Volume 26, Number 3, pages 72–76

Keywords: omega-6 fatty acids; omega-3 fatty acids; thermodynamics

Omega-3 Fatty Acids: A Novel Fat Burner

Brad Schoenfeld, CSCS
Scarsdale, New York

summary

The quest to get lean is an obsession with a large segment of the general public—an obsession that also extends to many athletes, body builders, and fitness competitors. By reducing body fat to low levels, physical performance and aesthetics can be enhanced, thereby providing a competitive edge in various sporting endeavors. This article will focus on the role of omega-3 fatty acids in lipolysis and explain how proper integration of these nutrients can lead to an improved body composition.

Dietary Principles

Studies have repeatedly shown that overall energy balance is the major determinant in weight management (3, 12). This is consistent with the first law of thermodynamics as applied to human energy balance: If you expend more calories than you consume, you will lose weight. For all intents and purposes, the first law of thermodynamics is immutable: Calories in versus calories out ultimately determines whether weight is gained, lost, or maintained.

However, despite claims to the contrary, all calories are not created equal. For example, it has been well documented that a percentage of a food's energy is burned off in the digestion process—a phenomenon known as the thermic effect of food (TEF). Of all the macronutrients, protein has the highest thermic effect, burning off approximately 25–30% of the calories consumed; approximately 6–8% of the calories from carbohydrates are burned off in digestion; and the thermic effect of fat is less than 3% (18). Thus, by altering dietary nutrient composition, a person can affect the expenditure side of the thermodynamic equation, thereby altering body composition.

It is also true that different types of the same nutrient also can have varying effects on metabolic processes. This is especially true of dietary fats. Although a thorough discussion of the structure and function of dietary fats is beyond the scope of this article, a brief review of physiology is in order (for a complete discussion on the topic, see "Advanced Nutrition and Human Metabolism," by Groff, Grouper, and Hunt).

Fats are classified into 2 basic types: saturated and unsaturated. Saturated fats are so-called because their carbon chain is completely saturated by hydrogen atoms (see Figure 1). These fats are solid at room temperature and, for the most part, are biologically inert. If not oxidized immediately for energy, they are stored in adipose tissue for future use.

Unsaturated fats, on the other hand, contain one or more double bonds in their carbon chain. For each double bond, there is a loss of 2 hydrogen atoms from the chain. The end result is a fat that is no longer saturated with hydrogen atoms (hence, the "unsaturated" moniker). Unsaturated fats can be categorized into monounsaturated fats (MUFAs), containing

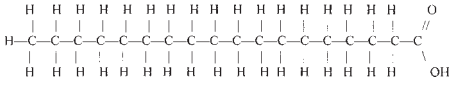

Figure 1. Structure of a saturated fatty acid (stearic acid). C = carbon, H = hydrogen, O = oxygen.

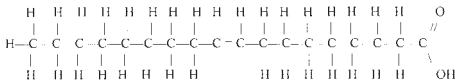

Figure 2. Structure of an unsaturated fatty acid (oleate). C = carbon, H = hydrogen, O = oxygen.

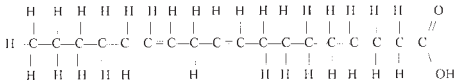

Figure 3. Structure of an omega-6 linoleate. C = carbon, H = hydrogen, O = oxygen.

Figure 4. Structure of an omega-3 alpha-linoleneate. C = carbon, H = hydrogen, O = oxygen.

1 double bond (see Figure 2), and polyunsaturated fats (PUFAs), containing 2 or more double bonds. There are numerous subtypes of PUFAs, the primary classes being linoleic omega-6 fatty acids, which have their first double bond at the sixth carbon from the methyl end of the carbon chain (see Figure 3), and alpha-linolenic omega-3 fatty acids, which have their first double bond at the third carbon from the methyl end of the carbon chain (see Figure 4). PUFAs are termed essential fats because they cannot be manufactured by the human body and are therefore an essential component in food. These fats can be further desaturated to form biologically active derivatives involved in a host of bodily functions.

