Review of the Effects of Glycerol-Containing Hyperhydration Solutions on Gastric Emptying and Intestinal Absorption in Humans and in Rats

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Glycerol-induced hyperhydration (GIH) has been shown to improve fluid retention and endurance performance compared with water-induced hyperhydration. The goal of this article is to report on what is known and unknown about how glycerol-containing hyperhydration solutions (GCHSs) are processed at the stomach and intestine level, propose strategies to improve the efficacy of GIH, and provide research questions for future studies. Through statistical analyses, it is demonstrated that the effectiveness of GCHSs in increasing fluid retention is maximized when fluid ingestion is in the upper range of what is normally administered by studies (~26 ml/kg body weight) and the duration of the protocol is no longer than the time it takes for the glycerol-fluid load to be totally or nearly completely integrated inside the body. The rate of gastric emptying and intestinal absorption of GCHSs is unknown. However, based on an analysis of indirect evidence obtained from human studies, it is proposed that most glycerol (~80 g) and fluid (~1,700 ml) ingested during a typical GIH protocol can be integrated inside the body within 60–90 min. Whether the stress associated with competition could alter these figures is unknown. Research in rats indicates that combining glycerol with glucose at a 3:1 ratio accelerates intestinal absorption of both glycerol and water, thereby potentially improving the efficacy of GIH. Human studies must be conducted to determine how GCHSs are processed by the gastrointestinal system and whether adding glucose to GCHSs could improve the technique’s efficacy.

Keywords: overhydration, ergogenic aid, gastrointestinal function, stomach, intestine

Although change in body weight (BW) is not always a reliable measure of changes in hydration status (Maughan, Shirreffs, & Leiper, 2007), it is suggested that an exercise-induced loss in BW of > 2% can impair endurance performance (American College of Sports Medicine et al., 2007; Cheuvront, Carter, & Sawka, 2007).
Evidence indicates that endurance athletes lose more than 2% BW during prolonged exercise (Noakes, 1993). The use of preexercise hyperhydration, by delaying or preventing the 2% BW loss threshold, could therefore be advantageous in situations in which it is anticipated that a BW loss >2% could occur if the exercise is started in a euhydrated state only.

In a recent meta-analysis (Goulet, Aubertin-Leheudre, Plante, & Dionne, 2007), glycerol-induced hyperhydration (GIH) was demonstrated to significantly enhance fluid retention and power output during prolonged exercise by 50% and 2.6%, respectively, compared with water-induced hyperhydration. Moreover, it has recently been shown that, compared with preexercise euhydration, GIH improved peak power output by 5% during an incremental test to exhaustion after 2 hr of cycling during which fluid consumption replaced only 33% of sweat losses (Goulet, Rousseau, Lamboley, Plante, & Dionne, 2008).

Some individual studies showed no effect of GIH on endurance performance compared with water-induced hyperhydration (Goulet, Robergs, Labrecque, Royer, & Dionne, 2006; Marino, Kay, & Cannon, 2003; Nishijima et al., 2007; Wingo et al., 2004). Moreover, the effects of GIH on cardiovascular and thermoregulatory functions during prolonged exercise are equivocal (Nelson & Robergs, 2007). The discrepancy between findings could result from the different GIH and exercise protocols used by studies.

Isotonic sodium-containing hyperhydration solutions have been shown to improve endurance performance compared with hypotonic solutions (Coles & Luetkemeier, 2005; Sims, Rehrer, Bell, & Cotter, 2007). However, the use of GIH may prove more advantageous than sodium-induced hyperhydration, because it has been shown to improve fluid retention significantly more (Griffin et al., 1999).

Three review articles found in the literature provide important insights into the physiology of GIH (Latzka & Sawka, 2000; Nelson & Robergs, 2007; Robergs & Griffin, 1998). Taken together, those articles cover most of the aspects of GIH, including a review of the biochemistry and pharmacokinetics of glycerol and how GIH affects thermoregulatory and cardiovascular functions, as well as fluid retention and plasma volume regulation, before, during, and after exercise. However, no review has been done reporting on how glycerol-containing hyperhydration solutions (GCHSs) are processed at the stomach and intestine level. This is an issue that merits particular attention, because the stomach and gut represent the two barriers that need to be crossed before any of the water and glycerol ingested during GIH can be integrated into the body fluid pools to produce their respective physiological effects. Therefore, understanding how GCHSs interact with the stomach and intestine should help optimize the use and, by extension, the efficacy of GIH. The goal of this article is to report on what is known and unknown about how the glycerol and water ingested during GIH are handled by the gastrointestinal system, propose strategies and hypotheses with the hope of improving the efficacy of this hydration strategy, and provide research questions for future studies.

The rates of gastric emptying and intestinal absorption of the glycerol and water ingested during GIH will be discussed. It is important to be aware of those rates, because, as will be demonstrated, the efficacy of GCHSs in increasing fluid retention is maximized when fluid ingestion is in the upper range of what is nor-
mally administered in studies and the duration of the protocol is no longer than the
time it takes for the fluid-glycerol load to be totally or nearly completely inte-
grated inside the body. Because of the lack of human studies, an attempt will be
made to predict those rates based on an analysis of indirect data obtained from
relevant human studies.

Research in rats that looked at the gastric absorption of glycerol, the intestinal
absorption rate of glycerol, and the intestinal absorption rate of the water ingested
with glycerol will be reviewed. Furthermore, the mechanisms involved in the
absorption of glycerol at the brush border of the intestine and how these facilitate
water absorption will also be discussed.

**Glycerol and Glycerol-Induced Hyperhydration**

**What Is Glycerol?**

Glycerol is also known as *glycerin*, *trihydroxypropane*, and *1,2,3-propanetriol*. It
is a naturally occurring 3-carbon alcohol metabolite (C₃H₈O₃) that constitutes the
structural core of the triglyceride molecules in humans (Frank, Nahata, & Hilty,
1981). Glycerol is an extremely sweet-tasting substance, which is colorless, odor-
less, and viscous. It is a potent osmotic agent, which has a molecular weight of
92.10 and a specific gravity of 1.26. Glycerol is usually available in pharmacies,
and its use is considered safe (Robergs & Griffin, 1998).

**Glycerol-Induced Hyperhydration**

**Volume of Fluid.** Studies have administered 20–29 ml of fluid/kg BW (1,400–
2,200 ml), with an average corresponding to 24 ml/kg BW, or 1,700 ml (Anders-
on, Cotter, Garnham, Casley, & Febbraio, 2001; Coutts, Reaburn, Mummery, &
Holmes, 2002; Freund et al., 1995; Goulet et al., 2006, 2007, 2008; Griffin et al.,
1999; Hitchins et al., 1999; Latzka et al., 1997, 1998; Lyons, Riedesel, Meuli, &
Chick, 1990; Magal et al., 2003; Marino et al., 2003; Montner et al., 1996, 1999;
Nishijima et al., 2007; O’Brien, Freund, Young, & Sawka, 2005; Riedesel, Allen,
Peake, & Al-Qattan, 1987; Wingo et al., 2004). As shown in Figure 1, there is a
significant positive correlation between the quantity of fluid administered and the
ability of GCHSs to increase fluid retention. Hence, the efficacy of GIH is maxi-
mized when the volume of fluid administered is in the upper range of the quanti-
ties normally provided. Goulet et al. (2007) indicated that a fluid dose of 26 ml/kg
BW should maximize fluid retention.

**Quantity of Glycerol.** Studies have administered 0.9–1.5 g glycerol/kg BW (69–
110 g), with an average corresponding to 1.1 g/kg BW, or 80 g (Anderson et al.,
2001; Coutts et al., 2002; Freund et al., 1995; Goulet et al., 2006, 2007, 2008;
Griffin et al., 1999; Hitchins et al., 1999; Latzka et al., 1997, 1998; Lyons et al.,
1990; Magal et al., 2003; Marino et al., 2003; Montner et al., 1996, 1999;
Nishijima et al., 2007; O’Brien et al., 2005; Riedesel et al., 1987; Wingo et al.,
2004). There is no significant relationship between the amount of glycerol
administered and the efficacy of GCHSs in increasing fluid retention. This might
be because of the lack of variation in the amount of glycerol administered by
studies. Nevertheless, Goulet et al. (2007) indicated that the ideal quantity of glycerol ingested to maximize fluid retention is 1–1.2 g/kg BW.

Some studies administered the glycerol as a bolus at the start of the ingestion protocol (Freund et al., 1995; Latzka et al., 1997, 1998; Lyons et al., 1990; Magal et al., 2003; Montner et al., 1996, 1999; Nishijima et al., 2007; O’Brien et al., 2005; Riedesel et al., 1987), whereas others mixed it with the total fluid load to be ingested throughout the protocol (Anderson et al., 2001; Coutts et al., 2002; Goulet et al., 2006, 2008; Griffin et al., 1999; Hitchins et al., 1999; Marino et al., 2003; Nishijima et al., 2007; Wingo et al., 2004). A statistical analysis (independent t test) reveals that the fluid retention provided by both types of protocols does not differ significantly (Figure 2). Because of glycerol’s sweetness, it must be noted that ingesting it as a bolus may produce side effects such as nausea and vomiting (Latzka et al., 1997).

**Length of Protocol.** Studies that looked at the effect of GIH before exercise used protocols ranging in length from 60 to 180 min (Anderson et al., 2001; Coutts et al., 2002; Goulet et al., 2006, 2008; Hitchins et al., 1999; Latzka et al., 1997, 1998; Lyons et al., 1990; Magal et al., 2003; Marino et al., 2003; Montner et al., 1996, 1999; Nishijima et al., 2007; Wingo et al., 2004). Four studies with no exercise period examined the effect of GIH on total body water during time periods ranging from 210 to 300 min (Freund et al., 1995; Griffin et al., 1999; O’Brien et al., 2005; Riedesel et al., 1987). The average length of a typical GIH protocol is 136 min (Goulet et al., 2007). There is a trend toward an inverse relationship between the effectiveness of GCHSs in increasing fluid retention and protocol length ($r = -.38, p = .09, n = 22$ studies).
Elapsed Time Between End of GCHS Ingestion and Start of Exercise. The interval of time between the end of ingestion of GCHSs and the start of exercise may significantly affect GCHSs’ ability to increase fluid retention. It varies a lot between studies, with values ranging from 0 to 120 min. As shown in Figure 3, there is a significant negative relationship between the fluid retention provided by GCHSs and the elapsed time between the end of ingestion of the fluid-glycerol load and the start of exercise. This suggests that the effectiveness of a GIH protocol in increasing fluid retention should be maximized when its duration equals the minimum time it takes for the fluid-glycerol load to be totally or nearly completely integrated inside the body. In fact, if the ingestion period and/or waiting time after ingestion are too long, a substantial part of the circulating fluid-glycerol load will be excreted through the kidneys before exercise. This will reduce fluid retention and diminish the physiological effect of the strategy during exercise. However, based on results shown in Figure 3, one should note that for any elapsed time between the end of ingestion of the fluid-glycerol load and the start of exercise there could be some variations in fluid retention between individuals.

Predictors of GCHSs’ Ability to Increase Fluid Retention. To determine the best predictors of the efficacy of GCHSs in increasing fluid retention, five key variables (glycerol and fluid dose, length of protocol, whether or not glycerol was administered as a bolus, and the time span between the end of ingestion and start of exercise) were entered in a stepwise regression analysis. Only the time span between the end of ingestion and start of exercise was retained in the model, explaining 25% of the variation in fluid-retention results. Using a hierarchical regression analysis with all five variables entered separately, the quantity of fluid administered along with the time span between the end of ingestion and start of exercise.
exercise explained 50% of the variation in fluid-retention results, with the other variables not contributing in significantly improving the model. Taken together, the previous findings emphasize the importance of trying to define as precisely as possible the rate of gastric emptying and intestinal absorption of the glycerol and water ingested during GIH to better define the optimal length of a GIH protocol.

**Handling of Glycerol by the Stomach and Intestine**

**Gastric-Emptying and Intestinal-Absorption Rates of Glycerol in Humans**

No study has yet evaluated the rates of gastric emptying and intestinal absorption of glycerol in humans and whether they are dose dependent. Therefore, research is needed on these topics. Despite the lack of human studies, indirect evidence indicates that glycerol is rapidly emptied from the stomach, absorbed by the intestine, integrated into the body, and distributed among the body fluid pools. For example, Freund et al. (1995) administered a single bolus of 0.9 g glycerol/kg BW followed by the ingestion of 22 ml of fluid/kg BW within a 30-min period and then observed the changes in plasma glycerol concentration over the next 150 min. A peak plasma glycerol concentration of 1,250 mg/L was reached 60 min after glycerol ingestion, followed by a steady and linear decrease over time because of glycerol turnover and urinary excretion. Using an identical GIH protocol, O’Brien et al. (2005) obtained a peak plasma glycerol value comparable to

![Graph](image_url)

**Figure 3** — Correlation between the elapsed time between end of ingestion of the fluid-glycerol load and the onset of exercise and the relative retention of fluid provided by the glycerol-induced hyperhydration. $N = 17$ studies.
that observed by Freund et al. 90 min after glycerol ingestion. Hitchins et al. (1999) had participants ingest 1 g glycerol/kg BW with 22 ml of fluid/kg BW within a 30-min period and observed a peak plasma glycerol concentration of 920 mg/L 60 min after the end of ingestion. Because glycerol elimination through urine and metabolism is a relatively slow process—for example, 3 hr after ingestion Freund et al. estimated that 62% of the ingested glycerol was still inside the body—the cited results taken together indicate that the bulk of the ingested glycerol can be integrated inside the body 60–90 min after ingestion. This assumption is reasonable, because if significant quantities of glycerol would still have been absorbed by the intestine after 60–90 min, plasma glycerol values should have remained at a peak much longer or even continued to increase over time, which was not the case.

Gastric Emptying and Intestinal Absorption of Glycerol in Rats

Gastric-Absorption and -Emptying Rates. I am aware of no study that determined in rats the rate at which glycerol is emptied from the stomach. However, it has been demonstrated that small quantities of glycerol can be absorbed by the rats’ stomach wall (Embree, Harris, & Herting, 1956). Those authors argued that the likely mechanism was passive diffusion, although this hypothesis was not directly tested. Whether glycerol can be absorbed (and at what rate) from the human stomach is not known and remains to be studied. However, because the time glycerol spends in the stomach during GIH is likely short (based on the observations reported herein), very little glycerol is likely to be absorbed.

Rate of Intestinal Absorption. Yuasa et al. (2003) observed that the rate of glycerol absorption in the rat’s small intestine is rapid. In fact, when they introduced into 5-cm closed loops of rat small intestine in situ 0.5 ml of glycerol solutions concentrated at 0.002, 1.0, and 40.0 mM, the fraction of the doses absorbed after 30 min reached 92%, 90%, and 73%, respectively.

Allen, Wingertzahn, Teichberg, and Wapnir (1999) perfused (10–12 ml/hr) 20- to 30-cm long segments of rat jejunum with a low-osmolality (228 mOsm/kg H₂O) solution containing 2.6 g NaCl/L and 7 g/L glycerol over a 3-hr period and observed a rate of glycerol absorption of 104 nmol · min⁻¹ · cm⁻¹. They also showed that the presence of glucose improves the rate of intestinal glycerol absorption. In fact, when they perfused a low-osmolality (225 mOsm/kg H₂O) solution containing 2.6 g NaCl/L, 6 g of glycerol/L, and 1.8 g of glucose/L, the rate of glycerol absorption increased threefold. Other glycerol:glucose ratios were tested, but none produced absorption rates as high as the one just reported. Thus, in rats a ratio of 3 g glycerol for 1 g glucose maximizes the entry of glycerol inside the body.

This finding could have important practical implications for the formulation of GCHSs. In fact, it suggests that adding some glucose to a fluid-glycerol load might speed up glycerol absorption into the body and, in turn, lead to a more rapid creation of the optimal osmotic gradient needed at the kidney level to maximize fluid retention (Nelson & Robergs, 2007). Human studies definitely must be conducted on this topic.
Kato, Hayashi, Inoue, and Yuasa (2004) showed that some glycerol can be absorbed by the colon, but at a rate that is about 10 times lower than that of the small intestine. As a result of the significant absorption of glycerol taking place in the small intestine and colon, all orally ingested glycerol is made available to the body (Sommer, Nau, Wieland, & Prange, 1993). The fact that studies on GIH did not report diarrhea supports this idea.

**Mechanisms of Glycerol Absorption.** Because glycerol is a small hydrophilic solute (Kato et al., 2004), it was assumed until recently that its absorption by the small intestine was occurring strictly by passive diffusion via the paracellular route (Yuasa et al., 2003). However, using the in vitro everted-sac method involving the rat small intestine, Kato et al. showed that the transport of glycerol through the epithelial cells is saturable and primarily mediated by sodium-dependent and secondary active carriers and, to a lesser extent, by passive diffusion. In the colon, glycerol uptake is also saturable and also likely governed by a sodium-dependent carrier-mediated transport system (Kato et al.). It is known that a group of aquaglyceroporins found in rat and human intestine acts as channels for small neutral solutes such as urea and glycerol (Kato et al.). However, it was argued by Kato et al. that it is unlikely these aquaglyceroporins are involved in the carrier-mediated transport of glycerol, because permeation through channels is expected to be a linear process (unsaturable), therefore kinetically different from the saturable transport found for glycerol.

Glucose is actively transported along with sodium from the lumen of the small intestine into the cytoplasm of the enterocytes (Leiper, 1998). The reason for glucose’s capacity to increase glycerol transport across the mucosa of the small intestine is likely that its presence allows for the recruitment of more sodium-dependent active carriers that are also needed for the absorption of glycerol (Allen et al., 1999). Another mechanism may explain the facilitating effect of glucose on glycerol absorption. Paracellular transport is coupled with sodium-dependent transcellular transport because the latter mechanism provides the osmotic force for solvent drag between the enterocytes and increases the permeability of absorptive cells (Leiper; Schedl, Maughan, & Gisolfi, 1994). Hence, the presence of glucose may enhance glycerol absorption through increased paracellular transport of glycerol.

**Processing of the Water Ingested Along With Glycerol by the Stomach and Intestine**

**Gastric-Emptying and Intestinal-Absorption Rates of the Fluid Ingested During GIH in Humans**

No study has yet determined in humans the rate at which the water ingested with glycerol is emptied from the stomach and absorbed by the gut. It will thus be important to shed some light on these issues in future studies. There has also been no study conducted examining the rate at which the fluid ingested with glycerol is emptied from the stomach of rats or any other animals.
Studies that evaluated the effect of carbohydrate-electrolyte solutions on the rate of gastric emptying and intestinal absorption in humans could provide some insights into the speed at which GIH could be integrated inside the body. The first barrier to the availability of ingested fluids is the rate of gastric emptying, which is a reflection of the rate at which fluid is delivered to the absorptive surface of the small intestine (Leiper, 1998). The primary determinants of the rate of gastric emptying are gastric volume and the energy density of the ingested fluid (Noakes, Rehrer, & Maughan, 1991).

Typical GCHSs composed of 80 g of glycerol and 1,700 ml of fluid have an energy density of 5%. According to Noakes et al. (1991), who estimated the effect of drinking pattern (volume) and energy density (via carbohydrate) on gastric-emptying rate, 60 min should be long enough for complete or nearly complete emptying of the water contained in a typical glycerol solution if an initial intake of 600 ml is followed by ingestion of 300 ml every 10 min for 40 min. Although the ingestion of such a fluid load in this short amount of time may seem physiologically unreasonable, it must be noted that Coutts et al. (2002) and Lyons et al. (1990) administered fluid-glycerol loads of 2 L within 60 min and observed no side effect other than minor gastrointestinal bloating that disappeared shortly after the end of ingestion. Hitchins et al. (1999) and Anderson et al. (2001) administered a fluid-glycerol load of ~1.5 L in 30 min and 15 min, respectively, and reported no symptoms of discomfort or gastrointestinal distress. On the other hand, up to, but not more than, 90 min should be required to empty such a fluid-glycerol load if a slightly more “manageable and conservative” drinking pattern would be employed (initial bolus of 600 ml, followed by 400 ml every 20 min for 60 min). Such a GIH protocol has been shown to be very well tolerated by athletes and to produce no side effects (Goulet et al., 2008). The absence of side effects in these protocols suggests that GIH likely produces very high rates of gastric emptying. The decision to use a 60- or 90-min protocol depends on individual tolerance of high gastric volume.

It is not easy from human studies to provide an approximation of the rate of intestinal absorption of typical GCHSs. The principal determinant of the rate of intestinal water absorption is the osmolality of the ingested solution (Leiper, 1998). Typical GCHSs have an osmolality of ~500 mOsm/kg H₂O. Using the triple-lumen tube technique with humans, Duchman et al. (1997) perfused a 40-cm duodenojejunum test segment with a glucose-electrolyte solution (energy density of 6% and osmolality of 400 mOsm/kg H₂O, both factors comparable to typical GCHSs) at rates equivalent to the purported rates of gastric emptying of a typical fluid-glycerol load when ingested in the ways reported previously—18 or 28 ml/min—and obtained on average a rate of fluid absorption of 13 ml · cm⁻¹ · hr⁻¹. Shi et al. (1994) perfused the duodenojejunum (40-cm test segment) of 6 male volunteers with a 6%, 400-mOsm/kg H₂O glucose/fructose electrolyte solution at a rate of 15 ml/min and observed a rate of fluid absorption of 16 ml · cm⁻¹ · hr⁻¹. Taken together, these results indicate a possible fluid-absorption rate of GCHSs of ~600 ml/hr, or 900 ml/90 min, over the distal duodenum and proximal jejunum. The duodenojejunum’s length is ~275 cm, so obviously fluid absorption will occur throughout this segment but at a rate that will be reduced compared with its proximal section because of the reduction in fluid flow rate and total
solute absorption (Lambert, Chang, Xia, Summers, & Gisolfi, 1997). After the first 75 cm of the duodenojejunum, fluid-absorption rates of a carbohydrate-electrolyte solution and a water solution have been shown to be similar because at this stage both solutions have reached isotonicity and, therefore, the osmotic gradient for fluid absorption is comparable between solutions (Lambert et al.). Santangelo and Krejs (1985) perfused human stomachs with water (22 ml/min) and examined water absorption at 140 cm into a jejunum test segment. Their results indicated a rate of fluid absorption on the order of 2 ml · cm⁻¹ · hr⁻¹. Soergel, Whalen, and Harris (1968) perfused human ileums (30-cm test segment) with an isotonic solution at a rate of 9–9.5 ml/min and observed a rate of fluid absorption of 1 ml · cm⁻¹ · hr⁻¹. Given the length of the duodenojejunum and ileum (300 cm), and if it is assumed that fluid absorption from the first 50 cm of the duodenojejunum is 600 ml/hr, for the remainder of the duodenojejunum is 500 ml/hr, and for the ileum is 300 ml/hr, then the maximal absorptive capacity of the small intestine for GCHSs could be ~1,400 ml/hr.

The change in plasma volume reflects the net effect of fluid absorption (Shi et al., 1994). Gisolfi, Summers, Lambert, and Xia (1998) showed that a hypertonic carbohydrate-electrolyte solution is absorbed as rapidly as water in the distal duodenum and proximal jejunum and that both solutions have a similar effect on plasma volume regulation during exercise. Over a 3-hr period, Freund et al. (1995) demonstrated that GCHSs do not alter plasma volume compared with water, indirectly suggesting that they are absorbed as fast as water or a carbohydrate-electrolyte solution.

Although the composition of GCHSs differs from that of a carbohydrate-electrolyte solution, based on the results reported here, it is reasonable to suggest that the water found in standard GCHSs is likely to be totally or nearly totally integrated inside the body within a period of 60 min if an aggressive drinking pattern is used and within 90 min when a more “relaxed” drinking pattern is used. This assumption obviously needs to be tested in futures studies.

It is important to note that the effect of GIH on endurance performance has been tested only under controlled laboratory conditions and not in real-world stressful situations such as before key competitions. This is an important factor that ultimately prevents us from knowing or advocating an optimal timing of ingestion before competition. For example, nervousness before competition might alter gastrointestinal kinetics. On the other hand, nobody has demonstrated that fluid retention and the ergogenic benefit of GCHSs could not be maximized for some competitions (e.g., cycle road race or ultradistance triathlon) when drinking is accelerated or completed at exercise onset. This would allow fluid-conservation responses to be initiated before the volume load takes full effect and thus might attenuate diuresis. Research is needed to delineate the effect of stress on gastrointestinal symptoms during GIH.

**Intestinal Absorption of Water Ingested With Glycerol in Rats**

**Rate of Absorption.** Previously, using procedures that have been reported here, Allen et al. (1999) determined the rate of intestinal absorption of water ingested with glycerol in rat jejunum. In this segment of the intestine, they observed a mean rate of water absorption of 0.96 μl · min⁻¹ · cm⁻¹ when a low-osmolality
(228 mOsm/kg H$_2$O) solution composed of 2.6 g NaCl/L and 7 g/L glycerol was perfused over a 3-hr period. On the other hand, the perfusion of a glucose solution (2.6 g NaCl/L with 13.5 g glucose/L, 243 mOsm/kg H$_2$O) allowed a rate of water absorption of 1.6 µl · min$^{-1}$ · cm$^{-1}$. However, when solutions (all containing 2.6 g NaCl/L) with the same osmolar load but composed of glycerol and glucose at a ratio of 25:50, 37.5:37.5, 50:25, and 65:10 (mmol/L) were perfused, the rate of water absorption increased to 1.9, 2.1, 2.8, and 2.6 µl · min$^{-1}$ · cm$^{-1}$, respectively. These latter rates of intestinal absorption are interesting in that they were significantly greater than those allowed by the perfusion of either the glycerol or glucose solution alone. Wapnir, Sia, and Fisher (1996) arrived at similar conclusions and showed that the presence of glycerol in a rehydration formula comprising carbohydrate, sodium citrate, and potassium enhances the rate of water absorption into the body compared with a rehydration formula devoid of glycerol.

Taken together, the foregoing results suggest that adding glucose to GCHSs could increase the rate of water absorption and, thus, potentially accelerate the hyperhydration process. On the other hand, adding glucose to GCHSs will further increase their energy density, which may slow gastric emptying and nullify the facilitating effect of glucose on fluid absorption. Studies must be conducted to determine how the combination of glucose and glycerol influences fluid absorption in humans.

**Mechanism of Glycerol-Induced Water Absorption.** Water movement into the absorptive cells is a passive process. The transport of solutes moves water into the enterocytes down the osmotic gradient produced by solute movements. As reported here, the absorption of glycerol is mediated via sodium-dependent and secondary active transport and, to a lesser extent, by passive diffusion. As glycerol is transported along with sodium, the excess water ingested during hyperhydration will follow those two solutes as the transport process creates a favorable osmotic gradient. Obviously, the fact that glycerol is a small molecule with hydrophilic property offers a clear advantage for water transport across the mucosal brush borders (Wapnir et al., 1996).

The combination of glucose and glycerol has been shown to improve fluid absorption in rats. The concomitant ingestion of glucose and glycerol likely potentiates water absorption by activating more and diverse active sodium-nutrient cotransporters, as well as allowing a greater efflux of water between enterocytes.

**Conclusions**

There are currently no studies that have been conducted that determined in humans the rate of gastric emptying and intestinal absorption of GCHSs. It is important to pursue studies on these issues in the future. It was demonstrated in this review that the best predictor of the efficacy of GCHSs in increasing fluid retention was the elapsed time between the end of ingestion of a GCHS and the onset of exercise: The shorter it is, the higher will be the retention of fluid. This suggests that the efficacy of GIH is maximized when the duration of the protocol is no longer than the time it takes for the fluid-glycerol load to be totally or nearly completely integrated inside the body. The amount of fluid ingested during GIH also has an important impact on the efficacy of GIH: The higher the amount of fluid ingested,
the higher the retention of fluid. However, this factor has less impact on the efficacy of GIH than the elapsed time between the end of ingestion of the fluid-glycerol load and the onset of exercise, at least with the fluid doses that have been administered in studies. These findings combined together emphasize the importance of being aware of the rate of gastric emptying and intestinal absorption of GCHSs. Based on indirect evidence provided by human studies, it is proposed that a GIH protocol lasting 60–90 min should be sufficient for the integration of all or nearly all water and glycerol inside the body. Whether this assumption makes sense needs to be tested in future studies. Research in rats suggests that combining glycerol with glucose at a 3:1 ratio can accelerate the rate of glycerol and fluid absorption in the intestine, thereby potentially speeding up the hydration process and further improving the efficacy of GIH. Whether similar results could be obtained in humans must be tested. The stress associated with competition may slow gastrointestinal function. Whether this could prolong the time required for the fluid-glycerol load to be integrated inside the body requires further investigation.

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References


