CHAPTER 17

Dietary supplements and nutritional ergogenic aids in sport

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17.1 INTRODUCTION

The sports world is filled with special foods, potions, pills and powders that promise to provide the athlete with a performance edge. Advertisements and testimonials for these products claim prolonged endurance, faster recovery, increases in muscle mass and strength, losses of body fat, and resistance to fatigue, illness or infection. Such promises are attractive to athletes and coaches, especially in elite competition where very small differences separate the fame and fortune of winning from the anonymity of the rest of the field. Yet external rewards provide only part of the drive to find a 'magic bullet', because even non-elite and recreational athletes show considerable interest in using sports supplements.

In the general community, supplement use continues to increase. In 1996, consumer spending on supplements in the United States was $6.5 billion, doubling the expenditure of 1990–91. In 1997 the market increased to $12.8 billion (Camire & Kantor 1999). Traditional markets of health food shops and pharmacies have been expanded to include sports shops, multilevel marketing, mail-order and Internet sales. Supplements targeted at athletes and sports performance provide an important niche in this market. Sales figures provided for some contemporary sports supplements illustrate the rapid response of consumers to marketing and word of mouth publicity for these products. Creatine supplements, first brought to public attention after the 1992 Barcelona Olympics, now have annual sales estimated at 2.7 million kilograms (Williams et al. 1998). Hydroxy-methyl butyrate (HMB), a

supplement which received its first mention in sports science literature in 1995–96, reached sales figures of $30–50 million in the United States during 1998 (Slater, in press), in the absence of clear proof of its success in increasing muscle mass and strength.

This chapter will review the supplement practices of athletes, discussing the science behind commonly used supplements. It will be seen that the evidence to support the claims of many products is absent, but that there are specific situations in which athletes may benefit from the use of nutritional supplements. To help simplify the vast array of products on offer to athletes we will continue to use a system that identifies two separate categories or applications of nutritional supplements, classifying these either as dietary supplements or nutritional ergogenic aids.

17.2 SUPPLEMENTATION PRACTICES OF ATHLETES

In a previous summary of the literature (Burke & Heeley 1994), we noted that few formal studies have focussed solely on the use of nutritional supplements by athletes. Most of the information about the supplementation practices of athletes is provided, in brief, from surveys of dietary intake of athletic populations. Exceptions to this include surveys of the supplementation practices of marathon runners (Nieman et al. 1989), swimmers (Baylis et al. in press) and high school athletes (Sobal & Marquart 1994; Krumbach et al. 1999). A large survey of drug use among Australian athletes (Australian Sports Medicine Federation 1983) also included a section on the use of nutritional supplements.

Table 17.1 summarises the prevalence of supplement use reported among a variety of athletic groups, and shows that more than half the athletic population are supplement users, although the prevalence ranges between sports. Of course, some of the variation in the supplement use reported by different athletic groups is due to methodological differences in collecting this information. First, there are differences between surveys with respect to the definition of 'nutritional supplements', with some surveys limiting supplements to vitamin and mineral preparations (Moffatt 1984) while others include items such as sports drinks and other sports food products (Australian Sports Medicine Federation 1983). The definition of 'regular', 'routine', 'irregular' and 'occasional' use may also differ. Finally, the method of collecting information (e.g. questionnaire/self-report versus actual record of intake) also influences the results. For example, Nieman and colleagues (1989) found a higher percentage of runners reported using vitamin or mineral supplements when responding to a frequency questionnaire (69% usage) than when recording actual use over a three-day period (48%). Methodological differences aside, the literature shows that supplement use by athletes is a popular and widespread activity, with considerable variability between and among different sports with regard to the number and type of products used. It is also apparent that

supplement use moves in cycles or trends, with new supplements quickly becoming popular and others disappearing from fashion.

Table 17.1 *Prevalence of supplement use by athletes*

Reference	Population	N	% use
Adams et al. 1982	College swimmers (F)	12	33
Australian Sports Medicine Federation 1983	All athletes (M, F)	4063	47
Barr 1986	Marathon runners (F)	104	75
	Fitness class runners (F)	105	64
Barr 1987	College athletes (F)	70	76
Barr & Costill 1992	College swimmers (M)	24	17
Barry et al. 1981	Mixed elite athletes (M, F)	143	55
Baylis et al. in press	Elite swimmers (M, F)	77	99
Bazzarre et al. 1993	Recreational athletes (M, F)	91	51
Berning 1986	Elite swimmers (M, F)	NA	63
Bobb et al. 1969	College athletes (M)	28	25
Brill & Keane 1994	Body builders (M, F)	309	100
Burke et al. 1991	Elite triathletes (M)	25	44
	Elite marathon runners (M)	19	95
	Australian footballers (M)	56	46
	Olympic weightlifters (M)	19	100
Campbell & MacFadyen 1984	Swimmers (M, F)	101	61
Clark et al. 1988	Elite runners (F)	93	71
Cross 1997	Olympic swimmers (M, F)	28	89
Deuster et al. 1986	Runners (F)	57	54
Douglas & Douglas 1984	Marathon runners (M, F)	943	58
Ersoy 1991	Competitive gymnasts (F)	20	45
Faber & Spinnler-Benade 1987	Body builders (M)	76	63
Faber & Spinnler-Benade 1991	National field athletes (M)	20	35
	National field athletes (F)	10	33
Felder et al. 1998	Elite surfers (F)	10	50
Frederick & Hawkins 1992	College track athletes (F)	13	62
Grandjean 1983	Elite athletes (M, F)	69	92
Grandjean 1985	Elite athletes (M, F)	150	52

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Reference	Population	N	% use
Grandjean et al. 1992	Elite road cyclists (F)	3	100
Houston 1980	Elite swimmers (M)	8	75
	Elite swimmers (F)	12	50
Jonnalagadda et al. 1998	Elite gymnasts (F)	33	92
Khoo et al. 1987	Ironman triathletes (M)	19	60
	Ironman triathletes (F)	10	80
Kleiner et al. 1990	Body builders (M)	8	90
	Body builders (F)	19	100
Krowchuk et al. 1989	High school athletes (M, F)	298	33
Krumbach et al. 1999	Collegiate athletes (M, F)	411	57
Lamar-Hildebrand et al. 1989	Competitive body builders (F)	6	100
	Non-competitive body builders (F)	4	50
Lawrence et al. 1975a and 1975b	Swimmers (M, F)	48	52
	Swimmers (M, F)	72	54
Loosli et al. 1986	Club gymnasts (F)	97	43
Moffatt 1984	Elite high school gymnasts (F)	13	23
Nieman et al. 1989	Non-elite runners (M)	285	86
	Non-elite runners (F)	54	70
Nowak et al. 1988	College basketball (F)	10	50
	College basketball (M)	15	6
Oppliger et al. 1993	High school wrestlers (M)	713	40
Parr et al. 1984	High school/college athletes (M)	1432	56
	High school/college athletes (F)	1547	33
Peters et al. 1986	Ultra-distance runners (M)	15	80
Sandoval et al. 1989	Body builders (M)	5	20
	Body builders (F)	6	50
Saris et al. 1989	Elite road cyclists (M)	5	100
Schulz 1988	College athletes (M, F)	127	44
Short & Short 1983	College football players (M)	40	43
	College basketballers (M)	12	42
	College wrestlers (M)	38	13
Singh et al. 1993	Ultra-marathoners (M)	10	66
	Ultra-marathoners (F)	2	100
Slavin et al. 1984	Bicycle racers (M)	76	32
	Bicycle racers (F)	32	64

Reference	Population	N	% use
Snyder et al. 1989	Elite speed skaters (M)	10	60
	Elite speed skaters (F)	7	86
Sobal & Marquart 1994	High school athletes (M, F)	742	38
Steel 1970	Olympic athletes (M, F)	80	49
Thibault et al. 1984	Marathon runners (M, F)	1123	20
Werblow et al. 1978	College athletes (F)	94	37
Worme et al. 1990	Triathletes (M, F)	71	39

Unfortunately, most studies reporting the supplementation practices of athletes fail to provide the most interesting information: the type of supplements used, the amounts taken and the rationale for their use. However, several authors have commented that at least some athletes use a large number of supplements concurrently, often resulting in nutrient intakes that are very high in comparison with normal dietary intakes (Grandjean 1993). The Australian Sports Medicine Federation Report (1983) expressed concern about the 'significant minority' of athletes who reported intakes of six to 15 supplement preparations daily. We also noted 'polypharmacy' in our survey of elite swimmers; 71% of swimmers reported the use of more than one vitamin and mineral preparation and one swimmer recorded 28 different supplement products in regular use (Baylis et al. in press).

There is little information about why athletes choose their supplement patterns. In a study of 347 marathon runners (n=347), Nieman et al. (1989) failed to find significant associations between supplement use and gender, race, marital status, education, dietary intake or training level. By contrast, Krumbach and colleagues found that male and female college athletes were motivated by different beliefs in deciding to use supplements (Krumbach et al. 1999). The Australian Sports Medicine Federation survey (1983) reported that the beliefs within a particular sport, particularly emanating from a coach, strongly influenced supplementation practices. The role of the coach as nutrition adviser has been highlighted in other studies, with 35% of coaches in one survey (Wolf et al. 1979) and 68% in another (Bentivegna et al. 1979) reporting that they had recommended their athletes to take supplements on some occasions. Despite often following supplement practices that are not generally supported by scientific evidence, elite swimmers nominated professional advice from dietitians, doctors, pharmacists and sports scientists as the most important information to consider before trying a new supplement product (Baylis et al. in press). In this survey, the advice of a coach was ranked highly as a supporting source of information, but expense was not considered to be very important (Baylis et al. in press).

Generally, there are three reasons put forward by athletes in support of their supplement use (Nieman et al. 1989):

1. to compensate for less than adequate diets or lifestyles;

2. to meet unusual nutrient demands induced by heavy exercise; and
3. to produce a direct (e.g. ergogenic) effect on performance.

Although some surveys have suggested that certain types of athletes use supplements to compensate for poor food intake, in our experience, the majority of current athletes are motivated by the direct performance or health claims made for various supplements. To better understand the variety of products marketed to athletes we have devised a simple classification system, dividing supplements into two categories.

17.3 CLASSIFICATION OF SUPPLEMENTS USED BY ATHLETES

17.3.1 Definition of dietary supplements

Previously we have proposed the following definitions for a 'dietary supplement' or 'sports supplement' (Burke & Read 1993):

- contains nutrients in amounts generally similar to the levels specified in the recommended dietary intakes or allowances (RDIs/RDAs), and similar to the amounts found in food;
- provides a convenient or practical means of ingesting these nutrients, particularly in the athletic setting;
- allows or aids the achievement of known physiological or nutritional requirements of an athlete;
- contains nutrient(s) in large amounts for use in treating a known nutrient deficiency;
- has been shown to meet a specific physiological or nutritional need that improves sports performance; and
- is generally acknowledged as a valuable product by sports medicine and science experts.

Sports supplements that meet the definition of the dietary supplement are summarised in Table 17.2. This table shows some of the specific applications of those products that have been demonstrated to assist in the achievement of optimal sports performance. More information on the composition and use of these products can be found in other chapters of this book, as shown in Table 17.2.

It is important for athletes to appreciate that sports supplements *per se* do not produce a performance enhancement. Rather, it is the use of a supplement to achieve sports nutrition goals or guidelines that allows the athlete to perform optimally. Nutrition education of athletes is needed to ensure that dietary supplements are used appropriately. In many cases, this information is specific to the athlete or the sports situation and may require one-on-one counselling. In most situations, the use of the supplement will be part of a larger plan of optimal sports nutrition or the clinical management of a nutritional problem. Effective education will not only ensure that dietary supplements are used for maximum benefit, but will also highlight the general importance of optimal nutrition for the athlete.

Table 17.2 *Dietary supplements and their use by athletes*

Supplement	Form	Composition	Sports-related use	Chapter
Sports drink	Powder Liquid	5–7% CHO 10–25 mmol/L Na	Optimum delivery of fluid + CHO during exercise Post-exercise rehydration	14 15
Sports gel	Gel 30–40 g sachets or larger tubes	60–70% CHO (~25 g CHO per sachet) Some contain MCTs or caffeine	Supplement high-CHO training diet Carbohydrate loading Post-exercise CHO recovery May be used during exercise when CHO needs exceed fluid requirements	15 13 15 14
High-CHO supplement	Powder Liquid	10–25% CHO (+ some B vitamins)	Supplement high-CHO training diet Carbohydrate loading Post-exercise CHO recovery May be used during exercise when CHO needs exceed fluid requirements	15 13 15 14
Liquid meal supplement	Powder (mix with water or milk) or liquid	1–1.5 kcal/mL 15–20% protein 50–70% CHO Low to moderate fat Vitamins/minerals: 500–1000 mL supplies RDIs/RDAs	Supplement high-energy/CHO nutrient diet (especially during heavy training/competition or weight gain) Low-bulk meal replacement (especially pre-event meal) Post-exercise recovery Portable nutrition for travelling athlete	5, 15 13 15 24
Sports bar	Bar (50–60 g)	40–50 g CHO 5–10 g protein Usually low in fat Vitamins/minerals: 50–100% of RDIs/RDAs	CHO source during exercise Post-exercise recovery Supplement high-energy/CHO/nutrient diet Portable nutrition (travelling)	14 15 15 24
Vitamin/mineral supplement	Capsule/tablet	Broad range 1–4 x RDIs/RDAs of vitamins and minerals	Micronutrient support for low-energy or weight-loss diet Micronutrient support for restricted variety diets (e.g. vegetarian diet) Micronutrient support for unreliable food supply (e.g. travelling athlete)	7, 12 9, 12, 21 24
Iron supplement	Capsule/tablet	Ferrous sulfate/gluconate/fumarate	Supervised management of iron deficiency	11
Calcium supplement	Tablet	Calcium carbonate/ phosphate/lactate	Calcium supplementation in low-energy or low dairy food diet? Treatment/prevention of osteopenia	10

Since the dietary guidelines for exercise are well documented, and their application can provide a substantial enhancement of performance, it should be relatively simple to demonstrate beneficial uses of common sports supplements. In fact, many products such as sports drinks, carbohydrate (CHO) gels and liquid meal supplements have been specially manufactured in response to needs identified by applied sport science research. Pharmaceutical and food companies that produce and market dietary supplements provide much of the financial support for this research. In this way, a beneficial relationship between the company, the sports science or medicine professional and the athlete is nurtured.

17.3.2 *Definition of nutritional ergogenic aids*

The second broad category of sports supplements might be termed nutritional ergogenic aids, described by the following characteristics (Burke & Read 1993):

- contain nutrients or other food components in amounts greater than nutrient RDI levels, or the amounts typically provided by food;
- propose a direct ergogenic (work-enhancing) effect on sports performance, often through a pharmacological rather than a physiological effect;
- often rely on theoretical or anecdotal support rather than on documented support from scientific trials; and
- are generally not supported by sports nutrition experts, except where scientific trials have documented a significant ergogenic effect.

It is the use of these supplements that continue to escalate, and to cycle in and out of fashion. It is difficult for scientists and practitioners to stay abreast of the number of new products that emerge onto the market each year. In this chapter, we review the use and scientific support for a number of nutritional ergogenic aids that are of current interest. However, before this summary is presented, it is useful to understand the processes of government regulation of sports supplements, and the processes needed to provide suitable proof of performance enhancements resulting from the use of a product.

17.4 *THE SPORTS SUPPLEMENT INDUSTRY*

In Australia, the production and sale of sports supplements falls under the jurisdiction of two government bodies: the Australian and New Zealand Food Authority (ANZFA), which controls food products, and Therapeutic Goods Administration (TGA), which controls pills and other formulations marketed as therapeutic goods (Baylis et al. in press). Sports foods such as sports drinks, sports bars, sports gels and liquid meal supplements generally fall within Standards R9 and R10 of the ANZFA Foods Standards Code. These standards make provision for a range of acceptable formulations and permitted additives, as well as a list of permitted or compulsory education messages for presentation on product

packaging. It is the responsibility of individual states and territories to adopt these standards within their Food Laws, and to check and regulate that these laws are upheld. In reality, there are some sports foods, available on the Australian market, that do not meet these Standards, either by containing ingredients that are in contravention to the Code, or by carrying illegal claims. This is not the case for the larger number of mainstream products such as commercial sports drinks and bars. However, there are some sports foods, usually produced by smaller manufacturers targeting a niche market of athletes, which fail to comply. As ANZFA codes move towards the goal of developing a largely self-regulated industry of food manufacture and marketing, there is a greater likelihood that sports foods will contain non-permitted substances and/or incomplete or inaccurate labelling information.

According to our recent review (Baylis et al. in press), the availability and marketing of dietary supplements fitting the description of pills, powders or other non-food forms, fall within the jurisdiction of the TGA, under the Australian Therapeutic Goods Act 1989. This Act distinguishes two classes of products: drugs and therapeutic devices. Although dietary supplements may be packaged in a way suggesting medical or scientific rigour, as therapeutic devices they are regulated at an entirely different level to prescription pharmaceutical products. Therapeutic devices are further classified into categories of 'registrable' and 'listable' products, with almost all dietary supplements falling within the 'listable' or less regulated category. Although they need to comply with relevant statutory standards, for example to exclude ingredients banned by Australian Customs laws, they are considered low-risk self-medications and are not subjected to a comprehensive review of quality, safety and efficacy. They are expected to comply with Good Manufacturing Practice, and to advertising regulations, making limited therapeutic claims. In practice, these products receive little investigation of quality and advertising claims, unless they are the subject of serious complaints regarding health and safety issues (Baylis et al. in press).

Since supplements that are imported via mail order, Internet sales or personal importation are not subject to any scrutiny in the country of destiny, it is important for athletes to have a global understanding of the regulation of supplements. In other countries, including the United States, there is less regulation of the production and marketing of supplements than under the Australian system. In the US, all supplements (food and non-food forms) fall under the jurisdiction of the Food and Drug Administration. The Dietary Supplement Health and Education Act, passed in 1994, reduced the regulation of dietary supplements and broadened the category to include new ingredients such as herbal and botanical products, and constituents or metabolites of other dietary supplements. This Act shifted responsibility from the manufacturer to the FDA to enforce safety and claim guidelines. Since then manufacturers have not been required to comply with Good Manufacturing Practice.

In the absence or minimisation of rigorous government evaluation, quality control of supplement manufacturing is trusted to supplement companies. Large companies that produce conventional supplements such as vitamins and minerals, particularly to manufacturing standards used in the preparation of pharmaceutical products, are likely to achieve good quality control. This includes precision with ingredient levels and labelling, and avoidance of undeclared ingredients or contaminants. However, this does not appear to be true for all supplement types or manufacturers. Independent testing of 16 commercial dehydroepiandrosterone (DHEA) products revealed that only half the products contained the amount of DHEA stated on the product label, with actual levels varying between 0–150% of the stated content (Parasrampuria et al. 1998). Melatonin supplements have been found to fail to meet quality claims or delivery profiles stated on their labels (Hahm et al. 1999). Investigation of supplements containing Ephedra Sinica (Ma Huang) showed variability in alkaloid content between various brands of supplements, failure to report the Ephedra content on product labels, and batch-to-batch variability within the same product of nearly 140% (Gurley et al. 1998).

Although manufacturers are guided not to make unsupported claims about health or performance benefits from the use of supplements, product advertisements and testimonials show ample evidence that this aspect of supplement marketing is unregulated and exploited. For example, a survey of five issues of body building magazines found 800 individual performance claims for 624 different products within advertisements (Grunewald & Bailey 1993). Similarly large numbers of claims were found in another survey of health and body building magazines (Philen et al. 1992). The enthusiasm and emotive nature of these claims provide a false sense of confidence about the products. Most consumers are unaware that such advertising is not regulated. Therefore, they are likely to believe that claims about supplements are medically and scientifically supported, simply because they believe that untrue claims would not be allowed to exist. The undeserved credibility of supplement products derived from such claims not only continues to promote sales, but lures athletes into a false sense of security about aspects of quality and safety of products.

Later in this chapter, we will examine the process of undertaking rigorous scientific research, and appreciate that it is costly in time, money and resources. In the case of pharmaceutical products, which need to meet stringent regulations for therapeutic use and for safety, a company can expect to spend two to ten years and two million dollars in testing to gain approval for a new drug (Bucci 1998). Few of even the largest supplement manufacturers have the resources to comprehensively study the existing range of ergogenic aids and their combinations of use. Of course, it may not always be in the interests of a company to test some of their supplements in case they find negative results. In any case, a company might consider it wasteful to spend money on research, which they are not forced to do and which the market does not appear to demand.

17.5 WHAT IS PROOF?

The process of substantiating the performance benefits or outcomes from supplementation is difficult. In exploring the concept of 'proof', it will be seen that there are different levels of support that appeal to different audiences.

17.5.1 Scientific theories

A hypothesis is a line of enquiry that attempts to uncover or explain important relationships between factors. Historically, the dietary practices of athletes have evolved from hypotheses based on the contemporary understanding of exercise metabolism. Centuries ago, athletes were guided to eat the flesh of 'athletic' animals on the superstition that they might gain whatever factors underpinned the animal's prowess. Later on, high-protein diets were recommended, based on the belief that exercise was fuelled by protein, the key component of muscle. As our understanding of exercise metabolism has become more sophisticated, it has produced a huge number of new theories about factors that might play an important role in various reactions.

The current focus of the sports supplement industry is on compounds and nutrients that act as cofactors, intermediary metabolites or stimulants of key reactions in exercise metabolism. The rationale behind supplementation is simple and attractive: when the system is 'supercharged' with additional amounts of these compounds, metabolic processes will proceed faster or longer, thus enhancing sports performance. The marketing of most contemporary supplements is accompanied by sophisticated descriptions of metabolic pathways and biochemical reactions whose enhancement will lead to athletic success. To the scientist, a theory that links an increased level of a compound with performance enhancement may be a hypothesis that is worthy of testing, but it does not constitute proof or support for the idea. However, to the public, a hypothesis can be made to sound like a *fait accompli*, and athletes can be induced to buy products on the strength of a 'scientific breakthrough' which exists only on paper. In an era when sports scientists feel challenged by the apparent sophistication of the scientific theories presented by supplement companies, it is unlikely that athletes will possess sufficient scientific knowledge to be critical of these proposals.

While a 'supercharging' hypothesis may appear plausible at first glance, there are many reasons why it may not occur. Other issues to be considered include:

1. Will oral ingestion of the compound increase concentrations at the sites that are critical?
2. Does the present level of compound fall below the critical level for optimal metabolism?
3. Is this reaction the rate limiting step in metabolism or are other reactions setting the pace?

A scientific theory or hypothesis should be developed and fine-tuned before setting up a supplementation study. Since studies are expensive in time, money and resources, it is important that ideas that make it to trial are based on sound logic. But while a scientific theory should be developed in preparation for a study (or to explain the data collected in a study), it cannot be touted or accepted as evidence or practice until verified by actual research.

17.5.2 *Anecdotal support*

Testimonials provide a powerful force in the advertising and marketing of sports supplements, particularly products that target the body building or resistance training industry. This is also true of supplements sold through multilevel marketing schemes. Advertisements highlight the successful health or performance outcomes that people have achieved, allegedly as a result of their use of a supplement product. Often famous athletes or media stars supply these testimonials, but sometimes they also feature the exploits of 'everyday' people. Although cynics may note that people receive payment for these endorsements, testimonials, nevertheless, provide an emotive argument in favour of the supplement involved. Some athletes, including elite sportspeople, have financial interests in the supplement industry. For example, the body building guru, Joe Weider, not only publishes the magazine *Muscle and Fitness*, which has a world-wide circulation of over seven million readers, but also owns a number of supplement lines such as the Weider and Victory ranges. Other athletes may have a smaller role, by acting as distributors for supplement ranges that are sold through multilevel marketing.

However, not all testimonials are paid for. Since the sports world lends itself to the swapping of ideas, it is not surprising for an athlete to become interested in a supplement on the direct recommendation or hearsay from another supplement user. Successful athletes and teams are perpetually asked to nominate the secrets of their success in the media. In the following reviews of well-known ergogenic aids, we will see that public interest in a product can often be traced back to the recommendation or testimonial of a winning sportsperson.

It is hard for athletes to understand that success in sport results from a complicated and multifactorial recipe, and that even the most successful athletes may not fully appreciate the factors behind their prowess. In many cases, it is likely that the athlete has succeeded without the effects of the supplements they are taking—and in some cases perhaps, in spite of them! Unsupported beliefs and superstition are key reasons behind many decisions to use supplements. The idea that 'everyone is doing it' provides a powerful motivation to the athlete contemplating a new product. Sometimes, this manifests as a fear that 'others may have a winning edge that I don't have'. The *ad hoc* and undiscriminating patterns of supplement use reported by some athletes are testament to the power of 'word of mouth'.

Of course, the anecdotal experiences of athletes may be useful when considering the scientific investigation of a supplement. These experiences may support the

case for expending resources on a study, or help in deciding on protocols for using the supplement or for measuring the outcomes. However, by themselves they provide very weak support for the benefits of a supplement. Many of the benefits perceived by athletes who try a new supplement result from the psychological boost, which accompanies a new experience or special treatment.

17.5.2.1 *Placebo and other effects*

People who participate in a study experience various psychological responses. One response, known as the 'Hawthorne effect', occurs as a result of the 'special treatment' or monitoring received by subjects who know they are participating in a study. This was first identified during a series of management studies undertaken in the 1920s at the Hawthorne works of an electric company in the United States. The studies monitored the work output of a group of individuals under varying conditions of light. First, the lighting was increased in intensity and work output was found to increase—even in conditions exceeding the levels that were typically tolerated. However, when the light was then progressively reduced to very low levels, the work output of the subjects also improved. The researchers concluded that subjects improved their performance simply as a result of being involved in the experiment, or more likely, having their output closely monitored.

The Hawthorne effect predicts that athletes will improve their training or competition performances if they receive extra interest or monitoring. This is a common scenario for athletes who 'test' a new supplement, especially when this is done under the scrutiny of the coach, the manufacturer or other athletes in the group. These athletes might undergo some new testing or monitoring processes which they tackle more enthusiastically because they are under scrutiny and being encouraged. While the improvement in performance is a welcome outcome for the athlete, it is not necessarily the result of the supplement. The effect of the supplement can only be isolated by comparing the outcome with changes that occur in a 'control' group of athletes, who are similarly encouraged and monitored without receiving a new treatment.

The 'placebo' effect describes a favourable outcome arising simply from the belief that you have received a beneficial treatment. In a clinical environment, a placebo is often given in the form of a harmless but inactive substance or treatment that satisfies the patient's symbolic need to receive a 'therapy'. Despite our belief that the placebo effect is real and potentially substantial, only a few studies have tried to document the size or characteristics of the effect. Beecher (1959) reported that an injection of saline solution was 70% as effective as morphine in reducing pain for hospital patients. In another study, weightlifters who received injections described as anabolic steroids increased their gains in lean body mass despite receiving an inert (water) treatment (Arial 1972). A recent investigation where athletes were given either a sports drink or a sweetened placebo during a one-hour cycling time trial found that performance was affected by the information provided

to the subjects (Clark et al. in press). The placebo effect caused by thinking they were receiving a CHO drink allowed the subjects to achieve a small but worthwhile increase in performance of 4%. Being unsure of which treatment was being received increased the variability of performance, illustrating that the greatest benefits from supplement use occur when athletes are confident they are receiving a useful product (Clark et al. in press). Further work is needed to better describe the potential size and duration of this effect, and whether it applies equally to all athletes and all types of performance testing.

In the meantime, we can be satisfied that the placebo effect exists and may explain, at least partially, why athletes have a positive experience when trying a new supplement or dietary treatment. However, while the experience of other athletes provides a powerful incentive (or fear) to promote the use of nutritional ergogenic aids, it does not provide sufficient proof of beneficial effects.

17.5.3 *The scientific trial*

The scientific trial remains the 'gold standard' for investigating the effects of dietary supplements and nutritional ergogenic aids on sports performance. Scientists undertaking scientific trials should test the effects of the supplement in a context that simulates sports performance as closely as possible. Additional studies might be needed to elucidate the mechanisms by which these effects occur, but overall, sports science research must be able to deliver answers to questions related to real-life sport.

There are many variables that interfere with the outcomes of research. A researcher must design a protocol that eliminates extraneous or confounding variables and monitors a set of carefully chosen independent and dependent variables. Factors to consider include:

1. subject variables (characteristics including age, gender, level of training, experience with test protocols, psychological effects, nutritional status);
2. measurement variables (taking into account the validity and reproducibility of techniques, costs, availability of equipment, subjective versus objective measures, and the application to the hypothesis being tested);
3. study design (acute versus chronic supplementation protocols, laboratory versus field settings, blinding of subjects and researchers, crossover versus parallel group design, placebo control);
4. supplementation protocols (timing and quantity of doses, duration of supplementation period); and
5. statistical procedures (how best to examine the data generated by the studies).

At times, a series of studies might need to be undertaken to systematically address the range of questions that must be answered. It is beyond the scope of this chapter to fully explore the characteristics of good research design. However, the following ideas are useful in designing trials to test the effects of supplements on sports performance.

1. Recruit well trained athletes as subjects, unless the aim of the study is to test the effects of supplementation at different levels of training. The level of training may alter the effect of the supplement. Most importantly, it will affect the precision of measurements of performance. A homogenous group of well-trained athletes will generally show less intra- and inter- subject variability in performance, thus increasing the statistical power of the study.
2. Incorporate the use of a placebo treatment to overcome the psychological effect of supplementation. If practical, add a control (no treatment) trial so that the magnitude of the placebo effect can be determined.
3. Where possible, use repeated measures or 'crossover' design, in which each subject acts as their own control by undertaking both the treatment and placebo trials. This offers the benefit of increasing statistical power (reduced variability between treatments) and/or decreasing the number of subjects required. Take care to allow a suitable wash-out period so that the effects of the supplement are removed before the group which received the experimental treatment first begins the placebo trial.
4. Randomly assign subjects to treatment and placebo groups, and counter-balance the order of treatment, to remove the effect of time or training on study outcomes.
5. Employ a double-blind allocation of treatments to remove the subjective bias of both researcher and subjects. Blinding of the researchers will help to control the occurrence of the 'halo effect' where an observer, who believes an effect is likely, 'marks up' or encourages the performance of subjects.
6. Standardise the pre-trial training and dietary status of subjects.
7. Design the experimental conditions to mimic real-life practices of athletes. For example, allow athletes to consume a pre-event meal or to consume fluid and CHO during the performance according to usual or recommended practices.
8. Choose measurement variables that are sufficiently reliable to allow changes due to the supplement to be detected, and that are applicable to the hypothesis being tested.
9. Choose a performance test that is highly reliable and applicable to the real-life performances of athletes.
10. Choose a supplementation protocol that maximises the likelihood of a positive outcome. If a positive effect is found, doses can be manipulated in further trials to refine the optimal supplementation protocol.
11. Interpret results in light of what is important to sports performance.

17.5.3.1 *Are we testing the athlete's definition of performance enhancement?*

Although some of the features of a well designed scientific study have been outlined above, we must also consider whether the conditions and issues that satisfy a scientist are shared by the athlete. There are a number of ways in which scientific testing fails to provide answers to the questions that are asked by athletes.

When traditional scientific testing is applied to sports science, performance enhancement is tested according to the statistician's viewpoint. In human studies which involve a large number of experimental variables, typical outcome measurements of performance are highly variable. When variability between and within subjects is considerable, a large change or difference in performance (effect size) will be required to meet the 0.05 level of probability that is considered statistically significant. Therefore, most traditional intervention studies are biased towards detecting treatments that cause large changes, and ignoring treatments that produce only small changes.

Dwyer and Brotherhood (1981) first drew attention to this issue in sport, calculating relationships between sample sizes and critical levels of performance change, modelled from existing experimental-placebo trials of vitamin supplementation. Accepting the typical variability in performance measurements seen in these studies, they estimated that a subject sample of ~5000 would be needed to allow a 1–2% change in performance to become statistically significant (e.g. two to four seconds in a four-minute event such as the 1500 m run or 400 m swim). Conversely, a 12.5% improvement (e.g. 30 seconds) would be needed in a sample size of 20 before significance would be achieved. Of course, the critical change would be substantially smaller if within- and between-subject variation was reduced in such trials, and the use of repeated-measures design would reduce subject numbers. However, the point is clearly made that athletes might still be interested in improving their performance by margins that are smaller than considered 'significant' in most scientific studies.

So what is a substantial or worthwhile improvement for an elite athlete? In a sophisticated review, Hopkins and colleagues (1999) used simulations and the results of recent elite competitions to define the magnitude of the smallest enhancement that might be of interest to an elite athlete. Although the tight finishes that are typical of elite sports suggest that a tiny improvement in performance would make a difference, in fact, the situation is complicated by two important factors. These are the variation in an athlete's performance between events (also known as within-athlete variation), and the variation in performance between athletes in the same event (also known as between-athlete variation). Modelling suggests that an improvement equal to 0.4–0.7 times the typical within-athlete co-efficient of variation (CV) of performance will be worthwhile in changing the outcome of an event. For example, a 0.6 CV improvement would lift the athlete who is a true 4th place getter into winning 19% of races instead of 9% of races. The true top performer, who statistically wins 38% of races, will win 48% of races after a 0.3 CV enhancement. Analysis of competition shows that the typical CV of elite sports events ranges from 1–4%, although the top athletes are likely to be the most reliable performers (Hopkins et al. 1999).

According to Hopkins and colleagues, researchers would need a sample of 16–65 athletes in crossover studies, and 65–260 in a controlled experimental-placebo

study to delimit performance enhancements of this size. Since most studies are undertaken in laboratories, and enhancements in laboratory tests and real-life events may differ, they point out the need for validity studies that combine reliability data for laboratory performances and actual competition. At the very least, studies should be conducted using performance tests of known and high reliability. This review concludes by recommending features of study design that reinforce the guidelines provided in this chapter. The authors argue against using the traditional measures of statistical significance to interpret the results of intervention studies. Instead, they encourage researchers to report their performance outcomes expressed as a percentage change, with confidence limits to define the true outcome in a similar population (Hopkins et al. 1999). Researchers should then consider whether such changes are meaningful to the outcome of an elite sports event, or calculate sample sizes necessary to delimit the size and direction of worthwhile changes.

By incorporating our recommended features into research design (see Section 17.5.3), sports scientists should be able to detect performance changes of 2–5% and greater. Dietary interventions which produce performance enhancements of this order include providing fluid or CHO in events of one hour or greater (Below & Coyle 1995; Jeukendrup et al. 1997) or CHO loading before endurance events (Hawley et al. 1997). However, without performance tests of greater reliability and sample sizes that are larger than are currently typical of sports science research, scientists will be unable to rule out the merits of interventions that produce smaller enhancements. It is likely that some supplements may produce changes in this 'grey' area. A final but important issue is that the results of studies should only be applied to populations that are similar to the test group.

17.5.3.2 *Individual responses*

Notwithstanding the general variability in performance, there is evidence that some treatments cause a range of different responses in individual athletes. In some cases, the same intervention can produce favourable responses in some individuals, neutral responses in others, and sometimes, detrimental outcomes to another group. For example, research has identified that some athletes are 'non-responders' to caffeine or creatine supplementation (Graham & Spriet 1991; Greenhaff et al. 1994). It is useful to have metabolic or other mechanistic data to substantiate real differences in response, and to differentiate these from the general variability of performance. For example, it has been shown that subjects whose muscle creatine levels did not increase by at least 20% as a result of creatine supplementation did not show the functional changes and performance enhancements seen by the rest of the experimental group (Greenhaff et al. 1994).

Studies employing simple group analysis and small sample sizes are hampered by situations where there is true variability in the size and direction of the response to an intervention. Such studies will fail to detect a difference in performance, even

though this is a real outcome for some subjects in the group. Ideally, studies employing large sample sizes and co-variate analysis should be used; this approach will allow real changes to be detected and may also identify the characteristics of individuals which predict 'response' and 'non-response'. At present, such studies are rare.

17.6 NUTRITIONAL ERGOGENIC AIDS WITH CLEAR SCIENTIFIC SUPPORT

Well-conducted scientific trials have produced evidence that some ergogenic aids can enhance sporting performance. In this section, we review several products which enjoy such support. It should be noted that each work within a specific and narrow set of exercise situations, and should not be considered a universal sports supplement. Athletes need to be educated on appropriate situations of use, and appropriate supplementation protocols. Even so, studies show that some athletes are 'non-responders' to these protocols and some may actually experience side-effects or negative outcomes. Ideally, athletes should experiment with supplements before using them during important competitions, and will benefit from the assistance and monitoring provided by sports scientists.

17.6.1 *Creatine*

At the 1992 Barcelona Olympic Games, testimonials and gold-medal performances by British sprinters propelled a new supplement, creatine, into the limelight. Scientists were intrigued by the timely publication of a study which showed increases in muscle creatine stores following the oral intake of large doses of creatine (Harris et al. 1992). Since then, creatine has become the fastest selling and best-researched ergogenic aid. It is not often that scientists and athletes are excited by the same product. The coincidental rise of the Internet has assisted the rapid spread of scientific and testimonial information.

Although some lay publications and manufacturers have labelled creatine as a 'legal steroid', this is an incorrect and unfair comparison. In fact, creatine is a muscle fuel, and the ability of creatine supplementation to increase muscle creatine stores makes it similar to CHO loading. Creatine (methylguanidine-acetic acid) is a compound derived from amino acids which is stored primarily in skeletal muscle at typical concentrations of 100–150 mmol/kg/dry weight (dw) of muscle. About 60–65% of this creatine is phosphorylated. Creatine phosphate (CrP) provides a rapid but brief source of phosphate for the resynthesis of ATP during maximal exercise, and is therefore an important fuel source in maximal sprints of 5–10 seconds. Other functions of creatine phosphate metabolism are the buffering of hydrogen ions produced during anaerobic glycolysis and the transport of ATP, generated by aerobic metabolism, from the muscle cell mitochondria to the cytoplasm where it can be utilised for muscle contraction. Creatine metabolism is covered in more detail in Chapter 2 and in the following reviews of creatine

metabolism and supplementation: Spriet 1997; Williams et al. 1998; Juhn & Tarnopolsky 1998a, 1998b; Kraemer & Volek 1999; Demant & Rhodes 1999; Greenhaff 2000.

The daily turnover of creatine, eliminated as creatinine, is approximately 1–2 g/d. This can be partially replaced from dietary creatine intake, found in animal muscle products such as meat and eggs, and typically consumed in amounts of ~1–2 g/d in an omnivorous diet. Additional creatine needs are endogenously synthesised from arginine, glycine and methionine, principally in the liver, and transported to the muscle for uptake. Creatine is transported into the muscle against a high concentration gradient, via saturable transport processes that are stimulated by insulin (Green et al. 1996a, 1996b). High dietary intakes temporarily suppress endogenous creatine production. Vegetarians who do not consume a dietary source of creatine are believed to have a reduced body creatine store, suggesting that they do not totally compensate for the lack of dietary intake (Green et al. 1997). The reason for the variability of muscle creatine concentrations between individuals is uncertain. There are some suggestions that females typically have higher muscle creatine concentrations (Fosberg et al. 1991), and it appears that creatine stores decline with ageing. The effect of training on creatine concentrations also requires further study.

In 1992, Harris and colleagues published the watershed study that showed that muscle creatine levels were increased as a result of supplementation with repeated doses of creatine, large enough to sustain plasma creatine levels above the threshold for maximal creatine transport into the muscle cell (Harris et al. 1992). They used a protocol providing four to six doses of 5 g creatine (monohydrate) for five days to increase total muscle creatine concentrations by 20%, and reach an apparent muscle threshold of ~150–160 mmol/kg dw. About 20% of the increased muscle creatine content was stored as CrP and saturation occurred after two to three days. Increases in muscle creatine stores were greatest in those who had the lowest pre-supplementation concentrations and when coupled with intensive daily exercise (Harris et al. 1992).

Although this discovery appears to be recent, in fact, studies showing that oral creatine doses are largely retained in the body were available over 70 years ago (Chanutin 1926). However, it is only now that muscle biopsy procedures and imaging techniques are available to enable scientists to monitor muscle stores of creatine and investigate the success of creatine-loading protocols. Over the past decade a number of studies have refined our knowledge of supplementation protocols. Rapid loading is achieved by consuming a daily creatine dose of 20–25 g, in split doses, for five days. Alternatively, a daily dose of 3 g/d will achieve a slow loading over 28 days (Hultman et al. 1996). Elevated muscle creatine stores are maintained by continued daily supplementation of 2–3 g (Hultman et al. 1996). Across studies there is evidence that the creatine-loading response varies between individuals, with ~30% of individuals being ‘non-responders’ or failing to significantly increase muscle creatine stores

(Spriet 1997; Greenhaff 2000). Co-ingestion of substantial amounts of CHO (75–100 g) with creatine doses has been shown to enhance creatine accumulation (Green et al. 1996a, 1996b) and to assist individuals to reach the muscle creatine threshold of 160 mmol/kg dw. Creatine appears to be trapped in the muscle: in the absence of continued supplementation, it takes ~4–5 weeks to return to resting creatine concentrations (Hultman et al. 1996). Many studies have reported an acute gain in body mass (BM) of ~1 kg during rapid creatine loading. This is likely to be primarily a gain in body water, and is mirrored by a reduction in urine output during the loading days (Hultman et al. 1996).

Many studies have investigated the effect of creatine supplementation on muscle function exercise and performance. Studies vary according to the characteristics of subjects (gender, age, training status), the mode of exercise, and whether supplementation involved an acute loading intervention, or a chronic effect on training adaptations. In the Appendix to this chapter we have summarised the results of studies fully published in peer-reviewed journals in Tables 17.5 (acute supplementation) and 17.6 (chronic supplementation). All studies were undertaken using an experimental-placebo design unless otherwise indicated. This is the most suitable design for such studies since the wash-out period is of a lengthy duration. We offer the following summary of this literature, and of recent reviews (Spriet 1997; Juhn & Tarnopolsky 1998a, 1998b; Williams et al. 1998; Demant & Rhodes 1999; Kraemer & Volek 1999; American College of Sports Medicine 2000; Greenhaff 2000).

1. The major benefit of creatine supplementation appears to be an increase in the rate of creatine phosphate resynthesis during the recovery between bouts of high-intensity exercise, producing higher creatine phosphate levels at the start of the subsequent exercise bout. Creatine supplementation can enhance the performance of repeated 6–30 sec bouts of maximal exercise, interspersed with short recovery intervals (20 sec–5 min), where it can attenuate the normal decrease in force or power production that occurs over the course of the session.
2. Oral creatine supplementation cannot be considered ergogenic for single-bout or first-bout sprints because the likely benefit is too small to be consistently detected.
3. The exercise situations that have been most consistently demonstrated to benefit from creatine supplementation are laboratory protocols involving isolated muscular efforts or weight-supported activities such as cycling.
4. Evidence that creatine supplementation is of benefit to endurance exercise or weight-bearing activities (e.g. running and swimming) is absent or inconsistent.
5. Performance responses to creatine supplementation vary considerably between subjects in a study, and between studies.
6. In theory, acute creatine supplementation might be beneficial for a single event in sports involving repeated high-intensity intervals with brief recovery periods. This description includes team games and racquet sports. Similarly, chronic creatine supplementation may enhance training performance and long-term

adaptation to exercise programs based on repeated high-intensity exercise. These benefits may apply to the across-season performance of athletes in team and racquet sports, as well as the preparation of athletes who undertake interval training and resistance training (e.g. swimmers and sprinters).

7. These benefits remain theoretical since few studies have been undertaken with elite athletes or as 'field studies'. Performance enhancements will only occur in weight-bearing and weight-sensitive sports (e.g. light-weight rowing and rock climbing) if gains in muscular output compensate for increases in body mass. Performance enhancements may not always occur in complex games and sports; even if changes in strength or speed are achieved by creatine-assisted training, these may not translate into improvements in game outcomes (i.e. goals scored).
8. Whether the long-term gains in muscle mass reported in studies of resistance training are caused by direct stimulation of increased myofibrillar protein synthesis by creatine, enhanced ability to undertake resistance training, or a combination of both factors, remains to be determined.

Whether there are side-effects from long-term use of creatine, particularly with the large doses associated with rapid loading, remains to be determined. To date, there are anecdotal reports of nausea, gastrointestinal upset, headaches and muscle cramping/strains linked to some creatine supplementation protocols. Some of these adverse effects are plausible, particularly in light of increased water retention within skeletal muscle (and perhaps brain) cells. At this time, however, studies have not found evidence of an increased prevalence or risk of these problems among creatine users. Some concern is directed to long-term creatine users, particularly those who self-medicate with doses far in excess of the recommended creatine usage protocols in this chapter. Since creatine may affect fluid balance or fluid distribution within various body compartments, athletes are warned to pay additional attention to fluid needs in hot weather. It may also be prudent to avoid rapid loading regimens in situations where there is a high risk of dehydration. Similarly, rapid creatine loading is unwise for athletes who need to meet weight (body mass) targets. Although it is commonly suggested that creatine supplementation may cause renal impairments, the only case report of such a problem occurred in a patient with pre-existing renal dysfunction (Prichard & Kalra 1998). Poortmans and Francaux (1999) found creatine intake had no detrimental effects on renal responses. Nevertheless, until long-term and large population studies can be undertaken, bodies such as the American College of Sports Medicine (2000) have taken a cautious view on the benefits and side-effects of creatine supplementation.

17.6.2 *Caffeine*

Caffeine is the best known member of the methyl xanthines: a family of naturally occurring stimulants found in the leaves, nuts and seeds of a number of plants. Major dietary sources of caffeine, such as tea, coffee, chocolate and cola drinks,

typically provide 30–100 mg of caffeine per serve, while some non-prescriptive medications contain 100–200 mg of caffeine per tablet.

The physiological actions of caffeine are well documented and include stimulation of the central nervous system, cardiac muscle, diuresis, and epinephrine release and activity. Caffeine has several effects on skeletal muscle involving calcium handling, sodium-potassium pump activity, elevation of cyclic-AMP and direct action on enzymes such as glycogen phosphorylase. Increased catecholamine action, and the direct effect of caffeine on cyclic-AMP, may both act to increase lipolysis in adipose and muscle tissue, causing an increase in plasma-free fatty acid concentrations, and increased availability of intra-muscular triglyceride. It has been proposed that an increased potential for fat oxidation during moderate-intensity exercise promotes glycogen sparing. Caffeine may also influence athletic performance via central nervous system effects, such as a reduced perception of effort or an enhanced recruitment of motor units. Breakdown products of caffeine such as paraxanthine and theophylline may also have actions within the body. Caffeine supplementation is a fascinating and complex issue to investigate due to the difficulty in isolating individual effects of caffeine, and the potential for variability between subjects. Further information on the effects of caffeine can be found in the following reviews: Graham et al. 1994; Tarnopolsky 1994; Graham 1997.

Interest in the effect of caffeine on exercise performance dates back almost a century, but it is only the last forty years that controlled studies have been conducted and extensively reviewed. Early research in the 1970s focussed on the effects of caffeine on metabolism and performance during endurance events. A resurgence of interest in caffeine supplementation during the 1990s expanded the focus of studies to include exercise performances such as sprints (< 90 secs), and short (~5 min) and long (~20 min) events involving high intensity effort. Studies of caffeine supplementation and performance have been summarised in Tables 17.7 and 17.8 in the Appendix to this chapter, with divisions into categories based on the type of exercise protocol and the timing of caffeine intake.

In 1984, caffeine was added back to the International Olympic Committee (IOC) list of banned substances, as a restricted substance. Caffeine usage is monitored via a single urine sample taken post-event, and concentrations of caffeine, representing the 1–5% of an ingested caffeine dose that is excreted unchanged, are measured. Athletes whose urinary concentrations of caffeine exceed 12 μg/mL are considered to have 'doped'. It has been noted that there is a wide intra- and inter-individual variability in urinary caffeine concentrations (Birket & Miners 1991). However, few people exceed these levels as a result of normal coffee drinking and dietary practices (Delbeke & Debackere 1984). Studies document that at caffeine doses of 5–6 mg/kg, which produce ergogenic benefits in a number of exercise protocols, positive drug test outcomes are unlikely (Pasman et al. 1995). Furthermore, increased doses of 9 mg/kg at which a substantial percentage of subjects begin to show 'illegal' urinary caffeine concentrations do not

produce an additional performance advantage. Therefore, athletes may be able to explore ergogenic benefits from caffeine supplementation while keeping well within the doping laws of elite sport.

We offer the following summary of our present knowledge about caffeine supplementation and exercise performance based on Tables 17.7 and 17.8 and reviews of caffeine (Graham et al. 1994; Tarnopolsky 1994; Graham 1997; Spriet 1997).

1. Caffeine supplementation causes various effects on a range of body tissues, at a range of doses. Overall, beneficial effects begin to be detectable at intakes of 3 mg/kg. The optimal dose for a variety of effects appears to be ~5–6 mg/kg. Above this intake no further benefits are seen, and there is an increased risk of side-effects or a 'positive' urinary caffeine level (> 12 μg/mL).
2. There appears to be a range of exercise activities that may benefit from caffeine supplementation. Further research is needed to clarify the applied situations in which benefits are seen and to fine-tune supplementation protocols.
3. Most studies of caffeine supplementation have used the protocol of ingesting caffeine one hour pre-event. Recently, benefits have been seen when caffeine was fed in association with CHO at modest doses throughout the exercise bout (Kovacs et al. 1998). Further research is needed to explore the timing of doses, particularly in events of 60 min or greater.
4. At present there is no clear mechanism to explain beneficial effects of caffeine supplementation. If glycogen sparing occurs during submaximal exercise events, it appears to be limited to the first 15–20 mins of exercise. Epinephrine changes also do not appear to be critical for performance changes.
5. The effects of caffeine supplementation differ between individuals. Some people are non-responders and some people experience negative side-effects such as tremors, increased heart rate and headaches.
6. Factors that may explain differences between performance outcomes in various studies include:
   - mode of exercise (cycling studies seem to be more likely to show benefits than running studies);
   - training status of subjects (exercise performances of well-trained subjects are more reliable and may allow smaller changes to be detected);
   - habitual caffeine intake of subjects;
     nutritional status of subjects (fasting versus pre-event CHO feeding, CHO intake during exercise versus water); and
   - gender.
     At present the role of each of these factors is not clear.
7. Individuals who want to try caffeine supplementation during their competition performances should experiment in training situations or less important competitions to determine whether there is a safe, legal and efficacious protocol for their event.

17.6.3 *Bicarbonate*

As far back as the 1930s, exercise scientists discovered that the intake of acid salts by runners decreased blood pH and impaired performance of high intensity exercise, while the addition of alkalotic therapies improved running time (Denig et al. 1931; Dill et al. 1932). Anaerobic glycolysis provides the primary fuel source for exercise of near maximal intensity lasting longer than approximately 20–30 sec. The total capacity of this system is limited. A progressive increase in the acidity of the intracellular environment, caused by the accumulation of lactate and hydrogen ions, results in muscular fatigue and an inability to maintain the exercise intensity. Although the precise mechanism is not fully clear, it is believed that the intracellular accumulation of hydrogen ions directly inhibits muscle contraction by impairing the role of calcium in this process. It may also reduce the activity of glycolytic enzymes such as phosphofructokinase (see Chapter 2, Figure 2.1). When intracellular buffering capacity is exceeded, lactate and hydrogen ions diffuse into the extracellular space, perhaps aided by a positive pH gradient. Bicarbonate represents one of the most important extracellular buffers.

In theory, an increase in blood bicarbonate levels should delay the onset of muscular fatigue during prolonged anaerobic metabolism, by increasing extracellular buffering capacity and the muscle's ability to dispose of excess H^+ ions. 'Soda loading' or 'bicarbonate loading', the ingestion of sodium bicarbonate to increase blood bicarbonate levels, has been trialled by athletes and studied by scientists for over 70 years. Sodium citrate has also been used as a buffering agent. The science and practice of ingesting buffering salts have been conflicting and inconsistent.

The general protocol for bicarbonate loading is to ingest 0.3 g of sodium bicarbonate per kilogram BM, one to two hours prior to the exercise task. Sodium bicarbonate is available as the household product 'bicarb soda' or as pharmaceutical urinary alkaliners such as 'Ural'; the typical athlete requires a dose of approximately 4–5 teaspoons. Bicarbonate loading is not considered to pose any major health risk, although some individuals suffer gastrointestinal distress such as cramping or diarrhoea. Consuming the sodium bicarbonate with plenty of water (e.g. a litre or more) may help to prevent hyperosmotic diarrhoea. Sodium citrate is also usually ingested in doses of 0.3–0.5 g/kg BM.

Theoretically, bicarbonate loading might enhance the performance of athletic events that are otherwise limited by excess hydrogen ion accumulation. These include events conducted at near maximum intensity for the duration of 1–7 min—for example 400–1500 m running, 100–400 m swimming, kayaking, rowing and canoeing events. Sports that are dependent on repeated anaerobic bursts may also benefit from bicarbonate loading. Neither the IOC, nor the governing bodies of sport, bans bicarbonate and citrate loading. However, it might be argued that it contravenes the spirit of anti-doping rules, which ban the use of 'any physiological substances taken in an attempt to artificially enhance performance'. Alternatively,

one could argue that bicarbonate loading is similar to CHO loading or creatine loading, and that it represents an extension of dietary practice. In any case, it is difficult to detect the use of bicarbonate- or citrate-loading strategies by athletes, since urinary pH varies according to dietary practices such as vegetarianism and high CHO intake (Heigenhauser & Jones 1991).

There have been at least 40 studies of the effects of bicarbonate loading on athletic or exercise performance in humans (for reviews see Heigenhauser & Jones 1991; Linderman & Fahey 1991; Maughan & Greenhaff 1991; McNaughton 2000). It is beyond the scope of this chapter to review all of these studies. An alternative approach to summarise a large but narrowly defined group of studies is to undertake a meta-analysis. This statistical treatment is able to integrate and quantify results to uncover trends or relationships that might not be evident in individual studies, or from the subjective bias of a narrative review. This is a particularly useful way to review the bicarbonate-loading literature since it compacts the large number of individual studies, overcomes the limitations of small sample sizes, and helps to overview the problem of inconsistent and contradictory findings. Such a meta-analysis was published in 1993, and included 29 randomised double-blind crossover trials, published in English, with a primary purpose of investigating the effect of bicarbonate loading on physical performance (Matson & Tran 1993).

Since several studies compared multiple doses or modes of exercise, a total of 35 effect sizes were available for study (Matson & Tran 1993). A total of 285 subjects were represented, with the vast majority being healthy male, college-aged students. There was some variation in the protocol of bicarbonate loading, with different doses and times of ingestion being employed. While cycling was the most frequently used mode of exercise, performance was measured in a variety of ways. Performance times varied from 30 sec to 5–7 min of near maximal intensity, and included repeated intervals of one minute with short rest times between. Performance outcomes included changes in power over a given time period, total work performed in a specified time, or time to exhaustion at a specific exercise intensity. Only five studies included in this meta-analysis measured performance in an outcome that would mimic real-life sport, using trained subjects. These studies are summarised in Table 17.9 (see Appendix 17) along with more recent studies which feature a sports-specific design. It is worth noting some recent studies featured in Table 17.9 that showed that bicarbonate loading enhanced cycling performance of approximately one hour duration (Potteiger et al. 1996; McNaughton et al. 1999).

The 1993 meta-analysis of bicarbonate literature concluded that the ingestion of sodium bicarbonate has a positive effect on exercise performance. The weighted effect size was 0.44, meaning that the mean performance of the bicarbonate trial was, on average, 0.44 standard deviations better than the placebo trial. In statistical terms, this is considered a moderate effect size. Factors that were associated with a larger effect size included mode of exercise (exercise producing a larger anaerobic component, measuring time to exhaustion, or involving repeated work intervals)

and large doses of sodium bicarbonate. In trials that measured time to exhaustion there was a mean increase in duration of 27% ± 20%. It was noted that strategies that reduced the variability in performance, such as using a homogenous subject pool, particularly of well-trained athletes, would significantly improve the strength of the statistical analysis.

There was only a weak relationship reported between the alkalinity (increase in pH and bicarbonate) attained in the bicarbonate trial and the performance outcome. However, the greater the level of metabolic acidosis achieved during the exercise, the greater the ergogenic effect. Thus, it was concluded that a key factor associated with an ergogenic effect is the attainment of a threshold pH gradient across the cell membrane, resulting from the accumulation of intracellular H^+ as well as the extracellular alkalosis. Significant variability within studies suggests that bicarbonate ingestion has an individual effect on different subjects, and that the effect on performance is more complicated than the simple mechanisms suggested above. It has been suggested that an anaerobically trained athlete would have better intrinsic buffering capacity, and would be less likely to show a positive effect from bicarbonate loading. However, the meta-analysis provided no clarification of this theory. In concluding their findings, Matson and Tran (1993) recommended that further research be undertaken, particularly with subjects matched in VO_{max} anaerobic capacity, fibre type and performance times, and with exercise protocols that are specific to the subjects' training as well as actual athletic performance.

It is worth considering that an improvement in actual athletic performance (as studied by the trials summarised in Table 17.9) might only be achieved if the athlete is able to pace their performance to exploit the increased buffering capacity. Studies that fail to show an enhancement effect may result from exercise modes that are too short or insufficient in intensity to produce a critical H^+ load, or from the failure of the athlete to judge a challenging pace. Sports performance that might benefit from bicarbonate loading may therefore be very specific to the type of event, the individual athlete, and their ability to challenge their buffering system. Until further research can clarify the range of exercise activities that are potentially capable of performance enhancement, the individual athlete is advised to experiment in training to judge their own case. Experimentation in a competition-simulated environment should be considered crucial; the athlete needs to discover not only the potential for performance improvement, but also the likelihood of unwanted side-effects.

17.7 NUTRITIONAL ERGOGENIC AIDS WITH MIXED SCIENTIFIC SUPPORT

17.7.1 Antioxidant supplements

Exercise has been linked with an increased production of free oxygen radical species capable of causing cellular damage. A sudden increase in training stress (such as an increase in volume or intensity) or a stressful environment (training in hot

conditions or at altitude) is believed to increase the production of these free oxygen radicals, leading to an increase in markers of cellular damage. Supplementation with antioxidant vitamins such as vitamin C or vitamin E is postulated to increase antioxidant status and provide protection against this damage (see Chapter 12, Section 12.6.2).

The literature on the effects of antioxidant supplementation on antioxidant status, cellular damage and performance is complex and confusing. Some, but not all, studies of acute supplementation during periods of increased stress may provide bridging protection until adaptation processes can increase the host antioxidant status (for review see Dekkers et al. 1996; Packer 1997). It is possible that benefits may occur at a subtle and cellular level that are too small to translate into detectable performance benefits. Whether ongoing supplementation is necessary for optimal training adaptations and competition performance of athletes is similarly unknown, and any benefits may be too small to detect.

17.7.2 *Protein and amino acids*

Protein metabolism and protein requirements for training and competition are summarised in Chapter 5. This chapter concluded that protein supplements are expensive and are unnecessary for achieving an increase in muscle mass or strength. However, mixed-macronutrient products, such as liquid meal supplements, could be useful in situations where a compact source of CHO and protein was desirable. Such scenarios might include:

1. meeting the high-energy needs of an athlete undertaking heavy training, a growth spurt or an increase in muscle mass; and
2. a post-exercise recovery meal promoting enhanced protein status and glycogen restoration simultaneously.

In both situations, a liquid meal supplement might provide a practical alternative or adjunct to everyday foods. The cost of such supplements is usually considerably less than high-protein or all-protein products, but is typically greater than normal foods. Nevertheless, this expense may be justified when convenience is an important issue in achieving nutrient intake goals.

Several individual amino acids, or amino acid groups, have been singled out for special attention in sports nutrition. During the 1980s, preparations of individual amino acids were the most successfully marketed 'designer' supplement, despite a lack of evidence that 'free-form' preparations of amino acids were superior in digestion/absorption than amino acids found in intact proteins (i.e. in everyday foods). In the 1990s, special forms of 'ion-exchanged whey protein powder' received a similar but unsubstantiated hype. Contemporary products include tablets or powders containing individual amino acids as the sole ingredients, as well as general sports supplements (sports drinks, liquid meal powders, bars) fortified with additional amino acids. Many of these specialised amino acid products are

expensive and provide amino acid intakes that can easily be consumed from everyday foods at more reasonable cost. We will now explore the evidence that particular amino acids have a special role in athletic performance and recovery from exercise.

17.7.2.1 *Branched-chain amino acids (BCAAs)*

Interest in the branched-chain amino acids (leucine, isoleucine and valine) is based on their important role in protein metabolism and on their hypothesised role in the development of central fatigue. BCAAs in the muscle are able to transaminate pyruvate to form alanine, which is recycled to glucose in the liver via the Cori cycle (see Chapter 5, Section 5.2.2). There is significant oxidation of these amino acids during exercise, and tracer studies that follow leucine kinetics are often used as an estimation of protein turnover. Supplements containing BCAAs are claimed to enhance recovery after exercise, although there is no proof that BCAAs are unique in promoting an enhanced reversal of protein catabolism. Instead, intake of CHO and protein, as provided by everyday foods and supplements such as liquid meal preparations, is the recommended dietary strategy for post-exercise recovery (see Chapter 15).

Supplementation with BCAAs during exercise has been claimed to reduce or delay the onset of 'central fatigue', described as fatigue emanating from the central nervous system rather than the muscle. Over the last decade, theories about the role of neurotransmitters in the psychological sensations of mood, drive, pain, weariness and fatigue have become popular. It has been suggested that neurochemicals such as serotonin, dopamine and norepinephrine play a role in the determination of exercise performance. Since it is difficult to undertake a direct examination of brain function during exercise in humans, we are reliant on monitoring indirect markers such as plasma levels of neurotransmitter precursors, or monitoring the effects of drugs that are known agonists or antagonists of neurotransmitter function.

Briefly, it has been hypothesised that central fatigue occurs due to increased brain levels of serotonin, which result from greater amounts of free (unbound) tryptophan being able to cross the blood–brain barrier (for review see Davis 1995). A key factor in this increased uptake is an increase in the plasma ratio of free tryptophan to BCAAs (tryptophan:BCAA), which compete for the same transporters into the brain. The ratio changes during exercise as BCAAs are oxidised by the muscle. However, it also changes because the rise in free fatty acids (FFAs), which occurs during exercise, displaces tryptophan from its binding site on the albumin molecule and increases the plasma concentration of free tryptophan. It has been theorised that supplementation of BCAAs during exercise might prevent the drop in plasma BCAAs, attenuate the rise in free tryptophan:BCAA, and reduce the likelihood of fatigue arising from increased brain serotonin concentrations.

Studies that have investigated the effect of BCAA supplementation immediately before or during endurance exercise are summarised in Table 17.10 (see Appendix 17). At first glance it seems that this might be a successful strategy to enhance

sports performance. However, several of the studies summarised in this table can be criticised on methodological grounds. For example, the running studies by Blomstrand and colleagues (1991) only found a performance enhancement by undertaking an artificial statistical procedure whereby 'randomly selected' groups of subjects were subdivided according to an arbitrary finishing time. There is no rationale to justify this classification and no proof that the random allocation of subjects would not have produced slight mismatches in the calibre of each group, independent of the intervention received. The application of the cognitive test to sports performance has also been questioned. Finally, in these studies the researchers failed to control factors such as fluid and CHO intake during exercise (Davis 1995). Other studies have failed to confirm an enhancement in the performance of prolonged exercise following BCAA supplementation.

There are several arguments against an endorsement of BCAA supplementation during exercise. The first issue is that many studies have only compared BCAA supplementation with a water placebo. Interestingly, the ingestion of CHO during exercise minimises the unfavourable change in plasma free tryptophan:BCAA. Carbohydrate ingestion during exercise suppresses the rise in FFA concentrations, thus attenuating the increase in free tryptophan concentrations (Davis et al. 1992). Thus, CHO intake during endurance exercise provides an effective strategy against both peripheral and central mechanisms of fatigue. Studies comparing the co-ingestion of CHO and BCAAs with the intake of CHO alone are needed to settle the issue of BCAA supplementation. To date, convincing evidence of this type is lacking. Furthermore, the rise in plasma ammonia concentrations that accompanies the intake of substantial amounts of BCAAs must be considered. Ammonia is known to be toxic to the brain and muscle and may produce its own effect on exercise capacity and performance. At present, despite an intriguing theory of potential performance benefits, there is no substantial proof that BCAA supplementation enhances exercise performance.

17.7.2.2 *Arginine, ornithine and lysine*

Arginine, ornithine and lysine have been claimed individually, and in combinations, to promote the release of growth hormone, leading to an increase in muscle mass and a decrease in body fat. Arginine and ornithine have also been purported to stimulate insulin release when consumed in combination with CHO, enhancing anabolic activities including glycogen storage. These amino acids have been marketed as 'legal anabolic compounds', recovery agents and stimulators of muscle growth. Two studies by Elam and associates (1988, 1989) are often cited in support of gains in muscle size and strength in subjects undertaking body building training while supplementing with arginine and ornithine (2 g/d). However, these studies are flawed in design, and lacking an appropriate measure of pre- and post-measures in the 'control' group. Therefore, it is not possible to demonstrate whether any purported changes in this study are due to the amino acid supplementation.

Data supporting the stimulation of growth hormone following oral intake of amino acids are sketchy and inconsistent. Lemon (1991) found only modest changes in growth hormone release following ingestion of large amounts of arginine and ornithine (up to 20 g/d). Furthermore, he reported that growth hormone release was greater following heavy resistance training, and was not further stimulated by the addition of these amino acids. Studies at the Australian Institute of Sport also failed to find improvements in growth hormone release following intake of 3–4 g/d of these amino acids (Fricker et al. 1988, 1991). In a series of studies investigating the interaction of training, food and supplements on acute or late-night release of growth hormone, researchers found that exercising in a fasted state produced the greatest growth hormone stimulus (Fricker et al. 1988, 1991). Finally, inconsistent effects on growth hormone release were observed over three hours following the intake of ~2 g of arginine/lysine and ornithine/tyrosine amino acid combinations by body builders (Lambert et al. 1993).

Effects of amino acid intake on insulin responses are equally unconvincing. Studies of ornithine supplementation (170 mg/kg) and post-exercise arginine supplementation (80 mg/kg/h) in combination with CHO feedings have failed to find an enhanced insulin response (Bucci et al. 1992; Yaspelkis & Ivy 1999). Chronic intake of arginine/lysine supplements (132 mg/kg LBM) for ten weeks failed to change oral glucose tolerance test parameters in inactive subjects or subjects undertaking resistance training (Gater et al. 1992a). It also failed to alter body composition or strength changes (Gater et al. 1992b).

Overall, it appears that the oral intake of amino acids fails to achieve the hormonal stimulation seen when amino acids are intravenously administered in clinical situations. Furthermore, very high intakes of some amino acids are associated with cramping and diarrhoea (Yaspelkis & Ivy 1999). To date there is no convincing evidence to prove that amino acids supplements promote an enhanced hormonal response and/or an increased response to resistance training. Furthermore, the 2–3 g doses recommended by many amino acid manufacturers can easily be obtained by eating common foods such as milk, yoghurt and eggs.

17.7.2.3 *Glutamine*

Glutamine is the most abundant amino acid in muscle and plasma. It is considered important or essential for many activities of the immune system such as lymphocyte-activated natural killer cell activity, lymphocyte proliferation and macrophage phagocytosis. Glutamine is used at a high rate by these cells as an oxidative fuel and as a source of purine intermediates. Plasma glutamine concentrations are maintained by the balance between glutamine utilisation and release. Plasma glutamine concentrations fall during prolonged exercise and other catabolic states such as trauma or surgery, possibly as a result of increased liver uptake for gluconeogenesis. Glutamine concentrations may remain lowered for a

period during the recovery phase, depending on the intensity and duration of the exercise (for review see Walsh et al. 1998).

It has been suggested that the acute effects of several bouts of exercise may be cumulative, since some researchers have found that over-trained athletes have lower plasma glutamine values than healthy athletes (Rowbottom et al. 1996). Indeed, it has been theorised that a chronic glutamine debt may be responsible for the immunosuppression suffered by some athletes, and that glutamine supplementation may overcome the problems of over-training, or the impaired immunity suffered by athletes undertaking repeated bouts of heavy training. However, the data showing a link between lowered glutamine levels and susceptibility to illness in athletes are not consistent (Mackinnon et al. 1996; Kingsbury et al. 1998). One study has reported that glutamine supplementation after a heavy exercise session decreased the incidence of infection suffered during the following week (Castell et al. 1996). However, the interpretation of these results should be carried out with caution since there are flaws in the methodology, such as the failure to monitor plasma glutamine concentrations and the reliance on self-reports of illness. At present the data suggest that glutamine supplementation is only of benefit for athletes who show a true glutamine deficiency, and that this problem is less common than originally proposed. Therefore, glutamine supplementation does not provide a general cure or prevention for immune problems suffered by athletes.

17.7.3 *Glycerol*

Glycerol, a three-carbon alcohol, provides the backbone to triglyceride molecules and is released during lipolysis (see Chapter 16, Sections 16.2 and 16.5). Within the body it is evenly distributed throughout fluid compartments and exerts an osmotic pressure. When consumed orally, it is rapidly absorbed and slowly metabolised via the liver and kidneys. When consumed in combination with a substantial fluid intake, the osmotic pressure will enhance the retention of this fluid and expansion of the various body fluid spaces. Effective protocols for glycerol hyperhydration are 1–1.5 g/kg glycerol with an intake of 25–35 mL/kg of fluid. Typically, this allows a fluid expansion or retention of ~600 mL above a fluid bolus alone, by reducing urinary volume. A thorough review of glycerol and its role as a hyperhydrating agent is provided by Robergs (1998).

Glycerol can be consumed by using commercially available glycerine solutions, or more recently, special hyperhydration supplements targeted at athletes. Glycerol hyperhydration may be useful as a preparation strategy for events that are likely to challenge fluid status and thermoregulation (see Chapter 14). This includes exercise of high intensity and/or hot and humid environments, where sweat losses are high and opportunities to replace fluid are substantially less than the rates of fluid loss. It may also be useful as a rehydration agent for situations requiring the quick recovery from a moderate to large fluid deficit. This includes situations where there is a short recovery period between events or important training sessions and

the athlete has a significant fluid loss from the first session. Athletes who dehydrate to make weight might also benefit by enhancing their fluid retention during the recovery period between the weigh-in and competition (see Chapter 8).

Although glycerol may provide a logical aid for the rapid reversal of dehydration in the situations described above, such protocols have not been studied. Therefore, we can not be certain of any benefits or side-effects, or ways in which protocols might be fine-tuned. Research is needed to examine this idea. However, glycerol hyperhydration strategies have received a small amount of attention from sports scientists, and studies investigating the effects on sports performance are summarised in the Appendix (Table 17.11). At present, there is insufficient evidence to make a decision about the value of glycerol hyperhydration on performance. The literature appears inconsistent, at least partly because of differences in study methodologies. For example, some studies have investigated the effect of glycerol in assisting the body to retain larger amounts of a fluid bolus consumed in the hours before exercise, while others have used protocols in which glycerol is consumed with only a modest fluid intake. Other protocols have added glycerol to fluids consumed during exercise. At present, the most promising scenario involves the use of glycerol to maximise the retention of fluid bolus just prior to an event in which a substantial fluid deficit cannot be prevented. In some, but not all, studies of this type, glycerol hyperhydration has been associated with performance benefits. However, the mechanism for this effect is not clear, since the theoretical advantages of increased sweat losses and greater capacity for heat dissipation, and attenuation of cardiac and thermoregulatory challenges, are not consistently seen. Further investigation is needed to replicate and explain performance benefits.

Finally, protocols need to be fine-tuned, and perhaps individualised for specific situations. As explained in Chapter 11, the benefits of hyperhydrating with additional fluid must be measured against the energy cost of the increase in body mass. Hyperhydration techniques which increase BM by ~0.6 kg may not present a problem to weight-supported sports such as cycling on flat courses or on a laboratory ergometer. However, this may not be the case in weight-sensitive sports such as running or uphill cycling. Finally, side-effects from the use of glycerol include nausea, gastrointestinal distress, and headaches resulting from increased intracranial pressure. These problems have been reported among some but not all subjects in the current studies. Fine-tuning of protocols may reduce the risk of these problems, however, some individuals may remain at a greater risk than others. At the present time, glycerol hyperhydration should remain an activity that is supervised and monitored by appropriate sports science/medicine professionals, and only used in competition situations after adequate experimentation and fine-tuning has occurred.

17.8 NUTRITIONAL ERGOGENIC AIDS LACKING SUBSTANTIAL SCIENTIFIC SUPPORT

17.8.1 Ginseng and related herbal products

Ginseng has enjoyed popularity as a health supplement for many centuries. The chemical composition of commercial supplement products is highly variable due to differences in the genetic nature of the plant source, variation in active ingredients with cultivation and season, and differences in the methods of drying and curing. Several species of ginseng are known to exist: American, Siberian, Korean and Japanese (Bahrke & Morgan 1994). Most of these belong to the Panax species and are related. However, Russian or Siberian ginseng is extracted from a different plant (Eleutherococcus senticoccus). The root of these plants is considered the most valuable part.

A number of chemically similar steroid glycosides or saponin chemicals, known as ginsenosides, have been identified as active ingredients in ginsengs. Unfortunately for the process of scientific study, the variability of active ingredients within and between species is great, and the processes involved in the preparation of supplement products exaggerates these differences. The bioavailability of supplements varies according to the method of administration (chewing gum, pill, capsule, tablet or liquid). Some ginseng preparations also provide additional agents such as vitamins, minerals or other herbal compounds.

Ginseng has been used widely in herbal medicines of oriental cultures to cure fatigue, relieve pain and headaches, and improve mental function and vigour. It is also claimed to increase non-specific resistance to various stressors, described by Russian and Eastern European scientists as an adaptogenic response. An adaptogen is a substance purported to normalise physiology after exposure to a variety of stresses. It exhibits a lack of specificity in its actions and can both reduce or increase a response that has been altered by a stressor. This theory represents a philosophy of physiology or medicine different from the traditional Western understanding.

Despite the history of use in Eastern or traditional medicine, ginseng has only recently emerged as a purported ergogenic aid for exercise performance. In athletes, ginseng is claimed to reduce fatigue, and improve aerobic conditioning, strength, mental alertness and recovery. Table 17.12 in the Appendix summarises the few controlled studies that have investigated the effect of ginseng and related products on exercise or sports performance. Other studies presented in reviews or discussions of ginseng supplementation (Bahrke & Morgan 1994; Dowling et al. 1996) have not been included due to flaws in research design (failure to include a control or placebo group) and lack of availability of details due to publication in a foreign language journal. Conference presentations, which have not been published in a peer-reviewed forum, have also been omitted. We included a study on a supplement containing ciwujia since the active ingredients are extracted from the leaves of the plant whose roots provide Siberian ginseng.

In view of the paucity of literature and failure to utilise trained subjects in most studies, it is fair to say that the effect of ginseng supplementation and athletic performance has not been thoroughly researched. However, the variability of the content of commercial ginseng supplements creates a difficulty in undertaking well-controlled research, as well as advising athletes about any favourable results. Chong and Oberholzer (1998) assayed 50 commercial ginseng preparations and noted that 44 products ranged in ginsenoside concentration from 1.9–9.0%, and six preparations failed to produce a detectable level of ginsenosides. Thus, even if scientific evidence showed that ginseng could enhance exercise performance, athletes could not be certain of receiving the appropriate dose and type of active ingredients from all preparations in the commercially available range. Furthermore, one product that was assayed by Chong and Oberholzer (1988) contained large amounts of ephedrine, confirming expert opinion that herbal preparations present an unknown risk of causing an inadvertent doping outcome for elite athletes (Baylis et al. in press). At the current time there is no substantial evidence to support testimonial claims that ginseng is of benefit to performance or recovery.

17.8.2 *Carnitine*

Carnitine is a non-essential nutrient, first described early in the 1900s. It was first considered to be an essential vitamin until the discovery that it could be manufactured in the liver and kidney from amino acid precursors (lysine and methionine). Dietary sources of carnitine include most animal foods, but due to losses in cooking and preparation of foods there are few data on the total content of the diet. Carnitine ingested or synthesised by humans is in the L-isoform, and is carried via the blood for storage, predominantly in heart and skeletal muscle.

One of the chief roles of carnitine is, as a component of the enzymes carnitine-palmityltransferase I, carnitine-palmityltransferase II and carnitine-acylcarnitine translocase, to transport long chain fatty acids (LCFAs) across the mitochondrial membrane to the site of their oxidation (see Chapter 16). Because of this function, it has been suggested that carnitine supplementation might enhance fatty acid transport and oxidation, potentially decreasing body fat levels. This claim has been widely embraced by body builders and other groups conscious of body-fat levels, where carnitine supplements are consumed for 'cutting up' or 'getting ripped'. Carnitine is an ingredient in many purported weight-loss products. Endurance athletes might also benefit from carnitine supplementation, if it can enhance fatty acid oxidation during submaximal exercise. Theoretically, a reduced reliance on glycogen stores and blood glucose oxidation could enhance endurance in events where CHO stores are otherwise limiting.

Another role of carnitine is to act as a 'sink' for acetyl-CoA units produced during high intensity exercise. By converting this to acetyl-carnitine and CoA, carnitine could help to maintain CoA availability and to decrease the ratio of acetyl-CoA:CoA. If supplementation could increase this function it might enhance

flux through the citric acid cycle. Furthermore, it could enhance the activity of the enzyme pyruvate dehydrogenase (PDH), which is otherwise inhibited by high levels of acetyl-CoA, thus increasing oxidative metabolism of glucose. If this results in lower lactate production, it might enhance exercise performance in situations that might otherwise be limited by excess lactate and hydrogen ion accumulation (see Chapter 2). Extensive reviews of carnitine function are available (Cerretelli & Marconi 1990; Wagenmakers 1991; Clarkson 1992; Heinonen 1996).

There are several inborn errors of metabolism leading to inadequate muscle carnitine activity. Individuals with such conditions experience lipid abnormalities and reduced exercise capacity, which can be attenuated by carnitine supplementation. Such uses of carnitine supplementation are established medical therapy. However, whether additional carnitine intake in healthy individuals enhances metabolism and exercise performance is another matter. Several issues must be considered if this is a possibility:

1. Does heavy training lead to a decrease and sub-optimal level of muscle carnitine?
2. Does carnitine supplementation lead to an increase in muscle carnitine concentrations?
3. Is carnitine the limiting factor in the transport of long-chain fatty acids (LCFAs) into the mitochondria?
4. Can enhanced carnitine levels enhance activities of PDH or citric-acid cycle flux?

Reviews by Wagenmakers (1991) and Heinonen (1996) cast doubt on the theoretical benefits of carnitine supplementation in healthy athletes. They summarise that there is no proof that fatty acid transport is the rate limiting step in fat oxidation, and that muscle levels of carnitine appear to be adequate for maximal function of carnitine palmityltransferase. Furthermore, PDH is believed to be fully active within seconds of high-intensity exercise, and additional carnitine is unlikely to further stimulate this activity. The issue of optimal muscle carnitine content for athletes is probably the most important issue to clarify. It is known that exercise results in increased carnitine excretion, and it is possible that muscle carnitine content may decrease during intense training. However, Clarkson (1992) maintains that a serious deficiency would not occur since carnitine and its amino acid precursors are easily obtained in the diet. Cerretelli and Marconi (1990) have summarised a series of studies of carnitine supplementation in human subjects. Although most studies show an increase in plasma carnitine levels following carnitine supplementation of 1–6 g/d, the effects on muscle carnitine content are less clear. Nevertheless, the consensus of most reviewers is that there is no compelling evidence that muscle carnitine levels are enhanced as a result of supplementation (Wagenmakers 1991; Heinonen 1996).

Studies that have investigated the effects of carnitine supplementation on exercise metabolism and/or performance are summarised in Table 17.13

(Appendix). Overall, there is little evidence that carnitine supplementation causes any change in metabolism during submaximal or high-intensity exercise. The few studies that report favourable metabolic outcomes, or an increase in exercise performance, are hard to explain. For example, it is hard to understand how a supplement taken 90 min before exercise has sufficient time to be absorbed through the gut into the bloodstream and taken up by the muscle (Siliprandi et al. 1990). However, on balance there is little evidence of increased performance resulting from carnitine supplementation. The effect of carnitine supplementation on body-fat levels, although widely publicised in supplement advertising, has not been studied.

Although studies of acute and long-term carnitine supplementation report that carnitine appears to be safe, it should be noted that this applies to L-carnitine preparations. D-carnitine, however, has been shown to cause depletion of L-carnitine in tissues, therefore creating a carnitine deficiency (Clarkson 1992). Athletes are advised to avoid commercial carnitine preparations that do not clearly specify that contents are > 99% L-carnitine.

17.8.3 *Coenzyme Q10*

Coenzyme Q10, also known as ubiquinone, is a non-essential lipid-soluble nutrient found predominantly in animal foods and in low levels in plant foods. In the body it is located primarily in the mitochondria, especially in skeletal and cardiac muscle. One of its well-known functions is as a link in the electron transport chain within the mitochondria, thus providing a part in the final production of ATP. It is also believed to have an antioxidant function, mopping up free oxygen radicals in the mitochondrial antioxidant defence system and preventing damage to DNA and cell membranes. It has been suggested that some cardiac and neuromuscular dysfunction is due to coenzyme Q10 deficiency; indeed, patients with ischaemic heart disease are shown to have lower plasma Q10 concentrations. Some studies have shown that these patients respond to coenzyme Q10 supplementation with increased exercise capacity. Coenzyme Q10 supplements have recently emerged as new products marketed in the general community to promote vigour. For athletes they are claimed to enhance energy production through the electron transport chain, and to reduce the oxidative damage of exercise.

Table 17.14 (see Appendix 17) summarises the studies, which have examined the effects of coenzyme Q10 supplementation on exercise metabolism, oxidative damage caused by exercise, and performance. Studies that have been reported only in the form of conference abstracts have not been presented. There are few data that support an ergogenic benefit of coenzyme Q10 on exercise performance. In fact, there are substantial data that show that coenzyme Q10 has an ergolytic effect on high-intensity performance and training adaptations. A series of studies undertaken at the Karolinska Institute in Sweden has produced consistent evidence that Q10 supplementation increases the oxidative damage produced by

high-intensity exercise (Malm et al. 1996; Malm et al. 1997; Svennson et al. 1999). Twenty-two days of supplementation, undertaken in conjunction with high-intensity training, was shown to increase oxidative damage, as indicated by higher plasma CK levels and increased malondialdehyde levels in response to exercise (Malm et al. 1996; Malm et al. 1997). In these circumstances, Q10 was believed to act as a pro-oxidant rather than an antioxidant. Training adaptations were impaired in healthy subjects who undertook high-intensity training while taking Q10 supplements, with the placebo group out-performing the Q10 group at the end of the supplementation phase (Malm et al. 1997).

Clearly, further work is required to investigate the effects of coenzyme Q10 supplementation on exercise performance and training. However, at present there is little to recommend Q10 supplementation to athletes undertaking high-intensity training, and we are reminded that some scientific theories are not as straightforward as they seem. The issue of antioxidant supplementation is complex and as yet unsolved.

17.8.4 *Inosine*

Inosine is a nucleic acid derivative, considered to be a non-essential nutrient. Major dietary sources include yeast and organ meats. Inosine is a precursor of the nucleotide inosine monophosphate (IMP), and may also lead to the production of ATP. In-vitro tests suggest that inosine may enhance the levels of 2,3-diphosphoglycerate (2,3-DPG) in red blood cells. Potential mechanisms by which inosine supplementation might enhance exercise performance include an increased ATP supply, and increased release of oxygen from erythrocytes to the muscle, via a shift in the oxyhaemoglobin curve mediated by increased 2,3-DPG concentrations. Inosine is believed to have vasodilatory effects and antioxidant properties, and may lead to the formation of fumarate, a citric acid cycle substrate. Further information on the functions of inosine is provided by Williams et al. (1990) and Starling et al. (1996). However, these are only hypothetical situations that have not been supported by research.

The major support for inosine supplementation comes from testimonials from successful athletes, with reports that it is a favourite supplement of Russian and Eastern European athletes. In 1988, *Muscle and Fitness*, a popular magazine, carried an article describing a six-week study of inosine supplementation on four trained athletes (Colgan 1988). The report claimed the study was undertaken using a double-blind crossover design, and found strength gains as a result of the supplementation. No data or statistical analyses were presented. This study has not appeared in a peer-reviewed publication or in adequate detail to judge the validity of these claims. Interestingly, the athletes were reported to suffer irritability and fatigue while on inosine treatment.

Only three well-controlled crossover designed studies of inosine supplementation have been published in the scientific literature (see Table 17.15 in Appendix 17).

It should also be noted that inosine was an ingredient in a multicompound ergogenic aid (CAPS) that failed to enhance performance of triathletes in a study by Snider and colleagues (1992); this study has been reviewed in the section on coenzyme Q10 above. The three studies of isolated inosine supplementation used well-trained athletes as subjects and all failed to find performance benefits following inosine supplementation (Williams et al. 1990; Starling et al. 1996; McNaughton et al. 1999).

Metabolic data from these studies failed to show any favourable enhancements that could improve sports performance, or support the theoretical actions of supplemental inosine. For example, 2,3-DPG levels were not increased following inosine supplementation and there was no evidence from blood metabolites or respiratory exchange data of enhancement of CHO metabolism. Although muscle substrates were not directly measured, purported changes to ATP concentrations are unlikely to enhance exercise performance, since ATP is not depleted by exercise, even at the point of fatigue (see Chapter 2). Interestingly, two of the studies reported that subjects performed the high-intensity tasks better on the placebo treatment than following inosine supplementation (Williams et al. 1990; Starling et al. 1996). This suggests that inosine might actually impair the performance of high-intensity exercise. Potential mechanisms for exercise impairment include an increased formation of inosine monophosphate (IMP) in the muscle, either at rest or during exercise. High IMP concentrations have been found at the point of fatigue in many exercise studies; furthermore, IMP has been shown to inhibit ATPase activity (Sahlin 1992). It is possible that increased resting muscle IMP concentrations could reduce the duration of high intensity exercise before critically high levels were reached, causing premature fatigue. Such a theory can only be investigated by direct measurements of muscle nucleosides.

Ultimately, inosine is degraded to uric acid for excretion. It is possible that inosine supplementation could lead to elevated uric acid levels, providing an alternative mechanism of performance impairment as well as an increased risk of gout. In the present studies, two days of inosine supplementation did not change uric acid levels; however, five days and ten days of intake doubled blood concentrations to levels above the normal range (Williams et al. 1990; Starling et al. 1996; McNaughton et al. 1999). Thus, chronic inosine supplementation may pose a health risk. In summary, since there is a lack of evidence of performance benefits, and the possibility of performance decrements and side-effects, there is little to recommend the use of inosine supplements by athletes.

17.8.5 *Chromium picolinate*

Chromium is an essential element, required in trace amounts. An Australian RDI for chromium has not been set, however, the US Food and Nutrition Board established an Estimated Safe and Adequate Daily Dietary Allowance (ESADDA) within the range of 50–200 μg/d (National Research Council 1989). Dietary

sources of chromium include yeast, nuts and legumes, some fruit and vegetables, chocolate, wine and beer. Dietary surveys often report the estimated chromium intake of many populations to be below this recommended range. However, in light of criticism that ESADDA ranges for chromium have been set artificially high, and the lack of reliable food composition data for chromium, it is difficult to assess the likelihood of an inadequate chromium intake (for review see Clarkson 1997). There is some evidence that daily training may increase urinary chromium losses, increasing chromium requirements and the risk of sub-optimal chromium intakes. However, adaptations may also occur to improve absorption or retention of chromium in compensation (see Clarkson 1997). As is the case for many micronutrients, athletes with restricted energy intakes are most at risk of low chromium intakes.

One of the best known roles of chromium in the body is to potentiate insulin action. This action enhances glucose uptake, as well as lipid and amino acid metabolism (for review see Stoecker 1996). Chromium may also have a role in immune function. Subjects with chromium deficiencies often show improvements in growth or glucose tolerance in response to chromium supplementation (Stoecker 1996). In the case of the athletic population, individuals with inadequate dietary intake of chromium may respond positively to supplemental chromium intake. However, the major market is focussed on claims that chromium supplements will enhance handling of glucose, amino acids and fatty acids, allowing dramatic gains in muscle mass and strength, while reducing body fat.

Chromium supplements are available in the form of chromium nicotinate, chloride and picolinate. Chromium picolinate is claimed to be the most biologically active form, and the claims for the efficacy of chromium picolinate have caused an interesting public debate between the patent holders (Evans and colleagues) and other trace element/mineral experts such as Levafi, and Lukaski. Examples of this discussion include a review on chromium supplementation (Levafi et al. 1992) followed by a series of letters published in *International Journal of Sport Nutrition* (Evans 1993; Levafi et al. 1993). One concern about chromium supplementation is that chromium potentially competes with trivalent iron for binding to transferrin, thus predisposing those with chronically high intakes of chromium to iron deficiency (Lukaski et al. 1996). Some (Lukaski et al. 1996), but not all (Campbell et al. 1997), studies have reported a reduction in iron status as a result of chromium picolinate supplementation.

Table 17.16 (Appendix 17) summarises studies of chromium piconate supplementation and effect on body composition and exercise performance. Not included in this table are the studies responsible for motivating the original interest in chromium picolinate. These studies claimed significant increases in lean body mass in subjects undertaking aerobic exercise classes (Evans 1993) and weight training (Evans 1989) while supplementing with chromium picolinate. However, Levafi has provided a thorough criticism of these studies (Levafi et al. 1992; Levafi

1993), identifying methodological flaws. He pointed out that the earlier study (Evans 1989) failed to control for training experience of subjects or their compliance with the study protocol. Furthermore, positive anabolic results were estimated through the use of anthropometric techniques that carry significant measurement and prediction error. With regard to the 1993 study claiming gains of 2 kg of lean body mass (Evans 1993), Lefavi suggests this is an unlikely outcome for subjects undertaking aerobics classes over 12 weeks. Most importantly, this study did not include a control group to verify that changes were simply the result of the chromium picolinate supplementation.

The studies summarised in Table 17.16 do not provide evidence of gains in strength and lean body mass or loss of body fat, other than what can be achieved through training alone. There is certainly no support for the dramatic claims made in some advertisements that position chromium picolinate as a 'legal anabolic' agent. The only situation in which chromium supplementation is likely to be useful is in treating individuals whose dietary intakes are inadequate.

17.8.6 *Medium chain triglycerides*

Medium chain triglycerides (MCTs) are fats composed of medium-chain fatty acids (MCFA) with a chain length of six to ten carbon molecules. As outlined in Chapter 16, Section 16.2, they differ from fats composed of LCFA in terms of digestion, absorption and uptake into the muscle mitochondria. Specifically, MCTs can be digested within the intestinal lumen with less need for bile and pancreatic juices than long-chain triglycerides, with MCFAs being absorbed via the portal circulation. MCFAs can be taken up into the mitochondria without the need for carnitine-assisted transport. In clinical nutrition, MCTs derived from palm kernel and coconut oil are used to supply energy to patients who have various digestive or lipid metabolism disorders. In the sports world, MCTs have been positioned as an easily absorbed and oxidised fuel source, and have been marketed to body builders as a fat source that is less likely to deposit as body fat. However the role of MCTs in the general diet of athletes has not been studied.

A more interesting role of MCTs in sport is as an additional fuel source during prolonged exercise (see Chapter 16, Section 16.3). If MCTs ingested during prolonged endurance and ultra-endurance events could spare glycogen, they might provide a performance advantage by prolonging the availability of important CHO stores. Although earlier studies of MCT ingestion immediately prior to or during exercise did not show much promise (Section 16.8.3), recent tracer techniques using stable isotopes have enabled a more sophisticated study of MCT metabolism during exercise. Jeukendrup and colleagues (1995) studied the ingestion of 29 g of MCT during three hours of cycling at 55% VO_2 max, alone, or with the addition of moderate and large amounts of CHO. The presence of CHO increased the rate of MCT oxidation, possibly by increasing its rate of absorption. The maximum rate of MCT oxidation was achieved in all trials between 120–180 min, at values of

~0.12 g/min. Although this suggests that MCT can supply a useful energy source, the researchers pointed out that the maximum amount of MCT that can be tolerated within the gastrointestinal tract is ~30 g in total. Therefore, they suggest that the contribution of MCTs is likely to be limited to a maximum of ~3–7% of the total energy expenditure during typical ultra-endurance events (Jeukendrup et al. 1995). In a separate study, this intake of MCTs was not shown to influence CHO utilisation or glycogen sparing (Jeukendrup et al. 1996).

Studies that have examined the effect of the co-ingestion of MCT and CHO on ultra-endurance performance are summarised in Table 17.17 (Appendix 17). These studies show inconsistent effects of MCT on fuel utilisation. In circumstances where the intake of large amounts of MCT raises plasma FFA concentrations and allows glycogen sparing, it may benefit the performance of exercise at the end of a prolonged bout (van Zyl et al. 1996). However, these metabolic (and performance) benefits may be over-ridden when exercise is commenced with higher insulin levels, as is the case following a CHO-rich pre-exercise meal (Goedecke et al. 1999; Angus et al. 2000). Critical to the whole issue is the ability of subjects to tolerate the substantial amount of MCT oils required to have a metabolic impact. Symptoms range in severity from insignificant (van Zyl et al. 1996) to performance-limiting (Jeukendrup et al. 1998). Differences in gastrointestinal tolerance between studies may reflect differences in the mean chain length of MCTs found in the supplements, or increased tolerance in some athletes due to constant exposure to MCTs. The intensity and mode of exercise may also affect gastrointestinal symptoms. For further reading on the ingestion of MCTs during exercise, see Chapter 16, Section 16.8.3.

In summary, although CHO gel supplements are marketed with the addition of MCTs, there are little data to support a beneficial use of these special products. Furthermore, the theoretical use is limited to the small population of athletes who undertake ultra-endurance sports. Further research may clarify whether MCTs can be a useful supplement for ultra-endurance sports, but significant investigation of gastrointestinal concerns is needed before any recommendations can be made.

17.9 NEW SUPPLEMENTS LACKING SUBSTANTIAL SCIENTIFIC SUPPORT

Many new ergogenic aids hit the sports world with considerable hype, testimonials, and clever marketing. The Internet has only served to exaggerate publicity about such products and accelerate the spread of unsupported information. It takes time for scientists to undertake well-controlled trials to investigate the claims made for these products, or substantiate the experiences claimed by satisfied customers. The process of peer-review and publication also adds to the time lag between the claims and the presentation of evidence. In this section we will review some of the newest and 'hottest' ergogenic aids in the sports world. By necessity we will need to present information from conference presentations and manuscripts that are still in review.

Therefore, we will be cautious in our judgement of any data that have not been fully reviewed by appropriate experts. However promising the claims and early evidence for these supplements might seem, we will need to consider that substantial scientific support is still lacking.

17.9.1 *Androstenedione, DHEA and pro-hormone supplements*

Anabolic steroids are controlled as pharmaceutical products and are listed as proscribed agents by the IOC. However, new supplements have recently appeared on the athletic market containing pro-hormones that can be converted in the body to testosterone (Blue & Lombardo 1999). These include androstenedione, dehydroepiandrosterone (DHEA), 19-norandrostenedione and other metabolites found in the steroid pathways. Theoretically, each has some androgen activity as well as being part of the pathway to testosterone production. Some herbal compounds such as Saw palmetto and Tribulus terrestris are also claimed to have anabolic activity.

Since these products are manufactured as dietary supplements rather than drugs, they enjoy a loose regulation of quality control and claims. We have already commented that independent analysis of some products has shown a variable content of the stated ingredients (Parasrampuria 1998). They are heavily promoted in the body building world where they are claimed to promote fat loss, gains in muscle mass and strength, increased libido, reversal of ageing and enhanced immunity. Some have shot to instant fame by being associated with successful performances or well-known athletes. For example, androstenedione received publicity as an ergogenic aid used by baseball player Mark McGwire during the 1998 season in which he broke the home-run record.

Pro-hormones are banned by the IOC, either directly by name, or indirectly under the umbrella of being a 'related substance' of anabolic-androgenic steroids. However, individual sporting organisations may not include these products within their own list of banned agents. There is some confusion about whether the use of these agents will cause a positive drug test. However, if they cause a large change in testosterone : epitestosterone ratio, or cause an increased excretion of banned substances such as metabolites of the anabolic steroid nandrolone, an athlete will record a positive doping outcome. To date, excretion studies have produced conflicting results with some, but not all, subjects who ingested common over-the-counter supplements experiencing differential changes in urinary testosterone and epitestosterone concentrations which increased the ratio above the legal cut-off of 6:1 (Bosy et al. 1998; Uralets & Gillette 1999). However, high concentrations of metabolites of nandrolone have been found in urine for seven to ten days following the ingestion of a single dose of 19-norandrostenedione (Uralets & Gillette 1999).

In the event that an athlete is not subject to doping restrictions, and has purchased a supplement that delivers the claimed dose of pro-hormones, they should then consider the evidence that such products can influence metabolism or

performance. To date, this information is scarce. In fact the rationale for the supplements is based on limited historical studies, for example, investigations in two female subjects (Mahesh et al. 1962).

A series of studies of androstenedione, DHEA and herbal 'anabolic' supplements has been undertaken by King and colleagues from the University of Iowa. In the first study, reported in *Journal of the American Medical Association*, they investigated the effects of acute (100 mg) and chronic (300 mg for eight weeks) supplementation with androstenedione in healthy males (King et al. 1999). Androstenedione is the immediate precursor of testosterone, and possesses its own weak androgenic properties. The composition of the androstenedione supplement used in the study, derived from wild yams, was verified by independent analyses and the amounts fed to subjects exceeded the maximum doses recommended by the manufacturers. The acute feeding trial found that serum androstenedione concentrations were elevated for six hours after the intake of the supplement, however, there was no change in either free or total serum testosterone concentrations. Similarly, serum androstenedione concentrations rose during the chronic supplementation trial and remained elevated above those of the placebo group after eight weeks. Again, there were no changes in free or total testosterone concentrations in the group receiving androstenedione over the eight-week period, and no differences between the treatment and placebo groups at any time.

Interestingly, serum oestradiol and oestrone concentrations increased in the treatment group, suggesting that the exogenous androstenedione was aromatised in peripheral tissues to oestrogens rather than converted to testosterone. Furthermore, serum concentrations of HDL cholesterol were significantly reduced within two weeks of androstenedione supplementation and remained below that of the placebo group throughout the trial. Supervised resistance training was undertaken during the eight-week study, and produced significant increases in lean body mass and muscle strength, and reductions in body-fat mass in both treatment and placebo groups. There were no differences in the gains made between groups, nor changes in the histology of muscle fibres collected by the muscle biopsy technique. The authors concluded that androstenedione supplementation is not useful to subjects with normal testosterone levels, and may only increase testosterone concentrations in hypotestosterogenic populations such as women and older men. They found that the supplement was of no benefit to healthy males in enhancing the benefits gained from a resistance training program. Furthermore, there were indications of negative health outcomes such as unfavourable lipid and oestrogen profiles, which might be associated with health problems such as cardiac disease and increased risk of prostate cancers.

A separate study by this group investigated the effects of acute and chronic supplementation with DHEA (Brown et al. 2000). DHEA is the steroid hormone of greatest natural abundance in the blood, and decreases with ageing. It can be converted to androstenedione, which in turn can be converted to testosterone. In

the study, young men took either a single 50 mg dose of DHEA, or an eight-week cyclical course providing 150 mg/d (two weeks on, one week off) while undertaking a supervised resistance training program. As in the androstenedione study, an independent analysis confirmed the purity of the supplements, and the chronic supplementation protocol exceeded the daily dose recommended by DHEA manufacturers. The acute intake of DHEA was found to raise serum androstenedione concentrations, peaking at 60 min and remaining elevated at six hours post-ingestion. There were no changes in serum concentrations of testosterone or other hormones, compared with the intake of a placebo. The chronic DHEA supplementation protocol resulted in increased serum androstenedione concentrations compared with baseline or placebo concentrations, at weeks two and five. However, by week eight these concentrations were not different to baseline values. There were no differences in serum testosterone or oestrogens as a result of training or supplementation with DHEA. The resistance training program increased strength and lean body mass significantly and similarly in DHEA and placebo groups. Therefore, the DHEA supplement did not enhance testosterone concentrations or adaptations associated with resistance training in young men.

The final study undertaken by the group (Brown et al. in press) investigated the effect of a combined supplement containing androstenedione (300 mg/d), DHEA (150 mg/d) and herbal ingredients (Tribulus terristris, Saw Palmetto, Chrysin and Indole-3-carbinol). The addition of the herbal extracts is claimed to promote testosterone production by reducing the aromatisation of androgens to form oestrogens. The supplement was taken in a cyclical protocol (two weeks on, one week off) during eight weeks of supervised resistance training. Again, the supplement was found to increase serum androstenedione concentrations but to have no effect on testosterone values. A rise in serum oestrogens and decrease in HDL-cholesterol was seen, replicating the results of the previous androstenedione supplementation study (King et al. 1999). The researchers concluded that the addition of the herbal extracts, at least in the dosages found in a common body-building supplement, are insufficient to reduce the aromatisation pathways for androgen disposal. Increases in strength and lean body mass achieved by the resistance training program were not enhanced by the supplement and the potential for negative health outcomes remains a concern.

The studies undertaken by this laboratory have received considerable scientific and lay comment. The editorial accompanying the publication of the first study in JAMA advised some caution in reviewing the results (Yesalis 1999). It pointed out that although the doses used in the study were in excess of the protocols recommended by the manufacturers, they are conservative in comparison to the doses recommended and used by some athletes. It also noted that the strength gains made by previously untrained men might be sufficiently large and variable as to mask any effects from androstenedione. Nevertheless, it called for the (US) federal government to remove androstenedione and other

pro-hormones from sale. Other critics have suggested that only well trained athletes who have reached a plateau in the results of their resistance training could be expected to benefit from androstenedione supplementation. Researchers were reminded that the first scientific position papers regarding anabolic steroids concluded that they were ineffective in enhancing strength or performance in sport, as a result of the failure of scientists to investigate the real practices undertaken by athletes.

However, other laboratories have begun to confirm the results reported by King and colleagues. Wallace et al. (1999) investigated the effects of 12-week supplementation with androstenedione (100 mg/d), DHEA (100 mg/d) or a placebo in middle-aged resistance-trained men. They found that the supplements failed to increase lean body mass, strength or testosterone concentrations above that seen in the placebo group. However, they saw no adverse side-effects in terms of liver function tests, lipid levels or prostate function.

Ten experienced male resistance trainers received 200 mg of androstenedione supplements or a placebo for two days in a crossover designed study undertaken by Ballantyne and colleagues (2000). Hormone levels were investigated at baseline, after the supplement and following a bout of resistance training on the second day of supplementation. Androstenedione supplementation was found to elevate plasma androstenedione concentrations, without any alteration in testosterone values. Exercise elevated testosterone equally in both supplement and placebo trials. Exercise in the supplement trial caused a significant elevation in plasma oestradiol. The researchers concluded that androstenedione supplementation is unlikely to provide male athletes with any anabolic benefit.

Finally, another group has employed tracer techniques to study muscle protein kinetics in six healthy men, before and after the intake of 100 mg/d androstenedione (Rasmussen et al. 2000). Muscle protein turnover was investigated before and after the supplementation, and was also compared to that of a control group. The results showed that androstenedione supplementation did not affect muscle protein anabolism. There was a trend towards increased muscle protein turnover (increased synthesis matched by increased breakdown) in the treatment group compared with control subjects. However, differences were small and did not lead to a net protein increase. Monitoring of blood hormone concentrations replicated the finding that androstenedione supplementation does not lead to increased plasma testosterone concentrations, but is associated with an increase in oestradiol concentrations (Rasmussen et al. 2000).

To date, there are no published reports of the effects of 19-norandrostenedione or related compounds on serum hormone concentrations, changes in body composition or exercise performance. In summary, although supplements containing pro-hormones and herbal ‘steroids’ are marketed emotively to athletes, there is no evidence to prove that they can enhance sports performance or adaptations to training. More importantly, there is evidence that these supplements

may cause negative health outcomes as a result of unfavourable changes in blood lipid profiles and oestrogen hormones. For elite athletes, these products are considered to be proscribed agents and may result in a positive doping result.

17.9.2 *Hydroxy-methyl butyrate (HMB)*

B-hydroxy B-methylbutyrate (HMB) is a metabolite of the amino acid leucine. As one of the latest fast-selling dietary supplements, it is claimed to enhance gains in strength and body mass associated with resistance training, enhance loss of body fat and to enhance recovery from exercise. It is claimed to act as an anti-catabolic agent, minimising protein breakdown and the cellular damage that occurs with high intensity exercise. Leucine administration is known to influence protein metabolism, specifically to reduce protein degradation during periods of stress or trauma that are associated with elevated protein catabolism. It has been proposed that it is the increase in leucine metabolites such as ketoisocaproate (KIC) or HMB that mediates this effect. In animals, some but not all studies have found that HMB supplementation increases gains in carcass weight or feed efficiency, defined as weight gain per unit feed, during growth (for review see Slater, in press). To date, only two studies of HMB supplementation in humans have appeared in the peer-reviewed literature.

In 1996, Nissen and colleagues reported on two studies of HMB supplementation in men (Nissen et al. 1996). The first study involved healthy but untrained males who were randomised into groups receiving 0, 1.5 or 3 mg of HMB per day for three weeks, while consuming a normal (117 g/d) or supplemented (175 g/d) protein intake and undertaking resistance training. HMB supplementation was found to reduce urinary 3-methyl histidine excretion (a marker of muscle protein degradation) and plasma creatine kinase concentrations (a crude indicator of cellular damage). HMB supplementation was associated with a dose-responsive increase in weight lifted during training (particularly in lower body strength), and there was a trend for increased gain in lean body mass with the increase in HMB dose. The second study investigated seven weeks of HMB supplementation (3 mg/d) and resistance training in previously trained subjects. No dietary control was imposed during this study. The results showed that all subjects increased body mass during the study period, but there were trends for greater increases in fat-free mass (assessed by total body electrical conductivity) in the HMB group. The increases in muscle strength measurements were greater for some (upper body) but not all (lower body) lifts in the HMB-supplemented group.

Although these results are supportive of benefits from HMB supplementation, there are several methodological concerns with these investigations. Firstly, dietary intake was not controlled in the second study and HMB was provided to subjects in a protein–CHO supplement that was not matched in the placebo group. This allows the possibility that dietary differences, or additional protein and nutrients provided in this supplement, were responsible for the small differences in responses

to the resistance training program between groups. Second, it is hard to explain differences in strength gains in various body parts in terms of differential effects of HMB supplementation. Examination of these data show that the groups were not equally matched for baseline values of upper body strength, despite random allocation of subjects to the treatment groups. The HMB group had lower upper body strength at the beginning of the study than the placebo group, and with a significant increase in strength over the seven-week study, still only reached the baseline values of the placebo group. Therefore, it is possible to explain the increased gains in the HMB group as the outcome of lower initial levels and a greater potential for change. Lower body strength, which was better matched at baseline, did not change over time between groups, thus providing additional support for this theory.

The other fully reported study compared changes in body composition, strength and marker of muscle damage in experienced resistance-trained males who consumed a daily supplement providing 0, 3 and 6 mg/d of HMB for 28 days (Kreider et al. 1999). All subjects throughout the study period were given individualised training programs. Changes in total body weight and body composition were not different between groups. There were no significant interactions between groups for gains in strength for bench press or leg press. There was a trend for lower plasma CK concentrations for subjects receiving the higher HMB dose compared with the placebo group. Otherwise, there were no differences in markers of muscle catabolism or damage between groups. The researchers concluded that HMB supplementation provides no ergogenic value to experienced resistance-trained athletes (Kreider et al. 1999).

There are several conference abstracts that report gains in strength and lean body mass when HMB is combined with resistance training in previously untrained men. Other studies have found variable effects on body composition and strength when HMB supplements were taken alone or in combination with creatine by college football players during pre-season training (for review see Slater, in press). However, it is difficult to fully interpret such data in the absence of details of the research design. Therefore, until further studies of HMB supplementation are conducted and reported in full, it is impossible to make a decision on the potential of this supplement.

17.9.3 *Colostrum*

Colostrum is a protein-rich substance secreted in breast milk in the first few days after a mother has given birth. It is high in immunoglobulins and insulin-like growth factors (IGFs). Unlike the adult gut, the gut of a baby has ‘leaky’ junctions which allows it to absorb whole proteins including immunoglobulins, thus developing the immuno-competence needed to survive outside the uterus. Recently, companies have developed supplements rich in the colostrum of cows for use by humans. In 1997, a paper published in the *Journal of Applied Physiology*

reported that colostrum supplements (Bioenervie™) increased plasma IGF-1 levels in sprinters and jumpers who undertook strength and speed training sessions during the supplementation period (Mero et al. 1997). Supplementation failed to change vertical jump performance in these athletes. This study raised several controversial issues. First, it appeared to show that humans could absorb intact proteins from a supplement, and second, it appeared to provide a dietary source of IGFs, an anabolic hormone whose intentional intake is banned by the IOC. Discussion of this paper suggested that if colostrum did provide a direct source of IGF it would contravene the doping laws of sport. However, more recently it has been suggested that the data showing increased IGF concentrations are spurious and the result of inaccurate techniques for measuring these growth factors.

To date, the only other studies of colostrum and athletic performance exist only as conference papers in abstract form. In a study presented in 1998, Buckley and colleagues reported on the effects of eight weeks of running training (3×45 min/week) in combination with 60 g/d of a colostrum powder (Intact™) or a whey placebo in two groups of previously untrained men (Buckley et al. 1998). The test set, consisting of two incremental treadmill runs to exhaustion, with a 20 min recovery interval, was undertaken at zero, four and eight weeks. They reported that after eight weeks the treatment group completed more work and ran further in the second of two treadmill runs than subjects in the placebo group (Buckley et al. 1998). No differences were seen at four weeks, and no measurements were taken to explain the performance improvements seen in the second run at eight weeks.

In 1999 Buckley and colleagues presented the results of a one-week supplementation program with 60 g/d of colostrum or whey protein placebo on the performance of well-trained rowers undertaking a supervised training program. At weeks zero and nine, the rowers undertook two four-step rowing ergometer tests separated by a 15 min recovery period. They reported that the colostrum supplementation resulted in a greater amount of work and distance achieved in the last stage of both incremental rowing ergometer tests (Buckley et al. 1999). Criticisms of this study include small subject numbers (three in the treatment group and five in the placebo group) and failure to measure any parameters to explain their findings.

It should be noted that the studies undertaken by Buckley and colleagues (1998, 1999) were funded by the manufacturers of the colostrum supplement and both have received considerable promotion and hype. The results of the studies have been interpreted aggressively by both the researcher and the manufacturer to include claims of enhanced recovery, superior muscle buffering capacity, and increased growth of muscle contractile proteins. The benefits have been transferred to other groups including manual workers and chronic fatigue sufferers. At the present time, we believe that the data from these studies cannot be commented on until it appears in press following the appropriate peer-review process. Furthermore, we note that the studies are limited by small subject numbers (rowing study) and the failure to investigate mechanisms to support and explain the observed

performance benefits (both studies). It is a concern that there does not appear to be a plausible explanation to underpin the effects of colostrum supplements on adults.

Therefore, although colostrum is a 'hot' supplement in the athletic world and merits further research, there is insufficient evidence at present to support any performance benefits. At the recommended price of $70 per week, and the suggestion that it may take at least four weeks to show benefits, athletes are reminded that colostrum supplementation involves a considerable expense.

17.10 BALANCING ADVANTAGES AND DISADVANTAGES OF SUPPLEMENT USE

Athletes should consider several factors before deciding to use a supplement. The likely benefits should be considered carefully, and weighed against the cost of a supplement program and the risk of negative outcomes. In the previous section of this chapter, we discussed supplements and the conditions of use that can lead to true performance benefits. We also acknowledged that placebo or psychologically mediated effects can achieve a worthwhile improvement in training or competition performance, at least in the short term. Supplement use, even when it provides a performance advantage, is an expense that the athlete must acknowledge and prioritise appropriately within their total budget. At times, it may be deemed money well spent, particularly when the supplement provides the most practical and palatable way to achieve a nutrition goal, or when ergogenic benefits have been well documented. On other occasions the athlete may choose to limit the use of expensive supplements to the most important events or training periods. The downside of supplement use includes the potential for side-effects, an inadvertent doping outcome and failure to consider other real performance-enhancing strategies. These problems are often forgotten.

17.10.1 Doping safety

In most sports, competition between elite athletes is conducted within a code of conduct, issued by the Governing Body of the sport or the Medical Commission of the IOC, that includes a ban against doping. Doping has been defined by the IOC as 'the administration or the use by a competing athlete of any substance foreign to the body or of any physiological substance taken in abnormal quantity or taken by an abnormal route of entry into the body, with the sole intention of increasing in an artificial or unfair manner his performance in competition'. The substances and methods that are banned by most sporting organisations are based on the list prescribed by the IOC (see Table 17.3).

Most countries have an anti-doping agency or program that provides education to athletes to distinguish between common pharmaceutical products that are permitted and those that contain substances that are banned, either totally or for competition use. Athletes, coaches and sports medicine/science professionals are responsible for applying this information to their own practice. Inadvertent doping

may occur, where an athlete records a positive drug test after unintentionally taking a banned substance as an unrecognised ingredient of a product they have consumed. However, most organisations now place full liability with the athlete who tests positive, regardless of circumstance, and full penalties can be expected.

Table 17.3 *List of substances or methods banned by the International Olympic Committee (IOC) 1999*

Category	Examples
A. Stimulants	Pseudo-ephedrine (e.g. Sudafed) Ephedrine Amphetamines High doses of caffeine—producing a urinary caffeine level ≥12 µg/mL
B. Anabolic agents Anabolic and androgenic steroids Non-steroidal anabolic agents	 Nandrolone DHEA Androstenedione Other agents with similar properties (e.g. 19-norandrostenedione and other pro-steroid hormones) Beta 2 agonists (except for inhalants of salbutamol and terbutaline)
C. Diuretics	Frusemide (Lasix) Spironolactone (Aldactone)
D. Narcotic analgesics	
E. Peptide and glycoprotein hormones and analogues	Human growth hormone (HGH) Human chorionic gonadotrophin Corticotrophin Erythropoetin (EPO)
F. Blood doping	
G. Pharmacological, chemical and physical manipulations	Catheterisation (drawing urine from bladder) Masking agents Swapping urine

Although pharmaceutical products, both prescription-regulated and over-the-counter products, are the most likely source of inadvertent doping problems, supplements must also be considered. Although there are no data recording the prevalence of inadvertent doping through drug use, there are isolated reports such as the case of a Dutch professional cyclist who recorded a doping positive after using a liquid herbal supplement that declared ephedra among 15 ingredients (Ros et al. 1999). Supplements containing herbal forms of ephedrine or caffeine, and

pro-hormones such as DHEA or androstenedione are either directly banned or may lead to a positive drug test (see Section 17.9.1). In fact, there has been speculation that dietary supplements, such as those containing 19-norandrostenedione, may be responsible for the recent spate of positive tests for the anabolic steroid Nandrolone among a wide range of athletes (Christie 1999).

An inadvertent doping outcome could arise from supplement use in a number of ways (Baylis et al. in press):

1. The supplement contains a banned substance as a stated ingredient but the athlete is not aware that the substance is banned or that it acts to cause a positive doping test.
2. The supplement contains a banned substance within stated ingredients but the athlete is unaware of the relationship between the products (for example, athletes may not recognise that guarana has a high caffeine level, or that Ma Huang herbal products contain ephedrine).
3. The supplement contains banned substances that are not declared as a stated ingredient. These ingredients may be added deliberately but not declared, or added inadvertently as by-products of other ingredients or contaminants of the production process. Examples include herbal preparations that inadvertently contain ephedra or other herbal alkaloid stimulants found in a common plant source, or multi-ingredient 'anabolic supplements' which have an undisclosed content of pro-hormones that convert into banned substances.

Despite the lack of data to report the actual prevalence of the problem, there is a small but real risk of inadvertent doping through supplement use (Baylis et al. in press). Admittedly, the problem exists for a minor percentage of people who undertake sport or exercise activities. However, the outcome for these athletes can be a substantial loss of success, reputation and earnings. In the case of pharmaceutical products, it is relatively easy and successful to prepare lists of proprietary products categorised into banned and permitted classifications. However, in view of the unknown and variable composition of at least some supplement formulations, such a system could not be applied to sports supplements. Understandably, laboratories are generally unwilling to test supplements because of the considerable liability involved with providing a false clearance. We have recently suggested a more suitable classification system would include four categories: low risk, unknown risk, restricted and banned. Examples of general classes of supplements that would fit these categories in Australia are summarised in Table 17.4. It would be useful to have an accredited program to test individual supplement products and perhaps place individual products within the classification system. However, any such program would need to be underpinned by the acceptance of liability by supplement manufacturers and the education of athletes about supplement use. The use of dietary supplements and some sports foods is never risk-free, and each athlete must make their own decision about whether or not to accept the risk (Baylis et al. in press).

Table 17.4 *Suggested categorisation of general groups of supplements and sports foods in Australia according to 'Sports safety'*

Low risk	Unknown risk	Restricted	Banned
The supplements in this category include only ingredients that are permitted in sport. These supplements are least likely to result in an athlete testing positive to a banned substance Examples: • Most sports foods (sports drinks, bars, gels, liquid meal supplements) • Vitamin and mineral supplements from pharmaceutical companies or established manufacturers	The supplements in this category contain ingredients about which there is insufficient information and, hence, cause for concern. With these supplements there is an athlete testing positive to a banned substance Examples: • Supplements obtained via Internet, mail-order, personal import from overseas	The supplements in this category contain ingredients that are banned when consumed in amounts that produce a urinary caffeine level of 12 µg/mL or greater Examples: • Supplements containing caffeine and guarana	The supplements in this category are known to contain banned substances Examples include supplements containing: • Ephedra and related compounds • Strychnine • DHEA • Androstenedione • 19-norandrostenedione, 19-norandrostenediol and other pro-hormones

The responsibility for the risk associated with the use of any supplements lies with the athlete
Baylis et al. In press

17.10.2 *Toxicity and side-effects*

Most supplements fall under the banner of foods or 'listable' therapeutic goods and are considered to be relatively safe. As such, there may be no official or mandatory accounting processes to document adverse side-effects arising from the use of these products. Nevertheless, some information about toxicological problems arising from the use of supplements and herbal medicines can be found in various medical registers. Reviews from around the world summarise that while the overall risk to public health from the use of supplements, herbal and traditional remedies is low, cases of toxicity and side-effects include allergic reactions to some products (such as royal jelly), overexposure as a result of self-medication, and poisoning due to contaminants (Perharic et al. 1994; Kozyrskyj 1997; Shaw et al. 1997). These reports call for better regulation and surveillance of supplements and herbal products, and increased awareness of potential hazards.

The problems that occurred with tryptophan supplements provide a good warning of the potential problems associated with poor regulation and heavy marketing of supplements. During 1989–90, regulatory bodies in most countries recalled the sale of supplements containing synthetic forms of the amino acid tryptophan. This occurred after a large number of cases of eosinophilia-myalgia syndrome were reported over the previous five years, leading to several deaths and chronic illness (Roufs 1992). The problem was linked to the use of certain tryptophan supplements produced by microbial fermentation processes (Roufs 1992). Whether these adverse effects were due to the amino acid *per se*, a contaminant in products, or a combination of these factors has not been resolved. However, according to the FDA (1998), impurities were recently found in some supplements containing 5-hydroxy-L-tryptophan, adding weight to the continuing caution regarding tryptophan supplements.

In summary, athletes should not regard supplements and herbal remedies as harmless substances. Although some of the solutions to problems lie with better regulation of manufacturing and marketing processes, the athlete must also bear some responsibility to promote safer use of products. Practices should include avoidance of unknown or 'backyard' brands in favour of products made by larger companies known to implement good manufacturing practice, and careful adherence to recommended doses.

17.10.3 *Misplaced priorities and use of resources*

The claims made for many supplements are emotive and tempting. The promise of instant and dramatic results is alluring to all athletes, regardless of their true talent. It is understandable that athletes want to use these supplements, especially when they hear reports that their opponents or other successful competitors are using them. In our experience, many athletes do not understand that the claims made for many supplements result from lack of regulation rather than the results of rigorous research. As a result, athletes can be drawn to products that are insubstantial and

faddish rather than strategies that provide a worthwhile and lasting contribution to sports performance.

Successful sports performance is the product of genetics, long-term training, optimal nutrition, state-of-the-art equipment, and a committed attitude. These factors cannot be replaced by the use of supplements. However, if these are all in place, then an athlete may 'fine-tune' their performance capacity through the use of certain well supported supplements. Since all athletes have limited resources of time and money, it is possible that they will be tempted to see supplements as a short cut to success. Unfortunately, the supplements that receive the most attention and publicity are those with little or no documented benefits for exercise performance. At best, the use of these supplements may be a waste of money. At worst, they may distract the athlete from the use of important training strategies and tools. As nutrition practitioners we commonly see evidence of athletes who do not eat well, and who shun scientifically supported supplements, such as the intake of sports drinks during prolonged training sessions, yet are fascinated by the latest new pill or potion on the sports scene. Equally we are concerned by coaches and parents who want to introduce supplements, including products with credible uses such as creatine, to young and adolescent athletes. We feel that young athletes can make important gains in sports performance through training and adoption of good eating habits. The use of such specialised supplement products should be left until a later stage of a sporting career when goals are to fine-tune rather than lay the foundation.

17.11 SUMMARY

Athletes and coaches are convinced that a performance edge can be found through the use of sports supplements. In our experience, in order to retain a 'real world' credibility, sports scientists need to accept this belief and work within such a framework. This does not mean abandoning critical thinking or downplaying the role of nutrition in optimal performance. Rather, the sports scientist should work with the athlete and coach to allow them to make informed choices about their use of supplements. Ideally, this will mean choosing supplements which have been shown to assist in achieving their specific training and competition goals, and incorporating the appropriate use of these supplements as part of their total nutritional program. In this chapter, we divided supplements into two categories: dietary supplements and nutritional ergogenic aids. We showed that dietary supplements, such as sports drinks, liquid meal supplements, and sports bars, have a variety of well supported roles in helping the athlete to achieve their nutritional goals for optimal performance. While research may continue to refine the composition of these supplements and, perhaps, add to the family of products, the greatest need is for education of athletes to ensure the appropriate use of these dietary supplements. In many cases the information is specific to the individual athlete or sports situation and will require one-to-one

counselling. In most situations, the use of the dietary supplement will simply be part of a large plan of optimal sports nutrition or the clinical management of a nutritional disorder. Effective education will not only ensure that the dietary supplement is used correctly, but will highlight the importance of optimal eating strategies.

By contrast, the role of most of the commonly sold nutritional ergogenic aids remains unsupported. There is good evidence that caffeine, bicarbonate and creatine offer the potential of performance benefits for specific athletes in specific situations. Well-conducted research is helping to produce better guidelines for the appropriate use of such supplements. Further research is needed to clarify the potential for glycerol and antioxidant vitamins. However, the majority of nutritional ergogenic aids sold to athletes seem unlikely to produce benefits, other than a placebo effect for the athletes that believe in their promises. The supplement industry is an extremely profitable business, relying mainly on testimonials and scientific theories to market the majority of the nutritional ergogenic aids. The production and marketing of such supplements is poorly regulated with respect to quality control and the scientific support for claims. Athletes appear to be unaware of these issues and are vulnerable to problems such as wasting money and overlooking superior performance-enhancing activities. The risk of side-effects and inadvertent doping arising from the use of many supplements is small but real.

A mutual benefit for all parties (athletes, supplement manufacturers and sports scientists) will only be achieved through co-operation and pooling of resources to undertake further well-designed research and to support appropriate education programs. However, such research needs to be carefully planned and executed so that it can answer questions that are relevant to athletes and real-life sports competition.

Even where the benefits of supplements to athletic performance can be proven, it is important to put their role into perspective. The effects of training and other factors (e.g. inherited talent, equipment, mental preparation and motivation) will provide a greater influence on sports performance than the effects of a dietary supplement or nutritional ergogenic aid. But when these issues are already optimised, as in the case of the well-prepared elite athlete, the effects of supplements might provide another small but significant improvement in performance. The nutrients that can be provided by supplements to greatest effect are fluid and CHO. Other supplements are unlikely to ever provide a substitute for the factors that are basic to sports performance.

17.12 PRACTICE TIPS

Glenn Cardwell

- Each month it seems there is a new supplement or ergogenic aid produced, which is widely and frantically marketed, before taking a back seat to the next product. This constant activity makes it difficult to keep up-to-date with what is available to athletes. It is wise to make regular visits to health stores, use the

Internet, read the latest sports magazines and ask athletes about their supplements in order to keep up-to-date with what is on the market. Many supplements will clearly be of little value, while a small proportion may have enough plausibility and merit to warrant further enquiries.

- It is vital to keep up-to-date with the latest research on products. Strategies include:
  - doing frequent searches for articles on new supplements through the Internet;
  - undertaking Medline searches for scientific references;
  - checking information from manufacturers and distributors of products, some of which list references which may be of assistance;
  - searching websites for universities and professional bodies such as sports dietitians Australia and other health professionals for their comments; and
  - looking at the National Institute of Health (USA) website on dietary supplements (http://www.odp.od.nih.gov/ods/databases/ibids.html).
- Dietitians are often stereotyped as being 'anti-supplements'. It is important to check that you have no such biases, or project this view to athletes and coaches. There are some sports supplements and sports foods that have a proven worth in sports performance. These products include sports drinks, creatine, and meal-replacement drinks. Although some useful products are more expensive than the supermarket equivalent, their superior taste and convenience might be an attraction to the athlete. Athletes should be aware that various products can be beneficial to their training and competition performance, but that the value of these products is specific to the conditions of use. It is important to remain up-to-date with research that illustrates the specific uses of these products. The sports dietitian can provide a valuable service to athletes by being the interface between the science and practice of this area of sports nutrition. It is good to have fact sheets and other educational tools on various supplements that can provide information to athletes about the conditions under which they might be used. Be prepared to be make your advice practical and specific to individual sports or individual athletes.
- Companies who make useful sports supplements and sports foods may also fund research, education resources or conferences and seminars. They may also employ sports dietitians or sports science teams who should not be overlooked as valuable contacts for nutrition information. Check for websites, education resources and other items that can keep you up-to-date, or provide information for your athletes.
- Athletes will frequently ask for your advice about a new supplement that has taken their interest. Ask them to bring in supplement packaging or information. Discuss what the label says, any performance-enhancing claims and each of the ingredients. Make every effort to find out if there is any

scientific validity for the product using the strategies outlined previously. Write to the company for scientific support for their product. If they don't answer, then tell the athlete that the manufacturer is unable to furnish any supporting evidence for their product. If they provide references, check their relevance and quality. If they provide only testimonials or non-peer-reviewed articles, then it is likely they have no quality research to back their claims.

- Provide copies of good research or quality reviews that discuss the merits or drawbacks of each product. Point out that many products are claimed to have almost drug-like properties, yet have not undergone the rigorous testing that is required for pharmaceutical products. Some products have been used in studies run over six weeks. Explain that this does not indicate the long-term effects of the product.
- Many products are sold through a multilevel marketing (MLM) process. Product distributors are recruited by friends and colleagues with the promise of an 'exciting business opportunity' that can make you 'financially independent'. Testimonials from the top salespeople suggest that MLM is a quick and efficient way of becoming rich, making it attractive to many people wanting extra income. It is very rare to find anyone with tertiary nutrition qualifications involved in MLM sales. Most MLM products specifically marketed to athletes are sold on their performance-enhancing properties, usually backed by testimonials from athletes who have received free product and sponsorship. Their sporting prowess is claimed to be due to the nutrition product, with little reference to their genetic background, the hours of training and the power of psychology. Distributors are generally encouraged to make verbal health or performance claims, claims that are often illegal to make in company brochures.
- Be careful when accepting supplement products from distributors, and particularly from MLM salespeople, as acceptance of the product can imply your endorsement. Distributors may use the line 'many sports dietitians are trying our product' when the dietitian has, in fact, only been given a free sample. It may be safer to get your product samples through other means, such as simply purchasing it from a store or as an anonymous sale. If you are comfortable with the benefits of the product and the ethics of the company, then you may choose to recommend the product to athletes.
- Frequently, sports supplements provide similar nutrient value to common supermarket items or other cheaper supplements. The glamour of sport is used as a hook to increase the price. Check the ingredients of products to see if they can be duplicated by a cheaper product. For example, sports bars may be replaced by breakfast bars or muesli bars, powdered weight-gain products may be similar to pharmaceutical liquid meal products or even skim milk powder, and amino acid supplements may provide the same dose of amino acids as a carton of yoghurt. While sometimes the packaging or form of the sports

supplement makes it particularly practical for use by an athlete, on many occasions a cheaper food product or alternative supplement can be found.

- Part of the marketing of products is to state that the product meets all of the health regulations of the country of sale. Sometimes this is given a twist in terminology, for example to state that the product is 'endorsed' by TGA or ANZFA. Although the product may meet the guidelines or codes of these agencies, it generally doesn't follow that the product has any therapeutic or performance-enhancing properties. These agencies are concerned mainly with issues of safety rather than efficacy.
- There are some groups of athletes that should be particularly careful before using sports supplements. Strongly dissuade children, growing teenagers, pregnant or breast-feeding women, and women attempting conception from taking new products. It is extremely unlikely that products have been tested on such groups, and we are unaware of their effect on the growth and development of the foetus, infants and children.
- There is a small but real risk that the use of some sports supplements will lead to a positive doping test. Elite athletes should never take any supplement without first consulting their physician or sports dietitian, as it may contain ingredients that have been banned by their sporting authority. Not all of the ingredients may be listed on a label.
- Since sports supplements are a challenging and often frustrating area of sports nutrition, it is important to be realistic about your goals. The role of a sports dietitian is to provide a balanced, rational and dispassionate evaluation of each product so that the athlete can make an informed choice. This information will need to be regularly updated. The athlete will make the final choice. Do not expect to change the views of every athlete. They have various reasons for wanting to try products. For example:
  - They are curious after hearing about the product.
  - 'Everyone else is using the product, so it must work.'
  - Everyone else is using the product, so they must take it to remain on level terms with the opposition.
  - It adds a little variety and interest to their sports preparation.
  - Your views may not change them. Whether or not they continue to use a product will be based on the perceived benefits to their sports performance.
- A supplement will always be an adjunct to, not replace, a well-chosen diet. It is unlikely that any supplement in the near future will be able to redress the harm of poor quality eating, or failure to address well-supported nutritional strategies for sport, such as achieving adequate fuel intake or fluid levels. As many athletes can't get the basics right, it is the duty of the sports dietitian to stress the value of good nutrition, before an athlete considers taking a supplement.

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APPENDIX: Summary of the research literature on common sports supplements

Table 17.5 *Studies of acute creatine loading on performance (< 10 d)*

Study	Event	Subjects	Creatine dose	Enhanced performance	Comments
Smith et al. 1998	Cycling 4 maximal bouts on ergometer	15 untrained M & F	20 g/d for 5 d	Yes	Improved time to exhaustion at shorter, higher-intensity exercise bouts
Vandebuerie et al. 1998	Cycling Progressive cycle to exhaustion (~2 h 30 min) + 5 × 10 sec maximal sprints	12 well trained amateur cyclists M	25 g/d for 4 d with or without 5 g at 0, 60 and 120 min of exercise	Yes	Improved power output for the 5 × 10 sec maximal sprints Creatine during exercise counteracted improvements caused by creatine loading
Snow et al. 1998	Cycling 20 sec maximal sprint	8 untrained M	30 g/d for 5 d	No	No change to any measurements of power ~1 kg ↑ BM
Engelhardt et al. 1998	Cycling 60 min submaximal exercise + 15 sec maximal sprints	12 regional class triathletes	6 g/d for 5 d	Yes	Endurance performance not affected. Interval power performance increased by 18%
Cooke & Barnes 1997	Cycling 2 sprints separated by either 30, 60, 90 or 120 sec of recovery	80 M	20 g/d for 5 d	No	No effect on maximum power, ability to maintain peak power (PP) output or restoration of PP after 30, 60, 90 or 120 sec ~1 kg ↑ BM
Jacobs et al. 1997	Cycling Cycle to exhaustion at 125% $VO_{2\ max}$ Maximal Accumulated Oxygen Deficit (MAOD) calculated	26 M & F of varying training status	20 g/d for 5 d	Yes	Increased MAOD and time to exhaustion. Large individual variation 0.7 kg ↑ BM
Schneider et al. 1997	Cycling (A) 5 × 15 sec maximal sprints (B) 5 × 1 min cycling bouts	9 untrained M	(A) 25 g/d for 7 d (B) 25 g/d for 9 d	Yes	(A) Improved work during each 15 sec bout of maximal cycling (B) No effect on work completed
Odland et al. 1997	Cycling 30 sec Wingate test	9 active but untrained M	20 g/d for 3 d	No	No effect on any recorded exercise measures
Casey et al. 1996	Cycling 2 × 30 sec sprints with 4 min recovery	9 healthy M (chosen for good reliability) Pre- and post-trial	20 g/d for 5 d	Yes	4% increase in total work done in both bouts
Barnett et al. 1996	Cycling 7 × 10 sec sprints	17 recreationally active M	280 mg/kg for 4 d	No	No effect on multiple sprint cycle performance

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Study	Event	Subjects	Creatine dose	Enhanced performance	Comments
Cooke et al. 1995	Cycling 2 × 15 sec maximal sprints	12 untrained M	20 g/d for 5 d	No	No effect on power output or fatigue
Balsom et al. 1995	Cycling 5 × 6 sec sprints + 1 × 10 sec sprint Counter-movement jump + squat jump	7 untrained M	20 g/d for 6 d	Yes	No difference in jump performance Subjects better able to maintain power output during 10 sec exercise period
Dawson et al. 1995	Cycling (A) 1 × 10 sec sprints (B) 6 × 6 sec sprints	(A) 18 active M (B) 22 active M	20 g/d for 5 d	Yes	(A) No effect on 1 × 10 sec sprint performance (B) Improved total work, work completed in sprint 1 and peak power
Febbraio et al. 1995	Cycling 4 × 1 min cycling bouts at 115–125% VO_2 max + fifth bout to fatigue	6 active but untrained M	20 g/d for 5 d Crossover design	No	No difference in exercise duration in the fifth work bout
Birch et al. 1994	Cycling 3 × 30 sec sprints	14 untrained M	20 g/d for 5 d	Yes	Work output increased during exercise bouts 1 and 2 but not 3
Theodorou et al. 1999	Swimming Interval set (5–10 repeats on 1 or 2 min, according to the event/distance of swimmer)	22 elite swimmers (M & F) Pre- and post-test in all swimmers (order effect)	25 g/d for 4 d	Yes	Improvement of 1.5% in mean swim time in interval set. ↑ BM. Order effect dismissed by failure to improve further with longer-term study
Peyrebrune et al. 1998	Swimming 1 × 50 yd maximal swim 8 × 50 yd maximal swims	14 elite M swimmers	9 g/d for 5 d	Yes	Reduced total sprint time for 8 × 50 yd swims. Percentage decline in performance times reduced
Grindstaff et al. 1997	Swimming 3 × 100 m freestyle sprints + 3 × 20 sec arm ergometer maximal sprints	18 M & F junior competitive swimmers	21 g/d for 9 d	Yes	Improved swim time in heats 1 and 2
Mujika et al. 1996	Swimming 25 m, 50 m,100 m swim	20 national and international level swimmers (M & F)	20 g/d for 5 d	No	No significant performance changes Significant ↑ BM
Burke et al. 1996	Swimming 25 m, 50 m, 100 m swim, 10 sec maximal leg ergometry test	32 national and international swimmers (M & F)	20 g/d for 5 d	No	No significant differences between group means for sprint swim times or 10 sec leg ergometry performance

Study	Event	Subjects	Creatine dose	Enhanced performance	Comments
Rossiter et al. 1996	Rowing 1000 m rowing ergometer time trial	38 competitive, club standard rowers (M & F)	0.25 g/kg for 5 d	No	Improved 100 m rowing performance by ~1% (P = 0.088)
Terrillion et al. 1997	Running 2 × maximal 700 m run on outdoor track	12 well-trained, competitive M runners	20 g/d for 5 d	No	No significant difference between placebo or supplemented groups
Redondo et al. 1996	Running 3 × 60 m sprints	18 trained team-sport athletes and sprinters (M & F)	25 g/d for 7 d	No	No significant difference
Balsom et al. 1993	Running Treadmill run at ~120% VO_2 max to exhaustion 6 km terrain run	14 habitually active to well-trained M	20 g/d for 6 d	No	No effect on treadmill run performance. Impaired terrain run due to ↑ BM
Harris et al. 1993	Running 4 × 300 m with 4 min rest plus 4 × 1000 with 3 min rest	10 trained M middle-distance runners	30 g/d for 6 d	Yes	Reduction in running time in the final 300 and 1000 m runs
Bosco et al. 1997	Jumping and Running 5 sec and 45 sec continuous jumping exercises Treadmill running at 20 km/hr to exhaustion	8 sprinters + 6 jumpers M	20 g/d for 5 d	Yes	Improved jumping performance in the first and second 15 sec period of the jumping test Improved intensive running time to exhaustion
McNaughton et al. 1998	Kayaking 90 sec, 150 sec, 300 sec kayak ergometer tests	16 elite surf-ski/ white-water kayak paddlers (M)	20 g/d for 5 d	Yes	Subjects completed significantly more work in all tests. ↑ BM
Volek et al. 1997	Resistance exercise Bench presses + jump squat to exhaustion	14 active M	25 g for 6 d		Significant increase in reps to exhaustion and peak power for squats ↑ BM
Van Leemputte et al. 1999	12 maximal isometric elbow flexions on isokinetic dynamometer	16 untrained M	20 g/d for 5 d	Yes	Relaxation time reduced following creatine
Urbanski et al. 1999	Maximal and submaximal isometric knee extension and handgrip exercise	10 active but untrained M	20 g/d for 5 d	Yes	Increased maximal knee-extension torque and time to fatigue during submaximal knee extension and submaximal handgrip. No significant ↑ BM

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Study	Event	Subjects	Creatine dose	Enhanced performance	Comments
Smith et al. 1999	Single leg knee extension exercise to exhaustion	9 active but untrained M & F	0.3 g/kg per d for 5 d	No	Muscle ATP cost of contraction not affected
Manganaris & Maughan 1998	Maximal knee extension + knee extension to exhaustion	10 weight-trained males	10 g/d for 5 d	Yes	Maximal voluntary contraction and endurance capacity increased ↑ BM
Smith et al. 1998	Single leg knee extension exercise to exhaustion	4 × > 50yr + 5 × < 40yr M & F	0.3 g/kg for 5 d	Yes	Time to exhaustion increased in both groups combined
Vandenberghe et al. 1996	Single leg knee extension exercise	9 active but untrained M	0.5 g/kg/d for 6 d	Yes	Dynamic torque production increased. No ↑ BM
Greenhaff et al. 1993	Maximal knee extension exercise	12 M	20 g/d for 5 d	Yes	Increased muscle peak torque production

M = male, F = female

Table 17.6 *Studies of chronic creatine supplementation on performance in athletes (> 10 d)*

Study	Event	Subjects	Creatine dose	Enhanced performance	Comments
Theodorou et al. 1999	Swimming Interval session according to usual stroke/event	22 elite swimmers (M & F)	5 g/d for 8 w (following loading dose)	No	No difference between groups or across time in mean interval swim times
Leenders et al. 1999	Swimming 6 x 50 m swims 10 x 25 yd swims	32 college swimmers (M & F)	20 g/d for 6 d + 10 g/d for 8 d	Yes (M only)	Mean overall swimming velocity improved in the 6 x 50 m intervals for M. (P = 0.02) Body composition not changed
Thompson et al. 1996	Swimming Resistance calf exercise to fatigue + 100 m and 400 m swim	10 university swimmers (F)	2 g/d for 6 w	No	No effect on calf-exercise duration or swim time No ↑ in LBM
Prevost et al. 1997	Cycling 4 x protocols on different days @ 150% peak work-load to exhaustion A = continuous, B = 30 sec: 60 sec recovery, C = 20 sec: 40 sec, D = 10 sec: 20 sec	18 active M & F	19 g/d for 5 d + 2 g for 6 d	Yes	Increase in time to exhaustion and work output in all protocols, with protocol D showing greatest increase and protocol A showing smallest improvement
Earnest et al. 1997	Running 2 x treadmill runs to exhaustion (~90 sec)	11 interval- and strength-trained M	20 g/d for 4 d + 10 g/d for 6 d	Yes	Mean time to exhaustion improved Running times improved more during second run trial than first No ↑ in BM
Stone et al. 1999	Resistance exercise 1-RM parallel squat and bench press + counter-movement vertical jump and static vertical jump + 15 x 5 sec cycle ergometer sprints	42 collegiate American football players (M)	0.22 g/kg for 7 w	Yes	Increased squat and bench press, static vertical jump power output ↑ BM and LBM

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Study	Event	Subjects	Creatine dose	Enhanced performance	Comments
Volek et al. 1999	Resistance exercise 1-RM bench press + squat strength, power production during jump squat, muscular endurance during bench press	19 resistance-trained M	25 g/d for 1 w + 5 g/d for 11 w	Yes	Improved bench press and squat. Greater increases in Type I, IIA and IIAB muscle fibre cross-sectional areas ↑ BM and FFM.
Kelly & Jenkins 1998	Resistance exercise 3-RM bench press, 85% of 1-RM bench press	18 M power lifters	20 g/d for 5 d + 5 g/d for 21 d	Yes	Improved 3-RM strength ↑ BM and LBM
Kreider et al. 1998	Resistance exercise Progressive resistance training + 12 x 6 sec maximal cycle ergometer sprints	25 NCAA division 1A American football players (M)	15.75 g/d for 4 w	Yes	Increased work during sprints 1–5. Increased bench press lifting volume and total lifting volume Total ↑ BM = 2.4 ±1.4 kg
Noonan et al. 1998	Resistance exercise 1-RM bench press, 40 yd dash time, vertical jump height	30 M college athletes	20 g/d for 5 d + 100 mg/kg FFM or 300 mg/kg FFM for 51 d	Yes	Improved bench press Improvement greatest in 300 mg/kg FFM group Improved 40 yd dash time in 100 mg/kg FFM group No significant differences in body composition
Vandenberghe et al. 1997	Resistance exercise Resistance training + intermittent right arm exercise test on isokinetic dynamometer	19 sedentary F	20 g/d for 4 d + 5 g/d for 10 w	Yes	No change in arm-flexion torque or body composition after high dose creatine. Improved leg press, leg extension, squat performance, and arm-flexion torque with long term creatine ↑ FFM
Earnest et al. 1995	Resistance exercise 3 x 30 sec Wingate cycle tests + 1-RM bench press + 70% x 1-RM bench press to fatigue	8 weight-trained M	28 d (dosage n.a.)	Yes	Improved anaerobic work for Wingate tests and bench press performance ↑ BM

M = male, F = female n.a. not available

Table 17.7 *Studies of caffeine intake and endurance performance (> 60mins)*

Reference	Event	Subjects	Caffeine dose	Performance benefits	Summary
			Caffeine taken 1 h pre-exercise		
Van Soeren & Graham 1998	Cycling 80–85% $VO_{2\ max}$ to exhaustion	6 M (habitual caffeine users)	6 mg/kg (after 0, 2 & 4 days' withdrawal)	Yes	Increased time to exhaustion in all caffeine trials regardless of period of withdrawal
Trice & Haymes 1995	Cycling Bouts of 30 min alternating 1 min exercise: 1 min rest to exhaustion	8 untrained M (caffeine naive)	5 mg/kg (taken in decaf coffee)	Yes	Time to exhaustion increased 29% with caffeine. Increased FFA and trend for decreasing RPE with caffeine
Spriet et al. 1992	Cycling 80% $VO_{2\ max}$ to exhaustion	8 recreational cyclists (M & F)	9 mg/kg	Yes	Increased time to exhaustion. Glycogen sparing by 55% in the first 15 min of exercise
Costill et al. 1978	Cycling 80% $VO_{2\ max}$ to exhaustion	9 recreational cyclists (M & F)	330 mg (in decaf coffee) (= 4.4 mg/kg for M, 5.8 mg/kg for F)	Yes	Increased time to exhaustion. Evidence of increased lipolysis. Reduced RPE
Cohen et al. 1996	Running Half-marathon in hot conditions	7 trained runners (M & F)	5 and 9 mg/kg	No	No effects on RPE or performance at either dose
French et al. 1991	Running 75% $VO_{2\ max}$ for 45 min then + incremental to exhaustion	6 M recreational runners	10 mg/kg (immediately before exercise)	Yes	Caffeine increased total distance run. Elevated glucose and lactate values only seen at exhaustion. No RER data collected
Berglund & Hemmingsson 1982	Cross country skiing Field study 23 km (high altitude) & 20 km (low altitude) races	High: 13 well-trained skiers (M & F) Low: 14 well-trained skiers (M & F)	6 mg/kg	High: Yes Low: No	Trend to improved performance in low-altitude study. No metabolic data collected. Difficult to standardise environmental conditions

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Reference	Event	Subjects	Caffeine dose	Performance benefits	Summary
			Caffeine taken 1 h pre-exercise and during event		
Wemple et al. 1997	Cycling 3 h (60% VO_2 max) + time trial	6 active subjects (M & F)	1 h before + every 20 min until 160 min of ride Total caffeine = 8.7 mg/kg	No	CHO intake during exercise No difference in performance, RPE or urine losses with caffeine
Ivy et al. 1979	Cycling 2 h isokinetic cycling @ 80 rpm	9 active cyclists (M & F)	Total caffeine = 500 mg 250 mg at 1 h then 7 x doses during exercise	Yes	Increase in total work Increased mobilisation and utilisation of fat RPE same despite increased work
Falk et al. 1989	Marching 8 h at 45–50% $VO_{2\ max}$ Followed by 90% $VO_{2\ max}$ cycle to fatigue	23 untrained M (caffeine naive) Experimental-placebo design	Total caffeine = 10 mg/kg (5 mg/kg prior, then 2.5 mg/kg at 3 & 5 h)	No	No elevation in FFA with caffeine RPE differences between groups were marginal (P = 0.05)
Ferrauti et al. 1997	Tennis (240 min of competition singles followed by hitting accuracy & tennis sprint test)	16 division II tennis players (M & F)	Total caffeine = 364 mg (M) & 260 mg (F) ~4–4.5 mg/kg	Yes (F only)	Caffeine suggested to improve metabolic transition from rest to play, but is unlikely to induce metabolic effects during event

M = male, F = female

Table 17.8 *Effects of caffeine supplementation on non-endurance performance (60 min or less)*

Reference	Event	Subjects	Dose	Performance benefits	Summary
			30–60 min event **Caffeine taken 1 h pre-event**		
Denadai & Denadai 1998	Cycling Cycling to exhaustion @ 10% below anaerobic threshold	8 healthy M	5 mg/kg	Yes	Increased time to exhaustion (46 vs 32 min) and reduced RPE
Cole et al. 1996	Cycling 30 min: 3 x 10 min @ RPE of 9, 12 & 15	10 healthy M	6 mg/kg	Yes	Average of 12.6% increase in work produced for same perception of effort No differences seen in RER despite higher intensity
Pasman et al. 1995	Cycling 80%W_{max} to exhaustion	9 well trained cyclists	0, 5, 9 & 13 mg/kg	Yes	Time to exhaustion was 27% longer in caffeine trials No greater gains with increasing caffeine doses Large individual variation in urinary caffeine response
Graham et al. 1998	Running 85% $VO_{2\ max}$ to exhaustion	9 well trained M	4.5 mg/kg Caffeine, decaf coffee and coffee	Yes	Increase in time to exhaustion with caffeine Other components of coffee antagonise the responses of caffeine
Graham & Spriet 1995	Running 85% $VO_{2\ max}$ to exhaustion	8 trained M runners	3, 6, 9 mg/kg	Yes (3 & 6 mg/kg only)	Highest dose of caffeine had the greatest effect on epinephrine and metabolites, yet had the least effect on performance
Graham & Spriet 1991	Running ~85% $VO_{2\ max}$ to exhaustion Cycling ~85% $VO_{2\ max}$ to exhaustion	7 well trained M runners	9 mg/kg	Yes (both running and cycling)	3 of 13 caffeine trials above IOC limit No metabolic explanation for performance improvement Non-responder noted

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Reference	Event	Subjects	Dose	Performance benefits	Summary
			30–60 min event **Caffeine intake before and during event**		
Kovacs et al. 1998	Cycling ~60 min time trial	14 well trained M	Total caffeine = 2.1, 3.2 & 4.5 mg/kg (equal doses at 75 min pre-exercise and at 20 and 40 min during time trial)	Yes (3.2 & 4.5 mg/kg < 2.1 mg/kg < Placebo)	Addition of caffeine to CHO/electrolyte drinks improved 60 min time trial performance Performance threshold at 3.2 mg/kg Dose well within legal limits
Saski et al. 1987	Running 80% $VO_{2\ max}$ for 45 min rest—then to fatigue	5 well trained males	Total caf. = 420 mg (mean ~7.25 mg/kg)	Yes	Caffeine + sucrose not significantly different from caffeine or sucrose alone, despite changes in substrate metabolism
			~20 min events		
Denadai & Denadai 1998	Cycling Cycling to exhaustion @ 10% above anaerobic threshold	8 healthy M	5 mg/kg	No	No difference in time to exhaustion (18.5 vs 19.2 min) or RPE
Mohr et al. 1998	Cycling Electronic stimulation of limbs	7 Tetraplegic ($C_{5\text{-}7}$) & 2 Paraplegic (T_4) males	6 mg/kg	Yes	Time to exhaustion 6% longer in caffeine trials Supports caffeine having a direct ergogenic effect on skeletal muscle
Bell et al. 1998	Cycling 85% VO_2 peak to fatigue	8 untrained M	5 mg/kg (90 min prior)	No	Caffeine elevated epinephrine, FFA, glycerol and glucose yet had no effect on performance
Fulco et al. 1994	Cycling 80% $VO_{2\ max}$: at sea level and altitude	8 untrained M	4 mg/kg	Yes	Caffeine increased time to exhaustion during acute altitude exposure only Influence of caffeine seemed to decrease with exposure to altitude

Reference	Event	Subjects	Dose	Performance benefits	Summary
Dodd et al. 1991	Cycling Incremental test to exhaustion	17 untrained M (8 caffeine naive & 9 habitual users)	3 & 5 mg/kg	No	Time to exhaustion unaffected by caffeine dose or intake history Caffeine-naive subjects showed heightened resting V_E, VO_2, HR & exercising FFA
Flinn et al. 1990	Cycling Incremental test to exhaustion	9 untrained M	10 mg/kg (3 h prior)	Yes	Increase in time to exhaustion & work completed Suggested taking caffeine 3 h prior to exercise to allow plasma FFA to peak
Bond et al. 1987	Cycling Incremental test to exhaustion	6 healthy M	5 mg/kg	No	Time to exhaustion unaffected by caffeine Caffeine failed to alter any respiratory or blood parameter other than slightly increasing blood glucose
Gaesser & Rich 1985	Cycling Incremental test to exhaustion	8 healthy M	5 mg/kg	No	Max values for work, VO_2, V_E, HR, RER and lactate along with lactate threshold were unaffected by caffeine
Powers et al. 1983	Cycling Incremental test to exhaustion	7 M recreational cyclists (2 w caffeine withdrawal)	5 mg/kg	No	Values for TTF, VO_2, HR, RER, FFA & lactate were all unaffected by caffeine. Plasma glycerol levels increased
MacIntosh & Wright 1995	Swimming 1500 m freestyle	11 well-trained M & F swimmers	6 mg/kg	Yes	23 sec improvement in swimming times Caffeine affected substrate and electrolyte balance
			~5 min events **Caffeine 1 h pre-exercise**		
Jackman et al. 1996	Cycling 100% $VO_{2\ max}$: 2 x 2 min, then to exhaustion	14 untrained (M & F) subjects	6 mg/kg	Yes	Increase in time to exhaustion for 10 subjects No glycogen sparing

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Reference	Event	Subjects	Dose	Performance benefits	Summary
Doherty et al. 1998	Running Time to exhaustion (3–4 min) MAOD	9 trained M	5 mg/kg	Yes	10–14% improvement in time to exhaustion Increased MAOD Mechanism unclear
Wiles et al. 1992	Running 1500 m time trial	18 trained M	3 g caffeinated coffee (~150–200 mg caffeine)	Yes	Av 4.2 sec faster over 1500 m
	1100 m constant speed + 1 min max: 'final burst' at self-selected pace	10 well trained athletes	As above	Yes	Caffeine improved speed of final 1 min 'burst', reduced RER and increased VO_2
	1500 m @ 0.5 km/h below max 1500 m pace	6 well trained M athletes	As above	Not measured	Caffeine increased VO_2 However RER, lactate and RPE unaffected by caffeine
			Sprint events		
Greer et al. 1998	Cycling 4 x 30 sec Wingate tests	9 untrained M	6 mg/kg	No	No effects on peak power, average power or rate of power loss Hence no indication for use of caf in sports requiring repeated supramaximal bouts of activity
Anselme et al. 1992	Cycling Repeated 6 sec sprints: force/velocity exercise test	14 untrained M & F subjects	250 mg (30 min prior to exercise av.~ 4 mg/kg)	Yes	Caffeine increased pedalling frequency and hence W_{max}. caffeine elevated lactate and lactate/W
Collomp et al. 1991	Cycling 1 x 30 sec Wingate test	6 untrained M & F subjects	5 mg/kg	No	No support for untrained individuals to take caffeine to enhance supramaximal exercise performance
Williams et al. 1988 `	Cycling 1 x 15 sec maximum power test	9 M (caffeine naive)	7 mg/kg	No	Caffeine failed to increase max power output or alter rate or magnitude of fatigue
Collomp et al. 1992	Swimming 2 x 100 m sprints	14 trained (M & F) and 7 recreational (M & F) swimmers	250 mg (~4 mg/kg)	Yes (in trained subjects)	Suggested that specific training is required for caffeine to produce improvements in anaerobic capacity

M = male, F = female

Table 17.9 *Studies of bicarbonate or citrate loading on sports specific performance: crossover designed studies*

Reference	Event	Subjects	Dose	Enhanced performance	Summary
McNaughton et al. 1999	Cycling 60 min time trial	10 well trained M cyclists	300 mg/kg sodium bicarbonate	Yes	14% more work completed with bicarbonate
Potteiger et al. 1996	Cycling 30 km time trial	8 M	500 mg/kg sodium citrate	Yes	Reduction in time trial time (57 min 36 sec vs 59 min 22 sec Sodium citrate raised pH values from 10 km onwards and improved power output in the initial 25 min
Tiryaki et al. 1995	Running 600 m	11 collegiate F runners + 4 untrained controls	300 mg/kg sodium citrate or sodium bicarbonate	No	No performance effect despite significant changes to acid/base status
Goldfinch et al. 1988	Running 400 m	6 trained M	400 mg/kg sodium bicarbonate	Yes	Improved running time [56.94 sec vs 58.63 sec (placebo) and 58.46 sec (control)] Elevated post-exercise values for pH and base excess
Wilkes et al. 1983	Running 800 m	6 varsity track M athletes	300 mg/kg sodium bicarbonate	Yes	Improved running time [2 min 2.9 sec vs 2 min 5.1 sec (placebo) and 2 min 5.8 sec (control)] Elevated post-exercise values for pH, lactate and blood bicarbonate
Kindermann et al. 1977	Running 400 m	10 healthy M	200 mg/kg infusion of bicarbonate	No	Questions the importance of pH as a limiting factor in performance of high intensity exercise
Gao et al. 1988	Swimming 5 x 100 yd swim: 2 min rest	10 US collegiate swimmers	250 mg/kg sodium bicarbonate	Yes	Faster times in 4th and 5th swim
McNaughton & Cedaro 1991	Rowing 6 min maximal effort on ergometer	5 elite M rowers	300 mg/kg sodium bicarbonate	Yes	Increased work and distance rowed (1861 m vs 1813 m) Increased lactate levels

M = male, F = female

Table 17.10 *Table of studies of BCAA supplementation and performance*

Study	Event	Subjects	BCAA dose	Enhanced performance	Comments
Mittleman et al. 1998	Cycling in the heat (34°C) Time to exhaustion at 40% $VO_{2\ max}$	13 moderately trained M & F Crossover design	9.4 g F or 15.8 g M	Yes	Increased time to exhaustion (153 vs 137 min) with BCAA trial Increase in plasma BCAA and decrease in plasma tryptophan: BCAA Trend to higher plasma ammonia No difference between sexes
Blomstrand et al. 1997	Cycling 60 min @ 70% $VO_{2\ max}$ + 20 min time trial Stroop Colour Word Test given after ride	7 trained M cyclists Crossover design	90 mg/kg (~6.5 g)	No	No differences in work done in time trial, however, RPE lower during BCAA trial during steady-state phase Index of cognitive function (Stroop Colour Word Test) improved after exercise on BCAA trial
Madsen et al. 1996	Cycling 100 km time trial	9 well trained M cyclists Crossover design	3.5 L @ 5% glucose or 5% glucose + 18 g BCAA	No	No performance differences between trials Plasma BCAA and ammonia levels higher with BCAA trial
Davis et al. 1999	Running Imtermittent shuttle run to exhaustion	8 active M & F Crossover design	CHO + 7 g BCAA or CHO or placebo consumed before/during/after trial	No	CHO or CHO + BCAA increased time to fatigue compared with placebo No further enhancement with addition of BCAA
Blomstrand et al. 1991	Running Marathon or 30 km cross-country race Run time, plus Stroop Colour Word Test (CWT) given after cross-country run	25 M cross-country runners, 193 M marathon runners Experimental-placebo design	16 g (marathon) or 7.5 g (cross-country)	Yes	CWT performance improved in BCAA trial after cross-country run 'Slower runners' ran faster in BCAA group Note that the methodology of dividing group into slower and faster runners according to arbitrary timepoint has been criticised on statistical grounds
Blomstrand et al. 1991	Soccer Soccer match: Stroop Colour Word Test given after match	6 F national soccer players Crossover design	6% CHO + 7.5 g BCAA or CHO alone	Yes	Improvement in CWT test after game with CHO + BCAA

M = male, F = female

Table 17.11 *Studies of glycerol hyperhydration and performance*

Study	Event	Subjects	Glycerol dose	Enhanced performance	Comments
Anderson et al. In press	Cycling 90 min @ 98% lactate threshold + 15 min time trial Hot environment (35°C)	6 well trained M cyclists Crossover design	1 g/kg with 20 mL/kg low-joule cordial (compared with low-joule cordial overload)	Yes	Glycerol allowed retention of additional 400 mL of fluid above hyperhydration with cordial alone 5% improvement in work done in 15 min time trial No change in muscle metabolism Reduced rectal temperature at 90 min with glycerol trial
Hitchins et al. 1999	Cycling 30 min @ fixed power + 30 min time trial Hot environment (32°C)	8 well trained M cyclists Crossover design	1 g/kg with 22 mL/kg dilute sports drink, 2.5 h pre-exercise (compared with sports drink overload)	Yes	Glycerol treatment expanded body water by 600 mL and increased (5%) work achieved in time trial This was achieved largely by preventing the drop in power seen at the start of placebo time trial No difference in power profile at end of time trials No difference in cardiovascular, thermoregulatory, RPE between trials despite differences in power output
Inder et al. 1998	Cycling 60 min @ 70% $V0_2$ max + incremental ride to exhaustion	8 highly trained M triathletes Crossover design	1 g/kg with 500 mL water, 4 h pre-exercise (compared with 500 mL water)	No	Glycerol was consumed with a modest fluid load No increase in pre-exercise hydration status, sweat losses or urine production during exercise No difference in time to exhaustion or workload reached 3 subjects experienced GI problems with glycerol
Montner et al. 1996	Cycling Cycling @ 60% W_{max} until exhaustion	11 active M & F 7 active M & F Crossover designs	1.2 g/kg with 26 mL/kg water, 1 h pre-exercise same pre-treatment + sports drink during exercise	Yes	Reduced HR and increased time to exhaustion with pre-exercise glycerol treatment by ~20%

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Study	Event	Subjects	Glycerol dose	Enhanced performance	Comments
Murray et al. 1991	Cycling 90 min cycling @ 50% $VO_{2\ max}$	9 active M & F Crossover design	0.9 g/kg (10%) glycerol solution or 4% glycerol + sports drink consumed during exercise. Total fluid load = 12 mL/kg (compared with sports drink or water of equal volumes)	Not measured No benefits to thermo-regulation seen	No differences in hydration status, urine production, HR, sweat loss or body temperature No benefits seen to suggest superior thermoregulatory or performance outcomes
Latzka et al. 1998	Running Treadmill running at 55% $VO_{2\ max}$ until exhaustion or high rectal temperature Hot environment (35°C) without further fluid intake	8 heat-acclimatised men Crossover design	1.2 g/kg LBM + 29 mL/kg water, 1 h pre-exercise (compared with water hyperhydration or control)	Yes (better than control, but equal to water hyper-hydration)	Both hyperhydration trials increased body fluid by ~1400 mL No advantages in either trial regarding sweat losses, cardiac output or temperature control; however, time to exhaustion longer in both trials compared with control Some GI and headache symptoms with glycerol
Lyons et al. 1990	Running 90 min treadmill run @ 60% $VO_{2\ max}$	6 M & F	1 g/kg with 25 mL/kg fluid, 2.5 h pre-exercise + 0.1 g/kg/h during exercise (compared with water hyperhydration and low fluid intake)	Not measured Benefits to thermo-regulation seen	Glycerol increased fluid retention by ~500 mL over water alone Increased sweat rate and decreased rectal temperature in glycerol trial suggest thermoregulatory benefits

M = male, F = female

Table 17.12 *Studies of ginseng supplementation and metabolism or performance*

Study	Event	Subjects	Ginseng dose	Enhanced performance	Comments
Ziemba et al. 1999	Cycling Incremental test to exhaustion Reaction time measured at each stage	15 M soccer players Experimental-placebo design	350 mg/d for 6 w	No (but enhanced reaction time)	No change in lactate threshold or $VO_{2\ max}$, however, enhanced reaction time at submaximal workloads
Allen et al. 1998	Cycling Incremental test to exhaustion	28 healthy subjects (M & F) Experimental-placebo design	200 mg/d for 3 w	No	No enhancement of total workload, RPE and lactate at submaximal loads or $VO_{2\ max}$
Engels & Wirth 1997	Cycling Submaximal and maximal cycling test	37 healthy M Experimental-placebo design	200 mg/d or 400 mg/d for 8 w	Not measured but no metabolic changes	No change in RPE, HR or RER at submaximal or maximal workloads
Morris et al. 1996	Cycling Time to exhaustion @ 75% $VO_{2\ max}$	8 active subjects (M & F) Crossover design	8 mg/kg/d or 16 mg/kg/d for 1 w standard ginseng preparation used	No	No change in time to exhaustion or metabolic parameters No change in RPE
Dowling et al. 1996	Running 10 min treadmill test at 10 km race pace, maximal treadmill test	20 highly trained runners (M & F) Experimental-placebo design	60 drops/d (maximum recommended dose) of Russian ginseng for 6 w	No	No change in metabolic characteristics at race pace, treadmill max, RPE Low statistical power may prevent small changes from being detected
Pieralisi et al. 1991	Running Incremental treadmill test to exhaustion	49 active subjects (M) Crossover design	2 capsules/d for 6 w Ginsana 115 (ginseng + vitamins, bitartrate + minerals)	Yes	Increased $VO_{2\ max}$ and reduced O_2 consumption at submaximal workloads
McNaughton et al. 1989	Physical testing $VO_{2\ max}$, grip, pectoral and quadriceps strength	30 active subjects (M & F) Crossover design	1 g/d x 6 w of Chinese ginseng or Russian ginseng	Yes	Significantly greater increase in $VO_{2\ max}$ and pectoral and grip strength with Chinese ginseng Trends for enhancement with Russian ginseng

M = male, F = female

Table 17.13 *Studies of carnitine supplementation and metabolism or performance*

Study	Event	Subjects	Carnitine dose	Enhanced performance	Comments
Barnett et al. 1994	Cycling 4 min ride at 90% $VO_{2\ max}$ + 5 x 1 min rides at 115% $VO_{2\ max}$	8 untrained M Crossover design– order effect	4 g/d for 14 d	Not measured No metabolic enhance-ments	Supplementaion failed to increase muscle carnitine content No change in lactate accumulation during submaximal and supramaximal performance
Vukovich et al. 1994	Cycling 60 min at 70% $VO_{2\ max}$ High-fat pre-trial diet to promote lipid availability	8 untrained M Crossover design	6 g/d for 7 and 14 d	Not measured No metabolic enhance-ments	No change in muscle carnitine content after supplementation No effect on VO_2, RER, HR or substrate utilisation Without glycogen sparing there is no mechanism to expect performance enhancements
Decombaz et al. 1993	Cycling CHO depletion regime + 20 min at 60% $VO_{2\ max}$	9 untrained M Crossover design	3 g/d for 7 d	Not measured No metabolic enhance-ments	Substrate metabolism not affected during submaximal exercise even after glycogen depletion and situation of high lipid flux
Siliprandi et al. 1990	Cycling Cycle to exhaustion	10 moderately trained M Crossover design	2 g @ 1 h before exercise (acute administration)	Yes	Increased time to exhaustion. Carnitine reduced the increase in plasma lactate and pyruvate after maximal progressive work, however, dose and timeframe for uptake into muscle seems unrealistic
Vecchiet et al. 1990	Cycling Incremental cycling to exhaustion	10 moderately trained M Crossover design	2 g @ 1 h before exercise (acute administration)	Yes	Increase in time (and work) until exhaustion Decrease in lactate production and oxygen consumption at same workload As for Siliprandi study, others have criticised the dose and timing of supplement
Gorostiaga et al. 1989	Cycling 45 min at 66% $VO_{2\ max}$ + 60 min seated recovery	10 endurance trained M & F Crossover design	2 g/d for 28 d	Not measured Metabolic enhance-ments	Reduced RER during exercise ($P < 0.05$). Non-significant trend for higher O_2 uptake, HR, blood glycerol and resting plasma FFA. Provides mechanism by which enhancement of endurance exercise might be seen

Study	Event	Subjects	Carnitine dose	Enhanced performance	Comments
Soop et al. 1988	Cycling 120 min at 50% $VO_{2\ max}$	7 moderately trained M Crossover design, with order effect	5 g/d for 5 d	Not measured No metabolic enhance-ments	No effect on muscle substrate utilisation during exercise or at rest
Oyono-Enguelle et al. 1988	Cycling 60 min at 50% $VO_{2\ max}$ + 120 min recovery	10 untrained males Crossover design with 2 control trials and 1 experimental trial. Order effect	2 g/d for 4 w	Not measured No metabolic enhance-ments	No change in in physiological parameters and blood metabolites. The increased demand for FA oxidation during exercise is adequately supported by endogenous carnitine
Greig et al. 1987	Cycling Progressive test to exhaustion	(A) 9 untrained M & F (B) 10 untrained M & F Crossover design	2 g/d for 2 w 2 g for 4 w	No	No significant physiological changes. Changes in performance were small and inconsistent
Trappe et al. 1994	Swimming 5 x 91.4 m swims	20 highly trained collegiate swimmers (M) Experimental-placebo design	4 g/d for 7 d	No	No difference in performance times between trials or between groups
Colombani et al. 1996	Running Marathon run + submaximal performance test day after marathon	7 endurance trained athletes (M) Crossover design	2 g @ 2 h before run and at 20 km mark	No	No change in exercise metabolism or marathon running time. No change in recovery and submaximal test performance on following day
Marconi et al. 1985	Running Supramaximal work (jumps) + treadmill $VO_{2\ max}$ + submaximal run	6 national class walkers Crossover design	4 g/d for 2 w	Yes	Increase in $VO_{2\ max}$ by 6%, but no effects on oxygen utilisation and RER at submaximal loads, or change in lactate accumulation with jumps. Results appear inconsistent

M = male, F = female

Table 17.14 *Studies of coenzyme Q10 supplementation and performance*

Study	Event	Subjects	Carnitine dose	Enhanced performance	Comments
Nielsen et al. 1999	Cycling Incremental $VO_{2\ max}$ test to exhaustion Energy status of the muscle measured by NMRS during contractions	7 well trained M triathletes	100 mg/d for 6 w (+ Vit E + Vit C)	No	No effect on maximal oxygen uptake or muscle energy metabolism
Svensson et al. 1999	Cycling 30 sec max cycle + 10 x 10 sec max cycles	17 well trained M Experimental-placebo design	110 mg/d for 20 d	Not measured No evidence of improved lipid peroxidation	Supplementation increased plasma but not muscle Q10 Q10 did not affect purine catabolism or plasma malondialdehyde (indication of lipid peroxidation) Combined with previous data from group, suggests that Q10 may reduce training adaptations*
Malm et al. 1997	Cycling Anaerobic training sessions + anaerobic + submaximal cycling tests undertaken throughout 2 d of supplementation	18 M Experimental-placebo design	120 mg/d for 22 d	No*	Q10 produced negative effect on anaerobic cycling performance—failure to achieve a training effect Increased CK levels with Q10 No effect on submaximal performance
Weston et al. 1997	Cycling Normal training undertaken during 28 d supplementation Incremental test to exhaustion undertaken pre- and post-exercise	18 trained cyclists and triathletes (M) Experimental-placebo design	1 mg/kg/d for 28 d	No	Q10 did not enhance any performance parameters
Malm et al. 1996	Cycling Anaerobic training sessions + anaerobic + submaximal cycling tests undertaken throughout 22 d of supplementation	15 healthy M Experimental-placebo design	120 mg/d for 22 d	No*	Q10 produced ergolytic effect on anaerobic cycling performance—failure to achieve a training effect No effect on submaximal performance

Study	Event	Subjects	Carnitine dose	Enhanced performance	Comments
Laaksonen et al. 1995	Cycling Prolonged endurance test to exhaustion	11 young and 8 older trained M Crossover design	120 mg/d for 6 w	No*	No change in muscle Q10 concentrations or plasma malondialdehyde as a result of Q10 supplementation Negative effect on time to exhaustion (placebo had greater endurance)
Snider et al. 1992	Cycling and running 90 min on treadmill @ 70% $VO_{2\ max}$ + cycling @ 70% $VO_{2\ max}$ to exhaustion	11 highly trained triathletes Crossover design	100 mg/d for 4 w (+ vitamin E, inosine, cytochrome C)	No	No difference in time to exhaustion between trials No differences in blood metabolites or RPE
Braun et al. 1991	Cycling Incremental test to exhaustion performance pre- and post-supplementation period Training continued during period	10 M cyclists Experimental-placebo design	100 mg/d for 8 w	No	Performance increased equally in both groups after training period Q10 had no effect on cycling performance or any measured parameters Malondialdehyde concentrations reduced in both groups after training
Kaikkonen et al. 1998	Running Marathon (field test)	37 moderately trained M runners Experimental-placebo design	90 mg/d for 3 w (+ vit E)	Not measured No change in muscle damage	No change in indices of oxidative or muscle damage following marathon run
Ylikoski et al. 1997	Cross-country skiing Treadmill pole-walking to exhaustion	25 national level cross-country skiers Experimental-placebo design	90 mg/d for 6 w	Yes	Improved $VO_{2\ max}$ with Q10 supplementation Increase in aerobic and anaerobic thresholds No control of exercise during supplementation periods

** Decrease in performance, M = male, F = female*

Table 17.15 *Studies of inosine supplementation and performance*

Study	Event	Subjects	Inosine dose	Enhanced performance	Comments
McNaughton et al. 1999	Cycling 5 x 6 sec, 30 sec and 20 min time trial	7 well-trained M Crossover design	10 000 mg for 5 and 10 d	No	No improvements in sprint times or time trial performance Increase in plasma uric acid concentrations
Starling et al. 1996	Cycling Wingate bike test, 30 min self-paced cycle, supramaximal sprint to fatigue	10 competitive M cyclists Crossover design	5000 mg/d for 5 d	No*	No difference in Wingate performance or 30 min cycle Negative effect on time to fatigue Increase in plasma uric acid concentration
Williams et al. 1990	Running Submaximal warm-up run, competitive 3-mile treadmill run, maximal treadmill run	9 highly trained M & F endurance runners Crossover design	6000 mg/d for 2 d (maximum recommended dose)	No*	No effect on 3-mile run time, $VO_{2\ peak}$ or other variables Negative effect on maximal run

** Decrease in performance, M = male, F = female*

Table 17.16 *Studies of chromium picolinate and changes in body composition*

Study	Event	Subjects	Chromium Picolinate dose	Enhanced performance	Comments
Walker et al. 1998	14 w resistance and conditioning training program and endurance testing Pre- and post-testing of strength, peak power, body composition, Wingate test, $VO_{2\ max}$ on run treadmill	20 M collegiate wrestlers Experimental-placebo design	200 µg/d for 14 w	No	Did not enhance body composition or performance variables beyond improvements seen with training alone
Grant et al. 1997	9 w supplementation with or without resistance training program Testing = BM, composition	43 obese F Experimental-placebo design	400 µg/d chromium picolinate or nicotinate for 9 w	No (↑ BM with chromium picolinate)	↑ BM in chromium picolinate group ↓ BM and insulin response with training and chromium nicotinate
Lukaski et al. 1996	8 w resistance training program Pre- and post-testing of strength, body composition and iron status	36 untrained M Experimental-placebo design	3.4 µmol/d (~200 µg/d) chromium picolinate or or chloride for 8 w	No	No beneficial effects on LBM, body fat or strength above training effect No difference between chromium preparations Trend for ↓ iron status (↓ transferrin status) with chromium picolinate
Hallmark et al. 1996	12 w resistance training program Pre- and post-testing of strength and body composition	16 untrained M Experimental-placebo design	200 µg/d for 12 w	No	No differences in body composition with training or supplement Strength increases independent of supplement

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Study	Event	Subjects	Chromium Picolinate dose	Enhanced performance	Comments
Trent & Thieding-Cancel 1995	16 w physical conditioning program Pre- and post-testing of body composition	95 active-duty Navy personnel (M & F) Experimental-placebo design	400 µg/d for 16 w	No	No differences in BM and fat changes between groups (note body composition measured by anthropometry)
Clancy et al. 1994	9 w pre-season resistance and conditioning training Pre-, mid- and post-testing of body composition, strength	36 M collegiate football (gridiron) players Experimental-placebo design	200 µg/d for 9 w	No	No enhancement of BM, body composition or strength above placebo group
Hasten et al. 1992	12 w resistance training program Pre- and post-testing of body composition and strength	59 M & F college students Experimental-placebo design	200 µg/d for 12 w	No (but ↑ BM in F)	No differences in strength changes due to chromium picolinate Greater ↑ in BM in F with chromium but no difference with M No difference with loss of body fat

M = male, F = female

Table 17.17 *Studies of MCT + CHO supplementation and ultra-endurance performance: crossover design*

Study	Event	Subjects	MCT dose	Enhanced performance	Comments
Angus et al. 2000	Cycling 100 km time trial (~3 h)	8 endurance trained M cyclists/triathletes	1 L per h of 6% CHO + 4% MCT (vs 6% CHO or placebo) (total intake of MCT = 42 g per or ~120 g)	No	CHO-enhanced performance over placebo, but addition of MCT does not provide further benefits 4 subjects experienced GI problems with MCT No differences in fat oxidation, plasma FFA between MCT and CHO + MCT Suppression of fat oxidation due to high exercise intensity or pre-trial CHO meal causing high insulin concentrations
Goedecke et al. 1999	Cycling 2 h @ 63% $VO_{2\ max}$ + 40 km time trial (~70 min)	9 endurance trained M cyclists	1.6 L of 10% CHO or 10% CHO + 1.7% MCT or 10% CHO + 3.4% MCT (total intake of MCT = 27 or 52 g)	No	No differences in time trial performance 2 subjects experience GI distress with higher MCT intake Higher FFA with MCT but no change in CHO oxidation
Jeukendrup et al. 1998	Cycling 2 h @ 60% $VO_{2\ max}$ + time trial (~15 min)	9 endurance trained M cyclists/triathletes	20 mL/kg of 10% CHO or 10% CHO + 5% MCT or 5% MCT or placebo (total intake of MCT = 86 g)	No	No difference between CHO, CHO + MCT or placebo (~14 min) but MCT alone impaired performance (17.3 min) MCT + CHO showed slightly higher fat oxidation than CHO alone No glycogen sparing seen
Van Zyl et al. 1996	Cycling 2 h @ 60% $VO_{2\ max}$ + 40 km time trial (~70 min)	6 endurance trained cyclists	2 L of 4.3% MCT or 10% CHO or 10% CHO+ 4.3% MCT (total intake of MCT = 86 g)	Yes	MCT + CHO-enhanced time trial performance times (65.1 min) compared with CHO (66.8 min) and MCT (72.1 min) Increase in FFA and glycogen sparing with MCT + CHO

M = male, F = female, GI = gastrointestinal