Theoretical Basis for Omega-3s and Fat Loss

Of all the types of dietary fat, it is the omega-3 class that has the greatest effect on enhancing lipolysis. The primary way in which omega-3s exert these effects is by acting as metabolic fuel partitioners, upregulating lipid oxidation. and downregulating lipid sythesis (5). This is accomplished on several different fronts:

First, omega-3s increase the fluidity of cell membranes (1). Cell membranes serve a critical function by regulating the passage of nutrients, hormones, and chemical signals into and out of cells. When cell membranes are fluid, they become more permeable, allowing substances and secondary messenger molecules to readily penetrate into the cytoplasm (19). This has wide-ranging effects, from increasing muscle protein synthesis to enhancing glycogen storage to improving insulin sensitivity to boosting leptin production (4, 21, 24)—factors that can have a tangible effect on lipolysis.

Second, omega-3s suppress production of malonyl-CoA (5). Malonyl-CoA is a precursor for fatty acid synthesis. Its primary mode of action is to impair the activity of the enzyme carnitine palmitoyltransferase (43). This enzyme transports existing fatty acids back into the mitochondrial matrix, where they can be utilized for fuel. By suppressing malonylCoA, levels of carnitine palmitoyl-transferase are increased, hence favoring the entry of fatty acids into the mitochondria for beta oxidation.

Third, omega-3s act as ligands for a specific hormone receptor called peroxisome proliferator-activated receptor-alpha (20) (PPAR-alpha). PPAR-alpha is located in the cell nucleus of many body tissues, predominantly those that exhibit high catabolic rates of fatty acids such as liver, heart, kidney, and muscle (9). One of its main functions is the management of glucose and fatty acid homeostasis (39). Specifically, it induces the expression of several gene-encoding proteins involved in lipid transport and oxidation, including hepatic carnitine palmitoyltransferase, and hepatic and skeletal muscle peroxisomal acyl-CoA oxidase (6). Therefore, through its synergistic effect on PPAR, omega-3s increase activity of these lipolytic enzymes, accelerating fat-burning processes.

PPAR-alpha also potentiates an increase in levels of a class of fat-burning compounds called uncoupling proteins (UCPs), especially UCP-3, a homolog found primarily in muscle tissue (2, 7). As the name implies, UCPs serve to uncouple cellular respiration from ATP synthesis. This makes the production of ATP less efficient, causing oxidation energy to be dissipated as heat. Because of its selective specificity in muscle tissue, UCP-3 has been cited as a central factor in the control of resting metabolic rate (14, 32) and the regulation of lipids as fuel substrate (31). Although the exact mode of action has not been fully elucidated, it is hypothesized that this thermogenic effect is related to proton leakage from the mitochondrion (28). So by mediating PPAR-alpha, omega-3s indirectly stimulate UCP-3 activity, thus promoting an increased lipolytic capacity.

Last, omega-3s decrease the nuclear content of hepatic sterol regulatory element binding protein (SREBP)-1, one of a family of transcription factors that activate genes encoding the expression of numerous hepatic enzymes involved in glucose metabolism and fatty acid biosynthesis (22, 26, 36). These enzymes include glucokinase, pyruvate kinase, acetyl-CoA carboxylase, stearoyl-CoA desaturase, pyruvate kinase, and fatty acid synthase. SREBP-1 exists in 2 isoforms, SREBP-1A and SREBP-1C, and the ratio of each varies by up to 100-fold in different tissues of the body (33). Supplementation with omega-3 fatty acids has been shown to reduce levels of both isoforms of SREBP-1 by up to 85%, significantly suppressing lipogenic gene transcription (41, 42). And by decreasing the presence of lipogenic enzymes, the body's ability to store fat is summarily diminished.

Studies on Omega-3s and Fat Loss

The effects of omega-3s on fat loss have been repeatedly demonstrated in animal studies. One such study examined the adiposity of rats after feeding them a diet of either omega-3s (fish oil) or saturated fat (lard). Although calories were kept constant, rats consuming fish oil had 77% less

fat in perirenal fat pads and 51% less fat in epididymal fat pads as compared with rats consuming lard (27). Another study found that, after 12 weeks, rats consuming a diet of 42% fish oil gained more lean body mass and had lower subcutaneous fat levels than those consuming a comparable amount of olive oil (a MUFA) or beef tallow (a saturated fat) (37). The study concluded that dietary fatty acid composition alters the efficiency of energy substrate accretion in rats. Other studies have produced similar findings, all showing a decrease in fat accumulation and increase in lean tissue growth when comparing consumption of omega-3s with saturated fats on a calorie-for-calorie basis (13, 25).

