No Effects of a Short-Term Gluten-free Diet on Performance in Nonceliac Athletes

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ABSTRACT

LIS, D., T. STELLINGWERFF, C. M. KITIC, K. D. K. AHUJA, and J. FELL. No Effects of a Short-Term Gluten-free Diet on Performance in Nonceliac Athletes. *Med. Sci. Sports Exerc.*, Vol. 47, No. 12, pp. 2563–2570, 2015. **Purpose**: Implementation of gluten-free diets among nonceliac athletes has rapidly increased in recent years because of perceived ergogenic and health benefits. The aim of this study was to investigate the effects of a gluten-free diet (GFD) on exercise performance, gastrointestinal (GI) symptoms, perceived well-being, intestinal injury, and inflammatory responses in nonceliac athletes. **Methods**: Thirteen competitive endurance cyclists (8 males, 5 females) with no positive clinical screening for celiac disease or history of irritable bowel syndrome (mean ± SD; age, 32 ± 7 yr; weight, 71.1 ± 13.4 kg; height, 177.0 ± 11.8 cm, $\dot{V}O_{2max}$ 59.1 ± 8.0 mL·kg^{-1}·min^{-1}) were allocated to a 7-d gluten-containing diet (GCD) or GFD separated by a 10-d washout in a controlled, randomized, double-blind, crossover study. Cyclists ate a GFD alongside either gluten-containing or gluten-free food bars (16 g wheat gluten per day) while habitual training and nutrition behaviors were controlled. During each diet, cyclists completed the Daily Analysis of Life Demand for Athletes (DALDA) and GI questionnaires (postexercise and daily). On day 7, cyclists completed a submaximal steady-state (SS) 45-min ride at 70% W_{max} followed by a 15-min time trial (TT). Blood samples were taken preexercise, post-SS, and post-TT to determine intestinal fatty acid binding protein (IFABP) and inflammatory markers (cytokine responses: interleukin [IL] 1β, IL-6, IL-8, IL-10, IL-15, tumor necrosis factor α). Mixed effects logistic regression was used to analyze data. **Results**: TT performance was not significantly different ($P = 0.37$) between the GCD (245.4 ± 53.4 kJ) and GFD (245.0 ± 54.6 kJ). GI symptoms during exercise, daily, and DALDA responses were similar for each diet ($P > 0.11$). There were no significant differences in IFABP ($P = 0.69$) or cytokine ($P > 0.13$) responses. **Conclusions**: A short-term GFD had no overall effect on performance, GI symptoms, well-being, and a select indicator of intestinal injury or inflammatory markers in nonceliac endurance athletes. **Key Words**: INTESTINAL PERMEABILITY, ATHLETES, PERFORMANCE, INFLAMMATION, DALDA

Gluten-free diets (GFDs) are a clinical necessity for 5% to 10% of the general population for health purposes including celiac disease, wheat allergy, and nonceliac gluten sensitivity (16). However, general population market reports indicate that the adoption of a GFD has far exceeded the requirement for clinical populations, with GFD uptake exploding among nonceliac athletic populations as well (14,27). Correspondingly, our recently published questionnaire-based study, which investigated the frequency, perceptions and beliefs surrounding GFDs, found that in 942 nonceliac athletes, over 40% reported following a GFD at

least 50% of the time (27). Startlingly, this group of nonceliac athletes mostly relied on self-diagnosis of a gluten-related disorder and subsequent self-treatment with a GFD (27).

General-population gluten avoidance has become prevalent due to a belief that a GFD is "healthier" or owing to self-diagnosed gluten-related gastrointestinal (GI) disorders (7). Nonceliac athlete populations adopt a GFD in the belief that it is not only healthier and augments weight loss, but it will also decrease GI distress and systemic inflammation and improve psychological well-being and athletic performance (27). This rise in GFD uptake may be further influenced by advertising campaigns around the medical necessity and health benefits, whereas athlete testimonies support the idea that this diet might provide an ergogenic performance edge (28). Although there is one study showing improved glucose metabolism and reduced obesity with gluten elimination in nonceliac rodents (38), there is no scientific evidence to date that shows a GFD positively influences elements of health or performance in nonclinical populations.

