More Questions than Answers
The Truth Behind Nutrition Science
Reflex Inhibition of Electrically Induced Muscle Cramps in Hypohydrated Humans

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ABSTRACT

MILLER, K. C., G. W. MACK, K. L. KNIGHT, J. T. HOPKINS, D. O. DRAPER, P. J. FIELDS, and I. HUNTER. Reflex Inhibition of Electrically Induced Muscle Cramps in Hypohydrated Humans. Med. Sci. Sports Exerc., Vol. 42, No. 5, pp. 953–961, 2010. Introduction: Anecdotal evidence suggests that ingesting small volumes of pickle juice relieves muscle cramps within 35 s of ingestion. No experimental evidence exists supporting the ingestion of pickle juice as a treatment for skeletal muscle cramps. Methods: On two different days (1 wk apart), muscle cramps were induced in the flexor hallucis brevis (FHB) of hypohydrated male subjects (~3% body weight loss and plasma osmolality ~295 mOsm kg⁻¹ H₂O) via percutaneous tibial nerve stimulation. Thirty minutes later, a second FHB muscle cramp was induced and was followed immediately by the ingestion of 1 mL kg⁻¹ body weight of deionized water or pickle juice (73.9 ± 2.8 mL). Results: Cramp duration and FHB EMG activity during the cramp were quantified, as well as the change in plasma constituents. Cramp duration (water = 151.9 ± 12.9 s and pickle juice = 153.2 ± 23.7 s) and FHB EMG activity (water = 60% ± 6% and pickle juice = 68% ± 9% of maximum voluntary isometric contraction EMG activity) were similar during the initial cramp induction without fluid ingestion (P > 0.05). During FHB muscle cramp induction combined with fluid ingestion, FHB EMG activity was again similar (water = 55% ± 9% and pickle juice = 66% ± 9% of maximum voluntary isometric contraction EMG activity, P > 0.05). However, cramp duration was 49.1 ± 14.6 s shorter after pickle juice ingestion than water (84.6 ± 18.5 vs 133.7 ± 15.9 s, respectively, P < 0.05). The ingestion of water or pickle juice had little impact on plasma composition 5 min after ingestion. Conclusions: Pickle juice, and not deionized water, inhibits electrically induced muscle cramps in hypohydrated humans. This effect could not be explained by rapid restoration of body fluids or electrolytes. We suspect that the rapid inhibition of the electrically induced cramps reflects a neurally mediated reflex that originates in the oropharyngeal region and acts to inhibit the firing of alpha motor neurons of the cramping muscle. Key Words: ACETIC ACID, DEHYDRATION, ELECTROLYTES, PICKLE JUICE, VINEGAR
weight of deionized water or pickle juice (73.9 ± 2.8 mL). **Results:** Cramp duration and FHB EMG activity during the cramp were quantified, as well as the change in plasma constituents. Cramp duration (water = 151.9 ± 12.9 s and pickle juice = 153.2 ± 23.7 s) and FHB EMG activity (water = 60% ± 6% and pickle juice = 68% ± 9% of maximum voluntary isometric contraction EMG activity) were similar during the initial cramp induction without fluid ingestion ($P > 0.05$). During FHB muscle cramp induction combined with fluid ingestion, FHB EMG activity was again similar (water = 55% ± 9% and pickle juice = 66% ± 9% of maximum voluntary isometric contraction EMG activity, $P > 0.05$). However, cramp duration was 49.1 ± 14.6 s shorter after pickle juice ingestion than water (84.6 ± 18.5 vs 133.7 ± 15.9 s, respectively, $P < 0.05$). The ingestion of water or pickle juice had little impact on plasma composition 5 min after ingestion. **Conclusions:** Pickle juice, and not deionized water, inhibits electrically induced muscle cramps in hypohydrated humans. This effect could not be explained by rapid restoration of body fluids or electrolytes. We suspect that the rapid inhibition of the electrically induced cramps reflects a neurally mediated reflex that originates in the oropharyngeal region and acts to inhibit the firing of alpha motor neurons of the cramping muscle. **Key Words:** ACETIC ACID, DEHYDRATION, ELECTROLYTES, PICKLE JUICE, VINEGAR
Gastric Emptying After Pickle-Juice Ingestion in Rested, Euthydrated Humans

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Context: Small volumes of pickle juice (PJ) relieve muscle cramps within 85 seconds of ingestion without significantly affecting plasma variables. This effect may be neurologic rather than metabolic. Understanding PJ’s gastric emptying would help to strengthen this theory.

Objective: To compare gastric emptying and plasma variables after PJ and deionized water (DIW) ingestion.

Design: Crossover study.

Setting: Laboratory.

Patients or Other Participants: Ten men (age = 25.4 ± 0.7 years, height = 177.1 ± 1.6 cm, mass = 78.1 ± 3.6 kg).

Intervention(s): Rested, euhydrated, and euanaemic participants ingested 7 mL·kg⁻¹ body mass of PJ or DIW on separate days.

Main Outcome Measure(s): Gastric volume was measured at 0, 5, 10, 20, and 30 minutes postingestion (using the phenol red dilution technique). Percentage changes in plasma volume and plasma sodium concentration were measured preingestion (−45 minutes) and at 5, 10, 20, and 30 minutes postingestion.