Research has also shown omega-3s to be more antilipogenic than omega-6s. One study put genetically obese mice on either a diet containing 100 g of primrose oil (rich in omega-6s) or 100 g of cod liver oil (rich in omega-3s). At the end of the protocol, mice on the cod liver oil diet experienced lower weight gain than those consuming primrose oil—an outcome unrelated to the quantity of food intake (8). Another study examined 2 groups of hamsters, one fed a diet of corn oil and the other fed fish oil. After 3 weeks, fish oil-fed animals demonstrated significantly lower carcass energy gains as fat compared with the corn oil-fed group (17).

To date, controlled experiments examining the effect of differing dietary fatty acid compositions on body fatness in human subjects have been lacking. However, studies on fatty acid oxidation rates in humans have shown that omega-3s are preferentially metabolized by the body after ingestion. One well-designed protocol found that the omega-3 fatty acid linoleate was oxidized at a much greater rate than other long-chain saturated and unsaturated fats, noting a high linear relation between oxidation and the number of double bonds (16). Because a reduced oxidation of fat is both predictive of greater weight gain (44) as well as an augur of relapse in those who have lost weight (11), these findings provide a sound rationale for the role of omega-3s in a fat-loss diet.

Practical Considerations

Given the wealth of theoretical and scientific data, it seems patently clear that consuming an adequate amount of omega-3 is essential for optimizing body composition. Unfortunately, research has yet to determine precisely how much omega-3 is needed for optimum nutritional effect. To date, there are not even any recognized clinical tests for omega-3 deficiency and sufficiency.

On the other hand, what has been well studied is the appropriate ratio of dietary omega-6 to omega-3. While Americans generally consume an abundance of omega-6 fats, most are woefully deficient in their consumption of omega-3s (the current Western diet has an omega-6 to omega-3 ratio that hovers in the range of about 20–30:1! (35)). This has been shown to promote the pathogenesis of many disease states as well as negatively impacting body composition (29, 38, 40).

Although no definitive research exists, scientists postulate that, from a health perspective, the ideal omega-6 to omega-3 ratio to be in the range of 4:1 or lower. However, taking body composition into account, a good case can be made that a ratio of 1:1 is best. Consider that our Paleolithic ancestors, on whom our genetic pool is based, maintained a fairly equal balance of omega-6s to omega-3s (34). Their diet consisted almost exclusively of lean wild meats, fish, vegetables, and fruits, all of which had a high omega-3 content. All things considered, there doesn't seem to be any downside to consuming an equal or greater amount of omega-3s vis-à-vis omega-6s, and the benefits are potentially significant, especially for those seeking to maximize fat loss.

Integrating Omega-3s into a Fat-Loss Diet

An optimum fat-loss diet should incorporate caloric restriction coupled with regular consumption of foods or supplements high in omega-3s, preferably substituted for those containing saturated and trans fats (and perhaps omega-6s, depending on the omega-6/omega-3 ratio). Two of the best sources of omega-3 are flax and fish oils.

Flax oil can be considered a staple omega-3 food. Like the seeds from which it is derived, flax oil contains about 50% omega-3 fatty acids in the form of alpha-linoleate. It also contains healthy amounts of vitamin E, which helps to reduce the potential for the PUFA to undergo lipid peroxidation (15). One tablespoon per day will go a long way to satisfying omega-3 needs. Because flax oil is very susceptible to degradation from light, air, and heat, it is important to buy flax oil that is mechanically (i.e., expeller) pressed (oils that use this approach state so right on the label) and packaged in an opaque container. Store it in a cool, dry place. Once opened, the oil should be refrigerated and used within a couple of months.

To complement intake of flax oil, it is beneficial to consume fish oil. Because fish oil contains longer chain derivatives of omega-3s, namely eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), it can provide utility over and above the omega-3 fatty acids obtained from flax. In order for the body to utilize omega-3s (i.e., alpha-linoleate), it must first desaturate them into EPA and DHA. But the desaturation process can be inefficient, resulting in a diminished conversion rate. Because fish oil has preformed EPA and DHA, the desaturation process is bypassed, allowing maximal utilization of its fats. Although the consumption of deep-colored cold-water fish (such as salmon, trout, mackerel, and sardines) will provide DHA and EPA, eating fish on a daily basis is unrealistic for most individuals. Hence, on days when cold-water fish is not consumed or for those who do not eat fish, supplementation with fish oil capsules is beneficial. A 5–10-g dose is recommended, using a formula of 1 g for every 20 pounds of body weight (each capsule is usually 1 g). Because of their susceptibility to undergoing lipid peroxidation, supplementation with antioxidants is advisable to prevent atherosclerotic buildup (10).