Dietary triggers such as wheat (which contains the protein gluten) have been shown to damage the intestinal barrier

in clinically sensitive individuals (e.g., celiac disease [36]). Conversely, high-intensity exercise also reduces the integrity of the GI barrier (41). A primary mechanism causing GI distress during exercise is gut ischemia, resulting from the redistribution of blood from the splanchnic area to tissue with increasing exercise intensities. Splanchnic hypoperfusion ultimately gives rise to a cascade of responsive events, including epithelial injury, increased permeability, bacterial translocation, and systemic inflammation (23). Recurrent GI stress and injury, which is common among endurance athletes, may create an environment resulting in greater susceptibility to adverse reactions to common dietary triggers (40). GI injury in response to gluten ingestion has been well classified in celiac disease patients, but in nonceliac gluten-sensitivity, this condition is less apparent and evidence varies (10,29).

Other nutritional changes that may take place subsequent to gluten elimination can either improve or compromise an athlete's diet (27). Athletes believe that GFD adherence increases conscientiousness of eating a healthy and balanced diet (27). However, adopting a GFD without appropriate nutrition counseling may be associated with increased expense (+242% [12]), inadequate intake of B vitamins, fiber and iron, as well as compromised gut health through reduced beneficial gut bacteria populations (12). More recently, Shepherd and Gibson (37) suggest that the inadequacies found in a GFD may be linked to dietary gluten-free food choices rather than the diet itself, which all need to be considered before adopting such a diet.

Given that our published observational data suggest that many nonceliac athletes have adopted a GFD because of perceived, yet unconfirmed, health and performance benefits (27), our primary aim was to determine the effects of a short-term GFD in nonceliac athletes on exercise performance. Secondary aims were to determine the effects of a GFD on several parameters that possibly influence performance, including 1) GI symptoms, 2) perceived well-being, 3) intestinal injury, and 4) systemic inflammation. Our a priori hypothesis was that a 7-d GFD would not affect time trial (TT) performance or associated parameters in nonceliac athletes.

METHODS

Participants

Thirteen competitive cyclists (inclusion criteria: 18–40 yr old, $\dot{V}O_{2max} > 60.0$ (male) and >50.0 (female) mL·kg^{-1}·min^{-1}, respectively) participated in this study. A mixed sex cohort was chosen to represent the population adhering to a GFD as presented in our questionnaire-based study (27). Exclusion criteria were celiac disease (determined by AGA, tTG IgA, tTG IgG screened by an accredited pathology laboratory); known familial history of celiac disease; history of wheat allergy; clinically diagnosed nonceliac gluten sensitivity or irritable bowel syndrome; were following a gluten-free or vegetarian diet, or; had any pre-existing medical condition that could be affected by dietary intervention. Ethics approval was obtained from the Tasmanian Health and Medical Human Research Ethics Committee (H0013244). Before inclusion, participants were informed about the study procedure, completed a physical activity readiness questionnaire, and provided signed informed consent.

Experimental Design

$\dot{V}O_{2max}$ test. Cyclists' maximal oxygen uptake ($\dot{V}O_{2max}$) and peak power (W_{max}) were determined using an incremental test to exhaustion on a calibrated cycle ergometer (Excalibur Sport Cycle Ergometer, Groningen, The Netherlands) approximately 10 d before the experiment trials. Following a 5-min warm up at 100 W, cyclists began an incremental protocol at 100 W with increases of 50 W for males and 25 W for females in 3-min stages until volitional fatigue. Every 15 s of the test, expired air was analyzed using a metabolic cart (Parvo Medics TrueOne 2400, UT, USA) to determine oxygen uptake ($\dot{V}O_2$). Heart rate (HR) (RS800CX, Polar Instruments Inc., Oy, Finland), cadence, and power output were recorded very 15 s and rating of perceive exertion (6–20 Borg scale) was recorded at the end of each stage (2).

Before study commencement, a one-time GI history questionnaire and a 24-h food recall was collected. Using a double-blind, placebo-controlled, crossover design, participants were randomized by an independent observer according to a computer-generated list to receive either a gluten-containing diet (GCD) or a GFD for seven days, separated by a 10-d washout, and then received the alternative diet. A registered dietitian provided dietary education to participants on label reading, gluten-free eating, and nutrition intake recording as participants were permitted to self-select gluten-free foods in addition to the study food provided (i.e., fresh fruits and vegetables, yogurt) stipulating that all food was replicated in the subsequent trial. Exercise performance testing took place on day 7 of each dietary intervention, and blood samples were taken immediately preexercise, post–steady state (SS), and post-TT.

