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Review article

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DIETARY FAT AND SPORTS NUTRITION: A PRIMER

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ABSTRACT

The general public's view of macronutrients has undergone sweeping changes in recent years. Dietary fats are a key example. Since the anti-fat health education initiatives of the 1980s and early 1990s, certain dietary fats have been increasingly recognized as actually beneficial to health. Athletes, like the mainstream populace, are now getting the message that wise dietary fat (triacylglycerol) choices offer essential fatty acids, blood lipid management, maintained endocrine and immune function, inflammation control, metabolic effects and even potential body composition and performance benefits. Toward this end, many companies now sell specialty dietary fat supplements and recognized health authorities have begun recommending them to certain populations. This review will cover data regarding the physiology, dietary needs, food sources, and potential benefits and risks most relevant to athletes. Practical suggestions for incorporating healthy fats will be made. Both food-source and supplemental intakes will be addressed with interrelationships to health throughout.

KEY WORDS: Dietary fat, triacylglycerol, fatty acids, metabolism, inflammation, recovery.

INTRODUCTION

Dietary fat has both suffered and enjoyed large swings in public and scientific consensus over recent decades. The fat-reduction public education initiatives of the 1980s and 1990s (Weinberg, 2004), although credited with lower cardiovascular mortality, (Astrup, 2001) have also been linked to over-consumption of dietary carbohydrate and the obesity epidemic facing Western culture (Weinberg, 2004).

The escalating incidence of Syndrome X (central obesity, dislipidemia and glucose intolerance) has helped bring a more "moderate" approach to the *Dietary Guidelines for Americans* regarding fat's percentage of total kcal (Gifford, 2002). Additionally, an increased recognition of the types of dietary fat has broadened scientific understanding beyond simply saturated and unsaturated fatty acids. Further, researchers have referred to the potency of various dietary lipids as pharmaceutical in nature (DeCaterina et al., 1996; Fauconnot and Buist, 2001; Watkins et al., 2001). For example, monounsaturated fatty acids, as common to the Mediterranean diet, may reduce cardiovascular risks beyond any effects on plasma lipids, such as via blood pressure normalized glucose tolerance (Perez-Jimenez et al., 2002; Rasmussen et al., 1995; Thomsen et al., 1995). Highly unsaturated omega-3 fatty acids found in cold water fish reduce inflammation (Browning, 2003; Calder, 1997, 2001; Endres et al., 1989; Endres, 1996; Kremer et al., 1987), mediate psychiatric function (Logan, 2003; Su et al., 2003), alter neuro-endocrine activity (Delarue et al., 2003), and decrease cardiac mortality (Richter, 2003). A

less common fatty acid found in dairy and beef, conjugated linoleic acid (CLA), has the ability to dramatically alter body composition in animal models (Belury and Koster, 2004). This type of understanding is leading to changes in both dietary recommendations (American Heart Association, 2002) and a wide variety of dietary lipid supplements.

Athletes have special interests and needs regarding dietary fat. Ironically, many are at risk of being hypocaloric (Burke, 2001; Economos et al., 1993; Venkatraman, 2000), yet they also seek glycogen sparing and fatigue prevention (Hargreaves et al., 2004). These situations are aided by available, energy dense fat (9 $kcal \cdot g^{-1}$). Athletes also commonly deal with joint, soft tissue, systemic and even airway inflammation, which may also be affected by fat choices (Browning, 2003; Calder, 1997; 2001; Curtis et al., 2000; Endres et al., 1989; Endres, 1996, Mickleborough et al., 2003). Additionally, overtraining and staleness occur in roughly one-third to one-half of athletes (Kentta et al., 2001). These disorders have established endocrine and psychiatric components such as depressed testosterone or testosterone:cortisol ratio (Roberts et al., 1993; Urhausen et al., 1995), increased epinephrine during intensity–type overtraining (Fry et al., 1994), and even major depression (Armstrong and VanHeest, 2002; Uutisalo et al., 2004). All of these maladies have been positively affected by various amounts and types of dietary fat in various settings (Delarue et al., 2003; Dorgan et al., 1996; Hamlainen et al., 1983; Logan, 2003; Reed et al., 1987; Su et al., 2003). It is also interesting to speculate that the effect of maintained total fat intake on sex hormones (Dorgan et al., 1996; Hamlainen et al., 1983, Reed et al., 1987) and the reported protective effects of omega-3 fats against bone catabolism (Albertazzi and Coupland, 2002; Fernandes et al., 2003; Watkins et al., 2001), may have future application to the "female athlete triad", in which energy balance, sex hormones and bone mass are compromised. As with all of the potential benefits resented in this review, either directly applied or indirectly associated to athletes, further research is needed.

This review will briefly address the general biochemistry and physiology of dietary fat, dietary needs and food sources of various fats, and potential benefits and risks of various dietary fat manipulations to athletes.

Part 1. General Biochemistry and Physiology

Biochemistry

The preponderance of lipid in the human diet is in the form of triacylglycerols (formerly "triglycerides"). These triacylglycerols are composed of a glycerol "backbone" of three carbons with three fatty acids attached. It is primarily these fatty acids, which range widely in length up to approximately 22 carbons that are broken down for energy. The sheer number of carbons in fatty acids, compared to carbohydrate for example, is the reason for the fact that dietary fats contain more than twice the energy (9 kcal) of carbohydrates (4 kcal) in the body. That is, they contain comparatively long *aliphatic chains* as opposed to a hexagonal or pentagonal-looking ring structure (of six carbons). Proteins are not a primary source of energy but do provide roughly the same amount of energy as carbohydrates in the body (4 kcal). In order to be fully oxidized, or "burned", fatty acids require a specific biochemical pathway called *beta-oxidation* in the *mitochondria* of cells. Fatty acid oxidation will be addressed in Part Four of this review. *Exercise Metabolism*.

Differences in fatty acid chain length, desaturation (the number of carbon-carbon double bonds), and position of these double bonds on the chain all contribute to a vast variety of biological effects beyond simple provision of energy. For example, a lack of carbon-carbon double bonds makes a *saturated fatty acid*, as is common to animal fats. Even though not all saturated fats appear equally "unhealthy" regarding heart disease risk and cell membrane effects (National Cattlemen's Association, 2000), these are generally considered less desirable. Arguably just as detrimental are *trans-fatty acids*, which are simply common "cis" fatty acids that have had the region about their carbon-carbon double bonds altered via commercial hydrogenation. This process improves commercial aspects such as reduced rancidity but creates an "unnatural", straightened fatty acid that has been linked to elevated heart disease risk, and inflammation (Lichtenstein, 2000; Popkin et al., 2004).

Conversely, some fatty acids are pharmacologically beneficial and even essential to health. That is, humans lack the enzymes to make them, so in their absence symptoms develop. Linoleic acid is an essential omega-6 (n-6) type of fatty acid that is perhaps too common in the Western diet (Mann et al., 1995; Simopoulos, 2002) but necessary for eicosanoid synthesis nonetheless. Linolenic acid is another essential fatty acid, this time of the under-consumed omega-3 (n-3) type (Simopoulos, 2002). It has specific effects of its own (DeCaterina et al., 1996) but can also elongate and desaturate to have further biological impact. EPA (eicosapentaenoic acid) and DHA (docosahexaenoic acid), commonly referred to as "fish oils", are considered more "potent" pharmacologically than

linolenic acid in some regards and are fellow omega-3 fatty acids (Ehringer et al., 1990; Su et al., 1999). Omega-3 fats have been shown to have a number of beneficial physiological effects in various settings; these include: reduced cardiac arrhythmias and mortality, (Richter, 2003) improved aspects of muscular recovery (Phillips et al.,2003), increased cell membrane fluidity (Ehringer et al, 1990), altered nerve chemistry and depression (Logan, 2003; Su et al., 2003), reduced inflammation (Browning, 2003; Endres, 1996; Endres et al., 1989; Calder, 1997, 2001), and decreased cartilaginous breakdown (Curtis et al., 2000). A portion of these effects stem from their ability to displace arachidonic acid from cell membranes (Kelly et al., 1999). Interestingly, it appears that the ratio of n-6: n-3 fatty acids in the diet, as opposed to gross amounts of either, is the determining factor in many of the described effects (Boudreau et al., 1991; Simopoulos, 2002). Toward this end, a ratio much lower than found in Western diets, of approximately 5-10:1 has been recommended (Institute of Medicine, 2002).