Cod liver oil is also a good omega-3 source but, because of a high concentration of vitamin D, it can be potentially toxic when taken in supplemental form. Additionally, it has a pungent taste that many consider off-putting. Hemp, walnut, and canola oil have modest amounts of omega-3s and can be integrated into a diet to increase consumption. However, these oils also contain fairly high levels of omega-6s and therefore should be used selectively to avoid negatively skewing the dietary ratio of omega-6 to omega-3.

Finally, it is important to understand that consuming supraphysiologic doses of fish oil will not improve body composition. Like all dietary fats, omega-3s are energetically dense, containing approximately 9 calories per gram. Because excess energy intake will be stored in adipose tissue, careful attention must be paid to caloric consumption. Further, there is some evidence that overconsumption of omega-3s can cause immunosuppression and prolong bleeding time (23, 30), outcomes that can have negative implications, especially for the hard-training athlete. Although an upper limit has not been determined, it would seem prudent to restrict consumption to no more than about 10% of total calories.

In summary, omega-3 fats are an important dietary nutrient possessing lipolytic and antilipogenic effects that can be harnessed to optimize body composition. To confer maximal benefits, it is best to obtain omega-3s from sources containing both alpha-linoleic acid as well as the omega-3 derivatives, EPA and DHA, keeping the omega-6/omega-3 ratio at approximately 1:1. Consuming regular portions of flax oil and fish oil is an excellent way to meet these goals. Aim for 1 tablespoon of flax and 5–10 g of fish oil on a daily basis. ♦

