

GINGER (*ZINGIBER OFFICINALE*) AS AN ANALGESIC AND ERGOGENIC AID IN SPORT: A SYSTEMIC REVIEW

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ABSTRACT

Wilson, PB. Ginger (*Zingiber officinale*) as an analgesic and ergogenic aid in sport: a systemic review. *J Strength Cond Res* 29(10): 2980–2995, 2015—Ginger is a popular spice used to treat a variety of maladies, including pain. Nonsteroidal anti-inflammatory drugs (NSAIDs) are frequently used by athletes to manage and prevent pain; unfortunately, NSAIDs contribute to substantial adverse effects, including gastrointestinal (GI) dysfunction, exercise-induced bronchoconstriction, hyponatremia, impairment of connective tissue remodeling, endurance competition withdrawal, and cardiovascular disease. Ginger, however, may act as a promoter of GI integrity and as a bronchodilator. Given these potentially positive effects of ginger, a systematic review of randomized trials was performed to assess the evidence for ginger as an analgesic and ergogenic aid for exercise training and sport. Among 7 studies examining ginger as an analgesic, the evidence indicates that roughly 2 g·d⁻¹ of ginger may modestly reduce muscle pain stemming from eccentric resistance exercise and prolonged running, particularly if taken for a minimum of 5 days. Among 9 studies examining ginger as an ergogenic aid, no discernable effects on body composition, metabolic rate, oxygen consumption, isometric force generation, or perceived exertion were observed. Limited data suggest that ginger may accelerate recovery of maximal strength after eccentric resistance exercise and reduce the inflammatory response to cardiorespiratory exercise. Major limitations to the research include the use of untrained individuals, insufficient reporting on adverse events, and no direct comparisons with NSAID ingestion. While ginger taken over 1–2 weeks may reduce pain from eccentric resistance exercise and prolonged running, more research is needed to evaluate its safety and efficacy as an analgesic for a wide range of athletic endeavors.

KEY WORDS exercise, nutrition, pain, dietary

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29(10)/2980–2995

Journal of Strength and Conditioning Research
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2980 *Journal of Strength and Conditioning Research*

INTRODUCTION

Ginger is a popular spice originating from the rhizomes of the plant *Zingiber officinale* (30). Ginger has been used for centuries to treat a variety of maladies, particularly in non-Western cultures. Today, ginger is used around the world as a dietary supplement and food ingredient. In the 2002 Health and Diet Survey sponsored by the Food and Drug Administration, roughly 1.4% of adult Americans reported using ginger as a dietary supplement over the past 12 months (92).

Several scientific reviews and meta-analyses have examined the use of ginger, including for nausea and vomiting (31), pain management (91), cancer prevention (84), and as an anti-inflammatory (36). Lacking to date, however, is a comprehensive review outlining the physiological effects and potential uses of ginger in the context of exercise training and sport. The sport nutrition supplement industry has expanded dramatically over the past several decades, with the global market reaching an estimated \$31.2 billion in 2008 (9). Athletes and sports practitioners considering whether to use ginger would benefit from a review of its physiological effects, potential benefits, and adverse effects in the context of exercise training and sport.

The purpose of this article was to provide an overview of ginger as a dietary supplement for athletes and individuals partaking in exercise training. Particular attention is paid to the analgesic properties of ginger, including plausible mechanisms because pain medications are commonly used by athletes and individuals undergoing exercise training and can cause substantial untoward effects (109). As such, a brief overview of analgesic use in sport is provided. In addition, the physiological effects of ginger on the gastrointestinal (GI) and respiratory systems are outlined because both are stressed during prolonged and intense exercise and are affected by ginger. Subsequently, the results of a PubMed search of studies examining the analgesic and ergogenic effects of ginger in exercise training and sport are outlined. Finally, adverse effects of ginger supplementation and research needs are discussed to help guide future work.

ANALGESIC USE IN SPORT

Ginger has received significant study as an analgesic in both human (8,75,91) and animal models (23,76,83). Identifying alternative analgesics for use in sport and exercise training is

an important issue given the plethora of adverse effects that accompany analgesic medications. Nonsteroidal anti-inflammatory drugs (NSAIDs) are the most widely available class of analgesics used by athletes (109). Consequently, a brief overview of the prevalence of use and adverse effects of NSAIDs in sport is provided.

Prevalence of NSAID Use in Sport

Nonsteroidal anti-inflammatory drug use is highly prevalent during international athletic competitions, with a substantial proportion of athletes using NSAIDs prophylactically and consuming multiple NSAIDs simultaneously (24,99,100). During the XV Pan American Games, for example, nearly 65% of athletes reported using NSAIDs (24). Estimates from the 2000 Sydney Olympics were lower at 25.6% for athletes undergoing doping control, but these athletes were asked to report medications used only during the past 3 days (22). During the 2002 and 2006 FIFA World Cups, 30.8% of athletes took NSAIDs before a match and 10.6% took NSAIDs before every match (100).

Sports that involve violent contact, such as American football, are also susceptible to frequent NSAID usage. Among collegiate footballers, 73% reported using NSAIDs for pain during the previous season, with approximately one third of players taking more than the recommended dose (117). Similarly, in a survey of high school footballers, 75% had used NSAIDs sometime over the past 3 months (111). While the prevalent use of NSAIDs in contact sports is somewhat expected, NSAID use is also widespread among individuals participating in noncontact activities. Between 50 and 70% of endurance athletes competing in triathlons, marathons, and ultra-marathons report using NSAIDs on a regular basis (35,45). Of equal concern, knowledge regarding the adverse effects of NSAIDs seems to be low and limited to GI side effects (35).

Adverse Effects of NSAIDs in Exercise Training and Sport

The overuse of NSAIDs raises a host of concerns, both acutely and chronically. Nonsteroidal anti-inflammatory drugs can cause GI pain, GI hemorrhaging, nephropathy, hepatitis, and anaphylactic reactions (87,90) and are frequently implicated in hospital admissions stemming from drug interactions (10).

While it has long been established that NSAIDs can cause adverse effects, recent research raises concern over their use in the context of exercise training and sport. In the short term, NSAIDs can suppress muscle protein synthesis and muscle regeneration following an exercise bout, at least in young individuals (55,97). It has been hypothesized that cyclooxygenase (COX) enzymes regulate muscle protein synthesis and that COX inhibition may blunt the rise in muscle protein synthesis that normally accompanies exercise (112). Nonsteroidal anti-inflammatory drugs inhibit COX enzymes and have therefore been implicated as possible negative modifiers of training adaptations (55). However, studies examining the long-term effects of NSAID consumption

on the functional and morphological adaptations to resistance training have failed to find negative effects (47,96). The effect of NSAIDs on mitochondrial adaptation to exercise has also received scientific attention, and although *in vitro* and animal models show that NSAIDs can have deleterious effects on mitochondrial function, data from *in vivo* human models are lacking (78).

In terms of GI functioning, NSAIDs reduce intestinal barrier integrity and increase GI permeability during exercise (50,105), which may increase the inflammatory response to prolonged exercise (68) and the risk of heat illness (49). The negative GI effects of NSAIDs also extend to endurance competition. Participants of the 2010 Bonn Marathon who used analgesics (with the majority being NSAIDs) had 5.1 times the odds of experiencing an adverse event and were 10 times more likely to experience GI cramps than nonusers (48). In addition, GI cramps were 2–3 times as likely to be blamed for race withdrawal in analgesic users. Of even greater concern, 9 cases of hospital admittance were reported, all of which occurred in analgesic users (48). Athletes commonly consume NSAIDs to minimize existing or anticipated pain during exercise, believing it will allow them to continue competing because of this reduced pain. Evidence for this benefit, however, is lacking in the scientific literature (109), and as a consequence, it is unknown whether GI symptoms caused by NSAIDs override their possible musculoskeletal pain relief effects, and ultimately, how the balance of these factors influences performance.

Nonsteroidal anti-inflammatory drugs have also been implicated in the development of hyponatremia during endurance competition (25,113), a potentially fatal condition causing disorientation, nausea, headache, and muscle weakness (66). They are thought to influence the development of hyponatremia, in part, by altering the action of arginine vasopressin (AVP). They decrease renal prostaglandin synthesis, and because prostaglandins inhibit AVP, NSAID ingestion leads to increased AVP action and decreased fluid excretion (53). It should be noted, however, that not all studies have found NSAIDs increase the risk of hyponatremia (2,38,76). The lack of an association between NSAIDs and hyponatremia in these studies may be due to the modest sample sizes (38), mild environmental conditions (2,76), and limited fluid availability during the race (76).

Regarding respiratory function, NSAIDs have bronchoconstrictive properties that may exacerbate symptoms in individuals with exercise-induced asthma and bronchoconstriction (67). COX-1 and COX-2 enzymes are expressed in the airway epithelium of humans (26) and are thought to play a role in respiratory function by influencing the production of prostaglandin E₂ (73). Prostaglandin E₂ acts on prostaglandin E receptors on respiratory mast cells, influencing the release of histamine and leukotrienes (37). Nonsteroidal anti-inflammatory drug ingestion inhibits COX enzymes, which, in turn, decreases the production of prostaglandin E₂, contributing to hypersensitivity of the airways

to antigens (73). While estimates vary depending on the population studied, 21% of adults and 5% of children with asthma demonstrate an asthmatic response when ingesting NSAIDs, most commonly in the form of aspirin (43). Surprisingly, the influence of exercise-induced bronchoconstriction on exercise performance remains largely unknown, and although it seems logical that treating exercise-induced bronchoconstriction would enhance exercise performance, this has not been consistently shown (74). Consequently, more research is needed to delineate the interrelationships between NSAID use, exercise-induced bronchoconstriction, and athletic performance.