Gastrointestinal and well-being monitoring. Throughout the study, three questionnaires were required to be completed each day: 1) postexercise GI questionnaire, 2) daily GI questionnaire, and 3) Daily Analysis of Life Demands (DALDA). The presence and severity of upper and lower abdominal and other symptoms were determined using a 10-point scale ranging from 0 "no problem at all" to 9 "the worst it has ever been." Section 1 of the questionnaire addressed upper abdominal symptoms: reflux/heartburn, belching, bloating, stomach cramps/pain, nausea, vomiting. Section 2 addressed lower abdominal symptoms: intestinal/lower abdominal cramps, flatulence, urge to defecate, side ache/stitch, loose stool, diarrhea, and intestinal bleeding. Section 3 addressed other symptoms (dizziness, headache, muscle cramp, and urge to urinate) (33). We analyzed the frequency of all levels of GI and other symptoms (GI symptoms score

0–9) (33). All standardized GI questionnaires have been used in prior exercise and GI symptom research (6,32,33).

To assess the general stress levels (part A) and to determine stress-reaction symptoms (part B) of the participants, the DALDA tool was used (35). This questionnaire requires participants to rate each variable as "worse than normal," "normal," and "better than normal." Scores were tabulated and the "worse than normal" scores compared between trials. Each questionnaire was completed at the same time of day except for the postexercise questionnaire, which was completed immediately after training.

Food preparation. Participants were provided with gluten-free food including lunch and dinner meals prepared and frozen in a gluten-free commercial kitchen (Birdseed Catering), breakfast provisions (gluten-free cereals, breads, muffin, pancake mix), baking staples and snack foods (Orgran, Brookfarm, PureBred). Participants were permitted to add gluten-free foods to their meals and self-select gluten-free snacks provided dietary intake was replicated for the subsequent trial. The prototype study menu presented a macronutrient profile based on $g \cdot kg^{-1}$ body weight containing carbohydrate 6 to 8 $g \cdot kg^{-1}$, protein 1.2 to 1.7 $g \cdot kg^{-1}$, and fat 0.8–1.2 $g \cdot kg^{-1}$ (FoodWorks Professional 7, Xyris, Brisbane, Australia) (34). Two quinoa-based food bars were consumed per day that contained either vital wheat gluten or whey protein. The bars were designed to deliver 16 g wheat gluten per day (Manildra Group, Gladesville, Australia) or the equivalent dose of whey protein isolate (Vital Strength, Marrickville, Australia). Wheat gluten and whey protein were weighed using a digital food scale accurate to one decimal place (Terraillon, Croissy-Sur-Seine, France). Two food bars containing 8 g gluten each were ingested and spread throughout the day to simulate typical gluten intake patterns. Pilot blinded analysis in 10 healthy individuals, and two pre-trial participants confirmed that the food bars containing gluten could not be differentiated from the gluten-free food bars.

Familiarization to performance test. Before the first dietary intervention, a familiarization session was undertaken to accustom participants to the testing protocol (26). Information from the incremental exercise test was used to prescribe the intensity of the steady-state (SS) exercise ride: 70% W_{max} for 45-min SS (40) (234 ± 56 W) followed by a 15-min TT; a well-established and validated TT performance measure (20). For the TT, the ergometer was set in linear mode where the linear factor was based on individual participant's 70% W_{max} and preferred cycling cadence during the $\dot{V}O_{2max}$ test (20). We also purposely chose participants were given 0.5 $mL \cdot kg^{-1}$ distilled water every 10 min throughout the SS ride.

Performance test. Preceding each performance testing session, participants were provided with guidelines for gluten-free preexercise fueling. Guidelines for a moderate carbohydrate load 24 h before the performance test were provided which included the study food, self-selected gluten-free food, and instruction for increasing carbohydrate intake.

