

# The Application of Lactose in Sports Nutrition

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1 Title: The Application of Lactose in Sports Nutrition

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26 **Abstract (150 words):**

27 Lactose is a disaccharide of glucose and galactose, found exclusively in milk. Carbohydrates  
28 represent an important fuel for endurance and prolonged exercise. Recommendations for  
29 athletes include high carbohydrate diets to maximise performance, especially before, during  
30 and after exercise. However, lactose does not feature in guidelines for carbohydrate intake for  
31 athletes, despite athletes likely consuming nutritionally relevant amounts. This review will  
32 explore possible applications for lactose in a sports nutrition context. These include lactose as  
33 a fuel source, for before and during exercise, where maximizing availability of readily  
34 oxidisable carbohydrate can optimise performance. Lactose could play a role in a post-exercise  
35 recovery setting, as a vehicle for the delivery of glucose and galactose, for the optimisation of  
36 muscle and liver glycogen. Lactose may also act as a prebiotic, possibly promoting beneficial  
37 changes to gut microbiota. A discussion of the possible risks associated with lactose over-  
38 consumption and intolerance will also be considered.

39

40 **Keywords:** sugar, carbohydrate, sports nutrition, exercise metabolism, milk sugar

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51 **1. Introduction**

52 Lactose is a disaccharide originating in mammalian milk, and is comprised of the  
53 monosaccharides glucose and galactose, bound by a  $\beta$  1-4 glycosidic linkage (Mattar, de  
54 Campos Mazo, & Carrilho, 2012). Lactose within milk-derived dairy products is an extrinsic  
55 sugar, meaning it is not bound into the cellular structure of food (unlike intrinsic sugars such  
56 as glucose as starch in vegetables), and is free in the foods it is found in (Edgar, 1993). Milk-  
57 derived dairy products – containing variable amounts of lactose – have been a constituent of  
58 the human diet for approximately 8 000 years and feature prominently in the nutritional  
59 recommendations of most countries (Rozenberg et al., 2016). Governmental dietary  
60 recommendations encourage dairy intake in healthy individuals in the preponderance of  
61 countries, notably for their high calcium content, along with other micronutrients (U.S.  
62 Department of Health and Human Services and U.S. Department of Agriculture, 2015; Wang,  
63 Lay, Yu, & Shen, 2016) demonstrating the extensive demand for milk and dairy products.  
64 Dietary sources of lactose are numerous, with bovine milk usually containing the greatest  
65 lactose content of common foods (See **Table 1**). Isolated lactose and whey permeate, (a high-  
66 lactose (76-85 %) dairy ingredient produced when proteins are removed from liquid whey in  
67 the manufacturing of whey protein isolate) are used extensively to improve technical aspects  
68 of product formulation in foodstuffs such as baked goods, confectionary and soups (U.S. Dairy  
69 Export Council, 2015). Such dairy ingredients are also widely used in non-specific roles, such  
70 as bulking agents, animal feed or as fillers in capsules. Thus, lactose may feature in the human  
71 diet from a variety of traditional dairy and non-dairy sources.

72

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75

76 **Table 1.** Lactose content of a variety of dairy products

77

Food	Lactose content (g) per 100 g / 100 ml
Cow's Milk	5.0
Lactose-free milk	<1.0
Ice cream	3.3 - 6.0
Cottage cheese	0.3 - 2.4
Cream cheese	2.5 - 3.0
Hard cheeses	≤0.1
Yoghurt	4.5 – 6.0
Whey protein concentrate	3.5

78

79 (Dieticians of Canada, 2013; Misselwitz et al., 2013; Tetra Pak, 2018)

80

81 Despite the widespread potential presence of lactose, surprisingly limited information on the  
 82 contribution of lactose to the total energy intake (EI) of different populations is available. Most  
 83 investigations into the sugar content of individuals' diets have been primarily concerned with  
 84 added sugars and therefore lactose predominantly originating from dairy-derived products has  
 85 often been excluded, typically being classed as a non-added sugar. However, a limited number  
 86 of more specific estimations of lactose intake have been made. In Swedish women average  
 87 lactose intake was  $12 \pm 8 \text{ g}\cdot\text{day}^{-1}$ , approximately 50 kcal or ~2% of EI (Larsson, Bergkvist, &  
 88 Wolk, 2004). Habitual lactose consumption in Canadian adults was reported to be similar, with  
 89 milk sugar comprising ~11% of total sugar intake, estimated at approximately  $\sim 12 \text{ g}\cdot\text{day}^{-1}$   
 90 (Brisbois, Marsden, Anderson, & Sievenpiper, 2014). Whilst lactose is present in the diet, it

91 represents a relatively small amount, particularly in comparison to the other major dietary  
92 disaccharide sucrose. By comparison, mean added sugar intake (predominantly sucrose) totals  
93 ~58 g·day<sup>-1</sup> far exceeding lactose intake (Brisbois et al., 2014) . Nonetheless, it is important to  
94 note that the presence of lactose in the diet varies considerably, depending on lactose tolerance  
95 status, food preference, and numerous other factors (Keith, Nicholls, Reed, Kafer, & Miller,  
96 2011).

97

98 Though it has not been directly investigated, it is possible that athletes and recreationally active  
99 individuals may have a higher lactose intake than the general population. Athletes have a high  
100 EI to permit high volumes and intensities of training, far exceeding the EI of non-athletic  
101 populations (Grandjean, 1997). Athletes also have specific macronutrient requirements to  
102 facilitate recovery (including carbohydrate and protein) which can be conveniently obtained  
103 from dairy foods such as milk (James, Stevenson, Rumbold, & Hulston, 2019), or dairy-derived  
104 products, such as whey or casein protein supplements, which can contain ~4% lactose (Tetra  
105 Pak, 2018). However, the specific role of lactose in the diets of athletes or recreationally active  
106 individuals has rarely been considered. Apart from guidance on the avoidance of lactose for  
107 those recommended low FODMAP (fermentable oligo-, di-, mono-saccharides and polyols)  
108 diets, in contrast to other carbohydrates lactose does not feature explicitly in sports nutrition  
109 guidelines, perhaps due to a paucity of research. Because athletes may be consuming  
110 nutritionally relevant quantities of lactose that exceed those consumed by the general  
111 population, it is important to elucidate its metabolic effects so that its specific application can  
112 be understood in the context of sports nutrition. Therefore, this review will briefly summarise  
113 lactose metabolism, critically review the literature relating to the utility of lactose for athletes,  
114 highlight potential risks of lactose consumption and suggest areas for future research.

115

116 **2. Lactose digestion, absorption and metabolism**

117 Lactose is digested exclusively by the disaccharidase lactase (lactase-phlorizin hydrolase),  
118 located on the brush border of the small intestine (See **Figure 1**). Lactose is hydrolysed at its  
119  $\beta$  1-4 glycosidic linkage, releasing the constituent monosaccharides; glucose and galactose  
120 (Mattar et al., 2012). Both glucose and galactose are then actively transported by sodium-  
121 glucose transporter proteins (SGLT1), followed by GLUT2 allowing entry to the hepatic portal  
122 vein (Thorens, 2014; Turk, Martingn, & Wright, 1994).

123

124 The glucose component of lactose reaches the liver, where first pass extraction occurs. Glucose  
125 is directed towards metabolic processes such as liver glycogen synthesis, or oxidised. However,  
126 much of the absorbed glucose will escape this fate and enter the systemic circulation.  
127 Divergence from blood glucose homeostasis results in insulin release (Koeslag, Saunders, &  
128 Terblanche, 2003). This leads to glucose uptake by tissues through GLUT4 transporter  
129 proteins, most notably in skeletal muscle leading to the formation of glycogen through the  
130 action of glycogen synthase (Boucher, Kleinridders, & Kahn, 2014). Plasma glucose that  
131 appears after ingestion can also be oxidised directly by peripheral tissues, including skeletal  
132 muscle and the brain (Kelley et al., 1988). This is the typical metabolism of ingested glucose,  
133 including free glucose and that derived after digestion of disaccharides or polysaccharides  
134 (such as sucrose or starch respectively) containing glucose. Ingested glucose is metabolised  
135 efficiently in healthy, insulin sensitive individuals, which minimises prolonged elevations in  
136 blood glucose to ~60 minutes, depending on the quantity of glucose and the form of ingestion  
137 (Abdul-Ghani, Lyssenko, Tuomi, Defronzo, & Groop, 2010). Glucose is the comparator  
138 carbohydrate used in the glycaemic index, an index of the 2-hr blood glucose response after  
139 carbohydrate ingestion *versus* the reference (100). Lactose has a lower glycaemic index of 46

140 due to the presence of a galactose molecule, and similarly the index of sucrose (61) is lowered  
141 by the presence of a fructose molecule (Wolever & Miller, 1995).

142

143 The metabolism of galactose is markedly different to glucose. Lactose is the primary dietary  
144 source of galactose, though it is also present in smaller amounts in legumes (e.g. garbanzo  
145 beans, lentils, soybean), and as bound galactose in other vegetables (Acosta & Gross, 1995).  
146 Like glucose, galactose is actively transported into the hepatic portal vein via SGLT1, and  
147 GLUT2 (Thorens, 2014; Turk et al., 1994). The hepatic portal vein transports galactose to the  
148 liver where first pass extraction occurs, and some galactose enters the Leloir pathway, the  
149 primary pathway for galactose metabolism in humans. The end-product of this pathway is  
150 glucose-1-phosphate, which is then available for glucose production or glycogen storage.  
151 However, the rate of gluconeogenesis from galactose is limited, which leads to the  
152 accumulation of galactose in the circulation in large amounts and in a sustained manner.  
153 Williams, Phillips, and Macdonald (1983) demonstrated that ingestion of  $0.5 \text{ g} \cdot \text{kg}^{-1}$  body mass  
154 of galactose caused substantial accumulation of plasma galactose, increasing from  $<0.1$   
155  $\text{mmol} \cdot \text{L}^{-1}$  at baseline to  $1.70 \pm 0.42 \text{ mmol} \cdot \text{L}^{-1}$  after 60 min. By feeding subjects  $\sim 22 \text{ g} \cdot \text{h}^{-1}$  of  
156 galactose, Sunehag and Haymond (2002) estimated the maximal rate of splanchnic uptake to  
157 be  $\sim 10 \text{ g} \cdot \text{h}^{-1}$ . Because the liver is the primary site for galactose metabolism, and there is little  
158 evidence for extra-hepatic metabolism, circulating galactose is ultimately removed via hepatic  
159 metabolism or renal losses.

160

161 It has been observed that glucose can augment the metabolism of galactose when the two free  
162 monosaccharides are co-ingested (Sunehag & Haymond, 2002). Co-ingestion of galactose and  
163 glucose more than doubles the first pass splanchnic clearance of ingested galactose compared  
164 to galactose alone and can eliminate or drastically reduce the rise in plasma galactose after



165 ingestion. Furthermore, glucose production was shown to be almost exclusively derived from  
166 galactose under these conditions, *via* the Leloir Pathway. However, the mechanisms explaining  
167 this increased splanchnic uptake have not been fully elucidated. It appears that intact lactose  
168 can exert a similar effect upon galactose metabolism to free glucose and galactose ingestion,  
169 both resulting in attenuated rises in plasma galactose (Williams et al., 1983). This occurs  
170 despite the requirement for digestion. In addition, Stellaard et al. (2000) demonstrated that the  
171 hydrolysis of lactose is not the rate-limiting step in its oxidation. Subjects ingested 40 g of  
172 naturally high <sup>13</sup>C lactose or pre-digested lactose (glucose and galactose) and showed that  
173 breath <sup>13</sup>CO<sub>2</sub> recovery over 4 hours was not significantly different indicating that both  
174 conditions oxidised the constituent monosaccharides of lactose at the same rate, even when  
175 delivered as the disaccharide. This may suggest that lactose and its constituent  
176 monosaccharides exert similar metabolic effects, without much impact of the digestion stage,  
177 at least at this dose. This augmentation of galactose metabolism by glucose has only been  
178 directly investigated under resting conditions.

179

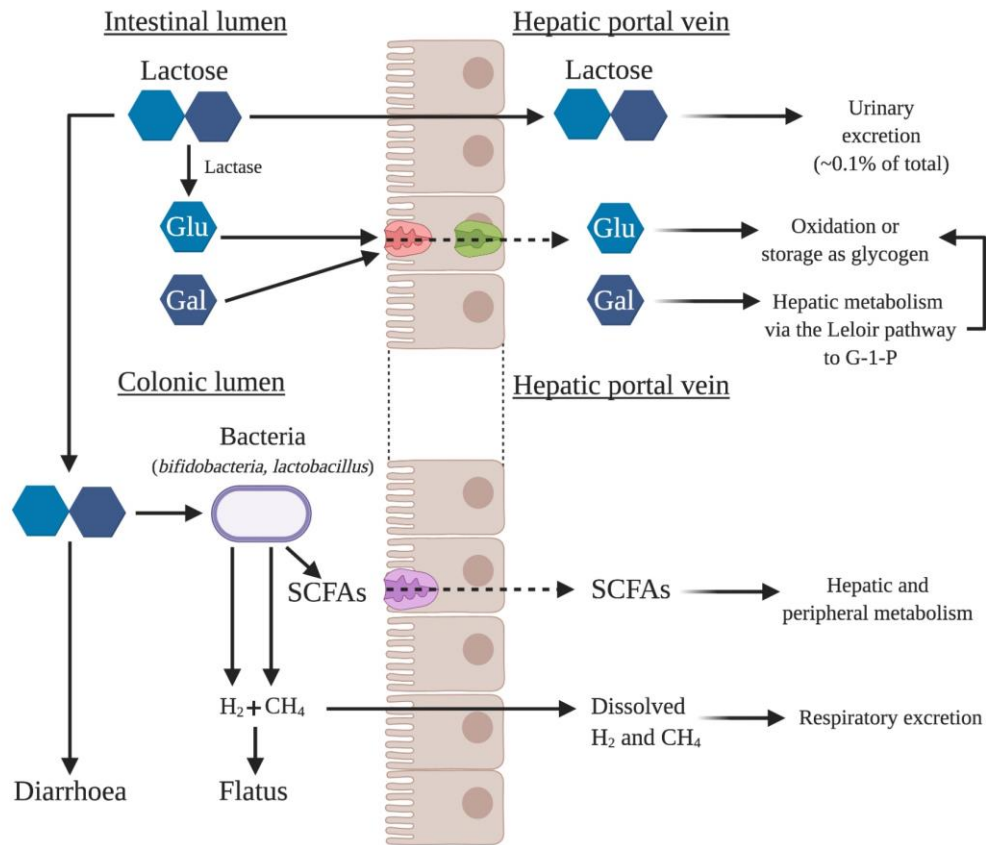
180 The metabolic fate of lactose described previously assumes that lactose hydrolysis has  
181 occurred. It has been demonstrated that small amounts of lactose (0.02 mmol·L<sup>-1</sup>) can appear  
182 in plasma following milk ingestion (~40g lactose; See **Figure 1**) in normal, non-pathological  
183 conditions (Pimentel et al., 2017). This was hypothesised to be due to passive diffusion through  
184 intercellular junctions in the intestinal cells at high lactose concentrations, which is  
185 subsequently excreted in the urine. If lactose is not digested in the small intestine (termed  
186 lactose maldigestion), it can pass from the small intestine into the colon (Misselwitz et al.,  
187 2013). Lactose may be fermented by a variety of local bacterial flora, which include  
188 *Bifidobacteria*, *Lactobacillus*, *Clostridium* and *Bacteroides* (Ito & Kimura, 2009). The  
189 fermentation of lactose by colonic bacteria can yield short-chain fatty acids (SCFA), lactic

190 acid, CH<sub>4</sub>, H<sub>2</sub> and CO<sub>2</sub> in different quantities depending on the bacteria (Jiang & Savaiano,  
191 1997a, 1997b; Rangel et al., 2016; Yazawa & Tamura, 1982). Some of these products of  
192 fermentation can trigger gastrointestinal (GI) symptoms, which is termed lactose intolerance,  
193 and can depend on the quantity of lactose ingested. Section 4.1 discusses in greater detail these  
194 pathologies related to lactose ingestion.

195

196 The majority of research on lactose ingestion and metabolism has been performed under resting  
197 conditions, with limited studies directly investigating lactose metabolism in an exercise  
198 context. Nonetheless, studies performed at rest, and those limited studies involving exercise  
199 will be used in this narrative review to explore the possible impact of lactose on salient aspects  
200 of sports nutrition, with a particular focus on lactose as an energy source for exercise, recovery  
201 from exercise and lactose as a prebiotic. There will also be a discussion of the possible  
202 considerations and risks of over-consumption of lactose, which must be considered before  
203 practical application in a sporting context.

204



205

206 **Figure 1.** The digestion and absorption of lactose. Glu – glucose. Gal – galactose. SCFAs –  
 207 short chain fatty acids. CH<sub>4</sub> – methane. H<sub>2</sub> – hydrogen. G-1-P – glucose-1-phosphate. Created  
 208 with BioRender.com

209

### 210 3. The role of carbohydrate in sports nutrition

211 During exercise the availability of endogenous carbohydrate (as plasma glucose, liver &  
 212 muscle glycogen) may be reduced to due to the high energy cost of training or competition. At  
 213 moderate to high exercise intensities ( $\geq 60\text{-}70\%$   $\dot{V}O_{2\text{max}}$ ) carbohydrate is the predominant  
 214 energy source for ATP resynthesis (Bergman & Brooks, 1999; van Loon, Greenhaff,  
 215 Constantin-Teodosiu, Saris, & Wagenmakers, 2001). Due to the limited quantities of stored  
 216 carbohydrate, availability can limit performance in submaximal or intermittent high-intensity  
 217 exercise of a duration  $>90$  min, and is also required for short-lived, high intensity exercise  
 218 performance (Burke, Hawley, Wong, & Jeukendrup, 2011). In addition to being an energy

219 substrate, glycogen can also regulate adaptive signalling pathways. Therefore, carbohydrate  
220 availability can be positive or negative with regards to adaptation. Although adaptations to  
221 resistance-type exercise may be impacted positively by high glycogen availability, the converse  
222 is true for endurance-type exercise (Mata et al., 2019). However, carbohydrate availability is  
223 pivotal to sustaining high exercise intensities, therefore high-intensity training sessions, and  
224 competitions should be performed with high carbohydrate availability.

225

226 Recommendations for maximising carbohydrate availability for exercise are typically divided  
227 into pre-exercise and during exercise. Carbohydrate intake in the hours preceding endurance,  
228 or extended exercise is important to ensure high endogenous carbohydrate availability, as both  
229 muscle and liver glycogen. This is imperative when multiple endurance exercise sessions are  
230 completed in one day, such as stage races, Ironman events, or in tournament settings. It is also  
231 necessary to ensure high carbohydrate availability following an overnight fast, during which  
232 liver glycogen may be reduced (Papin, König, Bulik, & Holzhütter, 2012). Ingesting  
233 carbohydrate during exercise (such as a drink or gel) is an effective strategy for improving  
234 performance in a variety of exercise scenarios, including endurance and intermittent exercise  
235 (Jeukendrup, 2011). Ingestion of additional carbohydrate provides an exogenous fuel source,  
236 which can reduce the reliance on stored carbohydrate, permit glycogen sparing, prevent  
237 hypoglycaemia, and positively impact the central nervous system (Burke et al., 2011).

238

### 239 **3.1 Lactose as an energy source for exercise**

240 Expert recommendations for pre-exercise carbohydrate intake suggest 1-4 g·kg<sup>-1</sup> body mass in  
241 the preceding 1-4 hours (Burke et al., 2011). The meal should contain little fibre, fat and  
242 protein, which all delay gastric emptying and could contribute to GI distress or discomfort  
243 (Nuttall & Gannon, 1991). Lactose ingestion pre-exercise may be advantageous due to its low

244 glycaemic index of 46 (Wolever & Miller, 1995). Current guidelines recommend a low  
245 glycaemic index meal before exercise to prevent post-prandial hyperglycaemia, which may  
246 promote better maintenance of carbohydrate availability during exercise (Burke et al., 2011).  
247 However, there does not appear to be a benefit to exercise performance with low glycaemic  
248 index carbohydrate ingestion before exercise, compared to high glycaemic index carbohydrate  
249 ingestion (Burdon, Spronk, Cheng, & O'Connor, 2017). Some athletes may avoid lactose-  
250 containing dairy foods in the meal before exercise due to concerns around GI discomfort.  
251 However, Haakonssen et al. (2014) demonstrated that a pre-exercise high carbohydrate dairy  
252 meal containing ~40g of lactose resulted in no differences in gut discomfort or performance  
253 during a cycling time-trial compared to a high carbohydrate dairy-free meal. Although there  
254 has been no direct investigation into lactose ingestion pre-exercise *per se*, there are no reasons  
255 to indicate that lactose could not form part of a pre-exercise carbohydrate ingestion regime.

256

257 Recommendations for carbohydrate intake during exercise, including type and amount, depend  
258 on intensity and duration. Endurance exercise lasting <30 min does not require carbohydrate  
259 intake, whilst exercise lasting 30-75 min may require small amounts of carbohydrate  
260 (Jeukendrup, 2011). Exercise lasting 60-120 min requires greater intake of 30-60 g·h<sup>-1</sup> of most  
261 carbohydrate forms, whilst durations of 120-180 min require up to 60 g·h<sup>-1</sup> of rapidly  
262 oxidizable carbohydrate (glucose, glucose polymers). Exercise lasting >150 min may require  
263 up to 90 g·h<sup>-1</sup> and necessitates the use of multiple transportable carbohydrates (glucose and  
264 fructose). Carbohydrate may be consumed in a liquid, gel or solid form, as all are oxidised at  
265 comparable rates (B. Pfeiffer, Stellingwerff, Zaltas, Hodgson, & Jeukendrup, 2010; B Pfeiffer,  
266 Stellingwerff, Zaltas, & Jeukendrup, 2010). The guidelines on carbohydrate ingestion during  
267 exercise do not mention lactose, and focus on ingestion of glucose, glucose polymers, sucrose

268 and fructose. This is likely due to a paucity of research into lactose and the limited data on its  
269 metabolism in an exercise context.

270

271 There are limited data on the metabolism of lactose and its monosaccharide constituents during  
272 exercise. However, pre- and during-exercise ingestion of free glucose and galactose in a 1:1  
273 ratio has been investigated as a method to improve time trial (TT) performance (Stannard,  
274 Hawke, & Schnell, 2009). Subjects consumed galactose, galactose/glucose (1:1 ratio) or  
275 glucose/fructose (4:1 ratio) 45 min before beginning cycling for 120 min, while consuming 1  
276 g·min<sup>-1</sup>, followed by a TT. TT performance was poorest in the galactose-only condition, and  
277 the glucose/galactose and glucose/fructose conditions were not different. Ingested galactose is  
278 oxidised ~50-60 % slower than glucose, due to the requirement for hepatic metabolism  
279 (Burelle, Lamoureux, Peronnet, Massicotte, & Lavoie, 2006; Leijssen, Saris, Jeukendrup, &  
280 Wagenmakers, 1995), which may explain the poorer TT performance with galactose ingestion.  
281 This demonstrates the potential for a blend of the free monosaccharides constituting lactose,  
282 both for improving TT performance, and for the maintenance of blood glucose concentration  
283 during exercise. Glucose production is almost exclusively derived from galactose when it is  
284 co-ingested with glucose at rest (Sunehag & Haymond, 2002), though it is not clear whether  
285 this is the case under exercise conditions when hepatic and GI metabolism are altered.

286

287 Lee, Maughan, Shirreffs, and Watson (2008) investigated the effects of ingestion of low-fat  
288 milk during exercise. Subjects exercised at 70%  $\dot{V}O_{2peak}$  to exhaustion, whilst ingesting water,  
289 a glucose drink (~36 g·h<sup>-1</sup> of carbohydrate), low-fat milk (~30 g·h<sup>-1</sup> of carbohydrate) or low-  
290 fat milk with added glucose (~36 g·h<sup>-1</sup> of carbohydrate). Whilst time to exhaustion (TTE) was  
291 not different between any conditions, when considered as percentage change *versus* water, the  
292 carbohydrate drink and milk ingestion resulted in an extended TTE. Although milk ingestion

293 does not isolate the lactose component and the confounding effects of other macronutrients in  
294 milk are not accounted for, this suggests that lactose could be a viable exogenous energy source  
295 during exercise. Additionally, Stellaard et al. (2000) compared ingestion of naturally high  
296 abundance  $^{13}\text{C}$  glucose and lactose before 4 hours of light physical activity, and showed  $^{13}\text{CO}_2$   
297 recovery was comparable between carbohydrates. This implies that the oxidation of lactose  
298 may be equal to glucose alone and also suggests that oxidation of galactose is likely to have  
299 been augmented by the presence of glucose. However, interpretation of this study is hampered  
300 by its small sample size ( $n=5$ ) and low exercise intensity (50 W).

301

302 Recently, Odell, Podlogar, and Wallis (2020) directly investigated the metabolic impact of  
303 lactose ingestion during endurance exercise. Subjects ingested  $0.8 \text{ g} \cdot \text{min}^{-1}$  of lactose or sucrose,  
304 or water during 150 min of steady-state cycling at 50%  $W_{\text{max}}$ . Ingested lactose and sucrose  
305 were oxidised at comparable rates ( $0.56 \pm 0.19 \text{ g} \cdot \text{min}^{-1}$  and  $0.61 \pm 0.10 \text{ g} \cdot \text{min}^{-1}$  respectively)  
306 during exercise. Despite the presence of galactose in the lactose condition, the exogenous  
307 carbohydrate oxidation rates were similar to sucrose, suggesting that the presence of glucose  
308 may have influenced the oxidation of galactose, which is known to occur at rest (Sunehag &  
309 Haymond, 2002). Exogenous lactose oxidation may have been slower than sucrose in the first  
310 ~30 minutes of the exercise bout, which may represent a delay in digestion, or possibly the  
311 slow metabolism of galactose via the Leloir pathway. However, this difference was non-  
312 significant, and lactose showed near-identical exogenous carbohydrate oxidation rates in the  
313 later stages of exercise. In addition, lactose ingestion promoted greater fat oxidation than  
314 sucrose, though not as high as that observed when water only was consumed. This may be due  
315 to lactose being less insulinemic than glucose at rest (Ercan, Nuttall, Gannon, Redmon, &  
316 Sheridan, 1993). Concurrently, oxidation of endogenous carbohydrate was lower with lactose  
317 ingestion than sucrose ingestion, which may represent the sparing of glycogen. Furthermore,

318 lactose ingestion was also effective at maintaining stable blood glucose during exercise, much  
319 like sucrose, unlike when water was consumed.

320

321 Lactose clearly has the potential to act as a viable exogenous energy source during, and  
322 potentially before exercise. It performs comparably to other carbohydrates in isolation and  
323 within milk, in terms of positive metabolic outcomes for exercise. Consideration of individual  
324 differences and lactose tolerance status should be made (see Section 4.1). Future investigations  
325 should seek to determine the source of the observed glycogen sparing that occurs with lactose  
326 ingestion (Odell et al., 2020), as well as its impact upon exercise performance or capacity  
327 *versus* other carbohydrates.

328

### 329 **3.2 Lactose in a post-exercise setting**

330 Prolonged endurance or intermittent exercise can deplete or reduce glycogen, stored in the liver  
331 and the muscle. Whilst muscle glycogen is hydrolysed and oxidised locally by the muscle, liver  
332 glycogen is utilised in the maintenance of blood glucose homeostasis (Taylor et al., 1996). The  
333 impact of pre-exercise muscle glycogen content on exercise performance is well established.  
334 Low muscle glycogen content causes reduced endurance performance and capacity, whilst high  
335 muscle glycogen content predicts better performance and capacity (Bergström, Hermansen,  
336 Hultman, & Saltin, 1967; Widrick et al., 1993). Therefore, post-exercise replenishment of  
337 muscle and liver glycogen is a priority to ensure adequate endogenous carbohydrate for later  
338 exercise. This is particularly relevant for athletes in sports with multiple events in a day, such  
339 as in tournaments, events over multiple days or stage races such as the Tour de France, or even  
340 when multiple training sessions per day are performed.

341



342 Current recommendations for post-exercise nutrition advocate carbohydrate ingestion as soon  
343 as is practical to maximise preparedness for the next exercise bout. Current guidelines  
344 recommend ingestion of carbohydrate at a rate of  $1.2 \text{ g}\cdot\text{kg}^{-1}\cdot\text{h}^{-1}$  in the initial hours after  
345 exercise, which is sufficient to maximise muscle glycogen replenishment in a post-exercise  
346 state (Burke et al., 2011). It has been extensively demonstrated that if sufficient quantities of  
347 carbohydrates of moderate to high glycaemic index are provided in the post-exercise period,  
348 muscle glycogen can be replenished optimally, though it has been observed that some  
349 carbohydrates perform poorly in the restoration of muscle glycogen, such as fructose (Blom,  
350 Høstmark, Vaage, Kardel, & Maehlum, 1987). However, more recently there has been an  
351 interest in carbohydrates with divergent paths for endogenous storage, namely fructose and  
352 galactose.

353

354 It has been observed that post-exercise glucose ingestion is a relatively poor strategy for  
355 optimising the resynthesis of liver glycogen, despite its positive effects on muscle glycogen  
356 replenishment (Décombaz et al., 2011). Both fructose and galactose have been demonstrated  
357 to be superior substrates for liver glycogen replenishment when co-ingested with glucose,  
358 which is likely due to their requirement for hepatic metabolism. Décombaz et al. (2011)  
359 compared combined maltodextrin and galactose, fructose or glucose ingestion, in total doses  
360 of 450 g ( $\sim 69 \text{ g}\cdot\text{h}^{-1}$ ) after glycogen depleting exercise. Liver glycogen content was assessed  
361 via  $^{13}\text{C}$  magnetic resonance imaging (MRI) over a 6.5h recovery period. Both galactose ( $8.6 \pm$   
362  $0.9 \text{ g}\cdot\text{h}^{-1}$ ) and fructose ( $8.1 \pm 0.6 \text{ g}\cdot\text{h}^{-1}$ ) ingestion effectively doubled the liver glycogen  
363 deposition compared with glucose-only ( $3.7 \pm 0.5 \text{ g}\cdot\text{h}^{-1}$ ). This increase in liver glycogen  
364 resynthesis can also be observed with sucrose ingestion compared to glucose (Fuchs et al.,  
365 2016). This demonstrates the requirement for carbohydrate types that require hepatic  
366 metabolism, in addition to glucose. To the author's knowledge, Décombaz et al. (2011)

367 performed the only investigation into liver glycogen replenishment post-exercise comparing  
368 galactose to other carbohydrates.

369

370 To promote optimal combined liver and muscle glycogen replenishment post-exercise, it is  
371 likely that glucose must be combined with a carbohydrate source that undergoes predominant  
372 hepatic metabolism (i.e. galactose, fructose). It is possible that lactose, as a disaccharide of  
373 glucose and galactose, may be sufficient to promote combined muscle and liver glycogen  
374 resynthesis, effectively acting as a blend of glucose and galactose. However, it remains to be  
375 investigated whether combined glucose and galactose feeding is as effective at replenishing  
376 muscle glycogen as glucose alone, which has been demonstrated for combined glucose and  
377 fructose ingestion (Fuchs et al., 2016; Trommelen et al., 2016; Wallis et al., 2008). This is an  
378 important factor determining whether glucose and galactose or lactose may be advantageous  
379 compared to glucose alone in the restoration of glycogen stores.

380

381 Whilst no studies have investigated muscle or liver glycogen replenishment and/or recovery of  
382 exercise performance or capacity with lactose ingestion, milk and chocolate milk have been  
383 extensively studied in recovery from exercise (Alcantara, Sanchez-Delgado, Martinez-Tellez,  
384 Labayen, & Ruiz, 2019; Amiri, Ghiasvand, Kaviani, Forbes, & Salehi-Abargouei, 2018). Milk  
385 ingestion has been shown to be equally effective at improving exercise capacity after an initial  
386 bout of exercise as carbohydrate-electrolyte beverages delivering similar amounts of  
387 carbohydrate in a number of investigations (Lee et al., 2008; Watson, Love, Maughan, &  
388 Shirreffs, 2008). Indeed, Loureiro, de Melo Teixeira, Pereira, Reis, and da Costa (2020) in a  
389 systematic review concluded that milk-based beverages result in similar effects on glycogen  
390 resynthesis and restoration of exercise performance to carbohydrate beverages. Chocolate milk  
391 has also been investigated for post-exercise recovery, as a convenient, carbohydrate and protein

392 rich recovery beverage. A systematic review and meta-analysis by Amiri et al. (2018) showed  
393 that chocolate milk ingestion after a variety of exercise types (including running and cycling)  
394 increased time-to-exhaustion (TTE) in a subsequent exercise bout, compared to placebos and  
395 mixed macronutrient beverages (containing carbohydrate, fat and protein). Furthermore, an  
396 investigation into chocolate milk ingestion and glycogen replenishment demonstrated that  
397 chocolate milk resulted in higher muscle glycogen restoration than an isocaloric carbohydrate  
398 beverage after an exercise bout, despite lower total carbohydrate content ( $0.84 \text{ g} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$  *versus*  
399  $1.2 \text{ g} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$ ) in the chocolate milk condition, which may be attributable to its protein content  
400 (Karfonta, Lunn, Colletto, Anderson, & Rodriguez, 2010). Ferguson-Stegall et al. (2011)  
401 showed that after an intense cycling exercise bout, chocolate milk was equally effective at  
402 replenishing muscle glycogen as an isocaloric carbohydrate beverage but resulted in improved  
403 TT performance.

404

405 One potential benefit to chocolate milk ingestion is that it contains a full spectrum of  
406 monosaccharides, including galactose (as lactose), fructose (as sucrose) and glucose from both  
407 disaccharides. Although it has not been directly investigated, the combination of two  
408 carbohydrates known to increase liver glycogen replenishment, as well as glucose, could have  
409 an additive effect over and above just one of these carbohydrates. This could explain the  
410 superior effects of chocolate milk ingestion compared to the carbohydrate (glucose) beverage  
411 on TT performance (Ferguson-Stegall et al., 2011). However, it is not possible to isolate the  
412 effects of the lactose component of milk or chocolate milk, compared to the other nutrients in  
413 milk such as protein, which can amplify insulin secretion and glycogen replenishment when  
414 carbohydrate intake is insufficient (Burke et al., 2011) as well as fat and electrolytes. A further  
415 benefit to chocolate milk in the post-exercise setting is its high protein content (~16 g per 500  
416 ml), and high protein quality, containing whey and casein (Amiri et al., 2018). Indeed, Phillips,

417 Tang, and Moore (2009) note that milk proteins are superior to carbohydrate alone for post-  
418 exercise nutrition in the promotion of skeletal muscle hypertrophy. Therefore, chocolate milk  
419 and other flavoured dairy beverages can be considered multifunctional foods in a post-exercise  
420 setting.

421

### 422 **3.3 Lactose as a prebiotic**

423 There is potential for lactose, in some scenarios to act as a prebiotic, which represents an  
424 additional application in sports nutrition. Prebiotics are indigestible compounds, that are  
425 metabolised by gut microbiota, which affect the microbiota composition or activity (Bindels,  
426 Delzenne, Cani, & Walter, 2015) in a manner beneficial to the host (Carlson, Erickson, Lloyd,  
427 & Slavin, 2018). This potential aspect of lactose is supported by only limited research, and so  
428 the potential for lactose to act as a prebiotic requires more thorough investigation to further  
429 understand any possible benefits that could be conferred. Lactose can be considered a prebiotic,  
430 as in some individuals, undigested lactose may pass from the small intestine to the colon, where  
431 it is available for fermentation by local bacteria, much like established prebiotics such as  
432 galactooligosaccharides (GOS) and fructooligosaccharides (FOS) (Carlson et al., 2018). This  
433 is likely most common in individuals with a limited ability to digest lactose, though undigested  
434 lactose could also appear in the colon of individuals with a normal ability to digest lactose,  
435 thereby also conferring a prebiotic effect. However, in both groups this is likely dependent on  
436 the amount of lactose ingested and the individual's lactose digestion capacity.

437

438 The mechanisms explaining the positive impacts of prebiotics are not fully understood but may  
439 be linked to the cultivation of beneficial colonic bacteria, capable of producing short chain fatty  
440 acids (SCFAs) which are favourable for bowel health and immune function (Parada Venegas  
441 et al., 2019). Prebiotics could offer athletes and recreationally active individuals a performance

442 benefit through a number of indirect mechanisms, though to the authors knowledge, the impact  
443 of prebiotic supplementation on athletic performance has not been assessed. However, some  
444 benefits have been observed in healthy individuals, which may be applicable to athletes.  
445 Supplementation with GOS is associated with reduced GI illness, as well as reduced cold and  
446 flu symptom severity (Hughes et al., 2011). Preventing illness in athletes may reduce  
447 interruption to training or attenuate the direct impact of illness upon exercise performance in  
448 competition. Another potential benefit is the suppression of appetite by FOS supplementation  
449 that has been observed, which may benefit energy-restricted athletes (Cani et al., 2009).  
450 Although different prebiotics exert their effects through similar mechanisms, it is not known  
451 whether certain effects are linked to specific prebiotics. Therefore, the benefits of certain  
452 prebiotics should not be ascribed to others without direct investigation.

453

454 Whether lactose exerts a prebiotic effect has not been investigated, and it is not clear whether  
455 any prebiotic effect might occur alongside GI symptoms due to the presence of lactose in the  
456 colon. However, ingestion of any prebiotic can lead to GI symptoms, including flatulence and  
457 osmotic diarrhoea, which can be modulated by individual factors, as well as the total dose  
458 (Livesey, 2001). It is possible that regular consumption of dairy products containing lactose  
459 may confer some prebiotic effect in both lactose digesters and maldigesters, though the quantity  
460 of lactose required to achieve this is not known. Although not directly related to sports  
461 nutrition, prebiotics may also exert a number of health benefits which include a protective  
462 effect on colon cancer and the reduction of certain risk factors for cardiovascular disease  
463 (Slavin, 2013). These health benefits are thought to be conferred by SCFA production, of which  
464 butyrate is likely the most beneficial for colonic health.

465

466 **4. Considerations for the application of lactose in sports nutrition**

#### 467 **4.1 Relevant conditions and pathologies, relating to lactose**

468 The application of lactose in sports nutrition may be confounded due to the presence of a  
469 variety of pathologies related to lactose digestion and absorption, including lactose  
470 maldigestion and subsequent intolerance. These conditions are a result of lactase non-  
471 persistence, the genetically determined, gradual decline in lactase activity to ~10 % of previous  
472 activity, after 2 – 3 years of age (Misselwitz et al., 2013). This condition is present in >90% of  
473 East Asians and ~70% of Mexicans and African Americans (Itan, Jones, Ingram, Swallow, &  
474 Thomas, 2010; Scrimshaw & Murray, 1988). However, in some individuals the activity of  
475 lactase does not reduce, remaining high into adulthood, which is termed lactase persistence,  
476 which is the normal condition in 80-95% of adults in Britain, Scandinavia and Germany (Itan  
477 et al., 2010). The development of lactase persistence is thought to be linked to the development  
478 of dairy farming over the past ~10 000 years (Ingram, Mulcare, Itan, Thomas, & Swallow,  
479 2009).

480

481 Lactose maldigestion refers to the inefficient or incomplete digestion of lactose, generally due  
482 to lactase non-persistence (Misselwitz et al., 2013). Lactose maldigestion may also be referred  
483 to as lactose malabsorption. However, this term does not accurately describe the condition, as  
484 the absorption of lactose is not impaired, whereas the digestion stage is limited. Therefore, the  
485 term maldigestion should be used when referring to the ineffective digestion of lactose and use  
486 of the term malabsorption should be avoided unless specifically describing reduced absorption  
487 of lactose (or its constituent's glucose and galactose) into the splanchnic circulation.

488

489 Lactose intolerance (LI) refers to the GI symptoms that result from lactose maldigestion, which  
490 are a result of unhydrolyzed lactose passing from the small intestine into the colon (Misselwitz  
491 et al., 2013). These can include diarrhoea due to the lactose increasing the osmotic load, which

492 draws water into the intestines, and can lead to abdominal cramping or pain (Rangel et al.,  
493 2016). Flatulence and bloating can also result from the fermentation of the lactose by colonic  
494 bacteria which may produce gases including CH<sub>4</sub>, H<sub>2</sub> and CO<sub>2</sub> (Jiang & Savaiano, 1997a,  
495 1997b; Rangel et al., 2016; Yazawa & Tamura, 1982). Certain bacteria favour reduced gas  
496 production (such as *Bifidobacteria* and *Lactobacillus*) compared to other colonic bacteria  
497 (Tsukahara, Azuma, & Ushida, 2009).

498

499 The symptoms of LI, and their severity vary widely between individuals, depending on the  
500 individual's lactase activity and bacterial flora. There may also be variation between LI  
501 symptoms in the same individual in response to different foods or meals. For example, high fat  
502 or high osmolality foods can slow gastric emptying, which can reduce symptoms of intolerance  
503 (Jellema, Schellevis, Van Der Windt, Kneepkens, & Van Der Horst, 2010). Ingesting yoghurt  
504 can improve GI symptoms and breath H<sub>2</sub> response compared to milk, which is likely due to the  
505 presence of bacteria capable of hydrolyzing the lactose (Pelletier, Laure-Boussuge, &  
506 Donazzolo, 2001). Consideration of the dairy 'matrix' is important when discussing LI and  
507 other issues surrounding dairy and health, rather a reductionist single-ingredient approach  
508 (Szilagyi & Ishayek, 2018).

509

510 It is important to note that many individuals with lactose maldigestion can tolerate small  
511 amounts of lactose without GI symptoms. Indeed 0.4 g of lactose ingested as a pill did not alter  
512 GI symptoms significantly compared to a placebo, nor did it trigger a positive (>20 ppm) breath  
513 H<sub>2</sub> test (Montalto et al., 2008). Shaukat et al. (2010) reported that dairy products containing up  
514 to 12-15g of lactose are well tolerated by most adults with lactose maldigestion, equivalent to  
515 250-300 mL of milk. Therefore, consumption of dairy products, as well as dietary supplements  
516 or medication capsules that use lactose as a filler should not necessarily be avoided by lactose

517 intolerant individuals, and the avoidance of all dairy products is no longer recommended  
518 (Szilagyι & Ishayek, 2018).

519

#### 520 **4.2 Diagnosis of lactase-related pathologies**

521 There are numerous tests to diagnose lactose maldigestion or intolerance, though none is  
522 considered a ‘gold standard’ test. The lactose tolerance test involves ingesting a bolus of 25-  
523 50g lactose, measuring blood glucose, and reporting acute GI symptoms (Hovde & Farup,  
524 2009). If the lactose is hydrolysed, serum glucose is expected to rise, whilst no change, and/or  
525 the presence of GI symptoms suggests LI. However, the test has very low sensitivity and  
526 specificity (Misselwitz et al., 2013). The National Institute of Health requires the diagnosis of  
527 LI to involve comparison to an inert placebo (Suchy et al., 2010), which can reduce the impact  
528 of self-reported food intolerances which are often unsubstantiated (Misselwitz et al., 2013).

529

530 The H<sub>2</sub> breath test is widely used to assess lactose maldigestion, as GI symptoms after a lactose  
531 challenge are better correlated with breath H<sub>2</sub> production than with changes in blood glucose  
532 (Hermans, Brummer, Ruijgers, & Stockbrügger, 1997). Expired breath is collected before and  
533 after ingestion of 25 g of lactose, and breath [H<sub>2</sub>] >20 ppm implies that the lactose has not been  
534 completely hydrolysed and has entered the colon where it is fermented by bacteria. The  
535 measurement of CH<sub>4</sub> alongside H<sub>2</sub> has been shown to improve the prognostic capability of the  
536 breath test (Hovde & Farup, 2009). However, false positives can be caused by small intestine  
537 bacterial overgrowth, whilst false negatives may be caused by H<sub>2</sub> or CH<sub>4</sub> non-producing  
538 subjects (between 2-43% of subjects; Gasbarrini et al. (2009)). A process known as ‘full  
539 colonic adaptation’ can also occur, where repeated lactose intake selectively favours flora that  
540 ferment lactose without the production of H<sub>2</sub> or CH<sub>4</sub> (Misselwitz et al., 2013). Inadequate  
541 dietary control in the day(s) preceding the test may artificially inflate pre-test values, due to



542 presence of fermentable substrates such as fibre in the colon. Furthermore, the boundary of  
543 breath [H<sub>2</sub>] of >20 ppm is arbitrary and does not relate to a threshold of lactase expression or  
544 the presence of GI discomfort.

545

546 Genetic testing can also be employed to diagnose of lactase non-persistence. Presence of the  
547 genetic polymorphism 13910C/C indicates lactase non-persistence. This test has a low false  
548 positive rate (<5 %) but generates false negatives if LI is caused by secondary factors. Testing  
549 for this polymorphism is only appropriate in Caucasian subjects (Misselwitz et al., 2013).  
550 Lactase activity in the jejunum can be assessed via biopsy, which is considered a ‘reference  
551 standard’ for the assessment of lactase deficiency. However, this method is extremely invasive  
552 and, lactase expression in enterocytes forms a ‘mosaic’ pattern meaning not all areas of the  
553 brush border express lactase strongly (Maiuri et al., 1991; Misselwitz et al., 2013). Therefore,  
554 biopsies can sample areas without lactase even in lactase persistent individuals, generating a  
555 false positive result.

556

557 It has been noted that it is possible for individuals to ‘adapt’ to lactose ingestion, showing  
558 decreased breath H<sub>2</sub> responses and GI symptoms in response to lactose feeding after chronic  
559 ingestion. Lactase activity does not increase in response to lactose exposure in humans (Gilat,  
560 Russo, Gelman-Malachi, & Aldor, 1972). However, adaptation of colonic bacteria in response  
561 to lactose ingestion is known to occur (for a review, see Szilagyi (2015)). ‘Full colonic  
562 adaptation’ is defined by Misselwitz et al. (2013) as a process in which “repeated intake of  
563 lactose selectively favours the growth of colonic flora that rapidly ferment lactose without  
564 producing hydrogen”. Colonic bacterial adaptation may also occur in such a manner that  
565 hydrogen-producing bacteria remain, but that lactose that reaches the colon is still fermented,  
566 without the presence of symptoms (Di Stefano et al., 2007; Johnson, Semanya, Buchowski,

567 Enwonwu, & Scrimshaw, 1993b). Indeed, only approximately one third of individuals with  
568 positive H<sub>2</sub> breath tests experience GI symptoms (Di Stefano et al., 2007), highlighting the  
569 disconnect between breath H<sub>2</sub> responses and LI symptoms.

570

571 It has been demonstrated that lactose intolerant individuals can adapt to increasing lactose  
572 intake without intolerance symptoms. Johnson, Semanya, Buchowski, Enwonwu, and  
573 Scrimshaw (1993a) provided 22 lactose intolerant maldigesters with progressively increasing  
574 daily doses of lactose. All subjects increased the threshold at which symptoms were induced,  
575 to at least 7 g lactose, and 17 subjects increased to  $\geq 12$  g. Lactose maldigesters regularly  
576 consuming dairy foods may not suffer from LI, except at high doses where the ability of colonic  
577 bacteria (and limited lactase) to metabolise lactose is exceeded, whilst maldigesters with low  
578 lactose intake may suffer intolerance even at very low doses. Most individuals with lactose  
579 maldigestion can tolerate ingestion of ~12-15 g of lactose (~250-300 mL of milk) without  
580 symptoms (Shaukat et al., 2010).

581

## 582 **5. Potential limitations of lactose in sports nutrition**

583 It is not entirely clear whether lactose can be a viable carbohydrate for exercise settings in light  
584 of the issues surrounding LI and maldigestion. A known or diagnosed intolerance generally  
585 precludes consumption of lactose, and although many maldigesters can tolerate small amounts  
586 of lactose without symptoms (Shaukat et al., 2010), these amounts are far below the quantities  
587 required for carbohydrate ingestion before ( $1-4 \text{ g}\cdot\text{kg}^{-1}$ ), during ( $30-90 \text{ g}\cdot\text{h}^{-1}$ ) or after ( $1.2 \text{ g}\cdot\text{kg}^{-1}\cdot\text{h}^{-1}$ )  
588 exercise (Burke et al., 2011). Therefore, lactose should not form the major part of a  
589 carbohydrate ingestion regimen for exercise in individuals with diagnosed LI, except up to  
590 quantities of ~12-15 g, if tolerable. Furthermore, individuals following FODMAP exclusion  
591 diets should also avoid lactose as required.

592

593 Most lactose tolerant individuals can ingest large quantities of lactose without GI symptoms.  
594 During endurance exercise, ingestion of 48 g·h<sup>-1</sup> of lactose did not result in worse mean GI  
595 symptoms than sucrose (Odell et al., 2020). However, one subject experienced mild LI  
596 symptoms in the hours post-exercise after lactose but not sucrose ingestion, despite being  
597 lactose tolerant and regularly ingesting dairy foods. It is not clear what the upper limit for  
598 lactose digestion is, though absorption of glucose and galactose via SGLT1 is limited to ~60  
599 g·h<sup>-1</sup>, therefore lactose ingestion rates during exercise should not exceed this (Jeukendrup,  
600 2010).

601

602 Individuals may have concerns about ingesting large quantities of lactose-containing dairy  
603 foods in the post-exercise period to achieve the required carbohydrate intake. It is unclear  
604 whether the hydrolysis of lactose in a post-exercise setting might limit the delivery of its  
605 constituent monosaccharides. However, investigations involving ingestion of large quantities  
606 of lactose (80-120 g) before and during exercise respectively, have not demonstrated any  
607 effects of maldigestion in the vast majority of lactose-tolerant subjects (Odell et al., 2020;  
608 Stellaard et al., 2000). Recommendations for post-exercise carbohydrate intake are higher than  
609 before or during exercise, but an investigation by Costa et al. (2020) involving participants  
610 ingesting 1.2 g·kg<sup>-1</sup> of carbohydrate from chocolate milk showed that neither GI symptoms nor  
611 breath H<sub>2</sub> were elevated with a high carbohydrate post-exercise dairy meal compared to water  
612 ingestion. Therefore, lactose could be an appreciable contributor to post-exercise carbohydrate  
613 requirements. Dairy foods such as milk or yoghurt may offer an additional benefit over isolated  
614 lactose, as the dairy matrix (as seen in yoghurt) is known to improve GI comfort and deliver  
615 relevant nutrients such as protein and electrolytes (Pelletier et al., 2001; Szilagy & Ishayek,  
616 2018; Thorning et al., 2017). Individuals should determine which carbohydrates and foods, and

617 in what quantities, are appropriate for them to use pre-, during and post-exercise, based on their  
618 personal preference and responses. This is the case for all carbohydrates, including fructose in  
619 light of the presence of fructose malabsorption in some individuals (Putkonen, Yao, & Gibson,  
620 2013).

621  
622 An impediment to the application of lactose in sports nutrition is the unfavourable perception  
623 of dairy and lactose. Dairy products are commonly included in a description of healthy eating  
624 by children and adolescents, but to a lesser extent by adults (Paquette, 2005). The potential of  
625 milk to cause allergic symptoms is considered to be high (*versus* soy milk), and it is thought to  
626 cause serious disease (including heart disease, high cholesterol and obesity) to a greater extent  
627 than soy milk (Bus & Worsley, 2003). Self-diagnosis of LI is also common, with 11-16% of  
628 North Americans self-diagnosing LI, in most cases without clinical diagnosis of intolerance or  
629 maldigestion (Barr, 2013; Nicklas et al., 2011). This leads to inappropriate avoidance and  
630 reduced consumption of dairy foods (Barr, 2013; Keith et al., 2011).

631

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637

638 **Table 2.** Summary of applications and considerations for lactose in sports nutrition

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Potential application	Comments
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Lactose as an energy source for exercise	<ul style="list-style-type: none"> <li>• Ingested lactose can be readily oxidised during exercise, and results in higher fat oxidation and reduced reliance on endogenous carbohydrate oxidation</li> <li>• Lactose can form part of a pre- or during exercise carbohydrate feeding regimen</li> </ul>
Lactose for glycogen restoration	<ul style="list-style-type: none"> <li>• Lactose is the primary source of dietary galactose, which has been shown to increase post-exercise liver glycogen resynthesis</li> <li>• Studies on milk and chocolate milk suggest that lactose can be a substrate for muscle glycogen resynthesis, resulting in similar subsequent exercise performance to carbohydrate beverages</li> </ul>
Lactose as a prebiotic	<ul style="list-style-type: none"> <li>• Lactose can exert a prebiotic effect on the purportedly beneficial bacterial flora <i>Bifidobacteria</i> and <i>Lactobacilli</i></li> <li>• It is not clear whether this may be concomitant with GI symptoms in lactose intolerant individuals</li> </ul>
<b>Considerations</b>	
Lactose intolerance	<ul style="list-style-type: none"> <li>• Lactose intolerant individuals should avoid large quantities of lactose, but 12-15 g is generally well tolerated</li> </ul>
Amount of lactose required	<ul style="list-style-type: none"> <li>• Nutritional recommendations for exercise settings involve large quantities of carbohydrate</li> <li>• Lactose and milk beverages do not trigger GI symptoms in most lactose tolerant individuals during, before or after exercise, even when ingested in large amounts.</li> </ul>

640

641 GI - gastrointestinal

642

## 643 **6. Summary and future directions**

644 Although lactose does not feature in guidelines for carbohydrate intake for sports nutrition, it  
645 is becoming increasingly apparent that the dairy carbohydrate could play a role in the diets of  
646 athletes and recreationally active individuals. Lactose or its free monosaccharide constituents  
647 represents a viable fuel source for endurance exercise and can be incorporated into  
648 carbohydrate feeding regimens for during, and possibly pre- and post-exercise. Lactose may  
649 be a good choice for combined replenishment of muscle and liver glycogen after exercise due  
650 to its galactose component, though this remains to be directly investigated. It is also not clear  
651 whether a combination of glucose, galactose and fructose ingestion (as monosaccharides or

652 through use of lactose and sucrose as found in chocolate milk) might confer the optimal  
653 enhancement of post-exercise liver glycogen resynthesis, potentially explaining reported  
654 performance recovery benefits of chocolate milk. Lactose could also be considered a prebiotic  
655 in some scenarios, and future research should aim to determine whether it can engender similar  
656 benefits to established prebiotics. Although GI symptoms with lactose ingestion may occur, in  
657 most lactose tolerant individuals the carbohydrate can be ingested at rates conducive to  
658 recommendations for sports nutrition, though the ingestion of higher quantities of lactose  
659 should be investigated.

660

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## References

- 676  
677  
678 Abdul-Ghani, M. A., Lyssenko, V., Tuomi, T., Defronzo, R. A., & Groop, L. (2010). The  
679 shape of plasma glucose concentration curve during OGTT predicts future risk of type  
680 2 diabetes. *Diabetes/Metabolism Research and Reviews*, 26(4), 280-286.  
681 doi:10.1002/dmrr.1084
- 682 Acosta, P. B., & Gross, K. C. (1995). Hidden sources of galactose in the environment.  
683 *European Journal of Pediatrics*, 154(7 Suppl 2), 87-92.
- 684 Alcantara, J. M. A., Sanchez-Delgado, G., Martinez-Tellez, B., Labayen, I., & Ruiz, J. R.  
685 (2019). Impact of cow's milk intake on exercise performance and recovery of muscle  
686 function: a systematic review. *Journal of the International Society of Sports Nutrition*,  
687 16(1), 22. doi:10.1186/s12970-019-0288-5
- 688 Amiri, M., Ghiasvand, R., Kaviani, M., Forbes, S. C., & Salehi-Abargouei, A. (2018).  
689 Chocolate milk for recovery from exercise: a systematic review and meta-analysis of  
690 controlled clinical trials. *European Journal of Clinical Nutrition*. doi:10.1038/s41430-  
691 018-0187-x
- 692 Barr, S. I. (2013). Perceived lactose intolerance in adult Canadians: a national survey.  
693 *Applied Physiology, Nutrition and Metabolism*, 38(8), 830-835.
- 694 Bergman, B., & Brooks, G. A. (1999). Respiratory gas-exchange ratios during graded  
695 exercise in fed and fasted trained and untrained men. *Journal of Applied Physiology*,  
696 86(2), 479-487.
- 697 Bergström, J., Hermansen, L., Hultman, E., & Saltin, B. (1967). Diet, Muscle Glycogen and  
698 Physical Performance. *Acta Physiologica Scandinavica*, 71(2), 140-150.
- 699 Bindels, L., Delzenne, N., Cani, P., & Walter, J. (2015). Towards a more comprehensive  
700 concept for prebiotics. *Nature Reviews Gastroenterology and Hepatology*, 12(5), 303-  
701 310.
- 702 Blom, P. C. S., Høstmark, A. T., Vaage, O., Kardel, K. R., & Maehlum, S. (1987). Effect of  
703 different post-exercise sugar diets on the rate of muscle glycogen synthesis. *Medicine  
704 and Science in Sports and Exercise*, 19(5), 491-496.
- 705 Boucher, J., Kleinridders, A., & Kahn, C. R. (2014). Insulin receptor signaling in normal and  
706 insulin-resistant states. *Cold Spring Harbor Perspectives in Biology*, 6(1).  
707 doi:10.1101/cshperspect.a009191
- 708 Brisbois, T., Marsden, S., Anderson, G., & Sievenpiper, J. (2014). Estimated Intakes and  
709 Sources of Total and Added Sugars in the Canadian Diet. *Nutrients*, 6(5), 1899-1912.  
710 doi:10.3390/nu6051899
- 711 Burdon, C. A., Spronk, I., Cheng, H. L., & O'Connor, H. T. (2017). Effect of Glycemic Index  
712 of a Pre-exercise Meal on Endurance Exercise Performance: A Systematic Review  
713 and Meta-analysis. *Sports Medicine*, 47(6), 1087-1101. doi:10.1007/s40279-016-  
714 0632-8
- 715 Burelle, Y., Lamoureux, M. C., Peronnet, F., Massicotte, D., & Lavoie, C. (2006).  
716 Comparison of exogenous glucose, fructose and galactose oxidation during exercise  
717 using 13C-labelling. *British Journal of Nutrition*, 96(1), 56-61. Retrieved from  
718 <https://www.ncbi.nlm.nih.gov/pubmed/16869991>
- 719 Burke, L. M., Hawley, J. A., Wong, S. H., & Jeukendrup, A. E. (2011). Carbohydrates for  
720 training and competition. *Journal of Sports Science*, 29 (Suppl 1), 17-27. Retrieved  
721 from <https://www.ncbi.nlm.nih.gov/pubmed/21660838>
- 722 Bus, A. E. M., & Worsley, A. (2003). Consumers' health perceptions of three types of milk: a  
723 survey in Australia. *Appetite*, 40(2), 93-100. doi:10.1016/s0195-6663(03)00004-7
- 724 Cani, P. D., Lecourt, E., Dewulf, E. M., Sohet, F. M., Pachikian, B. D., Naslain, D., . . .  
725 Delzenne, N. M. (2009). Gut microbiota fermentation of prebiotics increases

726 satietogenic and incretin gut peptide production with consequences for appetite  
727 sensation and glucose response after a meal. *American Journal of Clinical Nutrition*,  
728 90(5), 1236-1243. doi:10.3945/ajcn.2009.28095

729 Carlson, J., Erickson, J. M., Lloyd, B. B., & Slavin, J. L. (2018). Health Effects and Sources  
730 of Prebiotic Dietary Fiber. *Current Developments in Nutrition*, 2(3), nyz005.

731 Costa, R. J. S., Camões-Costa, V., Snipe, R. M. J., Dixon, D., Russo, I., & Huschtscha, Z.  
732 (2020). The Impact of a Dairy Milk Recovery Beverage on Bacterially Stimulated  
733 Neutrophil Function and Gastrointestinal Tolerance in Response to Hypohydration  
734 Inducing Exercise Stress. *International Journal of Sport Nutrition and Exercise  
735 Metabolism*, 30(4), 237–248.

736 Décombaz, J., Jentjens, R., Ith, M., Scheurer, E., Buehler, T., Jeukendrup, A., & Boesch, C.  
737 (2011). Fructose and Galactose Enhance Postexercise Human Liver Glycogen  
738 Synthesis. *Medicine and Science in Sports and Exercise*, 43(10), 1964-1971.  
739 doi:10.1249/MSS.0b013e318218ca5a

740 Di Stefano, M., Miceli, E., Mazzocchi, S., Tana, P., Moroni, F., & Corazza, G. R. (2007).  
741 Visceral hypersensitivity and intolerance symptoms in lactose malabsorption.  
742 *Neurogastroenterology and Motility*, 19(11), 887-895. doi:10.1111/j.1365-  
743 2982.2007.00973.x

744 Dieticians of Canada. (2013). Food Sources of Lactose [Press release]. Retrieved from  
745 <https://www.dietitians.ca/Downloads/Factsheets/Food-Sources-of-Lactose.aspx>

746 Edgar, W. M. (1993). Extrinsic and intrinsic sugars: a review of recent UK recommendations  
747 on diet and caries. *Caries Research*, 27, 64-67.

748 Ercan, N., Nuttall, F., Gannon, M., Redmon, J., & Sheridan, K. (1993). Effects of glucose,  
749 galactose, and lactose ingestion on the plasma glucose and insulin response in persons  
750 with non-insulin-dependent diabetes mellitus. *Metabolism: Clinical and  
751 Experimental*, 42(12), 1560-1567.

752 Ferguson-Stegall, L., McCleave, E., Ding, Z., Doerner, P., Wang, B., Liao, Y., . . . Ivy, J. L.  
753 (2011). Postexercise carbohydrate-protein supplementation improves subsequent  
754 exercise performance and intracellular signaling for protein synthesis. *Journal of  
755 Strength and Conditioning Research*, 25(5), 1210-1224.

756 Fuchs, C. J., Gonzalez, J. T., Beelen, M., Cermak, N. M., Smith, F. E., Thelwall, P. E., . . .  
757 van Loon, L. J. (2016). Sucrose ingestion after exhaustive exercise accelerates liver,  
758 but not muscle glycogen repletion compared with glucose ingestion in trained  
759 athletes. *Journal of Applied Physiology*, 120(11), 1328-1334.

760 Gasbarrini, A., Corazza, G. R., Gasbarrini, G., Montalto, M., Di Stefano, M., Basilisco, G., . .  
761 . st Rome, H. B. T. C. C. W. G. (2009). Methodology and indications of H2-breath  
762 testing in gastrointestinal diseases: the Rome Consensus Conference. *Alimentary  
763 Pharmacology and Therapeutics*, 29 Suppl 1, 1-49. doi:10.1111/j.1365-  
764 2036.2009.03951.x

765 Gilat, T., Russo, S., Gelman-Malachi, E., & Aldor, T. A. M. (1972). Lactase in man: a  
766 nonadaptable enzyme. *Gastroenterology*, 62(6), 1125-1127.

767 Grandjean, A. C. (1997). Diets of Elite Athletes: Has the Discipline of Sports Nutrition Made  
768 an Impact? . *The Journal of Nutrition*, 127(5), 874-877.

769 Haakonssen, E. C., Ross, M. L., Cato, L. E., Nana, A., Knight, E. J., Jenkins, D. G., . . .  
770 Burke, L. M. (2014). Dairy-based preexercise meal does not affect gut comfort or  
771 time-trial performance in female cyclists. *International Journal of Sport Nutrition and  
772 Exercise Metabolism*, 24(5), 553-558. doi:10.1123/ijsnem.2014-0069

773 Hermans, M. M., Brummer, R. J., Ruijgers, A. M., & Stockbrügger, R. W. (1997). The  
774 relationship between lactose tolerance test results and symptoms of lactose  
775 intolerance. *American Journal of Gastroenterology*, 92(6), 981-984.



- 776 Hovde, O., & Farup, P. G. (2009). A comparison of diagnostic tests for lactose  
777 malabsorption--which one is the best? *BMC Gastroenterology*, 9, 82.  
778 doi:10.1186/1471-230X-9-82
- 779 Hughes, C., Davoodi-Semiromi, Y., Colee, J. C., Culpepper, T., Dahl, W. J., Mai, V., . . .  
780 Langkamp-Henken, B. (2011). Galactooligosaccharide supplementation reduces  
781 stress-induced gastrointestinal dysfunction and days of cold or flu: a randomized,  
782 double-blind, controlled trial in healthy university students. *American Journal of*  
783 *Clinical Nutrition*, 93(6), 1305-1311. doi:10.3945/ajcn.111.014126
- 784 Ingram, C. J., Mulcare, C. A., Itan, Y., Thomas, M. G., & Swallow, D. M. (2009). Lactose  
785 digestion and the evolutionary genetics of lactase persistence. *Human Genetics*,  
786 124(6), 579-591. doi:10.1007/s00439-008-0593-6
- 787 Itan, Y., Jones, B. L., Ingram, C. J., Swallow, D. M., & Thomas, M. G. (2010). A worldwide  
788 correlation of lactase persistence phenotype and genotypes. *BioMed Central*  
789 *Evolutionary Biology*, 10, 36. doi:10.1186/1471-2148-10-36
- 790 Ito, M., & Kimura, M. (2009). Influence of Lactose on Faecal Microflora in Lactose  
791 Maldigestors. *Microbial Ecology in Health and Disease*, 6(2), 73-76.  
792 doi:10.3109/08910609309141564
- 793 James, L. J., Stevenson, E. J., Rumbold, P. L. S., & Hulston, C. J. (2019). Cow's milk as a  
794 post-exercise recovery drink: implications for performance and health. *European*  
795 *Journal of Sport Science*, 19(1), 40-48. doi:10.1080/17461391.2018.1534989
- 796 Jellema, P., Schellevis, F. G., Van Der Windt, D. A. W. M., Kneepkens, C. M. F., & Van Der  
797 Horst, H. E. (2010). Lactose malabsorption and intolerance: a systematic review on  
798 the diagnostic value of gastrointestinal symptoms and self-reported milk intolerance.  
799 *QJM*, 103(8), 555-572.
- 800 Jeukendrup, A. E. (2010). Carbohydrate and exercise performance: the role of multiple  
801 transportable carbohydrates. *Current Opinions in Clinical Nutrition and Metabolic*  
802 *Care*, 13(4), 452-457.
- 803 Jeukendrup, A. E. (2011). Nutrition for endurance sports: Marathon, triathlon, and road  
804 cycling. *Journal of Sports Sciences*, 29(sup1), S91-S99.  
805 doi:10.1080/02640414.2011.610348
- 806 Jiang, T., & Savaiano, D. A. (1997a). In Vitro Lactose Fermentation by Human Colonic  
807 Bacteria Is Modified by Lactobacillus acidophilus Supplementation *The Journal of*  
808 *Nutrition*, 127(8), 1489-1495.
- 809 Jiang, T., & Savaiano, D. A. (1997b). Modification of colonic fermentation by bifidobacteria  
810 and pH in vitro. Impact on lactose metabolism, short-chain fatty acid, and lactate  
811 production. *Digestive Diseases and Sciences*, 42(11), 2370-2377.
- 812 Johnson, A., Semanya, J. G., Buchowski, M. S., Enwonwu, C., & Scrimshaw, N. S. (1993a).  
813 Adaptation of lactose maldigesters to continued milk intakes. *American Journal of*  
814 *Clinical Nutrition*, 58, 819-881.
- 815 Johnson, A., Semanya, J. G., Buchowski, M. S., Enwonwu, C., & Scrimshaw, N. S. (1993b).  
816 Correlation of lactose maldigestion, lactose intolerance, and milk intolerance.  
817 *American Journal of Clinical Nutrition*, 57, 399-401.
- 818 Karfonta, K., Lunn, W., Colletto, M., Anderson, J., & Rodriguez, N. (2010). Chocolate Milk  
819 And Glycogen Replenishment After Endurance Exercise In Moderately Trained  
820 Males. *Medicine and Science in Sports and Exercise*, 42(8), 86.
- 821 Keith, J. N., Nicholls, J., Reed, A., Kafer, K., & Miller, G. (2011). The Prevalence of Self-  
822 reported Lactose Intolerance and the Consumption of Dairy Foods Among African  
823 American Adults Are Less Than Expected. *Journal of the National Medical*  
824 *Association*, 103(1), 36-45.

- 825 Kelley, D., Mitrakou, A., Marsh, H., Schwenk, F., Benn, J., Sonnenberg, G., . . . Gench, J.  
826 (1988). Skeletal Muscle Glycolysis, Oxidation, and Storage of an Oral Glucose Load.  
827 *Journal of Clinical Investigation*, 81, 1563-1571.
- 828 Koeslag, J. H., Saunders, P. T., & Terblanche, E. (2003). A reappraisal of the blood glucose  
829 homeostat which comprehensively explains the type 2 diabetes mellitus-syndrome X  
830 complex. *Journal of Physiology*, 549(Pt 2), 333-346.  
831 doi:10.1113/jphysiol.2002.037895
- 832 Larsson, S., Bergkvist, L., & Wolk, A. (2004). Milk and lactose intakes and ovarian cancer  
833 risk in the Swedish Mammography Cohort. *American Journal of Clinical Nutrition*,  
834 80(5), 1353-1357.
- 835 Lee, J. K., Maughan, R. J., Shirreffs, S. M., & Watson, P. (2008). Effects of milk ingestion  
836 on prolonged exercise capacity in young, healthy men. *Nutrition*, 24(4), 340-347.  
837 doi:10.1016/j.nut.2008.01.001
- 838 Leijssen, D. P. C., Saris, W. H. M., Jeukendrup, A. E., & Wagenmakers, A. J. M. (1995).  
839 Oxidation of exogenous [13C] galactose and [13C] glucose during exercise. *Journal*  
840 *of Applied Physiology*, 79(3), 720-725.
- 841 Livesey, G. (2001). Tolerance of low-digestible carbohydrates: a general view. *British*  
842 *Journal of Nutrition*, 85 Suppl 1, S7-16. doi:10.1079/bjn2000257
- 843 Loureiro, L., de Melo Teixeira, R., Pereira, I., Reis, C., & da Costa, T. (2020). Effect of Milk  
844 on Muscle Glycogen Recovery and Exercise Performance. *Strength and Conditioning*  
845 *Journal, Epub ahead of print*. doi:doi: 10.1519/SSC.0000000000000595
- 846 Maiuri, L., Raia, V., Potter, J., Swallow, D., Ho, M. W., Fiocca, R., . . . Auricchio, S. (1991).  
847 Mosaic pattern of lactase expression by villous enterocytes in human adult-type  
848 hypolactasia. *Gastroenterology*, 200(2), 359-369.
- 849 Mata, F., Valenzuela, P. L., Gimenez, J., Tur, C., Ferreria, D., Dominguez, R., . . . Martinez  
850 Sanz, J. M. (2019). Carbohydrate Availability and Physical Performance:  
851 Physiological Overview and Practical Recommendations. *Nutrients*, 11(5).  
852 doi:10.3390/nu11051084
- 853 Mattar, R., de Campos Mazo, D. F., & Carrilho, F. J. (2012). Lactose intolerance: diagnosis,  
854 genetic, and clinical factors. *Clinical and Experimental Gastroenterology*, 5, 113-121.  
855 doi:10.2147/CEG.S32368
- 856 Misselwitz, B., Pohl, D., Fruhauf, H., Fried, M., Vavricka, S. R., & Fox, M. (2013). Lactose  
857 malabsorption and intolerance: pathogenesis, diagnosis and treatment. *United*  
858 *European Gastroenterology Journal*, 1(3), 151-159. doi:10.1177/2050640613484463
- 859 Montalto, M., Gallo, A., Santoro, L., D'Onofrio, F., Curigliano, V., Covino, M., . . .  
860 Gasbarrini, G. (2008). Low-dose lactose in drugs neither increases breath hydrogen  
861 excretion nor causes gastrointestinal symptoms. *Alimentary Pharmacology and*  
862 *Therapeutics*, 28(8), 1003-1012. doi:10.1111/j.1365-2036.2008.03815.x
- 863 Nicklas, T. A., Qu, H., Hughes, S. O., He, M., Wagner, S. E., Foushee, H. R., & Shewchuk,  
864 R. (2011). Self-perceived lactose intolerance results in lower intakes of calcium and  
865 dairy foods and is associated with hypertension and diabetes in adults. *American*  
866 *Journal of Clinical Nutrition*, 94(1), 191-198.
- 867 Nuttall, F., & Gannon, M. (1991). Plasma glucose and insulin response to macronutrients in  
868 nondiabetic and NIDDM subjects. *Diabetes Care*, 14(9), 824-838.
- 869 Odell, O. J., Podlogar, T., & Wallis, G. A. (2020). Comparable Exogenous Carbohydrate  
870 Oxidation from Lactose or Sucrose during Exercise. *Medicine and Science in Sports*  
871 *and Exercise*, 52(12), 2663-2672. doi:10.1249/MSS.0000000000002426
- 872 Papin, J. A., König, M., Bulik, S., & Holzhütter, H.-G. (2012). Quantifying the Contribution  
873 of the Liver to Glucose Homeostasis: A Detailed Kinetic Model of Human Hepatic

874           Glucose Metabolism. *PLoS Computational Biology*, 8(6).  
875           doi:10.1371/journal.pcbi.1002577

876 Paquette, M. (2005). Perceptions of Healthy Eating: State of Knowledge and Research Gaps.  
877           *Canadian Journal of Public Health*, 96(3), 15-19.

878 Parada Venegas, D., De la Fuente, M. K., Landskron, G., Gonzalez, M. J., Quera, R.,  
879           Dijkstra, G., . . . Hermoso, M. A. (2019). Short Chain Fatty Acids (SCFAs)-Mediated  
880           Gut Epithelial and Immune Regulation and Its Relevance for Inflammatory Bowel  
881           Diseases. *Frontiers in Immunology*, 10, 277. doi:10.3389/fimmu.2019.00277

882 Pelletier, X., Laure-Boussuge, S., & Donazzolo, Y. (2001). Hydrogen excretion upon  
883           ingestion of dairy products in lactoseintolerant male subjects: importance of the live  
884           flora. *European Journal of Clinical Nutrition*, 55, 509-512.

885 Pfeiffer, B., Stellingwerff, T., Zaltas, E., Hodgson, A. B., & Jeukendrup, A. E. (2010).  
886           Oxidation of solid versus liquid CHO sources during exercise. *Medicine and Science  
887           in Sports and Exercise*, 42(11), 2030-2037.

888 Pfeiffer, B., Stellingwerff, T., Zaltas, E., & Jeukendrup, A. E. (2010). Carbohydrate oxidation  
889           from a carbohydrate gel compared to a drink during exercise. *Medicine and Science in  
890           Sports and Exercise*, 42(11), 2038–2045.

891 Phillips, S. M., Tang, J. E., & Moore, D. R. (2009). The role of milk- and soy-based protein  
892           in support of muscle protein synthesis and muscle protein accretion in young and  
893           elderly persons. *Journal of the American College of Nutrition*, 28(4), 343-354.  
894           doi:10.1080/07315724.2009.10718096

895 Pimentel, G., Burton, K. J., Rosikiewicz, M., Freiburghaus, C., von Ah, U., Munger, L. H., . .  
896           . Vergeres, G. (2017). Blood lactose after dairy product intake in healthy men. *British  
897           Journal of Nutrition*, 118(12), 1070-1077. doi:10.1017/S0007114517003245

898 Putkonen, L., Yao, C., & Gibson, P. R. (2013). Fructose malabsorption syndrome. *Current  
899           Opinion in Clinical Nutrition and Metabolic Care*, 16(4), 473-477.

900 Rangel, A. H. d. N., Sales, D. C., Urbano, S. A., Galvão Júnior, J. G. B., Andrade Neto, J. C.  
901           d., & Macêdo, C. d. S. (2016). Lactose intolerance and cow's milk protein allergy.  
902           *Food Science and Technology*, 36(2), 179-187. doi:10.1590/1678-457x.0019

903 Rozenberg, S., Body, J. J., Bruyere, O., Bergmann, P., Brandi, M. L., Cooper, C., . . .  
904           Reginster, J. Y. (2016). Effects of Dairy Products Consumption on Health: Benefits  
905           and Beliefs--A Commentary from the Belgian Bone Club and the European Society  
906           for Clinical and Economic Aspects of Osteoporosis, Osteoarthritis and  
907           Musculoskeletal Diseases. *Calcified Tissue International*, 98(1), 1-17.  
908           doi:10.1007/s00223-015-0062-x

909 Scrimshaw, N. S., & Murray, E. B. (1988). The acceptability of milk and milk products in  
910           populations with a high prevalence of lactose intolerance. *American Journal of  
911           Clinical Nutrition*, 48, 1079-1159.

912 Shaukat, A., Levitt, M. D., Taylor, B. C., MacDonald, R., Shamliyan, T. A., Kane, R., &  
913           Wilt, T. J. (2010). Systematic Review: Effective Management Strategies for Lactose  
914           Intolerance. *Annals of Internal Medicine*, 152, 797-803.

915 Slavin, J. (2013). Fiber and prebiotics: mechanisms and health benefits. *Nutrients*, 5(4),  
916           1417-1435. doi:10.3390/nu5041417

917 Stannard, S. R., Hawke, E. J., & Schnell, N. (2009). The effect of galactose supplementation  
918           on endurance cycling performance. *European Journal of Clinical Nutrition*, 63(2),  
919           209-214. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/17928803>

920 Stellaard, F., Koetse, H. A., Elzinga, H., Boverhof, R., Tjoonk, R., Klimp, A., . . . Liesker, J.  
921           (2000). 13C-Carbohydrate breath tests: impact of physical activity on the rate-limiting  
922           step in lactose utilization. *Scandinavian Journal of Gastroenterology*, 35(8), 819-823.

- 923 Suchy, F., Brannon, P., Carpenter, T., Fernandez, J., Gilsanz, V., Gould, J., . . . Wolf, M.  
 924 (2010). National Institutes of Health Consensus Development Conference: lactose  
 925 intolerance and health. *Annals of Internal Medicine*, 152(12), 792-796.
- 926 Sunehag, A., L., & Haymond, M., W. (2002). Splanchnic Galactose Extraction Is Regulated  
 927 by Coingestion of Glucose in Humans. *Metabolism: Clinical and Experimental*,  
 928 51(7), 827-832.
- 929 Szilagyi, A. (2015). Adaptation to Lactose in Lactase Non Persistent People: Effects on  
 930 Intolerance and the Relationship between Dairy Food Consumption and Evaluation of  
 931 Diseases. *Nutrients*, 7(8), 6751-6779. doi:10.3390/nu7085309
- 932 Szilagyi, A., & Ishayek, N. (2018). Lactose Intolerance, Dairy Avoidance, and Treatment  
 933 Options. *Nutrients*, 10(12). doi:10.3390/nu10121994
- 934 Taylor, R., Magnusson, I., Rothman, D., Cline, G., Caumo, A., Cobelli, C., & Shulman, G.  
 935 (1996). Direct Assessment of Liver Glycogen Storage by <sup>13</sup>C Nuclear Magnetic  
 936 Resonance Spectroscopy and Regulation of Glucose Homeostasis after a Mixed Meal  
 937 in Normal Subjects. *Journal of Clinical Investigation*, 97, 126-132.
- 938 Tetra Pak. (2018). *Dairy Processing Handbook Chapter 15 - Whey Processing*. Retrieved  
 939 from <http://dairyprocessinghandbook.com/chapter/whey-processing>
- 940 Thorens, B. (2014). GLUT2, glucose sensing and glucose homeostasis. *Diabetologia*, 58(2),  
 941 221-232. doi:10.1007/s00125-014-3451-1
- 942 Thorning, T. K., Bertram, H. C., Bonjour, J. P., de Groot, L., Dupont, D., Feeney, E., . . .  
 943 Givens, I. (2017). Whole dairy matrix or single nutrients in assessment of health  
 944 effects: current evidence and knowledge gaps. *American Journal of Clinical*  
 945 *Nutrition*, 105(5), 1033-1045. doi:10.3945/ajcn.116.151548
- 946 Trommelen, J., Beelen, M., Pinckaers, P. J., Senden, J., Cermak, N. M., & van Loon, L. J.  
 947 (2016). Fructose Coingestion Does Not Accelerate Postexercise Muscle Glycogen  
 948 Repletion. *Medicine and Science in Sports and Exercise*, 48(5), 907.
- 949 Tsukahara, T., Azuma, Y., & Ushida, K. (2009). The Effect of a Mixture of Live Lactic Acid  
 950 Bacteria on Intestinal Gas Production in Pigs. *Microbial Ecology in Health and*  
 951 *Disease*, 13(2), 105-110. doi:10.1080/08910600119621
- 952 Turk, E., Martingn, M. G., & Wright, E. M. (1994). Structure of the Human Na<sup>+</sup>/Glucose  
 953 Cotransporter Gene SGLT1. *The Journal of Biological Chemistry*, 269(21), 15204-  
 954 15209.
- 955 U.S. Dairy Export Council. (2015). *U.S. Whey and Milk Permeate*. Retrieved from Virginia,  
 956 US:
- 957 U.S. Department of Health and Human Services and U.S. Department of Agriculture. (2015).  
 958 2015 – 2020 Dietary Guidelines for Americans. 8th. Retrieved from [health.gov/our-](http://health.gov/our-work/food-and-nutrition/2015-2020-dietary-guidelines/)  
 959 [work/food-and-nutrition/2015-2020-dietary-guidelines/](http://health.gov/our-work/food-and-nutrition/2015-2020-dietary-guidelines/).
- 960 van Loon, L., Greenhaff, P., Constantin-Teodosiu, D., Saris, W., & Wagenmakers, A. (2001).  
 961 The effects of increasing exercise intensity on muscle fuel utilisation in humans.  
 962 *Journal of Physiology*, 536(1), 295-304.
- 963 Wallis, G. A., Hulston, C. J., Mann, C. H., Roper, H. T., Tipton, K. D., & Jeukendrup, A. E.  
 964 (2008). Postexercise muscle glycogen synthesis with combined glucose and fructose  
 965 ingestion. *Medicine and Science in Sports and Exercise*, 40(10), 1789-1794.
- 966 Wang, S. S., Lay, S., Yu, H. N., & Shen, S. R. (2016). Dietary Guidelines for Chinese  
 967 Residents (2016): comments and comparisons. *Journal of Zhejiang University*  
 968 *Science B*, 17(9), 649-656. doi:10.1631/jzus.B1600341
- 969 Watson, P., Love, T., Maughan, R., & Shirreffs, S. (2008). A comparison of the effects of  
 970 milk and a carbohydrate-electrolyte drink on the restoration of fluid balance and  
 971 exercise capacity in a hot, humid environment. . *European Journal of Applied*  
 972 *Physiology*, 104(4), 633-642.

973 Widrick, J. J., Costill, D. L., Fink, W. J., Hickey, M. S., McConell, G. K., & Tanaka, J.  
974 (1993). Carbohydrate feedings and exercise performance: effect of initial muscle  
975 glycogen concentration. *Journal of Applied Physiology*, 74(6), 2998-3005.

976 Williams, C. A., Phillips, T., & Macdonald, I. A. (1983). The influence of glucose on serum  
977 galactose levels in man. *Metabolism: Clinical and Experimental*, 32(3), 250-256.

978 Wolever, T., & Miller, J. (1995). Sugars and blood glucose control. *American Journal of*  
979 *Clinical Nutrition*, 62, 212-227.

980 Yazawa, K., & Tamura, Z. (1982). Search for Sugar Sources for Selective Increase of  
981 Bifidobacteria. *Bifidobacteria Microflora*, 1(1), 39-44.

982

983

984

985

986

987