References

1. Alexander, J.W. Immunonutrition: The role of omega-3 fatty acids. *Nutrition.* 14(7–8):627–633. (Review). 1998.
2. Boss, O., S. Samec, A. Paoloni-Giacobino, C. Rossier, A. Dulloo, J.Seydoux, P. Muzzin, and J.P.Giacobino. Uncoupling protein-3: A new member of the mitochondrial carrier family with tissue-specific expression. *FEBS Lett.* 12;408(1):39-42. 1997.
3. Bravata, D.M., L. Sanders, J. Huang, H.M. Krumholz, I. Olkin, and C.D. Gardner. Efficacy and safety of low-carbohydrate diets: A systematic review. *JAMA.* 289(14):1837–1850. (Review). 2003.
4. Cha, M.C. and P.J. Jones. Dietary fat type and energy restriction interactively influence plasma leptin concentration in rats. *J. Lipid Res.* 39(8):1655–1660. 1998.
5. Clarke, S.D. Polyunsaturated fatty acid regulation of gene transcription: A molecular mechanism to improve the metabolic syndrome. *J. Nutr.* 131(4): 1129–1132. (Review). 2001.
6. Clarke, S.D. and D. Jump. Polyunsaturated fatty acids regulate lipogenic and peroxisomal gene expression by independent mechanisms. *Prostaglandins Leukot. Essent. Fatty Acids.* 57(1):65–69. (Review). 1997.
7. Clarke, S.D. Polyunsaturated fatty acid regulation of gene transcription: A mechanism to improve energy balance and insulin resistance. *Br. J. Nutr.* 83:S59–S66. 2000.
8. Cunnane, S.C., K.R. McAdoo, and D.F. Horrobin. n-3 Essential fatty acids decrease weight gain in genetically obese mice. *Br. J. Nutr.* 56:87–95. 1986.
9. Diep, Q.N., R.M. Touyz, and E.L. Schiffrin. Docosahexaenoic acid, a peroxisome proliferator-activated receptor-alpha ligand, induces apoptosis in vascular smooth muscle cells by stimulation of p38 mitogen-activated protein kinase. *Hypertension.* 36(5):851–855. 2000.
10. Foulon, T., M.J. Richard, N. Payen, J.L. Bourrain, J.C. Beani, F. Laporte, and A. Hadjian. Effects of fish oil fatty acids on plasma lipids and lipoproteins and oxidant-antioxidant imbalance in healthy subjects. *Scand. J. Clin. Lab. Invest.* 59(4):239–248. 1999.
11. Froidevaux, F., Y. Schutz, L. Christin, and E. Jequier. Energy expenditure in obese women before and during weight loss, after refeeding, and in the weight-relapse period. *Am. J. Clin. Nutr.* 57:35–42. 1993.
12. Golay, A., A.F. Allaz, Y. Morel, and N. de Tonnac. Similar weight loss with low- or high-carbohydrate diets. *Am. J. Clin. Nutr.* 63(2):174–178. 1996.
13. Hainault, I., M. Carolotti, E. Hajduch, C. Guichard, and M. Lavau. Fish oil in a high lard diet prevents obesity, hyperlipidemia, and adipocyte insulin resistance in rats. *Ann. N. Y. Acad. Sci.* 683:98–101. 1993.
14. Harper, M.-E., R. Dent, S. Monemdjou, V. Bezaire, L. Van Wyck, G. Wells, G. Nihan Kavaslar, A. Gauthier, F. Tesson, and R. McPherson. Decreased mitochondrial proton leak and reduced expression of uncoupling protein 3 in skeletal muscle of obese diet-resistant women. *Diabetes.* 51(8): 2459–2466. 2002.
15. Huang, H.Y., L.J. Appel, K.D Croft, E.R. Miller, 3rd, T.A. Mori, and I.B. Puddey. Effects of vitamin C and vitamin E on in vivo lipid peroxidation: Results of a randomized controlled trial. *Am. J. Clin Nutr.* 76(3):549–555. 2002.
16. DeLany, J.P., M.M. Windhauser, C.M. Champagne, and G.A. Bray. Differential oxidation of individual dietary fatty acids in humans. *Am. J. Clin. Nutr.* 72:905–911. 2000.
17. Jones, P.J. Effect of fatty acid composition of dietary fat on energy balance and expenditure in hamsters. *Can. J. Physiol. Pharmacol.* 67:994–998. 1989.
18. Karst, H., J. Steiniger, R. Noack, H.D. Steglich. Diet-induced thermogenesis in man: Thermic effects of single proteins, carbohydrates and fats depending on their energy amount. *Ann. Nutr. Metab.* 28(4):245–252. 1984.
19. Kummerow, F.A. Modification of cell membrane composition by dietary lipids and its implications for atherosclerosis. *Ann. N. Y. Acad. Sci.* 414:29–43. (Review). 1983.
20. Lin Q., S.E. Ruuska, N.S. Shaw, D. Dong, and N. Noy. Ligand selectivity