Over the long term, frequent and heavy NSAID use increases the risk of cardiovascular events (61). This increased risk of cardiovascular events is partly attributable to the effects of NSAIDs on hypertension risk (33,34), a condition that is responsible for 7.6 million annual deaths worldwide (5). The increased risk of cardiovascular disease is of particular concern for athletes participating in sports that encourage a large body mass and that involve violent collisions, such as American football. Footballers with a high body mass index (BMI > 28) are simultaneously more likely to use NSAIDs (39) and show elevated cardiometabolic risk factors (7,17), and as a consequence, chronic NSAID usage in these athletes may be particularly deleterious for their long-term cardiovascular health.

Finally, NSAIDs may have undesirable effects on remodeling and repair of connective and bone tissue. In one study, NSAID consumption blunted the rise in patellar collagen synthesis following a 36-km bout of running (20), suggesting that NSAID usage for chronic tendonopathies could have detrimental effects on connective tissue remodeling. In support of this hypothesis, experimental animal research demonstrates that NSAIDs delay ligament (110) and tendon (21,32) repair, as well as interfere with bone fracture healing (11). While data from humans are limited, NSAID use was associated with a 44% higher fracture risk in a population of perimenopausal women (107) and regular NSAID users had a higher risk of nonvertebral fractures than nonusers among a population of men and women in the United Kingdom (103). Notably, the magnitude of fracture risk observed varies, for unknown reasons, between different types of NSAIDs (108).

Mechanisms of Ginger Analgesia

The mechanisms behind the analgesic properties of ginger remain an active area of research, with several separate mechanisms likely responsible. Like NSAIDs, ginger blocks the activity of COX enzymes (51,93) and leukotriene and prostaglandin synthesis (46). Beyond its effects on COX enzymes, ginger is an agonizer of the transient receptor potential vanilloid 1 (91), which can be found throughout the peripheral and central nervous systems and influences pain processing (63). Ginger has also shown the capacity to inhibit the release of proinflammatory cytokines from

macrophages in vitro (98). Proinflammatory cytokines are thought to, at a minimum, play a role in exacerbating exercise-induced muscle pain (18).

Gastrointestinal Effects of Ginger

Ginger, in contrast to NSAIDs, may act as a promoter of GI function. In a study of individuals with osteoarthritis, ginger extract was as effective as diclofenac for pain management but led to less GI discomfort and increased stomach mucosa prostaglandins (29). Animal studies confirm that ginger reduces gastric lesions with exposure to gastroerosive substances (3,56), which may be due to the inhibition of gastric H⁺ and K⁺-ATPase and reduction in gastric acid secretion (85). Additionally, ginger increases stomach mucosa prostaglandins (29), whereas NSAIDs deplete them (87), and these differential effects may be due to selective COX-2 inhibition by ginger constituents (102).

Ginger has long been regarded as an antiemetic agent. A review of randomized controlled trials examining the effectiveness of ginger for pregnancy-induced nausea and vomiting concluded that ginger reduced the frequency of vomiting and intensity of nausea (28). On the contrary, a review of randomized trials examining the efficacy of ginger for chemotherapy-induced nausea and vomiting found no conclusive evidence that ginger was an effective antiemetic agent (52). A review by Palatty et al. (70) confirmed that the research to date has been contradictory and suggested that the equivocal results may be due to, among other factors, variations in the bioactive compounds of the ginger treatments.

Despite clinical trial data showing contradictory findings, mechanistic studies strongly support the biological plausibility of ginger as an antiemetic. Specifically, ginger constituents act as serotonin receptor antagonists in the GI tract, preventing the overactivation of vagal afferent nerves involved in the pathogenesis of nausea and vomiting (40,44). Serotonin release in the GI tract can cause antral contractions that contribute to symptoms of nausea (86).

Nausea is commonly experienced during exercise, particularly intense endurance running. In one survey of marathoners, approximately 20% reported experiencing nausea during self-described "hard" runs, with the prevalence being even higher among females (77). As a result, ginger represents an intriguing compound for the prevention and management of exercise-induced nausea. However, the author is not aware of any published research that has examined the effects of ginger on exercise-induced nausea.

Respiratory Effects of Ginger

Preliminary evidence indicates that ginger acts as a bronchodilator and could potentially be used as a treatment for exercise-induced bronchoconstriction. The mechanisms behind the bronchodilatory effects of ginger are not entirely clear but seem to involve action on the β -adrenergic receptor (57) and alterations of intracellular Ca²⁺ in airway smooth muscle cells (94).

Experimental research in mice (94) and rats (57) has shown that ginger reduces airway hyper-responsiveness. Moreover, isolated human airway smooth muscle cells show significant and rapid relaxation with exposure to ginger (94), even when cells are simultaneously exposed to β -agonists (95). As β -agonists are a first-line therapy for asthma, ginger could be used as an adjunctive treatment and should be evaluated as such in future research. To date, only one controlled trial has assessed ginger in asthmatic humans, which demonstrated improvements in wheezing, chest tightness, and dyspnea with no changes in spirometry after 2 months of ginger supplementation (79).

Exercise-induced bronchoconstriction, which is a transient narrowing during or after exercise, affects up to 90% of individuals with asthma and up to 50% of elite athletes (80). Unfortunately, no published research has evaluated the effects of ginger supplementation on exercise-induced bronchoconstriction. Given the strong biological plausibility of ginger as a bronchodilator, more clinical trials are called for to evaluate the safety and efficacy of ginger as a primary and adjunctive treatment for exercise-induced bronchoconstriction.

GINGER AS AN ANALGESIC AND ERGOGENIC AID FOR EXERCISE TRAINING AND SPORT

Search Methodology

A PubMed literature search was conducted in April of 2015 to identify investigations that have examined ginger supplementation in the context of exercise training and sport. The following combinations of terms were used to identify

relevant articles: “ginger exercise,” “Zingiber exercise,” “ginger soreness,” “Zingiber soreness,” “ginger sport,” and “Zingiber sport.” Only randomized controlled trials were included in the analysis. Studies examining ginger as a part of a multi-ingredient supplement were not included due to an inability to differentiate the effects of ginger from other ingredients.

The results of the literature search are presented in Figure 1. In total, 6 articles (7 studies) examining the analgesic properties of ginger for exercise training and sport were identified (12–14,58,60,116). Eight articles (9 studies) examining the effects of ginger as an ergogenic aid were identified (6,12–14,58,60,116,118). The details of these investigations are subsequently outlined.

Ginger as an Analgesic for Exercise Training and Sport

Table 1 provides an overview of the 7 studies that have evaluated the analgesic properties of ginger for exercise training and sport. Black and O'Connor (13) were the first to evaluate the analgesic properties of ginger in the context of exercise-induced pain. Using a crossover design, 25 untrained college-age individuals ingested 2 g of ground ginger or placebo 30 minutes before cycling for 30 minutes at 60% $\dot{V}O_{2peak}$, while ratings of quadriceps pain were collected every 5 minutes. Although quadriceps pain ratings showed small increases from the start to end of exercise in both conditions, no significant differences in pain were found between conditions.

An additional study from Black and O'Connor (14) evaluated the effects of 2 g of ginger or placebo on pain stemming from eccentric elbow flexor exercises. Untrained

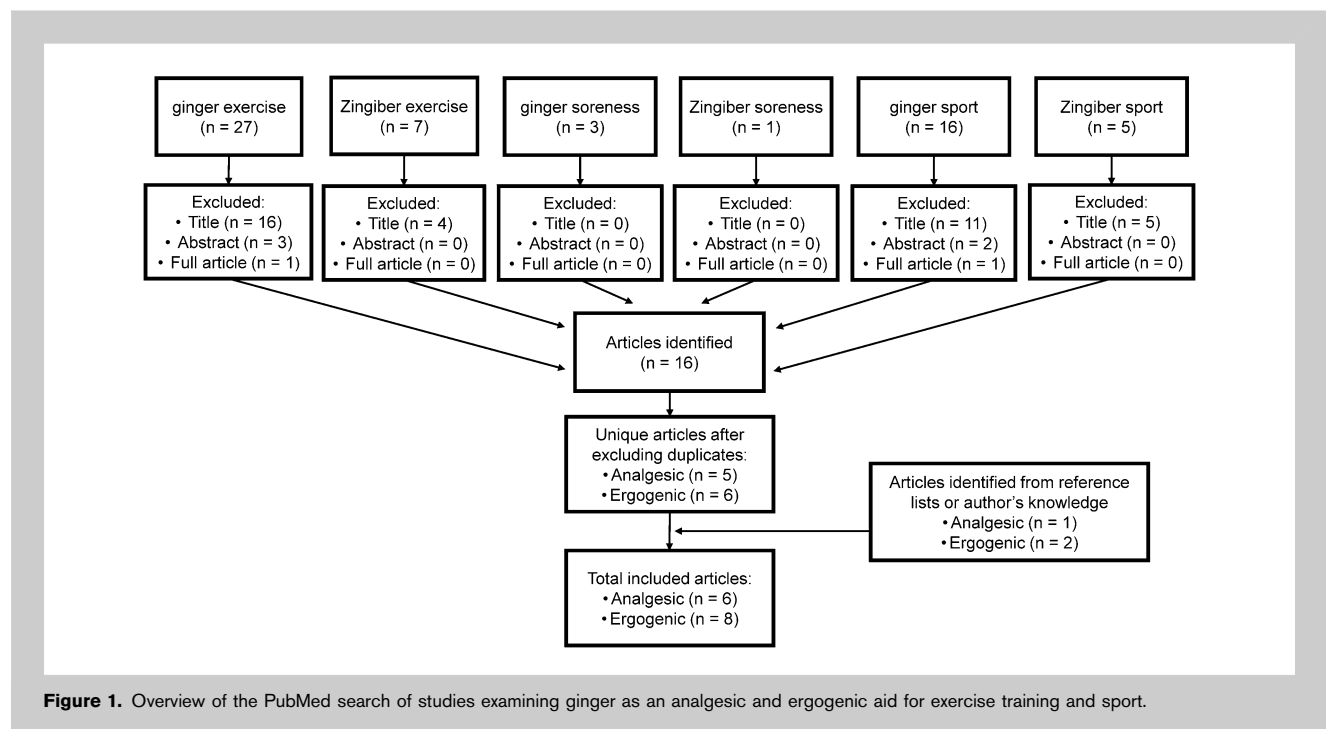


Figure 1. Overview of the PubMed search of studies examining ginger as an analgesic and ergogenic aid for exercise training and sport.