Thus, the energy density and differing pharmacologic effects of dietary fats makes them attractive to athletes from the perspectives of health, performance, weight control and possibly even overtraining and injury management. The body of dietary fat research relative to exercise is necessarily expanding. Examples of common and oft-researched dietary fats, their number of carbon atoms, dietary sources and intake recommendations are provided in Table 1.

Table 1. Types, composition, and sources of common dietary fatty acids and recommendations for intake.

Type and Examples of Fatty Acids	Number of Carbon Atoms	Sources of Fatty Acids	Suggested Intakes[†]
Saturated			
Medium-chain triacylglycerols (e.g. lauric acid: C12)	6-12	Medical nutrition formulas, dietary supplements; derived from tropical oils	↔
Palmitic			
Stearic	16	Animal fat, palm oil	↓
	18	Animal fat, cocoa butter	↕
Unsaturated			
Monounsaturated			
Oleic (*cis* form)	18	Olive oil, canola oil, peanut oil	↑
Elaidic *Trans* (form)	18	Some margarines, hydrogenated fats/oils (many processed foods)	↓
Polyunsaturated			
Linoleic (omega-6)*	18	Cakes, cookies, pastries, corn oil, soybean oil (~most vegetable oils)	↕
Conjugated linoleic acid	18	Beef, lamb, dairy products, dietary supplements	↕
Linolenic (omega-3)*	18	Walnuts, flaxseed oil, canola oil, soybean oil (some)	↑
Gamma-linolenic acid	18	Evening primrose, borage, and black currant plants/oils or dietary supplements, produced in body with use of other EFA's	↕
Eicosapentaenoic Acid (EPA; omega-3)	20	Salmon, herring, sardines, dietary supplements	↑
Docosahexaenoic Acid (DHA; omega-3)	22	Salmon, herring, sardines, dietary supplements	↑

*****Signifies an essential fatty acid (EFA)

[†]↑ consume *more* of; ↓ consumer *less* of; ↕ *debatable* (1, 2, 3); ↔ *no need* to add to diet (4).

Fat Digestion and Absorption

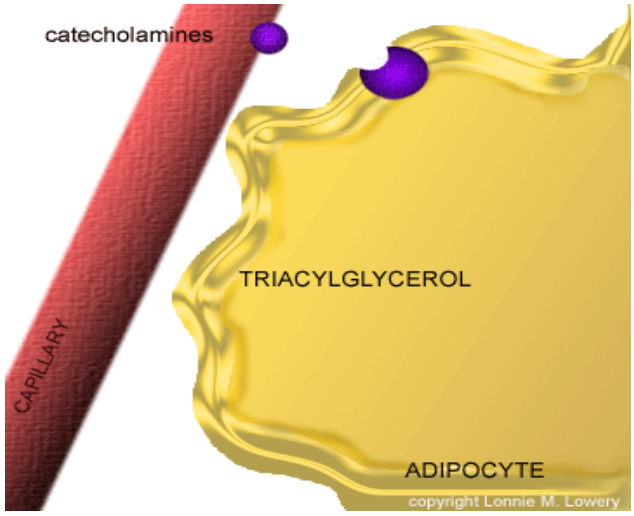
Dietary fat digestion starts in the mouth. Lingual lipase is secreted by the serous glands beneath the tongue and it is this enzyme that accounts for the limited breakdown of triacylglycerol proximal to the intestines (i.e. mouth and stomach). Upon reaching the stomach, fats can slow gastric emptying, providing a high satiety value (Groff et al., 1995) and tend to rise to the surface, "layering" gastric contents (Edelbroek et al., 1992; Horowitz et al., 1993).