A selection of gluten-free foods was provided to participants for each 7-d trial. In combination with this, participants were provided with guidelines for food and fluid intake before their performance test. These guidelines included a moderate ingestion of carbohydrate (1–4 $mL \cdot kg^{-1}$ body mass) 1 to 4 h before exercise and 5 to 7 $mL \cdot kg^{-1}$ body mass fluid in the 2-h period before exercise. Participants were permitted to self-select preexercise fuels (either provided study meal or snack foods) based on preference and this was evaluated preexercise and replicated for each testing session. Each testing session was performed at the same time of day and climatic conditions (20°C, 40% humidity, 767–769 mm Hg). Participants refrained from the use of nonsteroid anti-inflammatories, caffeine, alcohol, and strenuous exercise 24 h before testing.

Before the 45-min SS ride, cyclists completed a 5-min warmup at 100 W. The 45-min SS ride and 15 min TT were performed in the same manner as the familiarization and participants were encouraged to complete as many kJ in the TT as possible. During the SS ride, verbal feedback on time completed was provided every 5 min. During the 15-min TT verbal feedback on time completed was given at minute 3, 6, and 9 then every minute for the final 5 min, with no other information given. Standardized verbal feedback was provided with any feedback outside of the script recorded and replicated for the subsequent trial. All verbal feedback and encouragement were provided by the same investigator, standardized and replicated in each trial. Data were collected every 3 min for kJ completed, power, cadence, and HR.

Biochemical Measurements

At each exercise performance test, venous blood samples (5 mL lithium heparin and 5 mL EDTA) were collected from a forearm vein preexercise, post-SS, and post-TT. Full blood cell counts were obtained immediately via an automated cell analyzer (XS-1000i, Sysmex, Kobe, Japan), whereas hemoglobin and hematocrit were immediately determined in duplicate using a HemoCue Hb 20 (HemoCue, Angelholm, Switzerland) and the capillary centrifugation at 12,000g for 5 min, respectively. Blood samples were centrifuged at 1000g for 15 min and plasma was aliquoted and stored at −80°C until analysis. All plasma variables were adjusted for changes in plasma volume (9).

Intestinal fatty acid binding protein. Plasma intestinal fatty acid binding protein (IFABP), a sensitive and acute marker of small intestinal cell damage, was determined using an ELISA (Hycult Biotechnology, Uden, The Netherlands) according to manufacturer's instructions. All samples were analyzed in duplicate with a 5% intra-assay coefficient of variation.

Markers of inflammatory response. Plasma cytokines concentrations of IL (interleukin) 1β, IL-6, IL-8, IL-10, IL-15 and tumor necrosis factor alpha (TNF-α) were determined using a multiplex bead array assay (Millipore, MN, USA). The minimal detectable concentration of IL-1β was 0.8 $pg \cdot mL^{-1}$, IL-6 was 0.9 $pg \cdot mL^{-1}$, IL-8 was 0.4 $pg \cdot mL^{-1}$,

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IL-10 was 8.6 pg·mL^{-1}, IL-15 was 1.2 pg·mL^{-1}, and TNF-α was 0.7 pg·mL^{-1}. Samples were analyzed in duplicate, and the intra-assay coefficient of variation was 9% for IL-1β, 9% for IL-6, 5% for IL-8, 10% for IL-10, 8% for IL-15, 8% for TNF-α.

Statistical Analysis

Before analysis, all data were tested for normality using a Kolmogorov-Smirnov test. Where normally distributed, mixed-effects linear regression was performed. When assumptions of linear regression (heteroscedasticity, skewness, kurtosis, or linearity) were violated, data were analyzed using repeated-measures ordered logistic regression and all analysis were performed for intervention and order effect. Poisson regression was used to compare frequency of GI symptom severity between GCD and GFD daily and during exercise. Analysis was performed using Stata 13.0 (Statacorp LP, College Station, TX). A sport-specific Microsoft Excel spreadsheet (19) was used to estimate the likelihood that a GFD would be beneficial, negligible, or harmful based upon the smallest important change (5.17 kJ) (20).

Two methods for sample size calculation were applied, including magnitude-based inferential statistics for comparing performance (total kJ completed in 15-min TT) and power-based sample size calculations for postexercise IFABP and cytokines. Sample size analysis based on performance was determined using Hopkin's statistical spreadsheet, "estimating sample size for magnitude-based inferences" (18). The spreadsheet estimates sample size requirements when the typical error and smallest important change (Cohen's smallest important effect—0.2 of the between subject SD) are entered for the primary performance measure. The typical error (6.53 kJ) and smallest important change (5.17 kJ) were obtained from previously published 15-min TT reliability data (20). Sample size calculation using these values indicated the need for 12 participants. For the blood markers of IFABP and one chosen marker of inflammation (TNF-α), sample size was determined using power calculations to detect an intervention difference at a two-sided 5% significance level with a power of 80%. Assuming a postexercise IFABP value of 474 ± 74 pg·mL^{-1} (39), and detecting a 20% difference: a total of 10 participants were required. Assuming a postexercise TNF-α value of 28 ± 4 pg·mL^{-1} (4), and detecting a 10% difference a total of 11 participants were required. Thirteen participants were recruited to allow for one drop out.

RESULTS

Participants. Thirteen participants (8 males: $\dot{V}O_{2max}$ 63.7 ± 6.5 mL·kg^{-1}·min^{-1}, 5 females: $\dot{V}O_{2max}$ 51.6 ± 2.8 mL·kg^{-1}·min^{-1}; 32 ± 7 yr old; weight, 71.1 ± 13.4 kg; height, 177 ± 11.8 cm) completed the study. Blood results were available for 10 to 12 participants. There were no significant differences between males or females for any of the variables measured ($P > 0.05$).

Performance test. Exercise performance data are shown in Figure 1. There was no significant difference in total work completed over the 15-min TT on day 7 between the GCD and GFD (245.4 ± 53.4 kJ vs 245.0 ± 54.6 kJ, $P = 0.37$). Power (267 ± 60 W vs 267 ± 57 W, $P = 0.80$), HR (168 ± 9 bpm vs 167 ± 8 bpm, $P = 0.56$), and cadence (94 ± 8 rpm vs 95 ± 8 rpm, $P = 0.31$) were also similar during the TT for both the GCD and GFD trials. Analysis of the performance data (work completed) using magnitude-based inference indicated a 100% "negligible" effect of a GFD on performance.

Gastrointestinal well-being. Frequency of all GI symptoms ratings daily (outside of exercise) and during exercise for upper and lower GI symptoms are displayed in Figure 2. There were no significant differences in GI symptoms between GCD and GFD for daily upper ($P > 0.32$), lower ($P > 0.15$), and other ($P > 0.40$) symptoms. Similarly, during exercise, GI symptoms were not significantly different between dietary interventions for upper ($P > 0.27$), lower ($P > 0.11$), and other ($P > 0.08$) symptoms.

Overall well-being. DALDA scores were tabulated and the "worse than normal" scores were compared between each trial. No difference in the sum of 7-d DALDA scores between the GCD (26 ± 19) and GFD (27 ± 18) was found ($P = 0.26$).

IFABP. IFABP levels increased post-SS cycling and post-TT from preexercise for both groups (GCD and GFD, preexercise: 94 ± 83 pg·mL^{-1} and 99 ± 57 pg·mL^{-1}, post-SS: 233 ± 188 pg·mL^{-1} and 192 ± 159 pg·mL^{-1}; post-TT: 304 ± 191 pg·mL^{-1} and 301 ± 252 pg·mL^{-1}). There were no significant differences in IFABP concentration at any time point between the GCD and GFD ($P > 0.69$).

Cytokines. Plasma concentrations of IL-1β, IL-6, IL-8, IL-10, Il-15 and TNF-α, measured to determine systemic inflammatory responses, were not significantly ($P > 0.05$) different between the GCD and GFD (Table 1).

DISCUSSION

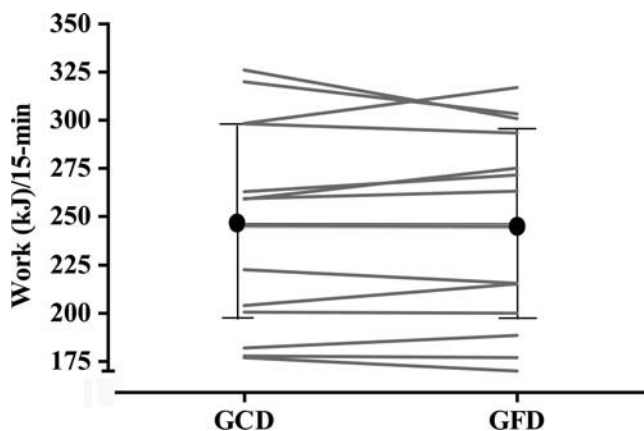This is the first study to examine the effects of dietary gluten removal on exercise performance and associated

FIGURE 1—Overall 15-min TT performance (kJ) in response to GCD and GFD. Solid grey lines – individual performance ● means (SD); *n* = 13.
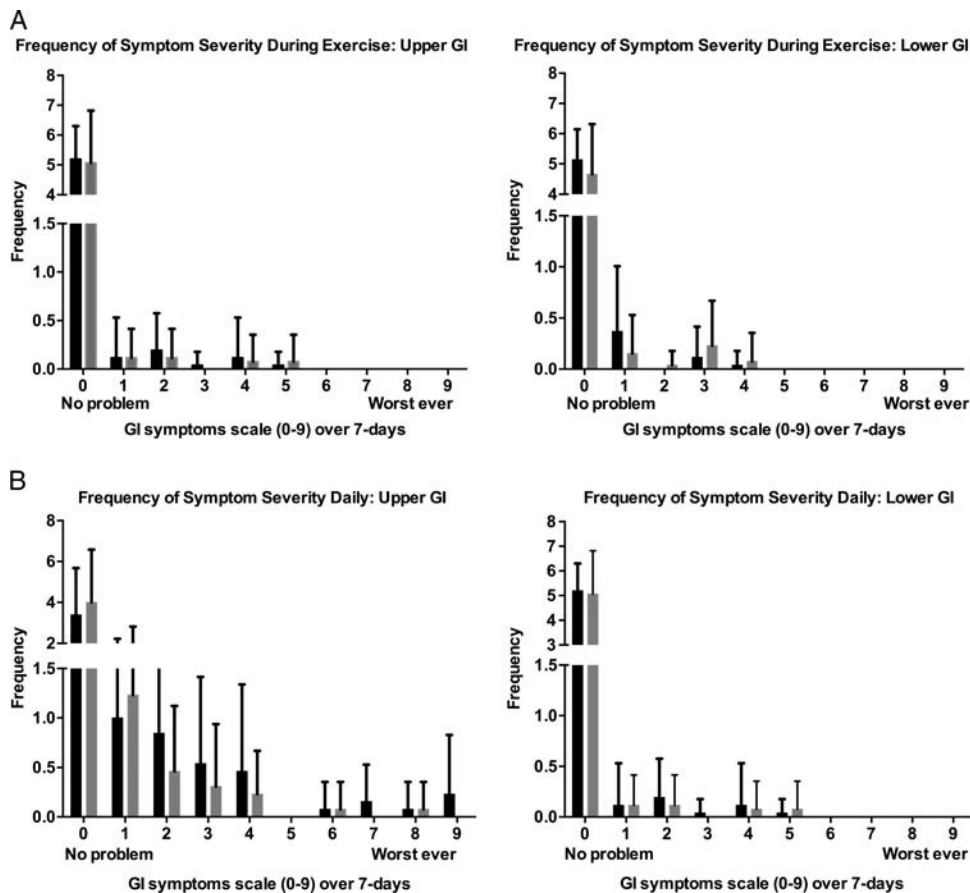
FIGURE 2—Frequency of GI symptoms daily and during exercise over 7-d period for gluten-containing diet (■GCD) and gluten-free diet (■GFD). Values are median (range); *n* = 13.

parameters in nonceliac athletes. Our previous observational data indicated that a much higher proportion of nonceliac athletes (>40% of endurance athletes, more females than males) follow a GFD than would be required for medical reasons (5%–10% of the general population) (16,27). Belief in a GFD being healthier and reducing GI symptoms and inflammation alongside self-diagnosed gluten-related conditions are the primary motivations for adopting this diet in athlete populations (27). In line with our a priori hypothesis, our double-blind, placebo-controlled, cross-over study found no effect of a 7-d GFD on exercise performance. We also found no difference in GI symptoms, overall well-being, markers of GI injury, or systemic inflammation.

A recent review by Halson and Martin summarized the "belief effect," which suggests that the belief in an intervention can contribute a 1% to 3% improvement in performance regardless if it actually has ergogenic mechanisms (17). We have recently shown a current belief in the performance-enhancing effects of gluten removal (27). Until findings of the present study, there have been no investigations that have determined the effect of a GFD on exercise performance. Through effective double-blinding, nonceliac athletes and researchers were unable to differentiate each diet and TT performance was similar between trials (Fig. 1). Accordingly, other physiologic parameters

such as HR, power, and cadence were not significantly different between diets. Given a mixed-sex cohort, the potential effects of menstrual cycle on performance were considered, and performance testing for female athletes was scheduled to avoid conflicting with early follicular or the midluteal phase. It is further pertinent to note that in undiagnosed celiac disease or gluten-related clinical conditions, dietary gluten removal would potentially yield a performance benefit through exhibited improvement in biochemical measures and GI symptoms; however, to our knowledge, no published data yet exist to support this.

TABLE 1. Cytokines responses from preexercise, post steady state (~70% W_{max}) and immediately after 15-min TT after 7-d of a gluten-containing diet vs gluten-free diet.

Cytokines (pg·mL^{-1})	Diet	Preexercise	Post-SS	Post-TT
IL-1β	GCD	7.64 ± 7.73	7.04 ± 6.76	8.17 ± 7.76
	GFD	9.71 ± 9.90	8.64 ± 8.72	9.04 ± 7.69
IL-6	GCD	4.33 ± 4.47	4.42 ± 4.11	6.39 ± 5.33
	GFD	7.21 ± 7.30	6.12 ± 5.73	7.93 ± 4.48
IL-8	GCD	8.83 ± 5.64	11.44 ± 11.34	8.00 ± 4.61
	GFD	10.11 ± 6.74	8.54 ± 3.88	8.75 ± 3.70
IL-10	GCD	14.71 ± 29.74	15.39 ± 24.80	18.53 ± 18.53
	GFD	24.68 ± 37.50	19.48 ± 34.37	18.50 ± 28.76
IL-15	GCD	12.65 ± 9.98	11.49 ± 9.22	12.04 ± 9.74
	GFD	15.17 ± 11.94	14.78 ± 13.94	12.56 ± 8.93
TNF-α	GCD	7.77 ± 2.59	7.47 ± 1.77	8.61 ± 1.74
	GFD	10.30 ± 4.88	9.21 ± 3.01	9.26 ± 2.78

Values and mean ± SD. There were no statistically significant differences between the GCD vs GFD preexercise, post-SS, and post-TT (*n* = 10).

Performance and training capacity can be affected by GI distress and a decrease in performance has also been shown as a consequence of this stress (30). No difference in GI symptoms was found during the performance test. Across each dietary trial, both exercise associated and daily GI symptoms were also similar (Fig. 2). It has been reported that up to 70% of endurance athletes commonly experience GI distress during intense exercise and that many athletes believe gluten removal might reduce these symptoms (33). Anecdotally, a short-term GFD is adopted before competition among some endurance athletes and many athletes follow this diet intermittently (27). Short-term clinical interventions in patients with reported GI distress have found that in true nonceliac gluten sensitivity symptoms triggered by gluten appear within a few hours to days after ingestion (1,5,8). Our findings do not support that gluten removal reduces the frequency or severity of GI symptoms daily, while training, or during a simulated competitive TT. GI symptom severity in the present study was lower than previously reported during endurance competition (33). Whether a difference in GI symptoms possibly related to gluten would manifest with a more jarring exercise modality, such as running (33), or in environments that further exacerbate GI stress such as prolonged endurance exercise in the heat with fluid restriction, is unknown (24,25).

Psychological well-being is an additional factor that can be influenced by dietary intake and further affect performance and training capacity. We used the DALDA tool to monitor the effects that this dietary intervention had on life stress and stress-reaction associated with athletic performance, and no significant difference in DALDA response was found over a 1-wk period (35). Although our study is the first to investigate the effects of a GFD on DALDA responses, previous literature has found alterations in psychological well-being with short-term dietary interventions (22). Observational data obtained from cyclists on a range of special diets by Burks et al. (3) summarized that 50% of respondents following a GFD reported increased feelings of tiredness/lethargy when deviating from this diet. A 9-d dietary intervention of low carbohydrate during a period of intensified cycling has also been shown to increase mood disturbances compared with a high-carbohydrate diet (22). The DALDA is as a sensitive tool to monitor well-being over a short-term dietary intervention (22), and given that nutritional intake for each trial in the present study was replicated, gluten does not appear to affect well-being in nonceliac athletes.

Gibson and Muir (13) have suggested that gluten itself may not be the sole nutrient regulating factor in the reported symptom improvement with a GFD, but that the subsequent reduction in fructans and galacto-oligosaccharides (fermentable oligo-, di- and monosaccharides and polyols; FODMAPs) associated with gluten removal may be a modulating factor (13). Although our study population was dissimilar to the clinical populations observed in the above research, we also wanted to design a study with a high degree of ecological validity. Dietary FODMAPs were included in the background diets of the participants due to the fact that the vast majority of athletes do not eliminate all sources of these short-chain carbohydrates when following a GFD. Our study design also selected a short-term intervention to minimize the interference with training regime and alongside the evidence that gluten-related symptoms appear, as previously mentioned, in a matter of hours to days in clinical assessment of nonceliac gluten sensitivity (11). Nonceliac athletes' GFD habits are shown to vary; however, a large cohort (42%) only eat gluten-free 50% to 75% of the time and sometimes only 1–2 wk before competition (27). Our data indicate that the pattern of short-term or periodic gluten avoidance common for athletes to adopt does not influence performance, GI symptoms, or well-being (27).

Endurance athletes predictably experience GI ischemia, which is proposed as a primary mechanism causing GI distress during exercise. GI ischemia can ultimately give rise to a cascade of responsive events including epithelial injury and both GI and systemic inflammation (15). In the current study, a submaximal exercise preload known to induce GI hypoperfusion was used before a 15-min TT to potentiate a high degree of GI stress (40). Increased epithelial injury also permits translocation of endotoxins across the gut barrier and into circulation, potentially contributing to increased systemic inflammatory responses (21,41). Preexercise IFABP levels were within expected ranges of healthy controls and increased in accordance with similar exercise studies across both dietary trials during the performance test (40). Increased IFABP levels are indicative of intestinal injury, known to occur under strenuous and acute exercise conditions. It is suggested that intestinal injury is a possible hindrance to training capacity, performance, and recovery through adverse GI symptoms and decreased nutrient absorption (40). Our investigation found gut injury to be increased during strenuous exercise; nonetheless, gluten ingestion did not seem to augment this response before, throughout or at the end of a strenuous exercise bought. It is further noteworthy to postulate if recurrent injury, as would occur in endurance training such as in the present study (average training sessions per week, 13), would facilitate an environment of enhanced susceptibility to dietary triggers or influence markers of systemic inflammation in nonceliac athletes.

Systemic inflammatory responses measured were also similar between dietary interventions before, during, and immediately after the performance test. Our data suggest that short-term gluten elimination in nonceliac athletes does not influence the cytokine response around this specific exercise bout (Table 1). Interestingly, aside from inflammatory mechanisms associated with strenuous exercise, Soares et al. (38) found that an 8-wk high-fat GFD attenuated inflammation associated with adiposity, reduced visceral fat, and improved glucose homeostasis in nonceliac rodents (38). Systemic inflammatory response patterns in both groups paralleled preceding literature with comparable exercise bouts (31); however, it is yet to be determined if inflammation localized to the GI tract would be different in nonceliac athletes.

Future research with a longer duration of GFD adherence may help account for differential gut flora habituation, which could be influential on GI health, performance, and other parameters. However, such outcomes may be difficult to monitor, as during a longer intervention, training adaptations would be likely to occur that may mask any dietary influenced performance changes. Lengthier interventions are also more intrusive for the athlete, compromise dietary adherence, and challenge the ability to control and replicate training and food intake.

CONCLUSIONS

In this tightly controlled study, our data suggest that a 7-d GFD does not have a beneficial or a negative effect on cycling performance, GI health, systemic inflammation, or overall well-being in nonceliac athletes. Based on these findings, it is recommended that athletes seek evidence-based advice before adopting a GFD for nonclinical reasons to ensure that nutrition intake supports individualised and optimal fueling for sport performance.

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