of the peroxisome proliferator-activated receptor alpha. *Biochemistry.* 38:185–190. 1999.
21. Lovejoy, J.C. The influence of dietary fat on insulin resistance. *Curr. Diab. Rep.* 2(5):435–440. Review. 2002.
22. Mater, M.K., A.P. Thelen, D.A. Pan, and D.B. Jump. Sterol response element-binding protein 1c (SREBP1c) is involved in the polyunsaturated fatty acid suppression of hepatic S14 gene transcription. *J. Biol. Chem.* 12;274(46): 32725–32732. 1999.
23. Meydani, S.N., S. Endres, M.M. Woods, B.R. Goldin, C. Soo, A. Morrill-Labrode, C.A. Dinarello, and S.L. Gorbach. Oral (n-3) fatty acid supplementation suppresses cytokine production and lymphocyte proliferation: Comparison between young and older women. *J. Nutr.* 121:547–555. 1991.
24. Nolan, B., J. Sentementes, and P. Bankey. Hepatocyte polyunsaturated fatty acid enrichment increases acute phase protein synthesis. *Surgery.* 124(2):471–476. 1998.
25. Okuno, M., K. Kajiwara, S. Imai, T. Kobayashi, N. Honma, T. Maki, K. Suruga, T. Goda, S. Takase, Y. Muto, and H. Moriwaki. Perilla oil prevents the excessive growth of visceral adipose tissue in rats by down-regulating adipocyte differentiation. *J. Nutr.* 127(9):1752–1757. 1997.
26. Osborne, T.F. Sterol regulatory element-binding proteins (SREBPs): Key regulators of nutritional homeostasis and insulin action. *J. Biol. Chem.* 20;275(42):32379–37382. (Review). 2000.
27. Parrish, C.C., D.A. Pathy, and A. Angel. Dietary fish oils limit adipose tissue hypertrophy in rats. *Metabolism.* 39(3):217–219. 1990.
28. Porter, R.K. Mitochondrial proton leak: A role for uncoupling proteins 2 and 3? *Biochim. Biophys. Acta.* 1;1504(1):120–127. (Review). 2001.
29. Rose, D.P. Dietary fatty acids and prevention of hormone-responsive cancer. *Proc. Soc. Exp. Biol. Med.* 216:224–233. 1997.
30. Saynor R., D. Verel, and T. Gillott. The long-term effect of dietary supplementation with fish lipid concentrate on serum lipids, bleeding time, platelets and angina. *Atherosclerosis.* 50(1):3–10. 1984.
31. Samec, S., J. Seydoux, and A.G. Dulloo. Role of UCP homologues in skeletal muscle and brown adipose tissue: Mediators of thermogenesis or regulators of lipids as fuel substrates? *FASEB. J.* 12:715–724. 1998.
32. Schrauwen, P., J. Xia, C. Bogardus, R.E. Pratley, and E. Ravussin. Skeletal muscle uncoupling protein 3 expression is a determinant of energy expenditure in Pima Indians. *Diabetes.* 48:146–149. 1999.
33. Shimomura, I., H. Shimano, J.D. Horton, J.L. Goldstein, and M.S. Brown. Differential expression of exons 1a and 1c in mRNAs for sterol regulatory element binding protein-1 in human and mouse organs and cultured cells. *J. Clin. Invest.* 1;99(5):838–845. 1997.
34. Simopoulos, A.P. Evolutionary aspects of omega-3 fatty acids in the food supply. *Prostaglandins Leukot. Essent. Fatty Acids.* 60(5–6):421–429. (Review). 1999.
35. Simopoulos, A.P. Essential fatty acids in health and chronic disease. *Am. J. Clin Nutr.* 70(3 Suppl):560S–569S. Review. 1999.
36. Stoeckman, A.K. and H.C. Towle. The role of SREBP-1c in nutritional regulation of lipogenic enzyme gene expression. *J. Biol. Chem.* 26;277(30):27029–27035. 2002. E-publication, accessed: 16 Ma6, 2002.
37. Su, W. and P.J. Jones. Dietary fatty acid composition influences energy accretion in rats. *J. Nutr.* 123(12):2109–2114. 1993.
38. Suchner, U. and U. Senftleben. Immune modulation by polyunsaturated fatty acids during nutritional therapy: Interactions with synthesis and effects of eicosanoids. *Infusionsther. Transfusionsmed.* 21:167–182. 1994.
39. Sugden, M.C., K. Bulmer, G.F. Gibbons, B.L. Knight, and M.J. Holness. Peroxisome-proliferator-activated receptor-alpha (PPARalpha) deficiency leads to dysregulation of hepatic lipid and carbohydrate metabolism by fatty acids and insulin. *Biochem. J.* 364(Pt 2):361–368. 2002.
40. Watkins, B.A., Y. Li, K.G. Allen, W.E. Hoffman, and M.F. Seifert. Dietary ratio of (n-6)/(n-3) polyunsaturated fatty acids alters the fatty acid composition of bone compartments and biomarkers of bone formation in rats. *J. Nutr.* 130(9):2274–2284. 2000.
41. Xu, J., M.T. Nakamura, H.P. Cho, and S.D. Clarke. Sterol regulatory element binding protein-1 expression is suppressed by dietary polyunsaturated fatty acids. A mechanism for the coordinate suppression of lipogenic genes by polyunsaturated fats. *J. Biol. Chem.* 13;274(33):23577–23583. 1999.
42. Xu, J., H. Cho, S. O'Malley, J.H. Park, and S.D. Clarke. Dietary polyunsaturated fats regulate rat liver sterol regulatory element binding proteins-1 and -2 in three distinct stages and by different mechanisms. *J. Nutr.* 132(11): 3333–3339. 2002.
43. Zammit, V.A. The malonyl-CoA-long-chain acyl-CoA axis in the maintenance of mammalian cell function. *Biochem. J.* 1;343(Pt 3):505–515. (Review). 1999.
44. Zurlo, F., R.T. