of stomach training could already improve stomach comfort.

Based on animal data an increase in dietary carbohydrate from 40% to 70% could result in a doubling of SGLT1 transporters over a period of 2 weeks. If we translate this to the diet of a football player, it probably means that if a player sticks to the recommendations (typically 5-8 g/kg), this should be sufficient to induce some adaptations, but it is also likely that higher intakes will be better for the upregulation of intestinal carbohydrate transport capacity.

In addition to an increased absorptive capacity it is essential to make sure that higher carbohydrate intakes can be tolerated and are also emptied from the stomach. Although it is generally believed that gastric emptying is not a limiting factor it is likely that a combination of factors (for example heat, high carbohydrate intake and high intensity exercise, which are all factors known to inhibit gastric emptying) will act together, thereby compromising gastric emptying. Therefore, it is important to practice a race nutritional strategy in training and get used to higher volumes of intake or higher carbohydrate intakes. (Jeukendrup, 2017, https://bit.ly/2soS2oQ)

Figure 7: A summary of methods to "train the gut", the adaptations that may occur in the gut and implications for performance



Source: Jeukendrup, 2017, https://bit.ly/2slxMEx

The study by Cox et al. (2010) suggests that these transporters can be upregulated in a relatively short period of time when a higher carbohydrate intake is maintained, mostly by taking in carbohydrate during and around training.

Although the exact magnitude of effects in athletes who are already on a high carbohydrate diet may be uncertain

it seems fair to conclude that those athletes who are not practicing a very high carbohydrate diet already can benefit substantially. The opposite is also true: when athletes are carbohydrate restricting, following a low carbohydrate, high fat or ketogenic diet or are reducing energy intake in order to lose weight, the reduced daily carbohydrate load will likely reduce the capacity to absorb carbohydrates during competition. This could be a reason why these athletes anecdotally seem to report more gastro-intestinal problems. These athletes would be advised to include some high carbohydrate days in their training as well.

Current guidelines for matches are that at least 60 grams is ingested just before and at half time. Ideally slightly greater amounts of carbohydrate (90 g/h) would be ingested and these carbohydrates should consist of a mix of multiple transportable carbohydrates e.g., glucose:fructose or maltodextrin:fructose. In order to obtain a carbohydrate intake of 90 g/h athletes could "mix and match" to fulfil their personal preferences and take into account their tolerance (Jeukendrup, 2013, 2014). Since the gut is so adaptable, it seems wise to include training with high carbohydrate intake into the weekly routine and regularly ingest carbohydrate during exercise (this simply would mean that once a week during hard training the same routine as match day is followed). With these strategies, the gut may be trained to absorb and oxidise more carbohydrate, which in turn should result in less gastro-intestinal distress and better performance. (Jeukendrup, 2017, https://bit.ly/2soS2oQ)

1.2.4 The gut microbiome

The term microbiota refers types of organisms (bacteria, viruses or eukaryotes) that are present in an environmental habitat and the microbiome is a collection of different microbes and their functions or genes found in an environmental habitat (Jeukendrup & Gleeson, 2018). Different parts of the body have different microbiomes, for example, the skin microbiome is different from the gut microbiome, but they are all part of the human microbiome. An adult human intestinal tract contains about 1 kg of various bacteria (colon bacilli) totalling over 100 trillion (10^{14}) cells which is 10 times the number of host cells in the human body.

The gastrointestinal tract contains an immensely complex

ecology of microorganisms. A typical person harbours more than 500 distinct species of bacteria. The composition and distribution of these microorganisms vary with age, state of health, and diet (Jeukendrup & Gleeson, 2018). The gut microbiome has received a lot of interest in the last few years.

The number and type of bacteria in the gastro-intestinal tract vary dramatically by region. In healthy people the stomach and proximal small intestine contain few microorganisms, largely a result of the bactericidal activity of gastric acid. In sharp contrast to the stomach and small intestine, the colon literally teems with bacteria, predominantly strict anaerobes (bacteria that survive only in environments virtually devoid of oxygen) (see Table 1). Between these two extremes is a transitional zone, usually in the ileum, where moderate numbers of both aerobic and anaerobic bacteria are found. (Jeukendrup & Gleeson, 2018, https://bit.ly/2LC9XB7)

Table 1: Microbial populations in the digestive tract of normal humans

	Stomach	Jejunum	Ileum	Colon
Viable bacteria per gram	$10{\cdot}10^{3}$	$10{\cdot}10^{4}$	$10{\cdot}10^{8}$	$10{\cdot}10^{12}$
pH	3.0	6.0_7.0	7.5	6.8_7.3

Source: Jeukendrup & Gleeson, 2018, https://bit.ly/2LC9XB7

The bacterial populations that comprise the microbiota of in the large intestine have a number of functions. They digest carbohydrates, proteins, and lipids that escape digestion and absorption in the small intestine. The bacteria are responsible for the fermentation of small amounts of cellulose but also produce vitamin K, vitamin B_{12}, thiamine, riboflavin, and other substances. Vitamin K is especially important because the daily vitamin K intake in foodstuffs is normally insufficient (Jeukendrup & Gleeson, 2018, https://bit.ly/2LC9XB7)

There are approximately 160 species in the large intestine of any individual and very few of these are shared between unrelated individuals. In contrast, the functions contributed by these species appear to be found in everybody's gastrointestinal tract, an observation that leads us to conclude that function is more important than the identity of the species providing it. Yet differences in the gut microbiota may matter because these may result

in differences in the effectiveness of a function. For example, while the ability to synthesise short chain fatty acids (SCFAs) is found in all humans, their amounts can vary (Jeukendrup & Gleeson, 2018).

Carbohydrate fermentation is a core activity of the human gut microbiota, driving the energy and carbon economy of the colon. Dominant and prevalent species of gut bacteria, including SCFA-producers, appear to play a critical role in initial degradation of complex plant-derived polysaccharides, collaborating with species specialised in oligosaccharide fermentation (e.g., bifidobacteria), to liberate SCFAs and gases which are also used as carbon and energy sources by other more specialised bacteria. The efficient conversion of complex indigestible dietary carbohydrates into SCFA serves microbial cross-feeding communities and the host, with 10% of our daily energy requirements coming from colonic fermentation.

Some of the SCFAs including butyrate and propionate can regulate intestinal physiology and immune function, while acetate acts as a substrate for lipogenesis and gluconeogenesis (i.e. fat and sugar synthesis, respectively). Recently, key roles for these metabolites have been identified in regulating various functions in the body. For example, there is a supporting role for immune function helping with the resolution of inflammation. In the colon, the majority of this carbohydrate fermentation occurs in the proximal colon, at least for people following a Western style diet. As carbohydrate becomes depleted as “food” moves distally through the intestine, the gut microbiota switches to other substrates, notably protein or amino acids. Fermentation of amino acids, besides liberating beneficial SCFAs, produces a range of potentially harmful compounds. Some of these may play a role in gut diseases such as colon cancer or inflammatory bowel disease (IBD). On the contrary, dietary fibre or intake of plant-based foods appears to inhibit this, highlighting the importance of maintaining gut microbiome carbohydrate fermentation (Jeukendrup & Gleeson, 2018, https://bit.ly/2LC9XB7)

In the past decade, interest in the human microbiome has increased considerably. A significant driver has been the realisation that the commensal microorganisms that comprise the human microbiota are not simply passengers in the host, but may actually drive certain host

functions as well.

> In germ-free rodents, the removal of the microbiota has a dramatic impact on nearly all aspects of the host's ability to function normally. By better understanding the mechanisms and the contribution of the microbiota to various diseases, it may be possible to develop novel therapeutics and strategies to modulate the microbiota to treat or prevent disease. In the healthy state, the microbiota contributes nutrients and energy to the host via the fermentation of non-digestible dietary components in the large intestine and influence both the host's metabolism and immune system. Furthermore, it is now clear that diet can have a major Influence on the composition of the microbiota which should open up new possibilities for health manipulation via diet (Jeukendrup & Gleeson, 2018, https://bit.ly/2LC9XB7)

Probiotics, and prebiotics and polyphenols

A number of dietary strategies are available for modulating either the composition or metabolic/immunological activity of the human gut microbiota and probiotics, prebiotics and polyphenols are among the most well established.

> Probiotics are potentially beneficial bacteria or yeasts. Probiotics are defined as live microorganisms that, when administered in adequate amounts, may confer a health benefit on the host. Probiotics can have multiple interactions with the host, including competitive inhibition of other microbes, effects on mucosal barrier function and interaction with immune cells and in particular antigen presenting dendritic cells. They can be found in certain foods or can be bought as a supplement. Examples include strains of the bacteria genera Bifidobacterium and Lactobacillus. The most common probiotics are the latter and are commonly referred to as lactic acid bacteria (LAB). These microbes have been used in the food industry for many years. LAB are able to convert sugars (including lactose) and other carbohydrates into lactic acid. This conversion not only provides the characteristic sour taste of fermented dairy foods such as yogurt but also by lowering the pH may create fewer opportunities for “bad bacteria” to grow, hence creating possible health benefits by preventing gastrointestinal infections. Strains of the Lacto-bacillus and Bifidobacterium are the most widely used probiotic

bacteria. Probiotic bacterial cultures are intended to help the body's naturally occurring gut microbiota flora, an ecology of microbes (the "good bacteria"), to re-establish themselves. They are sometimes recommended after a course of antibiotics. Claims are made that probiotics strengthen the immune system and gastrointestinal barrier function to help combat infections, allergies, excessive alcohol intake, stress, exposure to toxic substances, and other diseases. Indeed, there are many examples of positive results with different probiotic strains against a range of disease states in both animals and humans, but it is evident that their health-promoting traits are strain-specific. There is evidence in humans that some probiotic strains can help to reduce colonic inflammation, antibiotic-induced diarrhoea, some allergic conditions and both gut and respiratory infections. Some studies in athletes support the use of probiotics to reduce incidence and/or symptom severity of upper respiratory tract infections (Jeukendrup & Gleeson, 2018).

Instead of consuming probiotics, people can eat foods for the "good" bacteria to feed on. These foods, known as prebiotics, consist of indigestible food fibres and complex carbohydrates that specifically stimulate the growth of good bacteria in the bowel. Examples include inulin, oligofructose, galactofructose, galacto-oligosaccharides and xylo-oligosaccharides. It has been argued that it may be more effective to take prebiotics that boost growth of the good bacteria already present in the gut rather than take supplements of live bacteria that may be destroyed by the acidity of the stomach as soon as they are swallowed. Prebiotics are found naturally in small amounts in foods such as wheat, oats, bananas, asparagus, leeks, garlic, and onions. But to get an adequate daily dose, people may want to look for foods in the supermarket that have been enriched with prebiotics or even consider prebiotic supplements. As with probiotics, there is convincing evidence from animal studies showing efficacy in prevention or treatment of many diseases (e.g., IBD, colon cancer, obesity, type 2 diabetes and cardiovascular disease), but the data in humans remain ambiguous. (Jeukendrup & Gleeson, 2018, https://bit.ly/2LC9XB7)

Polyphenols are a diverse class of plant secondary metabolites, often associated with the colour, taste and defence mechanisms of fruit and vegetables. They have long been studied as the most likely class of

compounds present in whole plant foods capable of affecting physiological processes that protect against chronic diet-associated diseases. The gut microbiota plays a critical role in transforming dietary polyphenols into absorbable biologically active compounds and recent studies show that dietary intervention with polyphenol extracts, most notably de-alcoholised red wine polyphenol extract and cocoa-derived flavanols, modulate the human gut microbiota towards a more 'health-promoting profile' by increasing the relative abundance of bifidobacteria and lactobacilli. These data raise the possibility that certain functional foods tap into the underlying ecological processes regulating gut microbiome community structure and function, contributing to the health of the gut microbiota and its human host.

The latest research shows that both probiotics and prebiotics may have widespread health benefits. Likely mediated through immune influences, the effects of prebiotics and probiotics may reach beyond the gastrointestinal tract and include systemic effects such as reduced severity of colds or other respiratory conditions, lower incidence and reduced symptoms of allergy, and fewer absences from work or day-care. However, this field is in its infancy. There is an incomplete understanding of the role of the microbiome to performance. Any services that are offered that link the microbiome to performance outcomes are therefore also premature.

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