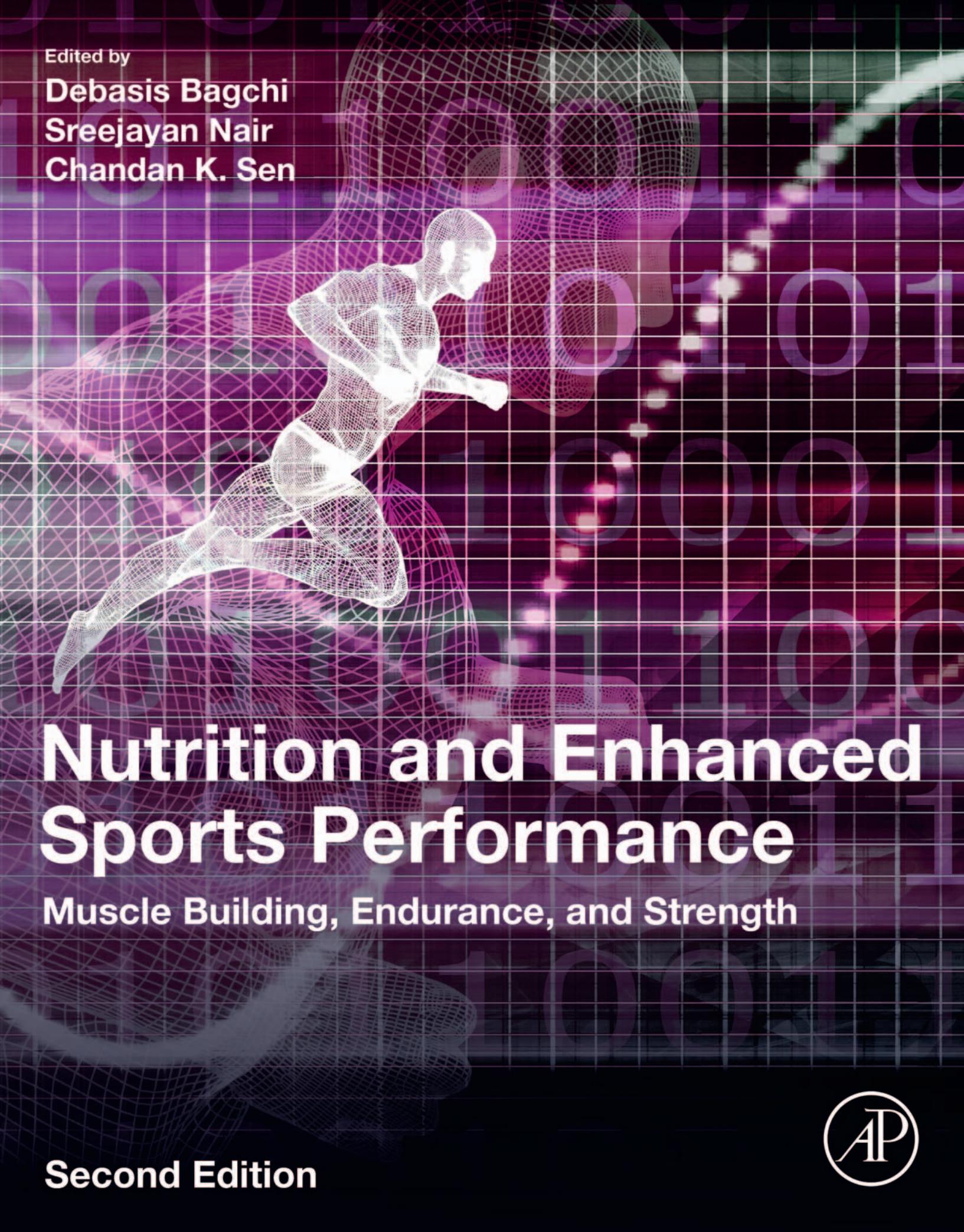
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Nutrition and Enhanced Sports Performance
Muscle Building, Endurance, and Strength
Second Edition

NUTRITION AND ENHANCED SPORTS PERFORMANCE

SECOND EDITION

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NUTRITION AND ENHANCED SPORTS PERFORMANCE

MUSCLE BUILDING, ENDURANCE, AND STRENGTH

SECOND EDITION

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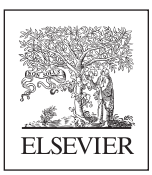

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Dedicated to my beloved ex-colleague-cum-dada, Mr. K.K. Biswas,
a dignified man of ethics, commitment, and inspiration.
My sincere regards and gratitude to KK-da.

Debasis Bagchi

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Preface

Outstanding interest of our audience has prompted the publisher to issue the second edition within 3 years of our initial launch. Enthused by your support we come back with renewed vigor to serve our colleagues in sports nutrition, exercise physiology and related health professionals and especially students. The first edition of this book had 55 chapters contributed by reputed scientists around the world. The second edition presents many of the same authors and adds new experts to cover emergent areas of broad interest. This updated second edition includes 66 cutting-edge chapters aiming to bring our readers to the forefront of nutrition and health in sports. This volume is divided into six thematic sections. The introductory section presents a general overview of the role of nutrition in human health. The second section is based on types of physical exercises. The third section addresses nutritional requirements in the context of sporting events. Molecular mechanisms implicated in muscle building constitute the theme of the fourth section. Section five addresses food products, minerals, supplements, phytochemicals, testosterone, amino acids, transition metals, small molecules, and other ergogenic agents that have been implicated in muscle building and human performance. The final section highlights the importance and significance of healthy cooking, physical training, lifestyle, and dietary recommendations.

Craving for a stronger and more able body has been inherent to humans from the dawn of civilization. *Ramayana (c.7500 BC)*, *Mahabharata (c.1000 BC)*, the Great Sphinx of Giza, the Stela of King Amenhotep II, and the ancient Olympic events all feature athletic activities as highlights. Archery, chariot racing, rowing, horse riding and racing, wrestling, swimming, and bull and lion fighting tested the limits of human strength, skills, and competitive psyche. Ancient paintings and sculptures narrate stories of valor and strength often depicting victorious muscular men. As a symbol of zeal and physical fitness of ancient Egyptians, murals exhibit Zoser actively engaged in the running program of Heb Sed festival (5000–3000 BC). Artists clearly represented the harmonious movements of muscles and exhibited the muscular movement and dimensions of Zoser's arms, trunk, and legs. Especially the most thrilling piece is the mural of the Zoser that displays the Pharaoh participating in the running program. On the wall of her sanctuary of the Karnak Temple, an ancient Egyptian temple located on the east bank of the Nile River, the renowned painting of 18th dynasty Queen Hatshepsut has been represented in a similar mindset of Heb Sed festival. In ancient India the practice of sports dates back to the pre-Vedic Ramayana/Mahabharata and Vedic era. The Vedic era (1200–500 BC) was a period in history during which the Vedas, the oldest scriptures of Hinduism, were composed. Martial arts such as *karate* and *kung fu* are rooted in India. In the 5th century a Buddhist monk from India named Bodhidharma (the Chinese called him Po-ti-tama) introduced Kalari into China and Japan in the 5th century. The temple in which he taught his art is now known as the Shaolin temple. The *Rigveda* (1200–900 BC) places emphasis on proper food and diet for good health. In 1889 Ralph T.H. Griffith published in London his translation as the Hymns of the Rigveda. In praise of food, Griffith translated the hymn "*In thee, O Food, is set the spirit of great Gods. Under thy flag brave deeds were done*." The Vedas placed equal emphasis on eating right as well as on fasting or caloric restriction.

In the history of Olympics, alcohol was commonly taken as ergogenic aids through the early 1900s. Wrestlers participating in the Olympic Games during 516 BC used to consume 20 pounds of meat, 20 pounds of bread, and 8.5 L of wine a day. The Olympic gladiators' meal plan inspired the discipline of sports nutrition. The Roman gladiator meal was rich in protein from different sources, cereals, and vegetables. Carbohydrate loading was achieved by eating fermented bread made of farro (a Roman cereal) and a soup made of farro and orzo. Barley was commonly regarded as a source of strength and stamina. Roasted meat, dry fruits, fresh cheese, goat milk, and eggs were the primary sources of protein. Onions, garlic, wild lettuce, and dill dominated among the vegetables. In particular, onions had a special place in ancient Greek sports nutrition as it would "lighten the balance of the blood." After Rome conquered Greece, onion became a staple in the Roman diet. Roman gladiators were rubbed down with onion juice to "firm up the muscles." Olive oil was used frequently with meals. Fried cakes, boiled meat, and cold drinks were prohibited. A great snack for energy in the Roman gladiator's diet was goat milk with honey and walnuts. At the public banquet *Coena Libera*, or the last dinner before the fight, athletes "stuffed themselves" so much that the dinner lasted long hours. They were advised to chew their food well to extract the maximum energy from it! Much of what was practiced in those days can be justified by today's science! Compared with the average inhabitant of Ephesus, gladiators

ate more plants and very little animal protein consistent with the advocacy for meat-limited diet in the Vedas. Those were the days when nutritional choices were driven by desirable health outcomes and not by commercial interests.

As the global sports nutrition industry rapidly approaches the multibillion dollar mark, most products seem to be driven more by marketing strategies than by rigorous research and development. Sports nutrition supplements (powders, pills, and "hardcore" bodybuilding ready-to-drink products), nutrition bars and gels, sports and energy drinks, and shots currently flood the market. Most solutions seem to be better marketed than developed through rigorous scientific research and clinical trials. Picking the most fitting solutions from an overcrowded marketplace represents a serious challenge and will depend on scientific awareness of the consumer. At a time when marketing budget seems to far outweigh the budget for research and development of most companies, it is our responsibility to verify the validity of claims made in advertisements. Of highest priority is to minimize potential risk to health in response to long-term use of any product. Next, potential benefits need to be critically evaluated. Both safety and efficacy can be only established through well-controlled studies. Original research articles in established high-impact peer-reviewed journals represent a reliable source of information. Look for registered clinical trial data in clinicaltrials.gov and do not be misled by attractively delivered marketing gimmicks not supported by peer-reviewed research publications. We are pleased to have been able to put together this digest aimed at providing general guidance to the physically active. Each chapter is supported with reference to the source article which readers may want to consult in developing their own opinion about a nutritional solution. We hope the readers enjoy this volume as much as we have enjoyed putting it together!

The editors extend their sincere thanks and gratitude to all the eminent contributors and especially to Billie-Jean Fernandez, Megan R Ball, and Nancy Maragioglio for their continued support, cooperation, and valuable suggestions.

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SECTION 1

NUTRITION AND HUMAN HEALTH

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CHAPTER

1

Nutritional Supplementation in Health and Sports Performance

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INTRODUCTION

The nutritional status of an athlete is a major determinant of health, fitness, and sports performance. Nutrition plays a central role in adaptation, rehydration, refueling, and repair as well as recovery from injury [1–7]. As a consequence, for optimal performance, it is essential that athletes be in the best possible nutritional and metabolically balanced state. It is important that athletes focus on nutrition basics such as vitamins and minerals, and not simply on carbohydrates, proteins, and fats as well as ergogenic dietary supplements. Vitamins and minerals constitute integral components of the coenzymes and cofactors that are responsible for energy production and the anabolic systems associated with tissue building and repair. As a consequence, optimal intake of basic nutrients is essential for optimal performance. One should not make the assumption that adequate calorie intake is associated with optimal intake of many other basic nutrients.

Athletes as well as the general population may be overfed and still be deficient in a wide range of essential nutrients, including vitamin A, vitamin B6, vitamin B12, vitamin C, vitamin D, vitamin E, vitamin K, folic acid, iodine, iron, zinc, calcium, magnesium, and selenium [7–14]. These nutritional deficiencies can be extrapolated to athletes, with some indications that the incidence of some deficiencies may be higher among athletes than the general population. Examples of nutritional deficiencies that have been specifically reported among athletes include iron [1,15], magnesium [13–16], sodium [1,5,17], zinc [1], calcium, vitamin D [18,19], vitamin C, vitamin E, and vitamin A [20]. As will be discussed in the following, much attention has been focused lately on the vitamin D status in athletes.

The primary reason for nutritional imbalance is consumption of refined foods and dietary supplements that are high in calories from sugars, starches, and fats and low in vitamins, minerals, trace elements, and fibers as the result of the refining and manufacturing processes. The net effect is that athletes may consume high levels of calories in conjunction with inadequate amounts of essential nutrients.

More than 90% of athletes and those engaged in regular exercise consume nutritional and dietary supplements on a daily basis [2]. Approximately two-thirds of the adult population consume dietary supplements daily, with women consuming supplements more regularly than men [7,21]. Other studies indicate that 72% of cardiologists, 59% of dermatologists, 91% of orthopedists and physicians, and 82% of nurses recommended dietary supplements to their patients, and also almost 70% of physicians and 89% of nurses at least occasionally used dietary supplements themselves [22,23]. Thus oral ingestion of nutrients in the form of dietary supplements is accepted by a large percent of athletes, the general population, and a wide range of health-care providers.

With respect to the regulation of dietary supplements, it should be kept in mind that dietary supplements are not regulated as drugs but rather are extensively regulated as a special category of foods. Many misconceptions exist regarding the regulation of dietary supplements.

In actuality, the FDA has greater substantive authority over dietary supplements than conventional foods [24]. Athletes, health-care providers, and the general public should be well informed regarding how dietary supplements are regulated [25] and what constitutes a dietary supplement.

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DEFINITIONS

The US Congress defined the term dietary supplement with the passage of the Dietary Supplement Health and Education Act (DSHEA) of 1994 [24]. Therefore a dietary supplement is defined as a product taken by mouth which contains a "dietary ingredient" intended to supplement the diet. Furthermore, dietary ingredients may include vitamins, minerals, amino acids, herbs, or other botanical products and substances such as enzymes, glandulars, organ tissues, and metabolites.

Dietary supplements may also be extracts or concentrates and can occur in forms such as capsules, tablets, powders, liquids, gelcaps, softgels, or bars. They must be labeled as dietary supplements because by law, they are a special category of "foods" and not drugs. The definition of dietary supplements used in the USA differs from the definition used in Europe where the term refers to vitamins and minerals, and herbal products are regulated separately as herbal medicines or herbal remedies [25,26].

Based on the DSHEA, structure/function claims can be made for dietary supplements which describe the role of a dietary ingredient or nutrient that is intended to affect normal structure or function in humans. Several examples of structure/function claims include statements such as "calcium builds strong bones," "fiber maintains bowel integrity," "omega-3 fatty acids support heart health," and "chromium helps maintain blood glucose levels in the normal range." When a dietary supplement includes a structure/function claim, it must state that the FDA has not evaluated the claim and must further affirm that the product is not intended to "diagnose, treat, cure, or prevent any disease" because legally only a drug can make these claims.

All nutrition facts panels on labels contain daily values (DVs) that represent the recommended daily intake (RDI) of each nutrient that is considered to be adequate to meet the requirements of 97%–98% of normal, healthy individuals in all demographics in the United States, based on 2000 kcal/day. RDIs are a reflection of the older recommended dietary allowances (RDAs) that were calculated based on estimated average requirements (EARs) or the amount of a nutrient believed to satisfy the needs of 50% of the people in a demographic. RDA values are usually about 20% higher than EARs. Finally, a system of nutrition recommendations entitled the dietary reference intake (DRI) values was introduced by the Institute of Medicine of the US National Academy of Sciences in 1997 to broaden the RDAs. DRIs have not been widely adopted.

Unfortunately, these multiple systems of recommended essential nutrient intake add much confusion and questionable clarity to the widely asked question "how much of each nutrient is needed for optimal physical performance and health?" In reality, these systems are based largely on the smallest amount of a nutrient needed to prevent a deficiency or disease state and do not reflect the amount of each nutrient required to provide optimal health and peak physical performance. Furthermore, they project the minimal needs of healthy individuals, with little or no allowance for stressful situations such as intense exercise or disease.

The widely held misconception that only 100% of the DV amount of each essential nutrient is required for good health clearly is not true. Supplementation with a multivitamin/mineral product containing 100% of the DVs may decrease the prevalence of suboptimal levels of some nutrients in athletes, but it will not provide optimal nutritional requirements. Furthermore, providing 100% of the DV for vitamins and minerals does not enhance the levels of various markers of antiinflammatory activity, antioxidant capacity, or immune response [9,27].

The RDA, also popularly known as the Recommended Daily Allowance, is the estimated amount of a nutrient (or calories) per day considered necessary for the maintenance of good health by the Food and Nutrition Board of the National Research Council/National Academy of Sciences.

An adequate intake (AI) is a recommended intake value based on observed or experimentally determined approximations of intake by a group or groups of healthy people which are assumed to be adequate when a recommended daily amount cannot be determined.

NUTRITIONAL SUPPLEMENT RECOMMENDATIONS FOR ATHLETES

Many factors are involved in determining how much of the various essential nutrients are required by an individual to meet daily needs and support optimal sports performance. These factors include age, weight, gender, stress levels, physical condition, daily physical activity, gastrointestinal health, general health, metabolic rate, disease states, and recovery from injury or surgery. Furthermore, the type or kind of physical activity in which an athlete is engaged can determine nutritional requirements. As a consequence, it is apparent that one size (amount) does not fit all, and as previously noted, supplementing with a product that contains 100% of DVs does not adequately meet the overall needs. Metabolism can be equated to a chain that is as strong as its weakest link.

With respect to the overall consumption of dietary supplements in the United States, the most widely consumed products are vitamins and minerals (43%), followed by specialty supplements (20%), botanicals (20%), and sports supplements (16%) that are designed to promote energy, enhance muscle, promote fat loss, contribute to body sculpting, and support sports performance [28]. It should be noted that a fraction of 1% of individuals consuming dietary supplements experience adverse events with the majority being classified as minor. The primary cause of adverse events is due to the adulteration of supplements with drugs and chemicals that are not classified as supplements. Among dietary supplements, the majority of adverse effects are associated with the use of caffeine, yohimbine, and other stimulant ingredients [28].

With these considerations in mind, what should be the approximate level of daily intake of essential nutritional supplements to facilitate optimal performance for an athlete who may be consuming 3000–6000 kcal/day, keeping in mind that DVs are based on a 2000-kcal/day intake? The average athlete should consume dietary supplements daily which contain at least 200%–300% of the DVs for vitamins and minerals and may require 400%–600% of the DVs, depending on the intensity and duration of daily activities. For example, consuming a product with 100% of the DVs for vitamins and minerals two to six times daily or a product with 200%–300% of the DVs twice a day with meals may be appropriate.

In recent years, much research has focused on the importance of vitamin D in athletes [29–31]. In addition to its role in calcium absorption and bone health, vitamin D has been shown to play a role in many other body functions, including skeletal muscle function, immune system support, cognition, cardiovascular health, prevention of some forms of cancer, and blood sugar regulation [18,19,30,32,33].

The current DV for adults for vitamin D is 400 IU while the RDA is 600 IU (NIH). Many athletes as well as the general public do not meet this minimal requirement [18,34]. Some studies have suggested that more than 70% of the general public [29] and up to 40% of athletes are vitamin D insufficient. Furthermore, wide seasonal variations occur in vitamin D sufficiency with greatest deficiencies occurring at the end of winter because of a lack of sun exposure combined with poor dietary practices [30]. However, for optimal overall health, blood levels of at least 40 ng/mL of the active intermediate 25-hydroxyvitamin D (25-OHD) are recommended, and 2000–5000 IU of vitamin D are needed daily to achieve this level [32]. As a consequence, the periodic determination of serum levels of 25-OHD is warranted, permitting appropriate adjustments of vitamin D intake.

The role of vitamin K, particularly vitamin K2 as menaquinone-7 (MK-7), in bone and cardiovascular health is increasingly being recognized based on preclinical, epidemiological, and clinical studies published over the past decade [35]. According to the NIH, the AI for vitamin K for adult males and females is 120 and 90 mcg, respectively. Few studies have specifically examined the role of vitamin K in athletic performance. However, as expected, because of increased nutrient demands, it has been suggested that higher doses than those currently recommended are required by athletes [31].

To avoid vitamin A toxicity, vitamin A should be used in the form of beta-carotene or mixed carotenoids and not retinol or its esters retinyl palmitate and retinyl acetate. Beta-carotene is converted into vitamin A as it is needed by the body [36] and exhibits a much greater safety profile.

The consumption of vegetarian diets has increased markedly in recent years, and if vitamin B12 supplements are not being used, deficiencies will occur. The most common foods that contain vitamin B12 are meats, seafood, and dairy products. Only microbes have the enzymatic systems that can synthesize this vitamin. Vitamin B12 plays a key role in the brain and nervous systems and the formation of red blood cells. Low vitamin B12 levels are associated with anemia, lack of endurance, weakness, neurological abnormalities, acidosis, elevated homocysteine levels, decreased HDL levels, and possibly platelet aggregation [37], all of which may contribute to decrements in athletic performance.

Typical multivitamin/mineral supplements do not contain adequate amounts of calcium, magnesium, or vitamin D. Products that contain only vitamin D and calcium with no magnesium are inadequate and should be avoided. High calcium intake in the absence of magnesium inhibits the absorption of magnesium and may be one of the reasons for the high incidence of magnesium deficiency in this country and the frequency of leg cramps among athletes and the general public [38,39]. Magnesium is involved in more than 300 metabolic reactions and is necessary for normal muscle function, bone formation, nerve and muscle function, heart rhythm, immune system, calcium absorption, and blood sugar regulation [16]. As a consequence, appropriate magnesium intake is essential for athletic performance as well as for the general public. The RDAs for magnesium for adult men and women are 410 and 360 mg, respectively. As noted previously, higher intakes are required depending on the caloric consumption and on the athlete and type of training involved. Green leafy vegetables are good sources of magnesium.

Calcium and magnesium products that are available in chelated and absorbable forms such as calcium citrate, fumarate, hydroxyapatite, aspartate or other amino acid chelates [40] and magnesium citrate, ascorbate, aspartate or other amino acid chelates should be used [41,42]. Products that contain magnesium oxide are poorly absorbed and should be avoided [42].

Omega-3 fatty acids are required for normal cell and organ function and are present in every cell in the body. As the result of widespread omega-3 fatty acid deficiency, an estimated 84,000 people die prematurely each year [43]. In the USA, vegetarians exhibit particularly low intake of this critical nutrient [37]. Omega-3 fatty acids exhibit numerous beneficial functions, including protective effects associated with muscles, joints, cardiovascular system, brain and nervous system, immune system, and gastrointestinal system, as well as bones, lungs, liver, skin, eyes, hair, and other organs and tissues [44–46]. Omega-3 fatty acids are believed to counteract the inflammatory and immumodulatory effects of exercise while improving exercise performance [45,46] and preventing sports-associated injuries [47].

Docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA) are the primary omega-3 fatty acids responsible for these health effects. They are derived primarily from fish oils although they may also be obtained from krill (zooplankton), whereas DHA may be extracted from algae and EPA from yeast. α-Linolenic acid (ALA) is another omega-3 fatty acid which is derived from plant sources such as flaxseed oil, canola oil, soybean oil, nuts, and some berries. Because ALA is poorly converted into EPA and DHA, it possesses only a fraction of the health benefits of DHA and EPA [48,49] and should not be relied on as the sole source of omega-3 fatty acids. Algal oils may be an appropriate source of omega-3 fatty acids for vegetarians.

The American Heart Association recommends 400–500 mg or DHA/EPA per week (2 servings of oily fish) for general health and wellness. Athletes, however, should consider consuming 1 g of DHA/EPA three to four times daily, and endurance athletes should consider ingesting higher amounts per day based on need [50,51]. A high-quality product from a reputable manufacturer should be used. Concerns expressed regarding possible contamination of fish oils with heavy metals and pesticides are unfounded and do not present a health threat [52].

Dietary protein intake has been shown to significantly increase muscle strength, muscle size, and performance during resistance exercise training in healthy adults [53]. The development, repair, and preservation of muscles are of paramount concern for athletes. The Institute of Medicine has established 0.8 g/kg body weight as the DRI for protein for adults [54]. However, this amount of protein is too low to support and preserve muscle mass in athletes, and as a consequence, the recommended daily protein intake for strength and speed in athletes is in the range of 1.2–1.5 g/kg body weight [55], assuming normal kidney function. Protein supplementation intake at amounts greater than approximately 1.6 g/kg day does not appear to further enhance gains derived at lower doses, although higher amounts are used by some athletes [56] and higher amounts may be required to maximize muscle retention under some conditions such as low calorie intake [57].

The branched chain amino acids (BCAA) L-leucine, L-valine, and L-isoleucine have been shown to enhance athletic performance and are effective ergogenic aids [58,59]. BCAAs inhibit breakdown of skeletal muscle and promote muscle repair. Of these three amino acids, L-leucine has been shown to stimulate muscle protein synthesis and is believed to play a primary regulatory role in muscle protein metabolism [60–63].

Supplementation with a mixture of the BCAAs or with L-leucine alone constitutes effective means of promoting and preserving muscle. A daily intake of 8–12 g/day of L-leucine or 12–18 g/day of a mixture of the BCAAs may be appropriate, particularly in athletes with compromised renal function or who are not able to tolerate high levels of protein [64]. Use of beta-hydroxy beta-methyl butyrate, a metabolite of L-leucine, constitutes an alternative approach to support muscle development [65]. Ingestion of protein levels that constitute approximately 30% of the total daily caloric intake is appropriate when not involved in competition.

The conditionally essential amino acids L-arginine and L-glutamine as well as L-citrulline should be considered also for nutritional supplementation of athletes. Best results with L-arginine and L-citrulline have been observed in moderately trained individuals as opposed to highly trained ones [66].

L-Arginine plays important roles in cell division, wound healing, support of the immune system, removal of ammonia from the body, synthesis of nitric acid, blood pressure regulation, mitochondrial respiration, and platelet function [67–70]. L-Arginine is the main source of nitric oxide via nitric oxide synthase. Supplementation can be provided with 10–14 g of L-arginine per day in divided doses.

L-Citrulline constitutes part of the arginine cycle and can be considered a second source of nitric oxide because it can be converted to L-arginine [66]. It is an ergogenic because it is not subject to presystemic elimination as is L-arginine, and as a consequence, it can effectively aid in the elevation of L-arginine levels. L-Citrulline at a daily dose in the range of 20–50 mg/kg may be appropriate.

L-Glutamine plays important roles in protein repair and synthesis, wound healing, acid–base balance, immune system support, gut barrier function, and cellular differentiation and serves as an energy source [71–73]. Supplementation can be provided with 8–12 g of L-glutamine per day in divided doses.

Finally, it should be noted that rehydration of athletes with water only after intensive exercise and dehydration is inadequate for recovery and subsequent performance [17]. Furthermore, rehydration with a product that contains

primarily water, sugar, and sodium chloride is superior to water alone but does not support optimal performance. A more complex product containing water, sodium, potassium, calcium, magnesium, carbohydrates, vitamins, and selected amino acids should be consumed [17].

SAFETY ISSUES

The US Poison Control Centers has repeatedly reported few deaths associated with vitamins, minerals, herbal ingredients, or dietary supplements in general [74]. Contrary to various publications and warnings, dietary supplements exhibit a very high degree of safety. Primary issues associated with the safety of dietary supplements are due to the adulteration of supplements with drugs and chemicals that are not classified as supplements. Furthermore, if one looks at the number of deaths that can be directly attributable to dietary supplements over the past 15 years, a number of these deaths have been due to the ingestion of massive amounts of caffeine [75,76]. Most deaths associated with dietary supplements are due to inappropriate use. Putting the number of deaths into perspective, more than 25,000 deaths occur annually from FDA-approved prescription drugs according to the NIH [77]. It should be kept in mind that all substances can be toxic, including salt, sugar, and water, if taken in sufficiently large and inappropriate doses.

The scientific literature does not support the premise that taking vitamin and mineral products in amounts that exceed 100% of the daily value results in toxicity. Excess amounts of water-soluble vitamins are readily excreted, and toxicities do not occur with consumption of vitamins in amounts up to 10–20 times the DVs. Greater caution is required for most minerals and fat-soluble vitamins that in general should not be used in amounts greater than approximately six times the DV unless specific deficiencies are documented. The recommendations presented previously are designed to address optimum nutritional needs while maintaining a wide margin of safety.

SUMMARY AND CONCLUSIONS

Approximately two-thirds of the adult population of the United States and most athletes consume nutritional supplements on a daily basis. Yet, large percentages of the populace, including athletes, are deficient in multiple vitamins and minerals because of poor dietary habits and consumption of refined foods with inadequate amounts of essential nutrients. The necessity for athletes to consume adequate amounts of vitamins and minerals cannot be over emphasized.

Consuming multivitamin/mineral products that contain up to 100% of the DV is inadequate to provide blood and tissue levels needed for good nutrition, let alone appropriate nutrition necessary to support optimal performance. In general, consuming up to 300%–600% of the DV for most common vitamins and minerals may be necessary to meet the nutritional needs of athletes. Intake will depend on the overall daily caloric consumption and the rigor of the training and exertion involved in a particular physical activity.

Omega-3 fatty acids impact the functions of all organs, and deficiencies are common. To appropriately address omega-3 fatty acid needs, athletes are encouraged to consume a minimum of 3–4 g of DHA/EPA daily, whereas larger amounts may be required by endurance athletes.

High protein intake and exercise are two factors that preserve and enhance muscle. A protein intake in the range of at least 1.2–1.5 g/kg day is recommended. Where renal function is an issue or an alternative approach is necessary to preserve and enhance muscle health, high intake of L-leucine (8–12 g/day) or a combination of the BCAAs L-leucine, L-valine, and L-isoleucine (12–18 g/day) may be appropriate. These BCAAs in general and L-leucine in particular promote protein synthesis.

L-Arginine and L-glutamine are two conditionally essential amino acids that support the immune system, cell division, and wound healing. Athletes can benefit from ingesting 8–10 g of L-glutamine per day and 10–14 g of L-arginine per day, along with 1.5–3.0 g of L-citrulline per day, particularly during periods of intense exercise and training.

Finally, hydration with a complex product that contains water, multiple minerals, vitamins, carbohydrates, and selected amino acids is superior to water alone or water plus sugar and sodium chloride.

In summary, nutritional status is a significant factor in determining overall performance and endurance of athletes. The recommendations mentioned previously are designed to assist in providing optimal nutritional support for athletes, recognizing that specific needs and the timing of nutritional intake will vary depending on the rigor of the physical activity and the goals that are involved.

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CHAPTER

2

Glycemic Index, Food Exchange Values, and Exercise Performance

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GLYCEMIC INDEX

Carbohydrates (CHOs) typically constitute the greatest percentage of all three energy-providing nutrients of a diet [1]. There is increasing evidence that the type of CHO being consumed is an important factor that influences exercise performance [2] and the risk of developing obesity and chronic diseases associated with it [3].

The concept of the glycemic index (GI) was developed in 1981 by Jenkins et al. to define the type of CHO ingested by estimating the responses of blood glucose (GLU) and insulin (INS) levels after the ingestion. GI is defined as the incremental area under the blood GLU response curve of a test food that provides a fixed amount of CHO (usually a 50-g CHO portion), expressed as a percentage of the response to the same amount of CHO from a reference food (glucose or white bread) consumed by the same subject [4,5]. Most often glucose is used as the reference food, which has been defined to have a GI value of 100 [6]. Table 2.1 illustrates the categorization of GI values.

Long-term compliance with a low-GI diet may induce favorable metabolic effects [7,8], but the planning of such a diet is complicated. Foods containing simple CHOs (monosaccharides, disaccharides, oligosaccharides) have a high GI. Conversely, foods containing complex CHO (starch, fiber) usually have a low GI. However, the GI of a food cannot be predicted by its CHO content alone as it depends on factors such as pH, cooking, processing, and other food components (fiber, fat, protein) (5 FAO/WHO 1998). There are some published studies on GI values of several food items as they were tested in healthy [4,9] or diabetic individuals [10–13]. Despite efforts to systematically tabulate published and unpublished sources of reliable GI values [14,15], no standardized method is currently available to determine dietary GI in national food databases. Considering that the GI value of a food may vary significantly from place to place because of the aforementioned factors (i.e., pH, cooking, etc.), it has already been demonstrated that "the need for GI testing of local foods is critical to the practical application of GI to diets" [16]. Moreover, there are different glycemic responses among individuals after consumption of a CHO meal. This may be attributed to the presence of diabetes [17,18] and different individual characteristics, such as age, sex, body weight, and race [19]. Therefore it would be prudent for researchers to consider all factors that can alter results, leading to erroneous conclusions, especially when investigating the association of GI in diseases such as type 2 diabetes mellitus (T2DM), where glycemic and insulinemic responses vary significantly.

GLYCEMIC LOAD

Glycemic load (GL) represents a ranking system for CHO ingested and reflects the total exposure to glycemia during a 2-h period, as first proposed by Salmeron et al. [20]. It constitutes a mathematical product incorporating both the CHO content in grams per serving and the GI score of the food and is calculated by the following equation:

$$GL = \text{CHO content} \times GI/100$$

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TABLE 2.1 Methods of Assessing Glycemia After Ingestion of a Meal

Method	Value
GLYCEMIC INDEX[a]	
Low	<55
Medium	55–69
High	≥70
GLUCOSE LOAD[b]	
Low	≤10
Medium	11–19
High	≥20

[a]*Aston [6].*
[b]*Brand-Miller et al. (2003) [21]*

Similar to the GI, foods are classified as having low, medium, or high GL. Owing to the limited experience with the use of GL values, Brand-Miller and colleagues [21] suggested, as a starting point, that the preliminary cutoffs be ≤10 for a low GL, between 11 and 19 for a medium GL, and ≥20 for a high GL.

As mentioned already, the GI concept is based on the ingestion of a fixed amount of available CHO, usually 50 g [4]. However, it has been demonstrated that the amount of CHO ingested accounts for 65%–66% of the variability in glucose response, and the GI of the CHO explains a similar degree (60%) of the variance [22]. Together the amount and the GI of CHOs account for approximately 90% of the total variability in the blood glucose response. Hence considering only the GI to explain the different metabolic responses in the postprandial phase, and not the amount of the CHO ingested, could lead to an incomplete estimation of these responses. Additionally, because of the broad ranges in the amounts of CHO consumed per serving [23], the development of practical recommendations for everyday settings would be inadequate.

The GL concept overcomes the previously mentioned restrictions of the GI concept as it takes into account not only the GI but also the serving sizes [24]. Hence GL is determined by the overall glycemic effect and INS demands of the diet and not just by the amount of CHO. Consequently GL may serve as a better predictor of glycemic response and INS demand compared with GI [24]. However, the incorporation of GL to examine the postprandial glycemic or inlulinemic effects or during subsequent exercise must be used with caution, especially in populations where controlling the glycemia and/or insulinemia is a critical issue for their health. From the equation of the GL, a low-GI/high-CHO food or a high-GI/low-CHO food can have the same GL [25]. Despite the fact that the effects on postprandial glycemia may be similar, there is evidence that the two approaches will have very different influences in β-cell function [26], triglyceride (TG) concentrations [26], free fatty acid (FFA) levels [26], and effects on satiety [27]. These influences should also be considered to effectively manage and control postprandial response to CHO with the use of the GL.

GI, GL, AND METABOLIC RESPONSES

The GI is an indicator of the effect of food on postprandial blood GLU and INS levels. Consumption of high-GI foods causes a sharp, large peak in postprandial blood GLU level and subsequently a large rise in blood INS level [28] and inhibition of glucagon release [6]. In normal individuals INS induces the uptake of ingested glucose by the liver and peripheral tissues (skeletal muscle and adipose tissue) [29]. Also, INS affects metabolism in many ways: increases glycogen synthesis in the liver and skeletal muscle, decreases gluconeogenesis and glycogenolysis in the liver, increases lipogenesis, and inhibits lipolysis in adipose tissue [6,30]. Elevated INS levels due to consumption of high-GI foods result in continuous uptake of nutrients and their suppressed mobilization from tissues, often leading to hypoglycemia. On the other hand, consumption of a low-GI food causes slower and smaller glycemic and insulinemic responses [6].

All these metabolic changes attributable to the GI value of foods lead to the suggestion that low-GI diets reduce the risk of metabolic diseases. Low dietary GI may improve health by contributing to decrease in body weight [3–33] and/or favorable changes in lipid profiles [34,35], whereas high dietary GI may have the opposite effect [7,36].

Some of the health benefits gained by avoiding high-GI diets include the prevention and control of obesity and chronic diseases associated to it [3], such as T2DM [37].

Similarly to GI, the GL of a diet is proposed to have an effect on metabolic responses in the postprandial state, and these responses have been associated with several chronic diseases and with altered metabolic responses during exercise.

GI, GL, and Chronic Disease Risk

Despite knowledge of the immediate metabolic benefits from compliance with a low-GI diet, a long-term metabolic effect of such diet has yet to be established [38]. The relationship of GI or GL to the risk for developing a chronic disease has been assessed through prospective cohort studies or metaanalyses, although the outcomes are inconsistent (Table 2.2). Several studies indicate that both GI and GL are associated with a risk of developing a chronic disease [39–41], whereas other studies support such association only for GI [7,42,43] or GL [20,44–48]. There are also studies that do not support any relationship between these two indices and disease risk [49–57].

A metaanalysis of observational studies by Barclay et al. [37] suggests that low-GI and/or low-GL diets are independently associated with a reduced risk of T2DM, heart disease, gallbladder disease, breast cancer, and all diseases combined [37]. It is notable although that the overwhelming majority of the subjects in the aforementioned metaanalysis were women (90%). Thus these findings may not be generalizable to men [37]. Nutritional interventions where the concept of GI is used are essential for the prevention [43] and management of T2DM [21]. Compliance with a low-GI diet has been suggested to be of benefit in improving insulin sensitivity and reducing glycated hemoglobin concentrations in T2DM patients [31,58–60]. Additionally, low-GI diets and diets with high cereal fiber content are shown to be independently associated with a reduced risk of developing type II diabetes in men [20,61] and women [43]. As for the GL, it has been demonstrated to be positively associated with increased risk of T2DM in men with low cereal fiber intake [20], but not in women [43]. More recently, Bhupathiraju et al. [62] also found a positive association between dietary GI and GL and the risk of T2DM.

The association of dietary GI and GL with the risk of cardiovascular disease (CVD) is not well established, and it may vary among different populations. Hardy et al. [63] has shown that high GI is associated with increased incident of coronary heart disease (CHD) in African Americans, while high GL is associated with an increased incidence of CHD in whites. Men with high GI and GL are at greater risk for CVD than women [64]. Among women though, the risk for CHD is greater with high GL with BMI > 23 [47] or BMI > 25 [48]. Dietary GI and GL may increase the risk of developing these diseases through adverse effects on blood lipids and systemic inflammation [65]. Indeed, dietary GI was associated with small increases in LDL cholesterol, LDL/HDL cholesterol ratio, TGs, and C-reactive protein (CRP) and with a small decrease in HDL cholesterol. Accordingly, dietary GL was associated with higher LDL/HDL cholesterol ratio and TG concentration, and lower HDL cholesterol [65]. Nevertheless, there are also reports that do not support any relation between high GI and high GL with the occurrence of CVD in men [66] or in women [56,57].

The occurrence of cancer has been linked with factors involving glucose metabolism [67]. Nevertheless, the evidence on the relationship between the GI and the risk of several types of cancer is inconsistent. Studies have shown that both high-GI and high-GL diets increase the risk of breast cancer [40,68], whereas others support the connection of high risk with high GI alone [42], or high GL alone [44–46]. Nevertheless, there are also studies that do not report an association between either high GI or high GL and increased risk for developing breast cancer [51,52,69]. Similarly, some investigators found an increased risk of endometrial cancer in women with the consumption of high-GI or high-GL foods [42], while others do not agree with these findings [53,70]. Similarly, some cohort studies do not support an association between either high GI or high GL and increased risk for developing colorectal cancer [49,71], despite the positive association that has been demonstrated [41,72]. Regarding pancreatic cancer, most of the data indicate that there is no relationship between GI and GL and increased risk for developing the disease in both men and women [54,55]; however, positive relationship has also been reported for high GL in overweight and sedentary women [73]. It is clear that published results have failed so far to establish a clear association between GI and elevated cancer risk. Therefore further long-term studies are needed to determine the role of GI in carcinogenesis.

Diets with high GI or high GL are also reported to independently increase the risk of gallbladder disease [37,39]. In regards with age-related cataract, no relative association has come up between the disease risk and either the GI or GL of the diet [50].

Obesity is a very common public health problem that increases the risk of developing several of the aforementioned diseases such as CVD, T2DM, and certain types of cancer, but also osteoarthritis [74]. Metabolic syndrome is also associated with CVD and T2DM [75] and characterized by abdominal obesity and insulin resistance along with elevated blood pressure, dyslipidemia, and elevated plasma GLU [75,76]. Therefore body weight loss is necessary in

TABLE 2.2 Glycemic Index, Glycemic Load, and Chronic Diseases

Study	Disease	Study Protocol	Estimation Method	Subjects	Average Follow-up (years)	Disease Incidences	Results
NEOPLASM							
Silvera et al. [42]	Breast cancer	Prospective Cohort; Data from the Canadian National Breast Screening Study	FFQ	49111 women (40–59 y)	16.6	1450	GI: ↑ risk in postmenopausal women; GL: no association with the disease risk
Jonas et al. [52]	Breast cancer	Cohort Study; Data from the Cancer Prevention Study-II	SFFQ	63307 postmenopausal women (40–87 y)	5	1442	GL, GI: no association with breast cancer
Sieri et al. [40]	Breast cancer	Cohort study; Data from the Hormones and Diet in the Etiology of Breast Tumors Study	SFFQ	8959 women (34–70 y)	11.5	289	GI: ↑ risk with HGI in postmenopausal women; GL: ↑ risk with HGL in postmenopausal women; ↑ risk with HGL in women with BMI<25
Larsson et al. [44]	Breast cancer	Data from the Swedish Mammography Cohort, a population-based cohort	FFQ	61433 women (39–76 y)	17.4	2952	GI, CHO: no association with risk of overall breast cancer; GL: positive association with risk of overall breast cancer
Wen et al. [45]	Breast cancer	Data from the Shanghai Women's Health Study, a population-based cohort study	FFQ	73328 Chinese women	7.35	616	GI: no association with breast cancer risk; GL, CHO: positive linear association with breast cancer risk in premenopausal women or women <50 y
Shikany et al. [51]	Breast cancer	Data from the Women's Health Initiative	FFQ	148767 postmenopausal women (50–79 y)	8	6115	GI, GL, CHO: no association with total breast cancer risk
Sieri et al. [46]	Breast cancer	Prospective cohort study; Data from the Italian section of the European Prospective Investigation into Cancer and Nutrition	FFQ, SLC	26066 women (not known)	11	879	GI, total CHO: no association with the risk of breast cancer; GL: positive association with the risk of breast cancer
Castro-Quezada et al. [69]	Invasive breast cancer	Prospective study; Data from the PREDIMED Cohort Study	SFFQ	4010 postmenopausal women (60–80 y) at high CVD risk	4.8 (17757 person-years)	32	GI, GL: no association with invasive breast cancer risk
Larsson et al. [49]	Colorectal cancer	Prospective study; Data from the Swedish Mammography Cohort Study	FFQ	36616 women born between 1914 and 1948	15.6	870	GI, GL, CHO: no association with colorectal cancer risk
Higginbotham et al. [41]	Colorectal cancer	Cohort study; Data from the Women Health Study	FFQ, RFQ	38451 women (≥45 y)	7.5	174	GI, GL: positive relationship with colorectal cancer
Sieri et al. [72]	Colorectal cancer	Cohort study; Data from the EPIC-Italy Study	VCFFQ	44225 men and women	11.7	421	GI, CHO from high-GI foods: positive relationship with colorectal cancer risk
Patel et al. [54]	Pancreatic cancer	Prospective Cohort; Data from the Cancer Prevention Study-II	FFQ	124907 men and women (62.7 ± 6.35 y)	9	401	GI, GL, CHO intake: no association with pancreatic cancer

Jiao et al. [55]	Pancreatic cancer	Prospective cohort study; Data from the NIH-AARP Diet and Health Study	FFQ	482362 (280542 men and 201820 women)	7.2	1151 (733 men and 418 women)	GI, GL: no association with the risk of pancreatic cancer
Michaud et al. [73]	Pancreatic cancer	Prospective cohort study; Data from the Nurses' Health Study	FFQ	88802 women	18	180	GI: no association with the risk of pancreatic cancer; GL: positive relationship with pancreatic cancer risk in sedentary and overweight women
Coleman et al. [70]	Endometrial cancer	Prospective cohort study; Data from the US Prostate, Lung, Colorectal and Ovarian Cancer Screening Trial	DHQ	36115 women (55–74 y)	9	386	GL, CHO: protective against endometrial cancer development
Silvera et al. [42]	Endometrial cancer	Prospective study; Data from the Canadian National Breast Screening Study	FFQ	34391 women (40–59 y)	16.4	426	GI, GL: overall positive association with endometrial cancer, particularly among obese women, premenopausal women, and postmenopausal women who use hormone replacement therapy
Larsson et al. [53]	Endometrial cancer	Prospective study; Data from the Swedish Mammography Cohort Study	FFQ	61226 women born between 1914 and 1948	15.6	608	GI, GL: no overall association with endometrial cancer; a 1.9 nonsignificant increase in disease risk in overweight women with low physical activity
DIABETES							
Salmeron et al. [20]	Type 2 diabetes	Prospective Study; Data from the Health Professionals Follow up Study	SFFQ	42759 men (40–75 y)	6	523	GL: ↑ risk with HGL in men with low cereal fiber intake
Schulze et al. [43]	Type 2 diabetes	Cohort Study; Data from the Nurses' Health Study II	SFFQ	91249 women (24–44 y)	8	741	GI: HGI ↑the risk of type 2 diabetes; GL: no association
CVD							
Liu et al. [47]	CVD	Cohort Study; Data from the Nurse's Health Study	SFFQ, MHQ	75521 women (38–63 y)	10	761	GL: positive association with CHD; ↑ risk with HGL in women with BMI > 23
Oh et al. [48]	Stroke	Prospective Cohort Study; Data from the Nurse's Health Study	SFFQ	78799 women (30–55 y)	18	1020	HCHO intake: positive association; GL: positive association with ↑ risk in women with BMI ≥ 25
Levitan et al. [66]	CVD	The Cohort of Swedish Men	FFQ	36246 men (46–79 y)	6-y follow-up for incidence of CVD and mortality; 8 y for all-cause mortality	2181 incidences of CVD, and 785 CVD deaths; 2959 deaths of all causes combined	GI: no association with ischemic CVD or mortality; GL: no association with ischemic CVD or mortality; positive association with the risk of hemorrhagic stroke
Hardy et al. [63]	CHD with or without type 2 diabetes	Data from the Atherosclerosis Risk in Communities Study	SFFQ	13051 whites and African Americans (1378 with diabetes and 11673 without diabetes; 45–64 y)	17	1683 (371 with diabetes, 1312 without diabetes)	GI: positive association with the risk of CHD in African Americans; GL: positive association with the risk of CHD in whites, more pronounced association in whites without diabetes

Continued

TABLE 2.2 Glycemic Index, Glycemic Load, and Chronic Diseases—cont'd

Study	Disease	Study Protocol	Estimation Method	Subjects	Average Follow-up (years)	Disease Incidences	Results
Levitan et al. [56]	Myocardial infarction	Participants from the Swedish Mammography Cohort	FFQ, MHQ	36234 women (48–83 y)	9	1138	GI, GL: no association with myocardial infarction in women
Levitan et al. [57]	Heart failure	Prospective, observational study with participants from the Swedish Mammography Cohort	FFQ, MHQ	36019 women (48–83)	9	639 (54 died and 585 hospitalized for heart failure for the first time)	GI, GL: no association with the risk of heart failure events in women
Burger et al. [64]	CHD and stroke	Data from the EPIC-MORGEN Study, a large prospective cohort study	FFQ	10753 women and 8855 men (21–64 y)	11.9	CHD: 300 women and 581 men. Stroke: 109 women and 120 men	GL: positive association with CHD risk in men only, after adjustment for established CVD risk factors. Slightly ↑CHD risk in men only, after inclusion of nutritional factors. No association with ↑ stroke risk in men or women; GI: no association with ↑CHD risk. Positive association with stroke risk in men only, after adjustment for CVD risk factors and nutrients
OTHER DISEASES							
Finley et al. [76]	Metabolic syndrome	Data from the Cooper Center Longitudinal Study	MHQ, Clinical examination	10912 (9137 men and 1775 women; 20–79 y)	-	2211 men and 153 women	GI: positive association with prevalence of the metabolic syndrome in men; positive association with prevalence of large waist girth, elevated TG, and low HDL-C in men and women; inverse association with high glucose in men; GL: positive association with prevalence of the metabolic syndrome in men; positive association with prevalence of large waist girth, elevated TG, and low HDL-C in men and low HDL-C in women
McKeown et al. [7]	Insulin resistance, metabolic syndrome	Data from the fifth examination cycle of the Framingham Offspring Study	SFFQ, MHQ	2834 (1290 men and 1544 women; 26–82 y)	4	2834	GI: positive association with prevalence of the metabolic syndrome; positive association with insulin resistance; GL: no association with prevalence of the metabolic syndrome; positive association with insulin resistance
Schaumberg et al. [50]	Age Related Cataract	Data from the Nurse's Health Study and the Health Professionals Follow-up Study	SFFQ	71919 women and 39926 men (>45 y)	10	3258 (women) 1607 (men)	GI, GL: no association with cataract extraction
Tsai et al. [39]	Gallbladder disease	Prospective Cohort Study; Data from the Health Professionals Follow up Study	FFQ, MHQ	44525 men (40–75 y)	12	1710	GL, GI, CHO intake: positive association with the disease risk

CHD, coronary heart disease; *CVD*, cardiovascular disease; *DHI*, diet history interviews; *DHQ*, diet history questionnaire; *FFQ*, food frequency questionnaire; *GDM*, gestational diabetes mellitus; *HCHO*, high carbohydrate; *HDL-C*, high density lipoprotein-cholesterol; *HGI*, high glycemic index; *HGL*, high glycemic load; *MHQ*, medical history questionnaire; *RFQ*, risk frequency questionnaire; *SFFQ*, semiquantitative food frequency questionnaire; *SLC*, standardized lifestyle questionnaire; *T2DM*, type 2 diabetes mellitus; *TG*, triglyceride; *UL*, uterine leiomyomata; *VCFFQ*, validated center-specific food frequency questionnaire.

both obesity and metabolic syndrome. A restriction in saturated fat intake for weight loss has had modest success, while a restriction in refined CHO is the most recent approach to weight loss and related reduction of disease risk [77]. Low dietary GI [31–33] and GL [78] may contribute to decrease in body weight in a most effective way in adults, but also in children [79]. This could be due to different substrate oxidation in the postprandial phase as a result of a low-GI meal consumption compared with a high-GI meal [31,80,81], which leads to different fuel portioning [31]. In addition, low-GI diets may assist with weight control by effecting satiety [31,80]. At the same time, high-GI foods may promote excessive weight gain due to hormonal responses that seem to lower circulating levels of metabolic fuels, stimulate appetite, and favor storage of fat [82]. BMI has been positively associated with the GI (long term) in women [33], while a low-GL dietary intervention has been shown to reduce body weight and improve the lipidemic profile in obese young adults [78]. Moreover, compliance with a low-GI/low-GL diet has shown to result in decreased C-reactive protein and fasting INS, indicating that it may also help in the prevention of obesity-associated diseases [83]. Similarly, with adults, reducing the GL of the diet may be also effective in improving BMI in children. Kirk et al. [79] report that reducing the GL of the diet for 3 months is equally beneficial with the reduction of CHO or a standard portion-controlled diet in improving BMI and several cardiovascular risk factors for up to 12 months after the intervention in children. Interestingly the adherence to the reduced GL diet in that study was higher compared with the low-CHO and standard portion-controlled diet, and this may indicate the promising effect of such an intervention for long-term weight management.

An important finding regarding the GI influence on health is that of the DECODE study [84]. According to this study, postprandial hyperglycemia is considered a risk factor for mortality not only for patients who suffer from chronic disease but also for people with normal fasting blood GLU. Therefore the prevention of hyperglycemic situations should also be targeted in healthy people.

GI, METABOLIC RESPONSES, AND EXERCISE PERFORMANCE

GI has recently gained interest in the field of sports nutrition. Different GIs have been shown to produce diverse metabolic responses during exercise [85,86], and some authors support the connection of preexercise low GI with enhanced physical performance [87–91], while others dispute such a connection [86,92,93].

The intensity of the glycemic and insulinemic responses in the postprandial phase and during exercise is considered a main contributor to achieved performance. The maintenance of euglycemia during exercise and the avoidance of rebound hypoglycemia have been shown to be of great importance in the delay of fatigue and improvement of performance, especially during prolonged exercise. The preservation of CHO and the use of fat as the main substrate for energy are of prime pursuit when it comes to endurance exercise. Because the GI represents the blood GLU response of CHO-containing foods, the perspective of controlling the glycemia and insulinemia and, as a consequence, substrate utilization during exercise by manipulating the GI of preexercise meals would be of critical significance for athletic performance. Glucose utilization during exercise is mediated by INS release by b-cells of the pancreas, which is stimulated by high GLU concentrations, as it happens after the consumption of a CHO-rich meal. Moreover, high-GI preexercise meals have been connected with increased CHO oxidation and diminished fat oxidation [1,81,91]. Such a connection is further indicated by elevated serum levels of GLU during exercise and suppression of circulating FFAs and TGs [85,91].

Substrate utilization during exercise is also dependent on the intensity of physical activity as CHOs are the preferred energy source in high-intensity exercise, whereas during low-to-moderate intensity physical activity, the energy comes mainly from the catabolism of FFA. Nevertheless, preexercise glucose ingestion suppresses lipolysis to a point at which it limits fat oxidation, even during low-intensity exercise [94]. It seems that preexercise CHO meal can affect the typical intensity-dependent substrate utilization, probably by the CHO-induced rise in INS that inhibits the mobilization and hence availability of circulating FFA. Additionally, the increased CHO oxidation that has been reported after high-GI preexercise meal and the subsequent inhibition of long-chain fatty acid entrance into the mitochondria [95] could also explain the reduced fat oxidation. However, such an inhibition of FFA entry into the mitochondria and a restricted energy production from fat oxidation could also result in earlier fatigue and deterioration of physical performance.

The Effect of Different GIs of Preexercise Meals on Physical Performance

As it has already been mentioned, different GI preexercise meals result in different metabolic responses. There is evidence that these responses affect in turn physical performance. However, despite the studies reporting an

enhancement in athletic performance, mainly due to low-GI preexercise meals [88–90], there are also reports that do not support any improvement [86,92,93]. Physical performance is mainly being estimated through the time trial (TT) that is the time needed for covering a predefined distance, time to exhaustion, or total work and power output, along with other physiological parameters such as heart rate (HR), rate of perceived exertion (RPE), VO_{2max}, and respiratory exchange ratio (RER). Table 2.3 summarizes all the studies that have been reviewed in this chapter.

Improvement of Physical Performance

An improvement by an average of 3 min in the time needed to cycle a 40-km distance was observed after the ingestion of a low-GI CHO meal 45 min before the onset of the exercise, when compared with the consumption of a high-GI meal in well-trained male cyclists [88]. Although improvement in exercise performance has also been reported in previous studies [91,96], the 3.2% improvement in performance in Moore's study appears somewhat lower than the 7.9%–59% range of improvement in performance previously observed. The authors attribute this lower improvement to a possible overestimation of the beneficial effect that low-GI meals are actually having on performance. Additionally, lower RPE during the TT and lower whole-blood GLU and INS concentration 45 min postprandial were demonstrated in the low-GI meal compared with the high-GI meal. Another interesting finding in Moore's study was the lower CHO oxidation during exercise in the high-GI trial, which is in contrast with the typical inhibition of FFA and increased CHO oxidation after the consumption of high-GI preexercise meals [87,89,91].

In the study by Karamanolis et al. [87] CHO oxidation during prolonged exercise till exhaustion was lower in the low-GI (GI = 29) group compared with the high-GI (GI = 83) and placebo (PL) group, whereas fat oxidation was similar among groups. In this study, the three different GI preexercise meals were consumed 15 min before exercise. The lower CHO oxidation was accompanied by an improvement in the performance, as the time to exhaustion was 23% greater in the low-GI trial compared with the PL trial. This seems to be the result of the prevention of hyperinsulinemia and a better maintenance of blood GLU concentration throughout the exercise and mainly due to significantly higher responses of blood GLU at the time to exhaustion. Lactate was lower in the low-GI than in the high-GI trial only at the time to exhaustion, when increased levels of anaerobic glycolysis were present. Glycerol mobilization was better in the low GI, which probably suggests that a better preservation of CHO was attained that led to less fatigue and improvement in exercise performance.

Wee et al. [85] investigated the effect of preexercise breakfast of either high or low GI on muscle glycogen metabolism 3 h postprandial and during 30-min submaximal running. Higher values of blood GLU and INS were reported in the postprandial state for both the high- and the low-GI meal, with GLU falling below baseline after the first 10 min of exercise in high-GI meal but been greater than those of low-GI meal at the end of exercise. INS appeared to be two-fold higher in the high GI compared with the low GI but only at the onset of exercise. FFA and glycerol were suppressed throughout the postprandial period for both meals, but the suppression was higher in the high GI compared with the low GI, which demonstrated higher levels of FFA and glycerol than the high GI during exercise. No differences in RER were observed between the two preexercise dietary approaches. However, higher levels of lactate were recorded in both serum and muscle in the high-GI meal. CHO oxidation during exercise was 12% lower in the low GI with a compensatory increase in fat oxidation compared with the high-GI meal, such that the overall energy expenditure was similar.

An attempt has also been made to ascertain whether a moderate-GI (M-GI) versus a high-GI preexercise meal could lead to different metabolic responses and performance. Kirwan et al. [91] used a M-GI (GI = 61) versus a high-GI (GI = 82) breakfast cereal to test this hypothesis. Six healthy active men were tested at ~60% VO_{2peak} cycling to exhaustion, 45 min after the consumption of the meals. Performance was improved in the M-GI meal, with a 23% improvement in time to exhaustion compared with a 5% improvement in the high-GI meal, which was no different from ingesting water. Elevated GLU and INS levels were observed postprandial in both meals, with GLU being higher at 60 and 90 min of exercise in the M-GI meal, whereas INS was elevated only at the start of exercise and returned to premeal values within 30 min of exercise. Circulating FFAs were suppressed before exercise and remained that way at 30, 60, and 120 min of exercise in both high- and M-GI meals compared with control. Total CHO oxidation was higher in the moderate GI than in control trial, and additionally, a significant association between total CHO oxidation and time to exhaustion were observed.

In a study by Wu and Williams [90], at 70% VO_{2max} an improvement by approximately 8 min in running time to exhaustion was observed 3 h after the consumption of a low-GI meal, when compared with an isocaloric high-GI meal trial in eight healthy male recreational runners. Plasma GLU level was higher during the high-GI trial than low-GI trial only for the first 30 min of exercise. FFA, glycerol levels, and fat oxidation rate were significantly higher, while RER and CHO oxidation rate were significantly lower in the low-GI trial compared with high-GI trial. These findings

TABLE 2.3 Glycemic Index and Metabolic Responses During Exercise

Studies	Study Protocol	Subjects	Estimated Indices	Results
Wee et al. [85]	HGI (GI = 80) and LGI (GI = 36) breakfast meals ingested 3h before 30-min running at ~70% VO_{2max}.	Seven male recreational runners (31 ± 4 y)	HR, RER, total CHO and fat oxidation, GLU, FFA, glycerol, glucagon, insulin, glucose AUC, insulin AUC	GLU: ↓ in HGI below baseline at 10 min of exercise, ↑ in HGI than LGI at the end of exercise; INS: two-fold ↑ in HGI than in LGI; Glucagon: ↑ in LGI than HGI postprandial; FFA: ↓ in both meals postprandial, ↑suppression in HGI than in LGI; LA: ↓ in LGI than in HGI in serum and in muscle after exercise; Muscle glycogen: 15% ↑ in the HGI, 46% ↑ net muscle glycogen utilization in the HGI than in the LGI
IMPROVEMENT OF PERFORMANCE				
Moore et al. [88]	Two standardized meals with HGI (GI = 72) or HGI (GI = 30) ingested 45 min before exercise. Each trial was separated by 7 days.	10 well-trained male cyclists (8 ± 6 y)	40-km TT, HR, RPE, fat and CHO oxidation, VO_{2max}, RER, GLU, INS, FFA, TG, LA	TT: ↑ performance in LGI than in HGI meal; RPE, fat oxidation, GLU, INS: ↓ in the LGI than in HGI; RER, CHO oxidation: ↓ in the HGI than in the LGI trial; HR, FFA, LA: ↑ in time than baseline for both GI trials; VO_{2max}, TG: no differences between trials
Karamanolis et al. [87]	HGI (GI = 83), LGI (GI = 29), and control meal ingested 15 min before exercise. Each trial was separated by 7 days.	Nine recreational runners (26 ± 3 y)	Treadmill TE, VO_{2max}, RER, HR, GLU, INS, glycerol, LA, fat and CHO oxidation	TE: 23% ↑ in LGI versus PL; VO_{2max}: ↑ in the HGI versus PL from 45 min until the TE; GLU: ↑ in the HGI versus PL and LGI 15-min postprandial; INS: ↑ in the HGI versus PL 15-min postprandial; Glycerol: ↑ in the PL versus LGI at 60 min and TTE; LA: ↑ in HGI versus LGI at TE; CHO oxidation: ↑ in the HGI versus LGI at 30 min and 45 min and versus PL at 30 min; RER, HR, fat oxidation: no differences between trials
Kirwan et al. [91]	HGI (GI = 82), moderate GI (GI = 61), and control (water) whole-food breakfast cereals, ingested 45 min before exercise. Each trial was separated by 7 days.	Six healthy active men (22 ± 1 y)	Cycling TE, VO_{2max}, RER, HR, GLU, INS, epinephrine, norepinephrine, glycerol, FFA, fat and CHO oxidation	TE: 23% ↑ in the MGI meal; GLU: ↑ postprandial in both HGI and MGI, ↑ at 60 and 90 min of exercise in the MGI; INS: ↑ postprandial in both HGI and MGI, ↑ at the start of exercise and returned to premeal at 30 min of exercise; FFA: ↓ before exercise and remained ↓ at 30, 60, and 120 min of exercise in both HGI and MGI; CHO oxidation: ↑ in the MGI than in control; significant association between total CHO oxidation and TE; Epinephrine, norepinephrine: not affected
Wong et al. [89]	LGI (GI = 37) or HGI (GI = 77) meal providing 1.5 g CHO/kg body mass, ingested 2 h before exercise. Each trial was separated by 7 days.	Eight endurance-trained male runners (33 ± 1.7 y)	21-km TT, HR, RPE, GLU, LA, INS, FFA, cortisol, glycerol, CHO and fat oxidation, hematocrit, Hb, PV, RER, RPT, RAD, and GFS, GLU IAUC, SL	TT: ↑ performance in the LGI trial; GLU: higher in the LGI trial; LA, RPE: ↑ in both trials; INS: no differences between trials; HR: ↑ in both trials, higher in the LGI trial at the end of exercise; FFA: ↓ until 10 km of exercise in both trials, higher in the LGI trial at the end of exercise; Glycerol: ↑ in both trials, higher in the LGI trial at 10 km until the end of exercise; PV, SL, RPT: no significant differences between trials; RER, CHO oxidation: ↑ in both trials, higher in the HGI trial at 5–10 km of exercise; Fat oxidation: ↑ in both trials, higher in the LGI trial at 5–15 km; RAD: ↑ at the end of the exercise in both trials; GFS: no differences between meals; GLU IAUC: higher in the HGI meal
Wu & Williams [90]	LGI (GI = 37) or HGI (GI = 77) meal providing 2 g CHO/kg body mass, ingested 3 h before exercise. Each trial was separated by 7 days.	Eight male recreational runners (28.9 ± 1.5 y)	TE, RER, RPE, RPT, GFS, BM, LA, FFA, GLU, INS, glycerol, hemoglobin, PV, GLU IAUC, INS IAUC, energy expenditure, CHO oxidation, fat oxidation	TE: higher in the LGI trial; GLU: higher in the LGI trial at 15–30 min of exercise; INS, LA, HR, RPE, PV, RPT, BM: no significant differences between trial; GLU IAUC, INS IAUC: postprandial higher in the HGI meal; FFA: higher in the LGI trial; Glycerol: higher in the LGI trial at 45 min of exercise until exhaustion; CHO oxidation, RER: higher in the HGI trial; Fat oxidation: higher in the LGI trial at 15 min and 45–90 min of exercise

Continued

TABLE 2.3 Glycemic Index and Metabolic Responses During Exercise—cont'd

Studies	Study Protocol	Subjects	Estimated Indices	Results
Moore et al. [88]	LGI or HGI meal providing 2.5 g CHO/kg body mass, ingested 3 h before cycling to exhaustion (60% VO_{2max}). Each trial was separated by 7 days.	10 untrained females (21.1 ± 1.1 y)	GLU, LA	TE: higher in the LGI than HGI trial; GLU: ↑ at 15 and 180 min after the HGI meal, at 20 min after the HGI exercise trial LA: ↑ at 15 min after the HGI meal, ↑ during the HGI exercise trial
NO EFFECT ON PHYSICAL PERFORMANCE				
Jamurtas et al. [92]	LGI (GI = 30) and HGI (GI = 70) meal providing 1.5 g CHO/kg of body weight or placebo (control) meal, ingested 30 min before 1 h of cycling (65% VO_{2max}) followed by cycling to exhaustion (90% VO_{2max}). Each trial was separated by 7 days.	Eight untrained healthy males (22.8 ± 3.6 y)	TE, RPE, HR, ventilation, GLU, INS, LA, β-endorphin, RER, CHO oxidation, fat oxidation	TE, RPE, HR, RER, ventilation: no differences between trials; INS: higher in the HGI than control at 20 min of exercise; β-Endorphin, RER: ↑ at the point of exhaustion in all trials; CHO oxidation, fat oxidation, GLU, LA: no differences between trials;
Febbraio et al. [86]	HGI and LGI meal providing 1 g CHO/kg body wt or placebo (control) meal, ingested 30 min before 120 min of submaximal exercise followed by a 30-min performance trial. Each trial was separated by 7 days.	Eight endurance-trained men (26 ± 6 y)	TW, VO_2, HR, GLU, FFA, INS, total glucose Ra and Rd, LA, glycogen, total CHO oxidation, glucose oxidation, fat oxidation	TW: no differences between trials during the performance trial; VO_2, HR, INS, LA, glycogen: no differences between trials during the submaximal exercise; FFA, GLU: lower in the HGI trial at some points of submaximal exercise; Glucose Ra, glucose Rd: higher in the HGI trial throughout submaximal exercise, lower in the control versus LGI trial at some points of submaximal exercise; CHO oxidation: higher in the HGI trial; Glucose oxidation: higher in the GI trials versus control, higher in the HGI versus LGI trial.
Kern et al. [93]	HGI (117 ± 15) or MGI (88 ± 13) meal providing 1 g CHO/kg body weight, ingested 45 min before 45 min of submaximal exercise followed by a 15-min performance trial. Each trial was separated by at least 7 days.	Eight endurance-trained male (n = 4) and female (n = 4) cyclists (30 ± 5 y)	GLU, INS, LA, TG, FFA, BHB, power output	GLU, LA, TG, BHB: no differences between trials during the submaximal exercise; INS: lower in the MGI trial; FFA: higher in the MGI trial; Power output: no differences between trials during the performance trial
Little et al. [101]	HGI (GI = 76) and LGI (GI = 26) meal providing 1.5 g CHO/kg body weight or placebo (control), ingested 2 h before 90 min of high-intensity, intermittent exercise including 15 min of a repeated-sprint test at the end of exercise. Each trial was separated by at least 7 days.	16 male athletes (22.8 ± 3.2 y)	15-min DC, VO_2, RER, RPE, fat oxidation, CHO oxidation, GLU, LA, INS, FFA, CAT, glycogen	15-min DC: ↑ of performance in both GI trials; VO_2, RER, GLU, LA: no differences between trials; Fat oxidation: lower in the HGI versus control at 33–40 min of exercise (collection period 2), lower in the LGI versus control at 63–70 min of exercise (collection period 3); ↑ in time for all trials; CHO oxidation: no differences between trials, ↓ in time for all trials; INS: lower in the control versus GI trials; FFA: higher in the control versus GI trials; CAT: higher in the HGI versus control at the end of exercise; Glycogen: higher in the GI trials after 75 min of exercise; RPE: lower in the LGI versus control
Bennett et al. [99]	Session 1: HGI (GI = 78) and LGI (GI = 42) meal providing 1.5 g CHO/kg body weight, ingested 2 h before 90 min of intermittent high-intensity treadmill running. Session 2: during 3 h of rest, HGI and LGI meal providing 2 g CHO/kg body weight, ingested >2 h before another treadmill running. Each trial was separated by at least 7 days.	10 male (27.2 ± 8.0 y) and 4 female (22.5 ± 4.4 y) recreational soccer players	GLU, INS, LA, NEFA, CHO oxidation, fat oxidation, sprint distance during five 1-min sprints at the end of each exercise session	GLU: higher in HGI versus LGI (peak postprandial), higher in LGI versus HGI (at the end of session 2); INS: higher in HGI versus LGI (peak postprandial); LA: higher in HGI versus LGI (at the end of session 2); NEFA: higher in HGI versus LGI (at the end of session 2) in females; CHO oxidation: higher in HGI versus LGI (at the start of session 1); Fat oxidation: no differences between trials; Sprint distance: no differences between trials