TABLE 1. Studies examining the analgesic properties of ginger for exercise training and sport (in chronological order).*

Study	Participants	Design	Blinding	Dosages	Outcomes	Significant findings
Black and O'Connor (13)	Untrained college-age men ($n = 10$) and women ($n = 15$); age 23.2 ± 4.2 y	Randomized crossover of 30-min cycling at 60% $\dot{V}O_{2peak}$	Double-blind; success not reported	2 g ground ginger or placebo (flour) 30 min before exercise	Quadriceps pain on a 0–10 scale every 5 min	No differences in pain ratings between conditions
Black and O'Connor (14)	Untrained men ($n = 12$; age 24.0 ± 4.9 y) and women ($n = 15$; age 21.8 ± 2.7 y)	Randomized crossover, with half of participants consuming ginger 24 h after elbow flexor exercises, while half consumed placebo. Participants consuming ginger at 24 h consumed placebo at 48 h and vice versa for placebo	Double-blind; success not reported	2 g dried ginger extract or placebo (flour) 24 and 48 h after exercise	Biceps pain on a 100-mm VAS during elbow flexor exercise at 50% 1RM; collected 24 and 48 h after exercise	Among those receiving ginger at 24 h, pain was reduced 13% relative to those receiving placebo at 24 h (Cohen's $d = 0.48$, $p = 0.21$)
Black et al. (12) study #1	Ginger: untrained men ($n = 3$) and women ($n = 14$); age 21.1 ± 0.7 y Placebo: untrained men ($n = 3$) and women ($n = 14$); age 20.9 ± 0.6 y	Randomized parallel trial, with participants consuming ginger or placebo for 7 d before, the day of, and 3 d after elbow flexor exercises	Double-blind; ginger group reported being 54% certain of their treatment, while placebo group was 37% certain	2 g·d ⁻¹ ground raw ginger or placebo (cornflower)	Biceps pain on a 100-mm VAS during elbow flexor exercise at 50% 1RM; collected before, 24, 48, and 72 h after exercise	Pain 24 h post-protocol was lower by 25% for ginger relative to placebo ($p = 0.041$)
Black et al. (12) study #2	Ginger: untrained men ($n = 7$) and women ($n = 13$); age 20.6 ± 0.6 y Placebo: untrained men ($n = 7$) and women ($n = 13$); age 21.4 ± 0.8 y	Randomized parallel trial, with participants consuming ginger or placebo for 7 d before, the day of, and 3 d after elbow flexor exercises	Double-blind; ginger group reported being 41% certain of their treatment, while placebo group was 47% certain	2 g·d ⁻¹ heat-treated ground ginger or placebo (brown sugar)	Biceps pain on a 100-mm VAS during elbow flexor exercise at 50% 1RM; collected before, 24, 48, and 72 h after exercise	Pain 24 h post-protocol was lower by 23% for ginger relative to placebo ($p = 0.049$)

Mashhadi et al. (58)	60 female (13–25 y) taekwondo athletes, with 49 completing the study. Exact sample size for each group not provided	Randomized parallel trial, with participants consuming ginger, cinnamon, or placebo for 6 wk	Double-blind; success not reported	3 g·d ⁻¹ ginger powder, cinnamon powder, or placebo (not specified)	General muscle soreness on a 7-point Likert scale before and after intervention	Soreness after 6 wk was lower with ginger vs. placebo (0.67 vs. 1.57, <i>p</i> < 0.01) No intention-to-treat analysis Number of dropouts for each group was not provided
Matsumura et al. (60)	Ginger: untrained men (<i>n</i> = 5) and women (<i>n</i> = 5); age 32 ± 9 y Placebo: untrained men (<i>n</i> = 5) and women (<i>n</i> = 5); age 27 ± 5 y	Randomized parallel trial, with participants consuming ginger or placebo for 5 d before elbow flexor exercises	Double-blind; success not reported	4 g·d ⁻¹ ginger powder or placebo (dextrose)	Biceps soreness on a 100-mm VAS collected before, 24, 48, 72, and 96 h after exercise	No treatment × time interactions were observed, indicating no effect of ginger on soreness
Wilson et al. (116)	Ginger: trained college men (<i>n</i> = 3) and women (<i>n</i> = 5); age 21 y Placebo: trained college men (<i>n</i> = 4) and women (<i>n</i> = 8); age 21 y	Randomized parallel trial, with participants consuming ginger or placebo for 3 d before, the day of, and day after a 20–22 mile training run	Double-blind; 5 correctly guessed ginger, while 4 correctly guessed placebo	2.2 g·d ⁻¹ ginger root or placebo (rice flour)	Soreness on a 100-mm VAS collected 4 d before and 24 h after run	Median soreness during jogging at 24 h after run was lower with ginger root than placebo (37 vs. 62 mm, <i>p</i> = 0.04)

*RM = repetition maximum; VAS = visual analog scale.

participants consumed dried ginger extract or placebo capsules 24 and 48 hours after eccentric exercises and rated muscle pain roughly 30 minutes later. The study used a cross-over design, such that half of the participants consumed ginger at 24 hours, while the other half consumed placebo at 24 hours. Subsequently, participants who consumed ginger at 24 hours consumed placebo at 48 hours, and vice versa for placebo. Overall, ginger had no effect on muscle pain relative to placebo. Among participants who received ginger at 24 hours, arm pain was reduced by 13% relative to participants who received placebo at 24 hours, although this difference was not statistically significant (Cohen's $d = 0.48$, $p = 0.21$).

Although the aforementioned studies found no clear benefit of acute ginger supplementation on exercise-induced muscle pain, additional studies from Black et al. (12) found that 11 days of ginger supplementation ($2 \text{ g} \cdot \text{d}^{-1}$) reduced pain stemming from eccentric actions of the elbow flexors. To induce muscle soreness, untrained participants completed 3 sets of 6 repetitions of elbow flexor exercises using a weight equal to 120% of concentric 1 repetition maximum (1RM). The article presented the results of 2 separate studies, one of which compared ground raw ginger with placebo while the other compared heat-treated ginger with placebo. In both studies, participants consumed ginger or placebo capsules for 7 days before, the day of, and for 3 days after the elbow flexor protocol. Raw ginger reduced muscle soreness by 25% ($p = 0.041$) relative to placebo, while heat-treated ginger reduced soreness by 23% ($p = 0.049$) relative to placebo. The duration of supplementation was the major difference between these and previous investigations, suggesting that the analgesic properties of ginger may require several days to take full effect.

The longest study identified was conducted over a period of 6 weeks (58). Sixty Iranian female taekwondo athletes were randomized in a double-blind fashion to $3 \text{ g} \cdot \text{d}^{-1}$ of ginger powder, cinnamon powder, or placebo for 6 weeks, and levels of interleukin 6 (IL-6) and muscle soreness (7-point Likert scale) were collected. A total of 49 athletes completed the 6-week intervention, and muscle soreness after 6 weeks was lower with ginger in comparison with placebo (0.67 vs. 1.57, $p < 0.01$). However, no intention-to-treat analysis was performed, and the number of dropouts for each group was not provided, casting doubt on the findings.

More recently, Matsumura et al. (60) examined the effects of 5 days of ginger powder supplementation ($4 \text{ g} \cdot \text{d}^{-1}$) on soreness resulting from eccentric elbow flexor exercises. Unlike the study from Black et al. (12) that also examined multiple days of ginger ingestion, supplementation was limited to the 5 days before exercise and was not continued during the postexercise evaluation period. No treatment \times time interactions were observed for biceps soreness on a 100-mm visual analog scale, indicating no significant effect of ginger supplementation.

Finally, Wilson et al. (116) tested whether 5 days of $2.2 \text{ g} \cdot \text{d}^{-1}$ ginger root reduced muscle soreness among college students undertaking a 20–22 mile training run. Muscle soreness was assessed at rest and during dynamic movements such as jogging. Although soreness at rest on a 100-mm visual analog scale was not significantly reduced with ginger root, median soreness during jogging 24 hours after the training run was lower with ginger root compared with placebo (37 vs. 62 mm; $p = 0.04$). Limitations to this investigation include the relatively small sample size ($n = 20$) and slight variations in distance covered during the training run between the ginger root and placebo groups.