After an encounter with bile (stimulated by cholecystokinin) and continued shearing forces in the duodenum, the partially hydrolyzed, fine lipid droplets are increasingly emulsified. At this time, they are also exposed to pancreatic lipases, which function in the higher pH environment supplied by pancreatic bicarbonate secretion into the duodenum. Pancreatic lipase activation is complex, requiring other duodenal contents including colipase, calcium ions, and bile salts. Diacylglycerols (DAG), monoacylglycerols (MAG) and free fatty acids (FFA) reach the mid-intestine (*ileum*) in micellar form – small enough to pass into the intramicrovillus spaces of enterocytes. During contact with the *brush border*, or *unstirred layer* DAG, MAG and FFA move down a concentration gradient into the enterocyte. Inside, they are re-esterified back into triacylglycerol. These newly reassembled triacylglycerols then enter the lymphatic system on the "other side" of the intestinal cell in a chylomicron package. Ultimately, the lymphatic circulation carrying these chylomicrons enters the thoracic duct and then empties into the left subclavian vein of the bloodstream. Peak lipid concentrations in the plasma occur from one to three hours post-meal and return to normal within six hours (Groff et al., 1995).

It is only after circulation in the bloodstream that triacylglycerol-containing chylomicrons can reach tissues that possess lipoprotein lipase (LPL) along their capillary beds and release their DAG and FFA for cellular fuel. Within muscle cells, the hormone sensitive lipase (HSL) finally hydrolyzes remaining DAG and any existing triacylglycerols into free fatty acids (and residual glycerol, which can serve as a gluconeogenic precursor). These free fatty acids can then be activated by coenzyme A and transported via carnitine into the mitochondria for (beta-) oxidation. The result of beta-oxidation and subsequent tricarboxylic cycle/electron transport system processing is nine kilocalories per gram of triacylglycerol.

In the adipocytes, conversely, the DAG and FFA released by passing chylomicrons are generally re-esterified into triacylglycerols for storage. These can later be hydrolyzed via HSL into FFA and glycerol for release directly back into the blood - where FFA bind to albumin and glycerol can enter the liver for formation of nascent glucose. Hydrolysis of stored triacylglycerol occurs during periods of fasting and exercise when insulin concentrations are low and catecholamines, growth hormone and cortisol are increased.

Animation 1 illustrates this process. It begins with activation of triacylglycerol breakdown via catecholamines in an adipocyte. After a pause, the animation then illustrates how cortisol and growth hormone also induce triacylglycerol hydrolysis. After another pause, the animation finally shows caffeine as a negative controller (inhibitor) of phosphodiesterase (PDE) – which facilitates triacylglycerol breakdown - and insulin as a positive controller of PDE, which suppresses triacylglycerol breakdown (Also see *Resting* and *Exercise Metabolism* sections of this review).



Animation 1. Adipocyte animation.

Part 2. Dietary Needs and Food Sources

Dietary fats are generally recommended to comprise a "moderate" 20-35 percent of energy in the diet (Gifford, 2002; Institute of Medicine, 2002). Although dietary fat proportions have been manipulated many times in attempts to improve performance, there presently appears to be little need to deviate from this 30 percent recommendation for health or ergogenic purposes. The composition of individual fatty acid types within that 35 percent "ceiling", however, has received recent attention. Based upon literature that the Western diet is too high in omega-6 fatty acids (mostly linoleic acid) and too low in omega-3 fatty acids (Boudreau et al., 1991; Simopoulos, 2002), the Institute of Medicine published Dietary Reference Intakes for Fatty Acids in September, 2002 suggesting that an omega-6:omega-3 ratio of 5-10 to one. This equates to 12 to 17 g per day of linoleic acid and 1.2 to 1.6 g per day of alpha-linolenic acid (women and men, respectively). Similarly, a safe intake of 3 g of fish

oil daily has been suggested by the United States Department of Agriculture (Morcos and Camilo, 2001). Food sources of various fatty acids are shown in Table 1.

Part 3. Resting Metabolism

Under resting fasting conditions, stored triacylglycerol is the primary source of human energy. The respiratory exchange ratio (RER), a ratio of carbon dioxide on the breath to oxygen consumed, is approximately 0.8, signifying that approximately 60-66% of the fuel being used by the body is from fat and perhaps 33% is from carbohydrate (Brooks, 1997; Wilmore and Costill, 1999). The RER is much less than 1.0 due to the large relative amount of inspired oxygen needed to "burn" fatty acids (See Part 4: Exercise Metabolism.).

As with exercise, resting metabolism is under energetic and hormonal control. In the relative absence of insulin and the presence of growth hormone, cortisol, sympathetic catecholamines and glucagon, hormone-sensitive lipase (HSL) is stimulated via cyclic-AMP and non-esterified fatty acids (NEFA) are either immediately oxidized (e.g. muscle) or released by adipose tissue into the blood for energy. A brief summary of the process is illustrated in Animation 1.

Body composition and weight control are important to many sports in addition to skill-related fitness. According to Burke (2001), "Many athletes are over-focused on reducing body mass and body fat below levels that are consistent with long-term health and performance." Hence, athletes periodize training goals throughout the year. Toward this end, dietary tactics may be undertaken to maximize fat loss and to build or preserve skeletal muscle. Although energy (kcal) balance is a widely accepted determinant of body mass, macronutrient manipulations allow for further management.

The choice of whether to principally restrict dietary fat or carbohydrate for weight control is an ongoing debate. Dietary fat reduction makes an energy deficit easier (Horvath et al., 2000) but dietary carbohydrate reduction reduces insulin concentrations (Sharman et al., 2002; Volek et al., 2002), facilitating lipolysis. Additionally, a higher dietary fat content appears to induce superior nitrogen sparing (McCargar et al., 1989). Both approaches appear to reduce body fat mass (Astrup, 2001; Hayes et al., 2004; Stern et al., 2004; Volek et al., 2002) but both types of restriction carry risks. Very low fat diets have been shown to reduce sex hormone concentrations (Dorgan et al., 1996; Hamalainen et al., 1983; Reed et al., 1987) and may suppress intake or absorption of fat-soluble vitamins and essential fatty acids. Very low carbohydrate intake retards glycogen resynthesis after exercise (Roy and Tarnopolsky, 1998), appears to increase protein breakdown in the body (Lemon and Mullin, 1980), and may reduce dietary fiber consumption (Kappagoda et al., 2004). Ketosis during low-carbohydrate diets, however, appears to be of less concern in non-diabetics due in part to basal concentrations of circulating insulin.

Performance, rather than body composition, however, is of primary interest to the competitive athlete (aside from bodybuilders), and thus weight control approaches must not unduly interfere. Ergogenesis is addressed in Part 4 of this review.

Part 4. Exercise Metabolism

The large, almost limitless fat stores of the body (e.g. 9 $kcal \cdot g^{-1}$ x 12 kg fat mass = 108 000 kcal) in relation to available carbohydrate stores (e.g. 4 $kcal \cdot g^{-1}$ x 450 g glycogen = 1 800 kcal) make it an attractive focus for extending endurance exercise. That is, increasing hydrolysis of stored triacylglycerol and subsequent oxidation of free fatty acids should spare limited glycogen stores, which have an impact on fatigue in moderately high intensity settings (e.g. a runner "hitting the wall" during a marathon). Hence, various attempts to manipulate the diet, and subsequently the body's substrate oxidation, are discussed later in this section.

Exercise affects fat metabolism greatly, not simply increasing it at a constant rate but rather controlling it in reciprocal proportion to carbohydrate. Both the intensity and the duration of a bout of exercise have an impact.

Intensity: The "Crossover Concept"

During the course of aerobic exercise, that is, physical activity utilizing the cardiorespiratory system and primarily large muscle groups for sustained periods of time (American College of Sports Medicine, 2000), multiple fuel sources (carbohydrate, fat, and to a small extent protein) are used depending on the intensity and duration of the activity. The point of intensity at which the body starts to rely more on carbohydrate than fat as a fuel is the "crossover". The metabolic control of this process is still under investigation. The process of hydrolyzing free fatty acids form stored triacylglycerol in adipose tissue, transporting them in the blood and oxidizing them within the muscle mitochondria appears to be too slow to keep pace with metabolic demand. It is also plausible that some aspect of rapid carbohydrate oxidation within the working muscle itself interferes with long-chain fatty acid transport into the mitochondria (Sidossis et al., 1997).

Fuel usage is commonly measured by the respiratory exchange ratio (RER) and the respiratory

quotient (RQ); the RER measures metabolism at the mouth (carbon dioxide produced vs. oxygen consumed as seen in air breathed) and the RQ looks at metabolism at the cellular level (e.g., in muscle tissue) (Houston, 2001; Wilmore and Costill, 1999). During light intensity exercise (RER ~8.5), fat still supplies nearly half of the energy needed in the body (Wilmore and Costill, 1999; Klein et al., 1994). With increasing intensity and oxygen consumption (VO_2), the body shifts toward carbohydrate as a primary fuel (mostly from glycogen but also from blood glucose; RER reaching 1.0), since it is most readily available for breakdown (Brooks, 1997).

Duration: The "Fat Shift"

Unlike the inverse relationship between exercise intensity and fat oxidation (that is, higher intensity = lower fat usage), is the "fat shift" that occurs with increasing duration of exercise. This direct relationship between the time spent at a given (moderate) intensity of aerobic exercise and the amount of stored fatty acids used as fuel is well documented (Brooks, 1997; Sidossis et al., 1997). It is measurable via both a reduced RER (oxidation) and a gradually increasing appearance of glycerol in the circulation (lipolysis).

After a prolonged period of exercising at a sustained moderate intensity, for example, jogging for more than 20 minutes (out to several hours), fat becomes increasingly available for use as an energy source. This is due in part to the fact that oxygen is now more accessible and able to be used to oxidize fat molecules (Abernethy et al., 1990).

Hence, long duration, low intensity exercise may appear to be superior for body fat reduction. It is in fact most effective for *direct* fat usage. Yet one must keep in mind the absolute kcal expended during a bout. An individual can workout for less time and at a higher intensity and expend the *same amount of kcal* as if he had exercised for a longer amount of time, at a lower intensity. Based solely on energetics, the impact on body fat stores should be virtually the same, since the absolute number of kcal has been expended. This is possible due to indirect metabolic processes beyond direct, mid-exercise fat oxidation, such as continued elevated metabolism and interactions among stored substrates.

Part 5. Dietary Fat, Ergogenesis and Athletic Recovery

The human body becomes better at mobilizing, transporting and oxidizing fat as an endurance training adaptation. Similarly, increased ingestion of fat as a proportion of total kcal enhances the body's ability to use it as fuel, in part due to fatty acid availability (Hawley et al., 2000; Schiffelers et al., 2001; Stepto, 2002; Zderic et al., 2004). In an effort to maximize both effects and spare glycogen, thus enhancing energy delivery to working muscles, attempts have been made to increase dietary fat during various periods prior to exercise. The results from these potential ergogenic manipulations are equivocal to date, however. In some studies, an increase in dietary fat resulted in elevated maximal aerobic capacity (VO_2max) and increased time-to-exhaustion (Venkatraman et al., 1997; 2000: 2001) but in others the result was either no effect, decreased performance and/ or increased rate of perceived exertion (Fleming et al., 2003; Hargreaves et al., 2004; Stepto, 2002).

Regarding fatty acid types and athletic recovery, there are mixed reports in the scientific literature. Although at least one study reported no effects on delayed-onset muscle soreness, hanging arm angle, creatine kinase (CK), cortisol or IL-6 after 30 days of 1.8 g fish oil daily, (Lenn et al., 2002), others have shown a fish oil-containing supplement to decrease eccentric exercise-induced IL-6 and C-reactive protein (Phillips et al., 2003). Dose and co-consumed nutrients may be a factor. Specific to the CK variable, the increased membrane fluidity induced by omega-3 fatty acids (Ehringer et al., 1990) may be a factor in studies resulting in increased release in older humans (Cannon et al., 1995) and rabbits (Chen et al., 1999). This elevation was considered beneficial in the human research, as it restored post-exercise CK in older adults to concentrations like those of young participants (Cannon et al, 1995).

Regarding more chronic recovery issues, data from Venkatraman (1997; 2000) suggests that higher total fat intakes are immunosupportive in aerobic athletes. Further, data on immune-challenged (ultra-violet radiation) mice supports the benefits of EPA on immunocompetence as well (Moison and Beijersbergen Van Henegouwen, 2001). Collectively, data on dietary fat amounts and types suggest an immuno-modulatory, rather than immuno-stimulating effect, as certain parameters of the *acute phase response* to stress and injury are reduced while other immune system aspects remain intact or enhanced.

Finally, it is also notable that the natural adaptations to exercise itself, without purposeful omega-3 ingestion or supplementation, increases oleic acid, DHA and total omega-3 fatty acids human muscle tissue of humans (Andersson et al., 2000; Helge et al., 2001).

Part 6. Dietary Fat Supplements

Throughout this review, evidence from dietary supplements and whole food manipulations has helped clarify the biological need for and

pharmaceutical possibilities of dietary fats. But in addition to efficacy, one must consider safety. A wide variety of dietary supplements have been investigated. Each of the following fats could constitute a review in itself; hence, they will be only briefly described here.

Fish oil (EPA and DHA)

Pollution affects fish (oil) quality. Polychlorinated biphenyls (PCBs) contaminate various species of fish and affect cognitive function in both children and adults (McCook, 2001). The risk of heavy metal (e.g. mercury) contamination is real with regards to fish consumption, particularly larger predatory fish like swordfish, which consume smaller fish, accumulating toxins in their flesh (Mendez et al., 2001). The controversy over whether ingested mercury negates the benefits of accompanying fish fats for humans is not settled, however (Guallar et al., 2002; Yoshizawa et al., 2002). In either case, dietary supplements may be a way to avoid the effects of such pollution. Over recent decades, a number of investigations screening fish oil products have resulted in negligible or absent mercury contamination (Bugdahl, 1975; ConsumerLab, 2001; Foran et al., 2003; Koller et al., 1989). At least one review has revealed a lack of "key ingredient" in fish oil products, however (ConsumerLab, 2001). A comprehensive screening of all available fish oil supplements for mercury contamination or even EPA and DHA content is not likely, however. Hence risks remain.

EPA and DHA themselves have been investigated for toxicity. Concerns over excessive clot inhibition, oxidative stress, red cell deformation and enlarged liver have been noted (Rabbani et al., 2001). Most of these effects are seen with extremely high doses - doses as high as $15ml \cdot kg^{-1}$ per day in rat studies. At least one human study has involved administration of 18 grams daily for six weeks and another up to 27 grams daily for one month without problems, however (Barber, and Fearon, 2001; Endres et al. 1989). Caution is nonetheless necessary. Any toxicity surrounding these fatty acids would likely be an extended situation, if any was found, as wash out periods for fish oil effects persist 10-18 weeks (Endres et al. 1989; Kremer et al., 1987). Peroxidative effects and physiological benefits appear to be improved by co-consumption of vitamin E (Hsu, 2001; Tidow-Kebritchi and Mobarhan, 2001).

In consideration of all available factors, the USDA has recommended a chronic intake of no more than three grams per day of EPA and DHA from all dietary sources (Morcos and Camilo, 2001).

Conjugated linoleic acid (CLA)

Conjugated linoleic acid (cis-9, trans-11 or trans-10, cis-12 octadecadieneoic acid) is actually an entire group of trans fatty acids that ironically appear to possess healthful aspects. The isomers, in singular or combined amounts, have dramatically reduced body fat in animal models (Belury and Koster, 2004, Pariza et al., 2001; Mougios et al., 2001) but human studies have been more modest and mixed in this regard (Belury and Koster, 2004). Similarly, investigations into muscle and strength gain have been mixed, perhaps confounded by methodological differences (Belury and Koster, 2004; Krieder et al., 2002; Lowery et al. 1998.) Clearly, the hypo-responsive and/ or hard-to-control nature of humans compared to animals warrants further investigation. Species and dose appear to influence effects. As with fish oil, toxicity appears generally low, with human studies generally involving 3.0-7.2 grams daily for 6-12 weeks (Belury and Koster, 2004; Blankson et al., 2000; Mougios, 2001), yet no upper limit of safety has been established.

Primrose, Borage, Black Cuurant oil (GLA)

As sources of gamma-linolenic acid (all cis-6,9,12 octadecatrieneoic acid), primrose, borage and black currant oils may have the capacity to reduce arthritic inflammation and improve aspects of diabetes (Salway, 1994; Belch and Hill, 2000). Situations in which the human delta-6 desaturase is relatively inactive, such as diabetes, alcoholism and advancing age, appear to be a particular target for GLA intervention (Horrobin, 1981; Salway, 1994). Dietary supplementation with GLA acts similar to EPA in that is proportionately enhances the less inflammatory Series 1 and Series 3 prostaglandins, in effect displacing the pro-inflammatory effects of the 2 Series (Salway, 1994). As with other types of fat, toxicity appears low (Belch and Hill, 2000; Yang-Yi and Chapkin, 1998).