Results: Initial gastric volume was 624.5 ± 27.4 mL for PJ and 659.5 ± 43.8 mL for DIW (P > .05). Both fluids began to empty within the first 5 minutes (volume emptied: PJ = 219.2 ± 39.1 mL, DIW = 305.0 ± 40.5 mL, P < .05). Participants who ingested PJ did not empty further after the first 5 minutes (P > .05), whereas in those who ingested DIW, gastric volume decreased to 111.6 ± 39.9 mL by 30 minutes (P < .05). The DIW group emptied faster than the PJ group between 20 and 30 minutes postingestion (P < .05). Within 5 minutes of PJ ingestion, plasma volume decreased 4.8% ± 1.6%, whereas plasma sodium concentration increased 1.6 ± 0.5 mmol·L⁻¹ (P < .05). Similar changes occurred after DIW ingestion. Calculated plasma sodium content was unchanged for both fluids (P > .05).

Conclusions: The initial decrease in gastric volume with both fluids is likely attributable to gastric distension. Failure of the PJ group to empty afterward is likely due to PJ’s osmolality and acidity. Cardiovascular reflexes resulting from gastric distension are likely responsible for the plasma volume shift and rise in plasma sodium concentration despite nonsignificant changes in plasma sodium content. These data support our theory that PJ does not relieve cramps via a metabolic mechanism.

Key Words: acetic acid, electrolytes, sodium, stomach, vinegar
plasma sodium concentration increased 1.5 ± 0.3 mmol L⁻¹ (P < .05). Similar changes occurred after DIW ingestion. Calculated plasma sodium content was unchanged for both fluids (P > .05).

**Conclusions:** The initial decrease in gastric volume with both fluids is likely attributable to gastric distension. Failure of the PJ group to empty afterward is likely due to PJ’s osmolality and acidity. Cardiovascular reflexes resulting from gastric distension are likely responsible for the plasma volume shift and rise in plasma sodium concentration despite nonsignificant changes in plasma sodium content. These data support our theory that PJ does not relieve cramps via a metabolic mechanism.

**Key Words:** acetic acid, electrolytes, sodium, stomach, vinegar
High rates of muscle glycogen resynthesis after exhaustive exercise when carbohydrate is coinjected with caffeine

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Pedersen DJ, Lessard SJ, Coffey VG, Churchley EG, Wootton AM, Ng T, Watt MJ, Hawley JA. High rates of muscle glycogen resynthesis after exhaustive exercise when carbohydrate is coinjected with caffeine. J Appl Physiol 105: 7–13, 2008. First published May 8, 2008; doi:10.1152/japplphysiol.01121.2007.—We determined the effect of coinjection of caffeine (Caff) with carbohydrate (CHO) on rates of muscle glycogen resynthesis during recovery from exhaustive exercise in seven trained subjects who completed two experimental trials in a randomized, double-blind crossover design. The evening before an experiment subjects performed intermittent exhaustive cycling and then consumed a low-CHO meal. The next morning subjects rode until volitional fatigue. On completion of this ride subjects consumed either CHO [4.5 g CHO/kg body mass (BM)] or the same amount of CHO + Caff (8 mg/kg BM) during 4 h of passive recovery. Muscle biopsies and blood samples were taken at regular intervals throughout recovery. Muscle glycogen levels were similar at exhaustion [~75 mmol/kg dry wt (dw)] and increased by a similar amount (~80%) after 1 h of recovery (133 ± 37.8 vs. 149 ± 48 mmol/kg dw for CHO and Caff, respectively). After 4 h of recovery Caff resulted in higher glycogen accumulation (313 ± 69 vs. 254 ± 50 mmol/kg dw, *P* < 0.001). Accordingly, the overall rate of resynthesis for the 4-h recovery period was 66% higher in Caff compared with CHO (57.7 ± 18.5 vs. 38.0 ± 7.7 mmol/kg dw·h⁻¹, *P* < 0.05). After 1 h of recovery plasma Caff levels had increased to 31 ± 11 μM (*P* < 0.001) and at the end of the recovery reached 77 ± 11 μM (*P* < 0.001) with Caff. Phosphorylation of CaMKThr286 was similar after exercise and after 1 h of recovery, but after 4 h CaMKThr286 phosphorylation was higher in Caff than CHO (*P* < 0.05). Phosphorylation of AMP-activated protein kinase (AMPK)Thr172 and AktSer473 was similar for both treatments at all time points. We provide the first evidence that in trained subjects coinjection of large amounts of Caff (8 mg/kg BM) with CHO has an additive effect on rates of postexercise muscle glycogen accumulation compared with consumption of CHO alone.

Akt: AMP-activated protein kinase; CaMK: calcium/calmodulin-dependent kinase; CHO: carbohydrate; dw: dry weight; BM: body mass; Caff: caffeine; dw·h⁻¹: dry weight·hour⁻¹; *P* < 0.05; *P* < 0.001.
of CHO + Caff (8 mg/kg DM) during 4 h of passive recovery. Muscle biopsies and blood samples were taken at regular intervals throughout recovery. Muscle glycogen levels were similar at exhaustion [\(\sim 75\) mmol/kg dry wt (dw)] and increased by a similar amount (\(\sim 80\%\)) after 1 h of recovery (133 \(\pm\) 37.8 vs. 149 \(\pm\) 48 mmol/kg dw for CHO and Caff, respectively). After 4 h of recovery Caff resulted in higher glycogen accumulation (313 \(\pm\) 69 vs. 234 \(\pm\) 50 mmol/kg dw, \(P < 0.001\)). Accordingly, the overall rate of resynthesis for the 4-h recovery period was 66\% higher in Caff compared with CHO (57.7 \(\pm\) 18.5 vs. 38.0 \(\pm\) 7.7 mmol·kg dw\(^{-1}\)·h\(^{-1}\), \(P < 0.05\)). After 1 h of recovery plasma Caff levels had increased to 31 \(\pm\) 11 \(\mu\)M (\(P < 0.001\)) and at the end of the recovery reached 77 \(\pm\) 11 \(\mu\)M (\(P < 0.001\)) with Caff. Phosphorylation of CaMK\(^{Thr286}\) was similar after exercise and after 1 h of recovery, but after 4 h CaMK\(^{Thr286}\) phosphorylation was higher in Caff than CHO (\(P < 0.05\)). Phosphorylation of AMP-activated protein kinase (AMPK)\(^{Thr172}\) and Akt\(^{Ser473}\) was similar for both.
Caffeine: Sleep and daytime sleepiness

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KEYWORDS
Caffeine;
Daytime sleepiness;
Sleep disturbance;
Caffeine dependence

Summary Caffeine is one of the most widely consumed psychoactive substances and it has profound effects on sleep and wake function. Laboratory studies have documented its sleep-disruptive effects. It clearly enhances alertness and performance in studies with explicit sleep deprivation, restriction, or circadian sleep schedule reversals. But, under conditions of habitual sleep the evidence indicates that caffeine, rather than enhancing performance, is merely restoring performance degraded by sleepiness. The sleepiness and degraded function may be due to basal sleep insufficiency, circadian sleep schedule reversals, rebound sleepiness, and/or a withdrawal syndrome after the acute, over-night, caffeine discontinuation typical of most studies. Studies have shown that caffeine dependence develops at relatively low daily doses and after short periods of regular daily use. Large sample and population-based studies indicate that regular daily dietary caffeine intake is associated with disturbed sleep and associated daytime sleepiness. Further, children and adolescents, while reporting lower daily, weight-corrected caffeine intake, similarly experience sleep disturbance and daytime sleepiness associated with their caffeine use. The risks to sleep and alertness of regular caffeine use are greatly underestimated by both the general population and physicians.

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documented its sleep-disruptive effects. It clearly enhances alertness and performance in studies with explicit sleep deprivation, restriction, or circadian sleep schedule reversals. But, under conditions of habitual sleep the evidence indicates that caffeine, rather than enhancing performance, is merely restoring performance degraded by sleepiness. The sleepiness and degraded function may be due to basal sleep insufficiency, circadian sleep schedule reversals, rebound sleepiness, and/or a withdrawal syndrome after the acute, over-night, caffeine discontinuation typical of most studies. Studies have shown that caffeine dependence develops at relatively low daily doses and after short periods of regular daily use. Large sample and population-based studies indicate that regular daily dietary caffeine intake is associated with disturbed sleep and associated daytime sleepiness. Further, children and adolescents, while reporting lower daily, weight-corrected caffeine intake, similarly experience sleep disturbance and daytime sleepiness associated with their caffeine use. The risks to sleep and alertness of regular caffeine use are greatly underestimated by both the general population and physicians.

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CHO Oxidation from a CHO Gel Compared with a Drink during Exercise

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ABSTRACT

PFEIFFER, B., T. STELLINGWERFF, E. ZALTAS, and A. E. JEUKENDRUP. CHO Oxidation from a CHO Gel Compared with a Drink during Exercise. Med. Sci. Sports Exerc., Vol. 42, No. 11, pp. 2038–2045, 2010. Recently, it has been shown that ingestion of solutions with glucose (GLU) and fructose (FRC) leads to 20%–50% higher CHO oxidation rates compared with GLU alone. Although most laboratory studies used solutions to deliver CHO, in practice, athletes often ingest CHO in the form of gels (semisolid). It is currently not known if CHO ingested in the form of a gel is oxidized as effectively as a drink. Purpose: To investigate exogenous CHO oxidation from CHO provided in semisolid (GEL) or solution (DRINK) form during cycling. Methods: Eight well-trained cyclists (age = 34 ± 7 yr, mass = 76 ± 9 kg, V̇O₂max = 61 ± 7 mL·kg⁻¹·min⁻¹) performed three exercise trials in random order. The trials consisted of cycling at 59% ± 4% V̇O₂max for 180 min while receiving one of the following three treatments: GEL plus plain water, DRINK, or plain water. Both CHO treatments delivered GLU plus FRC in a ratio of 2:1 at a rate of 1.8 g·min⁻¹ (108.8 g·h⁻¹). Fluid intake was matched between treatments at 867 mL·h⁻¹. Results: Exogenous CHO oxidation from GEL and DRINK showed a similar time course, with peak exogenous CHO oxidation rates being reached at the end of the 180-min exercise. Peak exogenous CHO oxidation rates were not significantly different (P = 0.40) between GEL and DRINK (1.44 ± 0.29 vs 1.42 ± 0.23 g·min⁻¹, respectively). Furthermore, oxidation efficiency was not significantly different (P = 0.36) between GEL and DRINK (71% ± 15% vs 69% ± 13%, respectively). Conclusions: This study demonstrates that a GLU + FRC mixture is oxidized to the same degree when administered as either semisolid GEL or liquid DRINK, leading to similarly high peak oxidation rates and oxidation efficiencies. Key Words: CHO INGESTION, CYCLING, EXOGENOUS CHO OXIDATION, CHO FORM
solutions with glucose (GLU) and fructose (FRC) leads to 20%–50% higher CHO oxidation rates compared with GLU alone. Although most laboratory studies used solutions to deliver CHO, in practice, athletes often ingest CHO in the form of gels (semisolid). It is currently not known if CHO ingested in the form of a gel is oxidized as effectively as a drink. **Purpose:** To investigate exogenous CHO oxidation from CHO provided in semisolid (GEL) or solution (DRINK) form during cycling. **Methods:** Eight well-trained cyclists (age = 34 ± 7 yr, mass = 76 ± 9 kg, VO_{2max} = 61 ± 7 mL·kg^{-1}·min^{-1}) performed three exercise trials in random order. The trials consisted of cycling at 59% ± 4% VO_{2max} for 180 min while receiving one of the following three treatments: GEL plus plain water, DRINK, or plain water. Both CHO treatments delivered GLU plus FRC in a ratio of 2:1 at a rate of 1.8 g·min^{-1} (108 g·h^{-1}). Fluid intake was matched between treatments at 867 mL·h^{-1}. **Results:** Exogenous CHO oxidation from GEL and DRINK showed a similar time course, with peak exogenous CHO oxidation rates being reached at the end of the 180-min exercise. Peak exogenous CHO oxidation rates were not significantly different (P = 0.40) between GEL and DRINK (1.44 ± 0.29 vs 1.42 ± 0.23 g·min^{-1}, respectively). Furthermore, oxidation efficiency was not significantly different (P = 0.36) between GEL and DRINK (71% ± 15% vs 69% ± 13%, respectively). **Conclusions:** This study demonstrates that a GLU + FRC mixture is oxidized to the same degree when administered as either semisolid GEL or liquid DRINK, leading to similarly high peak oxidation rates and oxidation efficiencies. **Key Words:** CHO INGESTION, CYCLING, EXOGENOUS CHO OXIDATION, CHO FORM
Effect of carbohydrate intake on half-marathon performance of well-trained runners.

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Abstract
Eighteen highly-trained runners ran two half marathons in mild environmental conditions, 3 wk apart, consuming either 426 +/- 227 mL of a flavored placebo drink (PLACEBO) or an equivalent volume of water (386 +/- 185 mL) and a commercial gel (GEL) supplying 1.1 +/- 0.2 g/kg body mass (BM) carbohydrate (CHO). Voluntary consumption of this fluid was associated with a mean BM change of approximately 2.4%. Runners performed better in their second race by 0.9% or 40 s (P = 0.03). Three runners complained of gastrointestinal discomfort in GEL trial, which produced a clear impairment of half-marathon performance by 2.4% or 105 s (P=0.03). The effect of GEL on performance was trivial: time was improved by 0.3% or 14 s compared with PLACEBO (P = 0.52). Consuming the gel was associated with a 2.4% slower time through the 2 x 200 m feed zone; adding a trivial approximately 2 s to race time. Although benefits to half marathon performance were not detected, the theoretical improvement during 1-h exercise with CHO intake merits further investigation.
Applied nutritional investigation

Delayed-onset muscle injury and its modification by wheat gluten hydrolysate

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Abstract

Objective: We investigated the pattern of delayed-onset muscle injury in well-trained athletes after a competitive half-marathon and the effects of post-race intake of wheat gluten hydrolysate (WGH).

Methods: Thirty well-trained college runners with a best time of 14–15 min over 5000 m raced in a half-marathon. Thereafter, they were divided into three groups based on finish times and given 0 (control), 10, or 20 g of WGH. Blood biochemical parameters were monitored at \(-1\) d, \(+1\) h, \(+1\) d, and \(+2\) d after the race. Data selected according to finish times and biochemical parameters were then analyzed.

Results: Plasma creatine kinase activity peaked at 1 d after the race in the control group and correlated with post-race white blood cell counts. The post-race elevation of creatine kinase activity was dose-dependently suppressed by WGH.

Conclusion: Delayed-onset muscle injury peaked in well-trained distance runners at 1 d after a half-marathon and was dose-dependently suppressed by a post-race intake of WGH. © 2009 Published by Elsevier Inc.
Female distance runners show a different response to post-workout consumption of wheat gluten hydrolysate compared to their male counterparts

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Abstract. Wheat gluten hydrolysate (WGH) is rich in glutamyl residue; glutamine is considered a conditionally essential amino acid under physical stress. WGH has been reported to suppress post-exercise rises in serum creatine kinase in male distance runners. This study aimed to reproduce the effects in female distance runners under similar conditions. The study was conducted in a double-blinded crossover manner. Six female collegiate distance runners ingested WGH or a placebo during recovery periods following half-marathons. When consumed after a half marathon, WGH suppressed increases in serum creatine kinase (CK) (4,5), suggesting the mitigation of muscle inflammation and promotion of recovery. Sawaki et al investigated changes in biochemical parameters among male runners with or without WGH intake after completing a half-marathon and found no changes. The present study investigated the effects of WGH in female distance runners. However, this was the first study to investigate the effects of WGH in female distance runners.
Dietary nitrate supplementation reduces the \( \text{O}_2 \) cost of low-intensity exercise and enhances tolerance to high-intensity exercise in humans

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Dietary nitrate supplementation reduces the \( \text{O}_2 \) cost of low-intensity exercise and enhances tolerance to high-intensity exercise in humans. J Appl Physiol 107: 1144–1155, 2009. First published August 6, 2009; doi:10.1152/japplphysiol.00722.2009. — Pharmacological sodium nitrate supplementation has been reported to reduce the \( \text{O}_2 \) cost of submaximal exercise in humans. In this study, we hypothesized that dietary supplementation with inorganic nitrate in the form of beetroot juice (BR) would reduce the \( \text{O}_2 \) cost of submaximal exercise and enhance the tolerance to high-intensity exercise. In a double-blind, placebo (PL)-controlled, crossover study, eight men aged 19–38 yr consumed 500 ml/day of either BR (containing 11.2 ± 0.6 mM of nitrate) or blackcurrant cordial (as a PL, with negligible nitrate content) for 6 consecutive days and completed a series of “step” moderate-intensity and severe-intensity exercise tests on the last 3 days. On days 4–6, plasma nitrate concentration was significantly greater following dietary nitrate supplementation compared with PL (BR: 273 ± 44 vs. PL: 140 ± 50 mM; \( P < 0.05 \)), and systolic blood pressure was significantly reduced (BR: 124 ± 2 vs. PL: 132 ± 5 mmHg; \( P < 0.01 \)). During moderate exercise, nitrate supplementation reduced muscle fractional \( \text{O}_2 \) extraction (as estimated using near-infrared spectroscopy). The gain in the increase in pulmonary \( \text{O}_2 \) uptake following the onset of moderate exercise was reduced by 19% in the BR condition (BR: 8.6 ± 0.7 vs. PL: 10.8 ± 1.6 ml·min\(^{-1} \cdot \text{W}^{-1} \); \( P < 0.05 \)). During severe exercise, the \( \text{O}_2 \) uptake slow component was reduced (BR: 0.57 ± 0.20 vs. PL: 0.74 ± 0.24 l/min; \( P < 0.05 \)), and the time-to-exhaustion was extended (BR: 675 ± 203 vs. PL: 583 ± 145 s; \( P < 0.05 \)). The reduced \( \text{O}_2 \) cost of exercise following increased dietary nitrate intake has important implications for our understanding of the factors that regulate mitochondrial respiration and muscle contractile energetics in humans.

ml·min\(^{-1} \cdot \text{W}^{-1} \); Ref. 36). During supra-GET exercise, \( \text{V}_\text{O}_2 \) dynamics become more complex, owing, in part, to the development of a delayed-onset \( \text{V}_\text{O}_2 \) “slow component”, which elevates the \( \text{O}_2 \) cost of exercise above 10 ml·min\(^{-1} \cdot \text{W}^{-1} \) (36, 64).

Whereas it is known that interventions such as training and the inspiration of hyperoxic gas can reduce the \( \text{O}_2 \) cost of heavy (above the GET but below critical power; Ref. 52) and severe (above critical power) exercise by reducing the amplitude of the \( \text{V}_\text{O}_2 \) slow component, the steady-state \( \text{V}_\text{O}_2 \) during moderate exercise is unaffected by these and other interventions in healthy humans (1, 15, 36, 51, 65). Surprisingly, however, it was recently reported that 6 days of dietary supplementation with pharmacological sodium nitrate reduced the \( \text{O}_2 \) cost of submaximal cycling at work rates expected to achieve 45–80% maximum \( \text{V}_\text{O}_2 \) (\( \text{V}_\text{O}_{\text{2max}} \)) (45). That this effect occurred without any increase in estimated nonoxidative energy production (as reflected by an unchanged blood lactate) (where brackets denote concentration) suggested that sodium nitrate ingestion improved the efficiency of muscle oxidative metabolism. It is known that tolerance to high-intensity exercise is, in certain respects, a function of \( \text{V}_\text{O}_{\text{2max}} \) and submaximal exercise economy (20). Therefore, assuming that \( \text{V}_\text{O}_{\text{2max}} \) is not altered, it is feasible that dietary nitrate supplementation might enhance exercise tolerance. However, this possibility has not been investigated.

The nitrate anion (\( \text{NO}_3^- \)) is relatively inert, and thus any biological effects are likely conferred via its conversion to the bioactive nitrite anion (\( \text{NO}_2^- \)). Inorganic nitrate is rapidly absorbed from the gut and is concentrated in saliva at least
Gastroprotective and blood pressure lowering effects of dietary nitrate are abolished by an antiseptic mouthwash

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ABSTRACT

Recently, it has been suggested that the supposedly inert nitrite anion is reduced in vivo to form bioactive nitric oxide with physiological and therapeutic implications in the gastrointestinal and cardiovascular systems. Intake of nitrate-rich food such as vegetables results in increased levels of circulating nitrite in a process suggested to involve nitrate-reducing bacteria in the oral cavity. Here we investigated the importance of the oral microflora and dietary nitrate in regulation of gastric mucosal defense and blood pressure. Rats were treated twice daily with a commercial antiseptic mouthwash while they were given nitrate-supplemented drinking water. The mouthwash greatly reduced the number of nitrate-reducing oral bacteria and as a consequence, nitrate-induced increases in gastric NO and circulating nitrite levels were markedly reduced. With the mouthwash the observed nitrate-induced increase in gastric mucus thickness was attenuated and the gastroprotective effect against an ulcerogenic compound was lost. Furthermore, the decrease in systemic blood pressure seen during nitrate supplementation was now absent. These results suggest that oral symbiotic bacteria modulate gastrointestinal and cardiovascular function via bioactivation of salivary nitrate. Excessive use of antiseptic mouthwashes may attenuate the bioactivity of dietary nitrate.
“Eat food. Not too much. Mostly plants”

- Michael Pollan
What is Food?
Proximates:
1. Moisture or Water
2. Protein
3. Fat
4. Energy
5. Carbohydrate
6. Dietary Fiber
7. Sugar

Minerals:
8. Calcium
9. Iron
10. Magnesium
11. Phosphorous
12. Sodium
13. Zinc
14. Copper
15. Manganese
16. Selenium

Vitamins:
17. Vitamin A
18. Alpha-Carotene
19. Beta-Carotene
20. Beta-Cryptoxanthin
21. Lycopene
22. Lutein
23. Vitamin D
24. Vitamin E
25. Vitamin K
26. Vitamin C
27. Thiamin
28. Riboflavin
29. Pantothenic Acid
30. Niacin
31. Vitamin B-6
32. Vitamin B-12
33. Folate
34. Choline

Lipids:
35. Cholesterol
36. Total Saturated Fatty Acids
37. Total Monounsaturated Fatty Acids
38. Total Polyunsaturated Fatty Acids

Other:
39. Caffeine
40. Alcohol
Proximates:
1. Moisture or Water
2. Protein
3. Fat
4. Energy
5. Carbohydrate
6. Dietary Fiber
7. Sugar
Minerals:
8. Calcium
9. Iron
10. Magnesium
11. Phosphorous
12. Sodium
13. Zinc
14. Copper
15. Manganese
16. Selenium
Vitamins:
17. Vitamin A
18. Alpha-Carotene
19. Beta-Carotene
20. Beta-Cryptoxanthin
21. Lycopene
22. Lutein
23. Vitamin D
24. Vitamin E
25. Vitamin K
26. Vitamin C
27. Thiamin
28. Riboflavin
29. Pantothenic Acid
30. Niacin
31. Vitamin B-6
32. Vitamin B-12
33. Folate
34. Choline
Lipids:
35. Cholesterol
36. Total Saturated Fatty Acids
37. Total Monounsaturated Fatty Acids
38. Total Polyunsaturated Fatty Acids

Other:
39. Caffeine
40. Alcohol
Nutrition Panels vs. Ingredients
<table>
<thead>
<tr>
<th>Nutrition Facts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serving Size: 1 pie (128.0 g)</td>
</tr>
<tr>
<td>Amount Per Serving</td>
</tr>
<tr>
<td><strong>Calories</strong></td>
</tr>
<tr>
<td><strong>Total Fat</strong></td>
</tr>
<tr>
<td>Saturated Fat</td>
</tr>
<tr>
<td>Trans Fat</td>
</tr>
<tr>
<td><strong>Cholesterol</strong></td>
</tr>
<tr>
<td><strong>Sodium</strong></td>
</tr>
<tr>
<td><strong>Total Carbohydrates</strong></td>
</tr>
<tr>
<td>Dietary Fiber</td>
</tr>
<tr>
<td>Sugars</td>
</tr>
<tr>
<td><strong>Protein</strong></td>
</tr>
<tr>
<td>Vitamin A</td>
</tr>
<tr>
<td>Calcium</td>
</tr>
<tr>
<td>Vitamin C</td>
</tr>
<tr>
<td>Iron</td>
</tr>
</tbody>
</table>

*Based on a 2000 calorie diet*
- Enriched Wheat Flour (Flour, Ferrous Sulfate, B-
Vitamins (Niacin, Thiamine (B1), Riboflavin (B2),
Folic Acid)]
- Diced Apple (Sulfite Treated)
- Partially Hydrogenated Vegetable and/or
Animal Shortening (Contains one or more of:
Soybean, Cottonseed or Canola Oil, Beef Fat)
- High Fructose Corn Syrup
- Corn Syrup
- Sugar
- Water
- Brown Sugar
- Modified Corn Starch
- Soy Flour
- Salt
- Sweet Dairy Whey
- Soy Protein Isolate
- Calcium Casseinate
- Sodium Casseinate
- Calcium Carbonate
- Calcium Sulfate
- Agar
- Locust Bean Gum
- Dextrose
- Sodium Phosphate
- Corn Flour
- Corn Dextrin
- Cinnamon
- Nutmeg
- Citric Acid
- Cornstarch
- Lemon Juice Solids
- Lemon Oil
- Potassium Citrate
- Sorbitol
- Tricalcium Phosphate
- Artificial Flavor
- Sodium Propionate
- Sorbic Acid
- FD & C Yellow 5
- Red 40
Flour
Apples
Butter
Egg Yolk
Salt
Brown Sugar
Water
Allspice
Nutmeg
Cinnamon
Vanilla Extract
Nuval - www.nuval.com

Nutrition Quality on a 0-100 score
What is Good Food?

Fiber
Folate
Vitamin A
Vitamin C
Vitamin D
Vitamin E
Vitamin B12
Vitamin B5
Potassium
Calcium
Zink
Omega 3 Fatty Acids
Bioflavonoids
Carotenoids
Magnesium
What is Bad Food?

- Saturated Fat
- Trans Fat
- Sodium
- Sugar
- Cholesterol
What Else to Consider?

Protein Quality
Fat Quality
Glycemic Load
Energy Density
Nuval Score = Pts for Good food / Pts for Bad Food
Everything good for you when you’re exercising is bad for you when you’re not.
Will it make me faster, healthier, better?

Where do you buy it?

Is it convenient?

Does it taste good?

How do you cook it?
Science vs. Practice
The Influence of Culture
Iron absorption from Southeast Asian diets

II. Role of various factors that might explain low absorption¹

L. Hallberg,² E. Björn-Rasmussen,³ L. Rossander,⁴ and R. Suwanik⁵

ABSTRACT  Previously reported levels of iron absorption from common Southeast Asian meals composed of rice, vegetables, and spices were too low to be consistent with the known prevalence of iron deficiency. In the present paper the cause of the low absorption was systematically sought. Variables investigated comprised methodological errors, factors in the diet such as certain foodstuffs, or contaminants inhibiting the absorption and characteristics of the subjects accompanied by malabsorption of dietary iron. The latter was excluded by comparing the absorption from both wheat rolls and a composite rice meal in Thai and Swedish women using the absorption of a small dose of ferrous ascorbate as a common basis of comparison. Two main factors were identified as causing the low absorption in the previous studies: the homogenization of the labeled meals before serving and the use of rice flour instead of rice. Iron absorption from nonhomogenized meals of identical composition as studied previously was many times higher (on an average 0.16 mg) and was consistent with the actual prevalence of iron deficiency in lower socioeconomic groups of Thais mainly consuming the simple meals studied. Recent modifications of the method to measure nonheme iron absorption from composite meals have thus not only made the determination simpler but also more accurate.  Am. J. Clin. Nutr. 30: 539-548, 1977.
basis for comparison. The similar ability to absorb iron in the two groups is illustrated in Figure 2.

Studies III and IV

Comparison of the absorption of iron from a composite Thai meal and from ferrous ascorbate in Thai and Swedish women. The results are given in Table 2. The Thai subjects absorbed more iron from the meals than the Swedish subjects. This could not be explained by a greater ability to absorb iron from the reference doses. On the contrary, the Swedish women absorbed more iron from the reference doses. The ratio between the absorption of food iron and ferrous iron was statistically significantly different in the two groups ($P < 0.01$). The difference between the two groups is evident from Figure 3.

Study V

Comparison of iron absorption from a composite Thai meal served in the usual way and in homogenized form. In eight of the 10 subjects a higher absorption was obtained from the nonhomogenized meals. On an average 70% more iron was absorbed from these meals. The effect of homogenization was statistically significant ($P < 0.01$). The results are shown as a graph in Figure 4.

Study VI

FIG. 3. Iron absorption from a simple composite Thai meal of rice, cooked vegetables, and spices and from ferrous ascorbate (3 mg of Fe) in Thai and Swedish women.

FIG. 4. Iron absorption from a simple composite Thai meal served in the usual way and in homogenized form.
from a composite Thai meal and from ferrous ascorbate in Thai and Swedish women. The results are given in Table 2. The Thai subjects absorbed more iron from the meals than the Swedish subjects. This could not be explained by a greater ability to absorb iron from the reference doses. On the contrary, the Swedish women absorbed more iron from the reference doses. The ratio between the absorption of food iron and ferrous iron was statistically signifi-
tion from the mix is probably related to consistency and/or appearance rather than to some physical factor as the hamburgers and the mashed potatoes were not further finely minced but simply mixed together.

The present finding of a low absorption from meals that were minced and mixed cannot alone explain the previously reported extremely low absorption from the Thai meals. It may, however, be an important contributory factor and this systematic error has to be considered in future studies on food iron absorption.

Ability to absorb food iron

In the systematic search for causes of the previously found low absorption the possibility of a malabsorption of dietary food iron in the subjects studied was also considered. As mentioned in the introduction, changes of the intestinal villi which might suggest a malabsorption syndrome have been reported to be prevalent in the Thai population (4). A general or marked malabsorption of dietary iron is not probable in the volunteers studied as judged from the hematological parameters.

The present studies comparing the absorption of food iron and an iron salt in Thai and in Swedish women were done to investigate whether a slight malabsorption of dietary iron might help to explain the previously found low absorption from a simple Thai meal. The present results clearly showed that the absorption of food iron in the Thai women was actually better than in the Swedish women. Wheat iron was absorbed to the same extent and a composite Thai meal seemed to be better absorbed by the Thai than by the Swedish women. The Swedish women liked the meal but considered it very spicy. It might be that the strong spices in some way interfered with the absorption of iron in the Swedish women (altered gastrointestinal motility, lowered secretion of gastric juice etc.).

Properties of the diet

The high iron content of the meals in the previously reported study—the basal meal contained about 8.4 mg instead of about 3 mg as expected from food tables—suggested that the meals were rich in iron which could be absorbed by the subjects without any malabsorption. Nevertheless, measurements showed that the main part of the excess iron was present in the rice flour. It should be noted that the water used contained no iron as distilled water was used in all studies.

Rice may be contaminated with soil, at least when dehusked by grinding in simple mills in the villages. Such soil would then, as we have observed in the villages, mainly consist of small lumps of clay from the rice fields. Usually the rice is washed before boiling. Rice flour, however, cannot easily be cleaned and may therefore have a higher content of contaminants. This was the reason why samples of clay from the rice field were studied in vitro. The iron content was high and the uptake of iron was as high as has been reported for some Turkish clays known to interfere with iron absorption (10). Of special interest in the present in vitro studies was the parallel uptake of iron and radioiron at neutral or slightly alkaline pH, which means that iron was adsorbed onto the clay and that in the in vitro system studied there was no isotopic exchange between the clay iron and the iron in the solution. At lower pH there was a more marked uptake of radioiron than of iron by the clay, which means that an isotopic exchange occurred between the tracer and the clay iron. It can be calculated that under the in vitro conditions used 70% of the clay iron exchanged with the radioiron. It is difficult to translate in vitro results to in vivo conditions. As clay might be a factor interfering with the absorption of iron it was considered important to carry out an in vivo study of the effect of an amount of clay on iron absorption which might give a detectable effect as judged from the in vitro studies. Assuming that there is no isotopic exchange in vivo between tracer and clay iron, it may be concluded that the percentage reduction in absorption observed when adding clay to the meals corresponds to a real reduction in the amount of iron absorbed as the specific activity of the meal would then be unaffected by the clay. Such a reduction would be small, however, from 0.14 to 0.12 mg. Therefore, only a slight isotopic exchange (more than 6%), which is to be anticipated from the in vitro studies, would be expected under the experimental conditions of the in vivo study.
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Nutrition vs. Nourishment
Emergence
The true science behind nutrition is that each person is their own scientific experiment.
- Ask a Question - e.g., Why does my gut hurt?
- Form a Hypothesis or theory to answer the question - e.g., The food I’m eating sucks...
- Make a Prediction - e.g., If I change my food, my gut won’t hurt. If I eat the same food my gut will keep on hurting
- Test or Experiment - e.g., Go on a ride with different food. Go on a ride with the same food.
- Analyze the Results - e.g., Did your gut still hurt?
Learn to cook & teach your athletes how to cook.

Enjoy what you eat.

If it makes you feel like crap, don’t eat it.

Eat Real Food unless it’s logistically impossible.

Not too much of it when you’re not training hard.

Mostly plants off the bike.
Nutrition on match day

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¹School of Sport and Exercise Sciences, Loughborough University, Loughborough, UK and ²Department of Sports Medicine, Real Madrid Football Club, Madrid, Spain

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Abstract
What players should eat on match day is a frequently asked question in sports nutrition. The recommendation from the available evidence is that players should eat a high-carbohydrate meal about 3 h before the match. This may be breakfast when the matches are played around midday, lunch for late afternoon matches, and an early dinner when matches are played late in the evening. The combination of a high-carbohydrate pre-match meal and a sports drink, ingested during the match, results in a greater exercise capacity than a high-carbohydrate meal alone. There is evidence to suggest that there are benefits to a pre-match meal that is composed of low-glycaemic index (GI) carbohydrate foods rather than high-GI foods. A low-GI pre-match meal results in feelings of satiety for longer and produces a more stable blood glucose concentration than after a high-GI meal. There are also some reports of improved endurance capacity after low-GI carbohydrate pre-exercise meals. The physical demands of soccer training and match-play draw heavily on players’ carbohydrate stores and so the benefits of good nutritional practices for performance and health should be an essential part of the education of players, coaches, and in particular the parents of young players.

Keywords: Soccer, football, carbohydrates, glycaemic index, fatigue, exercise capacity
The Truth Behind Nutrition Science