On the whole, $2 \text{ g} \cdot \text{d}^{-1}$ of ginger taken for a week before and 3 days after eccentric resistance exercise may modestly reduce muscle pain. Likewise, ginger taken for 3 days before and the day after a prolonged run may modestly reduce pain during jogging. However, a single dosage of 2 g of ginger does not substantially reduce muscle pain stemming from resistance or cardiorespiratory exercise. No conclusions can be drawn regarding the prolonged use of ginger (>2 weeks) for pain management during chronic exercise training.

Ginger as an Ergogenic Aid for Exercise Training and Sport

Table 2 provides an overview of the 9 studies that have evaluated ginger as an ergogenic aid for exercise training and sport. Black and O'Connor (13), in addition to assessing muscle pain during 30 minutes of moderate-intensity cycling, measured perceived exertion, heart rate, and oxygen uptake 30 minutes after 2 g of ground ginger ingestion. No significant differences were noted between any of the measures, indicating that acute ginger ingestion does not likely affect cardiorespiratory function during brief moderate-intensity exercise. The other study from Black and O'Connor (14) evaluated the effects of ginger on range of motion, arm volume, and metabolic rate after eccentric actions of the elbow flexors. A single 2 g dose of dried ginger extract had no significant effect on any of the outcomes.

The aforementioned 11-day study from Black et al. (12) assessed changes in isometric force and perceived exertion with $2 \text{ g} \cdot \text{d}^{-1}$ ginger supplementation. As noted previously, the article presented the results of 2 separate studies, one of which compared raw ground ginger with placebo while the other compared heat-treated ginger with placebo. Isometric force before and after the exercise protocol was assessed at 90 degrees of elbow flexion by having participants grasp a wooden bar connected to a force transducer. Range of motion, arm volume, and prostaglandin E_2 levels were also assessed, as was rating of perceived exertion during a series of 3 concentric/eccentric actions of the elbow flexors using a weight equal to 50% of concentric 1RM. No significant group \times time interactions were found in either study for elbow range of motion, isometric force, or prostaglandin E_2 levels. Although not statistically significant, effect sizes for perceived exertion at 24 and 48 hours post-protocol were

TABLE 2. Studies examining the ergogenic properties of ginger for exercise training and sport (in chronological order).*

Article	Participants	Design	Blinding	Dosages	Outcomes	Significant findings
Black and O'Connor (13)	College-age men ($n = 10$) and women ($n = 15$); age 23.2 ± 4.2 y	Randomized crossover of 30 min of cycling at 60% $\dot{V}O_{2peak}$	Double-blind; success not reported	2 g ground ginger or placebo (flour) 30 min before exercise	Work rate, O_2 use, heart rate, and RPE every 5 min	No differences in any outcomes between conditions
Black and O'Connor (14)	Untrained men ($n = 12$; age 24.0 ± 4.9 y) and women ($n = 15$; 21.8 ± 2.7 y)	Randomized crossover; with half of participants consuming ginger 24 h after elbow flexor exercises, while half consumed placebo. Participants consuming ginger at 24 h consumed placebo at 48 h and vice versa for placebo	Double-blind; success not reported	2 g dried ginger extract or placebo (flour) 24 and 48 h after exercise	ROM and arm volume before, 24 and 48 h after exercise; metabolic rate 24 and 48 h after exercise	Pre-to-post changes were not significantly different between conditions for arm volume or ROM No significant group \times time interaction for metabolic rate
Black et al. (12) study #1	Ginger: untrained men ($n = 3$) and women ($n = 14$); age 21.1 ± 0.7 y Placebo: untrained men ($n = 3$) and women ($n = 14$); age 20.9 ± 0.6 y	Randomized parallel trial, with participants consuming ginger or placebo for 7 d before, the day of, and 3 d after elbow flexor exercises	Double-blind; ginger group reported being 54% certain of their treatment, while placebo group was 37% certain	2 g \cdot d ⁻¹ ground raw ginger or placebo (cornflower)	ROM, arm volume, PGE ₂ levels, isometric force, and RPE before, 24, 48, and 72 h after exercise	No significant group \times time interactions were found for any outcomes Effect sizes for some outcomes were moderate (ROM Glass's $d = 0.6$ to 0.7 ; RPE Glass's $d = -0.5$ to -0.6) <i>(continued on next page)</i>

Black et al. (12) study #2	Ginger: untrained men ($n = 7$) and women ($n = 13$); age 20.6 ± 0.6 y	Randomized parallel trial, with participants consuming ginger or placebo for 7 d before, the day of, and 3 d after elbow flexor exercises	Double-blind; ginger group reported being 41% certain of their treatment, while placebo group was 47% certain	2 g·d ⁻¹ heat-treated ground ginger or placebo (brown sugar)	ROM, arm volume, PGE ₂ levels, isometric force, and RPE before, 24, 48, and 72 h after exercise	No significant group × time interactions were found for any outcomes
	Placebo: untrained men ($n = 7$) and women ($n = 13$); age 21.4 ± 0.8 y					Effect sizes for outcomes were small (Glass's $d \leq 0.3$), except RPE at 24 h (Glass's $d = -0.57$)
Atashak et al. (6)	Ginger: obese men ($n = 8$; age 23.7 ± 3.4 y)	Randomized factorial design, with participants allocated to 1 of 4 groups (placebo, ginger, placebo + resistance training, or ginger + resistance training) for 10 wk	Double-blind; success not reported	1 g·d ⁻¹ ginger root powder or placebo (maltodextrin)	FFM, FM, and CRP	FFM increased and FM decreased in both resistance training groups
	Ginger + resistance: obese men ($n = 8$; age 23.7 ± 4.4 y)					Ginger reduced CRP by 21% vs. baseline
	Placebo: obese men ($n = 8$; age 25.4 ± 2.2 y)					No significant changes in CRP were observed for placebo-only
	Placebo + resistance: obese men ($n = 8$; age 23.7 ± 3.8 y)					
Mashhadi et al. (59)	60 adolescent-to-adult (13–25 y) female taekwondo athletes, with 49 completing the study. Exact sample size for each group not provided	Randomized parallel trial, with participants consuming ginger, cinnamon, or placebo for 6 or 8 wk†	Double-blind; success not reported	3 g·d ⁻¹ ginger powder, cinnamon powder, or placebo (not specified)	MDA, exercise performance, and skinfolds	Not clear in results which statistic was used for comparisons
						Authors state, “No significant changes in MDA, exercise performance, and BMI were observed between groups over time”

Zehsaz et al. (118)	28 male runners (age 23 ± 2 y) divided into 2 groups. Exact sample size for each group not provided	Randomized parallel trial, with participants consuming ginger or placebo during the last 6 wk of a 12-wk training period	Double-blind; success not reported	1.5 g · d ⁻¹ dried ginger powder or placebo (toast powder)	Inflammatory markers (TNF-α, IL-6, IL-1β) after a Bruce treadmill test	At the end of intervention, TNFα, IL-6, and IL-1β levels after the Bruce treadmill test were lower (~20 to 45%) for ginger
Matsumura et al. (60)	Ginger: untrained men (n = 5) and women (n = 5); age 32 ± 9 y	Randomized parallel trial, with participants consuming ginger or placebo for 5 d before elbow flexor exercises	Double-blind; success not reported	4 g · d ⁻¹ ginger powder or placebo (dextrose)	Biceps 1RM, ROM, arm circumference, and creatine kinase collected before, 24, 48, 72, and 96 h after exercise	Ginger improved 1RM during the 24- to 48-h period, while placebo improved between the 48- and 96-h period
	Placebo: untrained men (n = 5) and women (n = 5); age 27 ± 5 y					Creatine kinase increased over the postexercise period in ginger but not placebo
Wilson et al. (116)	Ginger: trained college men (n = 3) and women (n = 5); age 21 y	Randomized parallel trial, with participants consuming ginger or placebo for 3 d before, the day of, and day after a 20–22 mile training run	Double-blind; 5 correctly guessed ginger, while 4 correctly guessed placebo	2.2 g · d ⁻¹ ginger root or placebo (rice flour)	Squat VJ 4 d before and 24 h after run	No between-group differences in jump height, peak force, or rate of force development
	Placebo: trained college men (n = 4) and women (n = 8); age 21 y					

*RPE = rating of perceived exertion; ROM = range of motion; PGE₂ = prostaglandin E₂; FFM = fat-free mass; FM = fat mass; CRP = C-reactive protein; MDA = malondialdehyde; BMI = body mass index; TNF = tumor necrosis factor; IL = interleukin; 1RM = 1 repetition maximum; VJ = vertical jump.
 †Trial duration was reported as 6 weeks at several points in the article but as 8 weeks at other points.

modest-to-moderate in size (Glass's delta = -0.35 to -0.59), eluding to a possible benefit of ginger.

The observation from Black et al. (12) that ginger reduced muscle pain but had no effect on muscle function raises the possibility that ginger may differentially affect the pathways involved in these 2 types of outcomes. In support of this possibility, research with other nutritional compounds has shown reductions in muscle soreness without parallel improvements in muscle function (16,41). COX inhibition, a primary pathway of ginger, may reduce muscle soreness but have little effect of muscle function (71), providing a mechanistic rationale for why ginger may differentially affect muscle soreness and function outcomes.

Atashak et al. (6) used a randomized factorial design to study the effects of 10 weeks of $1 \text{ g} \cdot \text{d}^{-1}$ of ginger root powder alone or in combination with resistance training on body composition changes. Thirty-two obese men were randomized to 1 of 4 groups (placebo, ginger, placebo + resistance training, or ginger + resistance training), and body composition was assessed using skinfold measurements. Both resistance training groups showed decreases in fat mass and increases in lean mass, with no effect of ginger supplementation. However, the study was statistically underpowered to detect small-to-moderate effects given the small sample size ($n = 8$ group) and the lack of sensitivity of skinfolds.