Olive, Canola oil (Monounsaturates)

By no means the only source of monounsaturated fatty acids (MUFA), olive and canola oils do offer a plentiful source of omega-9 *oleic acid* (C18:1) in proportion to other fatty acids in their makeup. The benefits of MUFA on blood pressure, serum lipids, and glucose metabolism (Perez-Jimenez et al., 2002; Rasmussen et al., 1995; Thomsen et al., 1995) as well as antioxidant status (Sola et al., 1997), coupled with minimal toxicity, generally acceptable taste and heavy consumption in long-lived cultures, has made it an attractive replacement for a portion of carbohydrate in the diet. There is no need to purchase MUFA in supplement form; they are

plentiful in the noted common oils.

Diacylglycerols

A diacylglycerol (DAG) has just two fatty acids attached to its glycerol "backbone". Recent reports suggest increased oxidation as opposed to storage and a potential for reduced body weight gain (Flickinger and Matsuo, 2003; Murase et al., 2002). An absence of any fatty acid at the middle, sn-2 position of the glycerol molecule reportedly alters DAG metabolism, eliciting these effects (Flickinger and Matsuo, 2003; Murase et al., 2002). Further research is necessary to determine the anti-obesity efficacy of DAG in free living humans. Widely available in Japan since 1999, DAG oils are now sold commercially in some U.S. cities (Flickinger and Matsuo, 2003) and are likely to garner future research interest.

Structured Triacylglycerols

Using technology similar to DAG, structured triacylglycerols strategically place fatty acids onto a host glycerol molecule. Further, a fatty acid of interest can be esterified directly onto the middle sn-2 position for optimal biological delivery (Bell et al., 1997). Structured triacylglycerols appear to have superior effects to simple physical mixtures of fats, such as nitrogen retention in burn patients (Babyan, 1986).

Medium Chain Triacylglycerols

The shorter length (6-12 carbons) of medium chain triacylglycerols (MCTs) results in biological usage more similar to carbohydrate than longer fatty acids. Being less hydrophobic, MCTs enter the blood stream directly at the portal vein as opposed to taking an initial circulation and are more readily oxidized than longer fatty acids (Berning, 1996; Delany et al., 2000; Jeukendrup et al., 1998). Although these factors have led to speculation over ergogenic properties and glycogen sparing due to rapid energy delivery, research is generally negative (Berning, 1996) and gastric distress has been reported due to the small amounts that are tolerable (Jeukendrup et al., 1998).

Nutrient-nutrient and Nutrient-Drug Interactions

Due to the thrombolytic effects of omega-3 lipids, other substances that reduce clotting such as vitamin E, ginkgo biloba, aspirin, and garlic) could conceivably result in hemorrhage. Despite this, at least one investigation has demonstrated safety during co-administration of fish oil and garlic (Morcos and Camilo, 2001). More research is needed to explore the existence and severity of such interactivity.

CONCLUSIONS

Despite wide differences in opinion among researchers and the general public over recent years, dietary fats remain a potent regulator of physiological function. This calls for caution in supplementing more than a few grams of any uncommon fat per day. Food sources remain the preferred method of intake in most situations. The various fatty acids provide perhaps the most important aspect of dietary fat manipulations, although increasing the percentage of fat in the diet also has an impact on exercise and resting metabolism. Acute ergogenesis, per se, does not appear to be the most promising area of research but rather longer-term investigations into effects supportive to athletic functioning such as body composition and recovery.

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KEY POINTS

- Nutrition education initiatives over recent years have sent contrasting messages on dietary fat to the public.
- Variations in chemical structure among triacylglycerols and their component fatty acids induce very different biological effects.
- Manipulating fat as a percentage of total kcal affects athletes.
- Athletes have special needs for which dietary fat may prove beneficial.

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