Mashhadi et al. (59) assessed the effects of $3 \text{ g} \cdot \text{d}^{-1}$ of ginger powder in comparison with cinnamon or placebo on exercise performance, body composition, and oxidative stress in a sample of Iranian female taekwondo athletes. Forty-nine of 60 athletes completed the intervention, which curiously, was reported to last 6 weeks at several points in the article but reported as 8 weeks at other points. Additionally, the exercise performance test was not clearly outlined in the methods nor were the units of measurement in the results. Furthermore, it was not clear in the results which type of statistic was used for each comparison, making any meaningful interpretation of the findings difficult.

In a similar fashion, Zehsaz et al. (118) examined ginger supplementation in trained male runners over a multiweek training period. Twenty-eight runners were randomized to $1.5 \text{ g} \cdot \text{d}^{-1}$ of dried ginger powder or placebo, which was consumed during the last 6 weeks of a 12-week training period. Of note, specific sample sizes for each group were not provided. Inflammatory markers, tumor necrosis factor (TNF)- α , IL-6, IL-1 β , were measured at rest and after a Bruce treadmill test to establish whether ginger induced anti-inflammatory properties in the context of endurance training. At the end of the 6-week supplementation period, levels of TNF- α , IL-6, and IL-1 β after the Bruce treadmill test were lower by roughly 20–45% in the ginger group relative to placebo. No differences between the 2 groups were observed for resting inflammatory marker levels. It should be noted that some research suggests that blocking oxidative stress and inflammatory pathways may, in certain situations, blunt the functional adaptations to exercise (72),

and because the authors did not include any data comparing functional adaptations between the groups, strong conclusions cannot be made as to whether the reduced inflammatory response should be viewed favorably.

The aforementioned study from Matsumura et al. (60) evaluated changes in biceps strength, range of motion, arm circumference, and creatine kinase levels with 5 days of ginger powder supplementation ($4 \text{ g} \cdot \text{d}^{-1}$). The ginger group showed a recovery of elbow flexor 1RM strength over the 24- to 48-hour postexercise recovery period, whereas a significant improvement was not observed in the placebo group until 48- to 96-hour postexercise (60). These effects resulted in a significant time \times treatment interaction, suggesting that ginger accelerated the recovery of 1RM strength. Despite the accelerated recovery of strength, no clear benefits for range of motion or arm circumference were observed with ginger supplementation. Interestingly, creatine kinase increased over the postexercise period in the ginger group but not placebo. The authors speculated that ginger supplementation could have improved muscular function, allowing participants to perform more work during the eccentric elbow flexor exercise protocol. However, the authors did not provide direct data to support this suggestion.

Finally, Wilson et al. (116) assessed whether 5 days of $2.2 \text{ g} \cdot \text{d}^{-1}$ ginger root maintained muscle function to a better degree than placebo after a 20–22 mile training run. A squat jump from a force plate was used to evaluate muscle function before and after the training run, and overall, no differences in jump height, peak force, or rate of force development were detected between the ginger root and placebo groups. These results showing a reduction in muscle soreness without any substantial benefit for muscle function support the results from the study by Black et al. (12).

Overall, the available research indicates no clear ergogenic benefit of ginger on oxygen use, heart rate, metabolic rate, body composition, isometric force generation, or perceived exertion. A single investigation suggests relatively high-dose ginger supplementation ($4 \text{ g} \cdot \text{d}^{-1}$) may accelerate recovery of upper body strength after eccentric resistance exercise. In addition, chronic ginger supplementation may reduce the inflammatory response to cardiorespiratory exercise. More research is needed to determine whether ginger supplementation affects other performance outcomes such as the functional adaptations to chronic resistance and cardiorespiratory training.

ADVERSE EFFECTS OF GINGER

Ginger is generally recognized as safe by the Food and Drug Administration (101). Despite this designation, clinical trials with ginger reveal the occurrence of several types of adverse events, albeit nearly all mild in nature. Heartburn has been observed in a number of studies (4,19,62,75,114,115) and has been reported as a cause of study withdrawal (114,115). The frequency of heartburn varies with study duration and dosage of ginger but has ranged from 3 to 27% (4,62). Other

adverse effects of ginger on the GI tract include exfoliation of gastric epithelial cells at high doses (6 g), indicating that ginger has the potential to act as a GI irritant when taken in large amounts (27).

Warnings regarding the potential for ginger to increase the risk of bleeding and to interact with anticoagulant medications are present in the literature (64,82). Suggestions that ginger may increase bleeding time are primarily based on *in vitro* research showing that ginger inhibits thromboxane synthesis and platelet aggregation (69,88). Ginger supplementation trials in humans, however, have been equivocal; several studies have found no significant effects on thromboxane production (42,54) or bleeding time (54), with others showing significant decreases in platelet aggregation (15,106) and thromboxane production (89).

It is yet to be determined whether ginger, like NSAIDs, influences hyponatremia risk and connective and bone tissue remodeling. Human studies examining the effects of ginger on renal physiology and function are needed to establish whether ginger is a potentially nephrotoxic agent that should be avoided during prolonged endurance exercise. Likewise, additional research is needed to determine whether ginger supplementation modifies the connective and bone tissue remodeling process that normally occurs with weight-bearing exercise.

Finally, the effects of ginger on muscle protein synthesis and adaptations to endurance and resistance training require examination. It has been hypothesized that COX enzymes regulate muscle protein synthesis and that COX inhibition may blunt the rise in muscle protein synthesis that normally accompanies exercise (112). Given ginger's inhibition of COX enzymes, it is possible that chronic ginger consumption may modify the functional and morphological responses to resistance and endurance training.

FUTURE RESEARCH NEEDS

A common limitation of the human research on ginger is a lack of reporting and/or standardization of bioactive compounds in treatments (70,91). Ginger contains many bioactive compounds, such as gingerols and shogaols, which are known to have varying effects on physiological pathways, including COX enzymes (1,102). Variations in the concentrations of these bioactive compounds make between-study comparisons and clinical application challenging. Additionally, the myriad of ways ginger can be prepared (e.g., powder, oil, extract, ground, heat-treated) further complicates between-study comparisons.

Reporting of adverse events among studies examining ginger as an exercise- and sport-specific supplement has been absent (6,13,14,58–60,118) or lacking in detail (12,116). Failure to systematically collect data on adverse events makes it difficult to properly evaluate the benefit-to-risk ratio of ginger. Guidelines for the reporting of adverse events are outlined in the Consolidated Standards of Reporting Trials (CONSORT) statement (65). Among other recommendations, CONSORT

advises that adverse events should be listed and defined, the methods used to collect adverse events should be described, and the absolute risk of each adverse event and number of withdrawals should be reported (65).

The pungent flavor and aroma of ginger are substantial obstacles to designing placebo-controlled double-blind trials. This may be particularly problematic for studies supplementing ginger before prolonged endurance exercise, given the relatively frequent incidence of reflux experienced during endurance exercise and with ginger supplementation. Although all of the articles identified from this review's PubMed search reported double-blinding, only 2 collected information on the success of blinding (12,116). While the utility of formally assessing the success of blinding has been questioned (81), ascertaining reasons why a participant believes they were consuming ginger or placebo (e.g., ginger smell or taste) may provide insight as to whether the sensory characteristics of the treatment and placebo were well-matched. Interestingly, ginger may be easier to blind if it is administered in blister packs as opposed to bottles, due to the odor arising from the storage of numerous ginger capsules in a single container (104,119).

Notably, several of the studies included in this review recruited untrained participants (12–14,60), including one that found ginger reduced soreness (12) and another that showed ginger accelerated recovery of strength (60). While it is common for muscle damage studies to use untrained individuals (presumably because it is easier to induce muscle soreness), this practice limits generalizability of the findings. Consequently, studies including trained individuals will be needed to further elucidate the analgesic properties of ginger in athletic populations.

PRACTICAL APPLICATIONS

The evidence indicates that 2 g·d⁻¹ ginger may modestly reduce muscle pain stemming from eccentric resistance exercise, particularly in untrained individuals and if taken for 1–2 weeks. Likewise, 5 days of ginger supplementation (2 g·d⁻¹) may modestly reduce soreness stemming from prolonged running, whereas 5 days of high-dose supplementation (4 g·d⁻¹) may accelerate recovery of muscular strength after eccentric resistance training. On the contrary, a single dose of ginger has little-to-no discernable effects on muscle pain, metabolic rate, oxygen use, isometric force generation, or perceived exertion. In addition, chronic ginger consumption does not seem to modify body composition responses to resistance training. However, chronic ginger supplementation may reduce the inflammatory response to exercise, although the implications of these effects are unknown.

Despite shortcomings in the research, tentative and circumstantial evidence suggests that ginger may offer a favorable combination of analgesic, GI, and respiratory effects, and thus, it seems reasonable to conduct additional research evaluating ginger as an analgesic and ergogenic aid for exercise training and sport. Future research should directly compare the analgesic properties and side effects

of ginger with NSAIDs in athletes and individuals undergoing exercise training. These future trials should include trained individuals and pay particular attention to blinding effectiveness and reporting of adverse events, as these details have been lacking in the research to date.

ACKNOWLEDGMENTS

The author discloses no conflicts of interest. The entirety of this article was conceived and drafted by P. B. Wilson.

REFERENCES

1. Ali, BH, Blunden, G, Tanira, MO, and Nemmar, A. Some phytochemical, pharmacological and toxicological properties of ginger (*Zingiber officinale* Roscoe): A review of recent research. *Food Chem Toxicol* 46: 409–420, 2008.
2. Almond, CS, Shin, AY, Fortescue, EB, Mannix, RC, Wypij, D, Binstadt, BA, Duncan, CN, Olson, DP, Salerno, AE, Newburger, JW, and Greenes, DS. Hyponatremia among runners in the Boston Marathon. *N Engl J Med* 352: 1550–1556, 2005.
3. Anosike, CA, Obidoa, O, Ezeanyika, LU, and Nwuba, MM. Anti-inflammatory and anti-ulcerogenic activity of the ethanol extract of ginger (*Zingiber officinale*). *Afri J Biochem Res* 3: 379–384, 2009.
4. Arfeen, Z, Owen, H, Plummer, JL, Ilesley, AH, Sorby-Adams, AC, and Doecke, CJ. A double-blind randomised controlled trial of ginger for the prevention of postoperative nausea and vomiting. *Anaesth Intens Care* 23: 449–452, 1995.
5. Arima, H, Barzi, F, and Chalmers, J. Mortality patterns in hypertension. *J Hypertens* 29: S3–S7, 2011.
6. Atashak, S, Peeri, M, Azarbayjani, MA, Stannard, SR, and Haghighi, MM. Obesity-related cardiovascular risk factors after long-term resistance training and ginger supplementation. *J Sports Sci Med* 10: 685–691, 2011.
7. Baron, SL, Hein, MJ, Lehman, E, and Gersic, CM. Body mass index, playing position, race, and the cardiovascular mortality of retired professional football players. *Am J Cardiol* 109: 889–896, 2012.
8. Bartels, EM, Folmer, VN, Bliddal, H, Altman, RD, Juhl, C, Tarp, S, Zhang, W, and Christensen, R. Efficacy and safety of ginger in osteoarthritis patients: A meta-analysis of randomized placebo-controlled trials. *Osteoarthritis Cartilage* 23: 13–21, 2015.
9. BCC Research. Sports nutrition and high energy supplements: The global market. Available at: <http://www.bccresearch.com/market-research/food-and-beverage/sports-nutrition-energy-supplements-fod043a.html>. Accessed January 10, 2015.
10. Becker, ML, Kallewaard, M, Caspers, PW, Visser, LE, Leufkens, HG, and Stricker, BH. Hospitalisations and emergency department visits due to drug–drug interactions: A literature review. *Pharmacoevidenciol Drug Saf* 16: 641–651, 2007.
11. Bergenstock, M, Min, W, Simon, AM, Sabatino, C, and O'Connor, JP. A comparison between the effects of acetaminophen and celecoxib on bone fracture healing in rats. *J Orthop Trauma* 19: 717–723, 2005.
12. Black, CD, Herring, MP, Hurley, DJ, and O'Connor, PJ. Ginger (*Zingiber officinale*) reduces muscle pain caused by eccentric exercise. *J Pain* 11: 894–903, 2010.
13. Black, CD and O'Connor, PJ. Acute effects of dietary ginger on quadriceps muscle pain during moderate-intensity cycling exercise. *Int J Sport Nutr Exerc Metab* 18: 653–664, 2008.
14. Black, CD and O'Connor, PJ. Acute effects of dietary ginger on muscle pain induced by eccentric exercise. *Phytother Res* 24: 1620–1626, 2010.
15. Bordia, A, Verma, SK, and Srivastava, KC. Effect of ginger (*Zingiber officinale* Rosc.) and fenugreek (*Trigonella foenumgraecum* L.) on blood lipids, blood sugar and platelet aggregation in patients with coronary artery disease. *Prostaglandins Leukot Essent Fatty Acids* 56: 379–384, 1997.
16. Bryer, SC and Goldfarb, AH. Effect of high dose vitamin C supplementation on muscle soreness, damage, function, and oxidative stress to eccentric exercise. *Int J Sport Nutr Exerc Metab* 16: 270–280, 2006.
17. Buell, JL, Calland, D, Hanks, F, Johnston, B, Pester, B, Sweeney, R, and Thorne, R. Presence of metabolic syndrome in football linemen. *J Athl Train* 43: 608–616, 2008.
18. Cheung, K, Hume, PA, and Maxwell, L. Delayed onset muscle soreness. *Sports Med* 33: 145–164, 2003.
19. Chittumma, P, Kaewkiattikun, K, and Wiriyasiriwach, B. Comparison of the effectiveness of ginger and vitamin B6 for treatment of nausea and vomiting in early pregnancy: A randomized double-blind controlled trial. *J Med Assoc Thai* 90: 15–20, 2007.
20. Christensen, B, Dandanell, S, Kjaer, M, and Langberg, H. Effect of anti-inflammatory medication on the running-induced rise in patella tendon collagen synthesis in humans. *J Appl Physiol* (1985) 110: 137–141, 2011.
21. Cohen, DB, Kawamura, S, Ehteshami, JR, and Rodeo, SA. Indomethacin and celecoxib impair rotator cuff tendon-to-bone healing. *Am J Sports Med* 34: 362–369, 2006.
22. Corrigan, B and Kazlauskas, R. Medication use in athletes selected for doping control at the Sydney Olympics (2000). *Clin J Sport Med* 13: 33–40, 2003.
23. Darvishzadeh-Mahani, F, Esmaeili-Mahani, S, Komeili, G, Sheibani, V, and Zare, L. Ginger (*Zingiber officinale* Roscoe) prevents the development of morphine analgesic tolerance and physical dependence in rats. *J Ethnopharmacol* 141: 901–907, 2012.
24. Da Silva, ER, De Rose, EH, Ribeiro, JP, Sampedro, LB, Devos, DV, Ferreira, AO, and Krueel, LF. Non-steroidal anti-inflammatory use in the XV Pan-American Games (2007). *Br J Sports Med* 45: 91–94, 2011.
25. Davis, DP, Videen, JS, Marino, A, Vilke, GM, Dunford, JV, Van Camp, SP, and Maharam, LG. Exercise-associated hyponatremia in marathon runners: A two-year experience. *J Emerg Med* 21: 47–57, 2001.
26. Demoly, P, Crampette, L, Lebel, B, Campbell, AM, Mondain, M, and Bousquet, J. Expression of cyclo-oxygenases 1 and 2 proteins in upper respiratory mucosa. *Clin Exp Allergy* 28: 278–283, 1998.
27. Desai, HG, Kalro, RH, and Choksi, AP. Effect of ginger & garlic on DNA content of gastric aspirate. *Indian J Med Res* 92: 139–141, 1990.
28. Ding, M, Leach, M, and Bradley, H. The effectiveness and safety of ginger for pregnancy-induced nausea and vomiting: A systematic review. *Women Birth* 26: e26–e30, 2013.
29. Drozdov, VN, Kim, VA, Tkachenko, EV, and Varvanina, GG. Influence of a specific ginger combination on gastropathy conditions in patients with osteoarthritis of the knee or hip. *J Altern Complement Med* 18: 583–588, 2012.
30. El-Ghorab, AH, Nauman, M, Anjum, FM, Hussain, S, and Nadeem, M. A comparative study on chemical composition and antioxidant activity of ginger (*Zingiber officinale*) and cumin (*Cuminum cyminum*). *J Agric Food Chem* 58: 8231–8237, 2010.
31. Ernst, E and Pittler, MH. Efficacy of ginger for nausea and vomiting: A systematic review of randomized clinical trials. *Br J Anaesth* 84: 367–371, 2000.
32. Ferry, ST, Dahners, LE, Afshari, HM, and Weinholt, PS. The effects of common anti-inflammatory drugs on the healing rat patellar tendon. *Am J Sports Med* 35: 1326–1333, 2007.
33. Forman, JP, Rimm, EB, and Curhan, GC. Frequency of analgesic use and risk of hypertension among men. *Arch Intern Med* 167: 394–399, 2007.
34. Forman, JP, Stampfer, MJ, and Curhan, GC. Non-narcotic analgesic dose and risk of incident hypertension in US women. *Hypertension* 46: 500–507, 2005.

35. Gorski, T, Cadore, EL, Pinto, SS, da Silva, EM, Correa, CS, Beltrami, FG, and Kreul, LF. Use of NSAIDs in triathletes: Prevalence, level of awareness and reasons for use. *Br J Sports Med* 45: 85–90, 2011.
36. Grzanna, R, Lindmark, L, and Frondoza, CG. Ginger-an herbal medicinal product with broad anti-inflammatory actions. *J Med Food* 8: 125–132, 2005.
37. Harrington, LS, Lucas, R, McMaster, SK, Moreno, L, Scadding, G, Warner, TD, and Mitchell, JA. COX-1, and not COX-2 activity, regulates airway function: Relevance to aspirin-sensitive asthma. *EASEB J* 22: 4005–4010, 2008.
38. Hew, TD, Chorley, JN, Cianca, JC, and Divine, JG. The incidence, risk factors, and clinical manifestations of hyponatremia in marathon runners. *Clin J Sport Med* 13: 41–47, 2003.
39. Holmes, N, Cronholm, PF, Duffy, AJ III, and Webner, D. Nonsteroidal anti-inflammatory drug use in collegiate football players. *Clin J Sport Med* 23: 283–286, 2013.
40. Huang, QR, Iwamoto, M, Aoki, S, Tanaka, N, Tajima, K, Yamahara, J, Takaishi, Y, Yoshida, M, Tomimatsu, T, and Tamai, Y. Anti-5-hydroxytryptamine₃ effect of galanolactone, diterpenoid isolated from ginger. *Chem Pharm Bull* 39: 397–399, 1991.
41. Jackman, SR, Witard, OC, Jeukendrup, AE, and Tipton, KD. Branched-chain amino acid ingestion can ameliorate soreness from eccentric exercise. *Med Sci Sports Exerc* 42: 962–970, 2010.
42. Janssen, PL, Meyboom, S, van Staveren, WA, de Vegt, F, and Katan, MB. Consumption of ginger (*Zingiber officinale* roscoe) does not affect ex vivo platelet thromboxane production in humans. *Eur J Clin Nutr* 50: 772–774, 1996.
43. Jenkins, C, Costello, J, and Hodge, L. Systematic review of prevalence of aspirin induced asthma and its implications for clinical practice. *BMJ* 328: 434, 2004.
44. Jin, Z, Lee, G, Kim, S, Park, CS, Park, YS, and Jin, YH. Ginger and its pungent constituents non-competitively inhibit serotonin currents on visceral afferent neurons. *Korean J Physiol Pharmacol* 18: 149–153, 2014.
45. Joslin, J, Lloyd, JB, Kotlyar, T, and Wojcik, SM. NSAID and other analgesic use by endurance runners during training, competition and recovery. *S Afr J Sports Med* 25: 101–104, 2013.
46. Kiuchi, F, Iwakami, S, Shibuya, M, Hanaoka, F, and Sandaw, U. Inhibition of prostaglandin and leukotriene biosynthesis by gingerols and diarylheptanoids. *Chem Pharm Bull* 40: 387–391, 1992.
47. Krentz, JR, Quest, B, Farthing, JP, Quest, DW, and Chilibeck, PD. The effects of ibuprofen on muscle hypertrophy, strength, and soreness during resistance training. *Appl Physiol Nutr Metab* 33: 470–475, 2008.
48. Küster, M, Renner, B, Oppel, P, Niederweis, U, and Brune, K. Consumption of analgesics before a marathon and the incidence of cardiovascular, gastrointestinal and renal problems: A cohort study. *BMJ Open* 3: e002090, 2013.
49. Lambert, GP. Role of gastrointestinal permeability in exertional heatstroke. *Exerc Sport Sci Rev* 32: 185–190, 2004.
50. Lambert, GP, Boylan, M, Laventure, JP, Bull, A, and Lanspa, S. Effect of aspirin and ibuprofen on GI permeability during exercise. *Int J Sports Med* 28: 722–726, 2007.
51. Lantz, RC, Chen, GJ, Sarihan, M, Sólyom, AM, Jolad, SD, and Timmermann, BN. The effect of extracts from ginger rhizome on inflammatory mediator production. *Phytomedicine* 14: 123–128, 2007.
52. Lee, J and Oh, H. Ginger as an antiemetic modality for chemotherapy-induced nausea and vomiting: A systematic review and meta-analysis. *Oncol Nurs Forum* 40: 163–170, 2013.
53. Liamis, G, Milionis, H, and Elisaf, M. A review of drug-induced hyponatremia. *Am J Kidney Dis* 52: 144–153, 2008.
54. Lumb, AB. Effect of dried ginger on human platelet function. *Thromb Haemost* 71: 110–111, 1994.
55. Mackey, AL. Does an NSAID a day keep satellite cells at bay? *J Appl Physiol* (1985) 115: 900–908, 2013.
56. Mahmood, AA, Philip, K, and Salmah, I. Anti ulcerogenic effect of the rhizomes of *Zingiber officinale* against ethanol induced gastric ulcers in rats. *J Anim Vet Adv* 5: 122–125, 2006.
57. Mangprayool, T, Kupittayanant, S, and Chudapongse, N. Participation of citral in the bronchodilatory effect of ginger oil and possible mechanism of action. *Fitoterapia* 89: 68–73, 2013.
58. Mashhadi, NS, Ghiasvand, R, Askari, G, Feizi, A, Hariri, M, Darvishi, L, Barani, A, Taghiyar, M, Shiranian, A, and Hajishafiee, M. Influence of ginger and cinnamon intake on inflammation and muscle soreness endured by exercise in Iranian female athletes. *Int J Prev Med* 4: S11–S15, 2013.
59. Mashhadi, NS, Ghiasvand, R, Hariri, M, Askari, G, Feizi, A, Darvishi, L, Hajishafiee, M, and Barani, A. Effect of ginger and cinnamon intake on oxidative stress and exercise performance and body composition in Iranian female athletes. *Int J Prev Med* 4: S31–S35, 2013.
60. Matsumura, MD, Zavorsky, GS, and Smoliga, JM. The effects of pre-exercise ginger supplementation on muscle damage and delayed onset muscle soreness. *Phytother Res* 29: 887–93, 2015.
61. McGettigan, P and Henry, D. Cardiovascular risk with non-steroidal anti-inflammatory drugs: Systematic review of population-based controlled observational studies. *PLoS Med* 8: e1001098, 2011.
62. Meyer, K, Schwartz, J, Crater, D, and Keyes, B. *Zingiber officinale* (ginger) used to prevent 8-Mop associated nausea. *Dermatol Nurs* 7: 242–244, 1997.
63. Mezey, É, Tóth, ZE, Cortright, DN, Arzubi, MK, Krause, JE, Elde, R, Guo, A, Blumberg, PM, and Szallasi, A. Distribution of mRNA for vanilloid receptor subtype 1 (VR1), and VR1-like immunoreactivity, in the central nervous system of the rat and human. *Proc Natl Acad Sci U S A* 97: 3655–3660, 2000.
64. Miller, LG. Herbal medicinals: Selected clinical considerations focusing on known or potential drug-herb interactions. *Arch Intern Med* 158: 2200–2211, 1998.
65. Moher, D, Hopewell, S, Schulz, KF, Montori, V, Gøtzsche, PC, Devereaux, PJ, Elbourne, D, Egger, M, and Altman, DG. CONSORT 2010 explanation and elaboration: Updated guidelines for reporting parallel group randomised trials. *J Clin Epidemiol* 63: e1–e37, 2010.
66. Mountain, SJ, Sawka, MN, and Wenger, CB. Hyponatremia associated with exercise: Risk factors and pathogenesis. *Exerc Sport Sci Rev* 29: 113–117, 2001.
67. Morton, AR and Fitch, KD. Australian Association for Exercise and Sports Science position statement on exercise and asthma. *J Sci Med Sport* 14: 312–316, 2011.
68. Nieman, DC, Henson, DA, Dumke, CL, Oley, K, McAnulty, SR, Davis, JM, Murphy, EA, Utter, AC, Lind, RH, McAnulty, LS, and Morrow, JD. Ibuprofen use, endotoxemia, inflammation, and plasma cytokines during ultramarathon competition. *Brain Behav Immun* 20: 578–584, 2006.
69. Nurtjahja-Tjendraputra, E, Ammit, AJ, Roufogalis, BD, Tran, VH, and Duke, CC. Effective anti-platelet and COX-1 enzyme inhibitors from pungent constituents of ginger. *Thromb Res* 111: 259–265, 2003.
70. Palatty, PL, Haniadka, R, Valder, B, Arora, R, and Baliga, MS. Ginger in the prevention of nausea and vomiting: A review. *Crit Rev Food Sci Nutr* 53: 659–669, 2013.
71. Paulsen, G, Egner, IM, Drange, M, Langberg, H, Benestad, HB, Fjeld, JG, Hallén, J, and Raastad, TA. COX-2 inhibitor reduces muscle soreness, but does not influence recovery and adaptation after eccentric exercise. *Scand J Med Sci Sports* 20: e195–e207, 2010.
72. Peternej, TT and Coombes, JS. Antioxidant supplementation during exercise training. *Sports Med* 41: 1043–1069, 2011.

73. Picado, C. Aspirin-intolerant asthma: Role of cyclo-oxygenase enzymes. *Allergy* 57: 58–60, 2002.
74. Price, OJ, Hull, JH, Backer, V, Hostrup, M, and Ansley, L. The impact of exercise-induced bronchoconstriction on athletic performance: A systematic review. *Sports Med* 44: 1749–1761, 2014.
75. Rahnama, P, Montazeri, A, Huseini, HF, Kianbakht, S, and Naseri, M. Effect of *Zingiber officinale* R. rhizomes (ginger) on pain relief in primary dysmenorrhea: A placebo randomized trial. *BMC Complement Altern Med* 12: 92, 2012.
76. Reid, SA, Speedy, DB, Thompson, JM, Noakes, TD, Mulligan, G, Page, T, Campbell, RG, and Milne, C. Study of hematological and biochemical parameters in runners completing a standard marathon. *Clin J Sport Med* 14: 344–353, 2004.
77. Riddoch, C and Trinick, T. Gastrointestinal disturbances in marathon runners. *Br J Sports Med* 22: 71–74, 1988.
78. Robinson, MM, Hamilton, KL, and Miller, BF. The interactions of some commonly consumed drugs with mitochondrial adaptations to exercise. *J Appl Physiol* (1985) 107: 8–16, 2009.
79. Rouhi, H, Ganji, F, and Nasri, H. Effects of ginger on the improvement of asthma [the evaluation of its treatment effects]. *Pakistan J Nutr* 5: 373–376, 2006.
80. Rundell, KW and Slee, JB. Exercise and other indirect challenges to demonstrate asthma or exercise-induced bronchoconstriction in athletes. *J Allergy Clin Immunol* 122: 238–246, 2008.
81. Sackett, DL. Commentary: Measuring the success of blinding in RCTs: Don't, must, can't or needn't? *Int J Epidemiol* 36: 664–665, 2007.
82. Saw, JT, Bahari, MB, Ang, HH, and Lim, YH. Potential drug-herb interaction with antiplatelet/anticoagulant drugs. *Complement Ther Clin Pract* 12: 236–241, 2006.
83. Sepahvand, R, Esmaeili-Mahani, S, Arzi, A, Rasouljan, B, and Abbasnejad, M. Ginger (*Zingiber officinale* Roscoe) elicits antinociceptive properties and potentiates morphine-induced analgesia in the rat radiant heat tail-flick test. *J Med Food* 13: 1397–1401, 2010.
84. Shukla, Y and Singh, M. Cancer preventive properties of ginger: A brief review. *Food Chem Toxicol* 45: 683–690, 2007.
85. Siddaraju, MN and Dharmesh, SM. Inhibition of gastric H⁺, K⁺-ATPase and *Helicobacter pylori* growth by phenolic antioxidants of *Zingiber officinale*. *Mol Nutr Food Res* 51: 324–332, 2007.
86. Smith, HS, Cox, LR, and Smith, EJ. 5-HT₃ receptor antagonists for the treatment of nausea/vomiting. *Ann Palliat Med* 1: 115–120, 2012.
87. Sostres, C, Gargallo, CJ, Arroyo, MT, and Lanás, A. Adverse effects of non-steroidal anti-inflammatory drugs (NSAIDs, aspirin and coxibs) on upper gastrointestinal tract. *Best Pract Res Clin Gastroenterol* 24: 121–132, 2010.
88. Srivastava, KC. Effects of aqueous extracts of onion, garlic and ginger on platelet aggregation and metabolism of arachidonic acid in the blood vascular system: In vitro study. *Prostaglandins Leukot Med* 13: 227–235, 1984.
89. Srivastava, KC. Effect of onion and ginger consumption on platelet thromboxane production in humans. *Prostaglandins Leukot Essent Fatty Acids* 35: 183–185, 1989.
90. Stevenson, DD. Diagnosis, prevention, and treatment of adverse reactions to aspirin and nonsteroidal anti-inflammatory drugs. *J Allergy Clin Immunol* 74: 617–622, 1984.
91. Terry, R, Posadzki, P, Watson, LK, and Ernst, E. The use of ginger (*Zingiber officinale*) for the treatment of pain: A systematic review of clinical trials. *Pain Med* 12: 1808–1818, 2011.
92. Timbo, BB, Ross, MP, McCarthy, PV, and Lin, CT. Dietary supplements in a national survey: Prevalence of use and reports of adverse events. *J Am Diet Assoc* 106: 1966–1974, 2006.
93. Tjendraputra, E, Tran, VH, Liu-Brennan, D, Roufogalis, BD, and Duke, CC. Effect of ginger constituents and synthetic analogues on cyclooxygenase-2 enzyme in intact cells. *Bioorg Chem* 29: 156–163, 2001.
94. Townsend, EA, Siviski, ME, Zhang, Y, Xu, C, Hoonjan, B, and Emala, CW. Effects of ginger and its constituents on airway smooth muscle relaxation and calcium regulation. *Am J Respir Cell Mol Biol* 48: 157–163, 2013.
95. Townsend, EA, Zhang, Y, Xu, C, Wakita, R, and Emala, CW. Active components of ginger potentiate β -agonist-induced relaxation of airway smooth muscle by modulating cytoskeletal regulatory proteins. *Am J Respir Cell Mol Biol* 50: 115–124, 2014.
96. Trappe, TA, Carroll, CC, Dickinson, JM, LeMoine, JK, Haus, JM, Sullivan, BE, Lee, JD, Jemiolo, B, Weinheimer, EM, and Hollon, CJ. Influence of acetaminophen and ibuprofen on skeletal muscle adaptations to resistance exercise in older adults. *Am J Physiol Regul Integr Comp Physiol* 300: R655–R662, 2011.
97. Trappe, TA, White, F, Lambert, CP, Cesar, D, Hellerstein, M, and Evans, WJ. Effect of ibuprofen and acetaminophen on postexercise muscle protein synthesis. *Am J Physiol Endocrinol Metab* 282: E551–E556, 2002.
98. Tripathi, S, Bruch, D, and Kittur, DS. Ginger extract inhibits LPS induced macrophage activation and function. *BMC Complement Altern Med* 8: 1, 2008.
99. Tscholl, P, Feddermann, N, Junge, A, and Dvorak, J. The use and abuse of painkillers in international soccer: Data from 6 FIFA tournaments for female and youth players. *Am J Sports Med* 37: 260–265, 2009.
100. Tscholl, P, Junge, A, and Dvorak, J. The use of medication and nutritional supplements during FIFA World Cups 2002 and 2006. *Br J Sports Med* 42: 725–730, 2008.
101. U.S. Food and Drug Administration. *Code of Federal Regulations Title 21*. Available at: <http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfcfr/CFRSearch.cfm?fr=182.20>. Accessed January 3, 2015.
102. van Breemen, RB, Tao, Y, and Li, W. Cyclooxygenase-2 inhibitors in ginger (*Zingiber officinale*). *Fitoterapia* 82: 38–43, 2011.
103. van Staa, TP, Leufkens, HG, and Cooper, C. Use of nonsteroidal anti-inflammatory drugs and risk of fractures. *Bone* 27: 563–568, 2000.
104. van Tilburg, MA. Efficacious blinding of ginger for randomized controlled trials. *Alt Med Stud* 2: e1, 2012.
105. Van Wijck, K, Lenaerts, K, van Bijnen, AA, Boonen, B, van Loon, LJ, Dejong, CH, and Buurman, WA. Aggravation of exercise-induced intestinal injury by ibuprofen in athletes. *Med Sci Sports Exerc* 44: 2257–2262, 2012.
106. Verma, SK, Singh, J, Khamesra, R, and Bordia, A. Effect of ginger on platelet aggregation in man. *Indian J Med Res* 98: 240–242, 1993.
107. Vestergaard, P, Hermann, P, Jensen, JE, Eiken, P, and Mosekilde, L. Effects of paracetamol, non-steroidal anti-inflammatory drugs, acetylsalicylic acid, and opioids on bone mineral density and risk of fracture: Results of the Danish Osteoporosis Prevention Study (DOPS). *Osteoporos Int* 23: 1255–1265, 2012.
108. Vestergaard, P, Rejnmark, L, and Mosekilde, L. Fracture risk associated with use of nonsteroidal anti-inflammatory drugs, acetylsalicylic acid, and acetaminophen and the effects of rheumatoid arthritis and osteoarthritis. *Calcif Tissue Int* 79: 84–94, 2006.
109. Warden, SJ. Prophylactic use of NSAIDs by athletes: A risk/benefit assessment. *Phys Sportsmed* 38: 132–138, 2010.
110. Warden, SJ, Avin, KG, Beck, EM, DeWolf, ME, Hagemeyer, MA, and Martin, KM. Low-intensity pulsed ultrasound accelerates and a nonsteroidal anti-inflammatory drug delays knee ligament healing. *Am J Sports Med* 34: 1094–1102, 2006.
111. Warner, DC, Schnepf, G, Barrett, MS, Dian, D, and Swigonski, NL. Prevalence, attitudes, and behaviors related to the use of nonsteroidal anti-inflammatory drugs (NSAIDs) in student athletes. *J Adolesc Health* 30: 150–153, 2002.
112. Weinheimer, EM, Jemiolo, B, Carroll, CC, Harber, MP, Haus, JM, Burd, NA, LeMoine, JK, Trappe, SW, and Trappe, TA. Resistance exercise and cyclooxygenase (COX) expression in human skeletal muscle: Implications for COX-inhibiting drugs and protein synthesis. *Am J Physiol Regul Integr Comp Physiol* 292: R2241–R2248, 2007.

113. Wharam, PC, Speedy, DB, Noakes, TD, Thompson, JM, Reid, SA, and Holtzhausen, LM. NSAID use increases the risk of developing hyponatremia during an Ironman triathlon. *Med Sci Sports Exerc* 38: 618–622, 2006.
114. Wigler, I, Grotto, I, Caspi, D, and Yaron, M. The effects of Zintona EC (a ginger extract) on symptomatic gonarthrosis. *Osteoarthritis Cartilage* 11: 783–789, 2003.
115. Willetts, KE, Ekangaki, A, and Eden, JA. Effect of a ginger extract on pregnancy-induced nausea: A randomised controlled trial. *Aust N Z J Obstet Gynaecol* 43: 139–144, 2003.
116. Wilson, PB, Fitzgerald, JS, Rhodes, GS, Lundstrom, CJ, and Ingraham, SJ. Effectiveness of ginger root (*Zingiber officinale*) on running-induced muscle soreness and function: A pilot study. *Int J Ath Ther Train* 2015. In press.
117. Wolf, DA, Miller, TW, Pescatello, LS, and Barnes, C. National collegiate athletic association division I athletes' use of nonprescription medication. *Sports Health* 3: 25–28, 2011.
118. Zehsaz, F, Farhangi, N, and Mirheidari, L. The effect of *Zingiber officinale* R. rhizomes (ginger) on plasma pro-inflammatory cytokine levels in well-trained male endurance runners. *Cent Euro J Immunol* 39: 174–180, 2014.
119. Zick, SM, Blume, A, Normolle, D, and Ruffin, M. Challenges in herbal research: A randomized clinical trial to assess blinding with ginger. *Complement Ther Med* 13: 101–106, 2005.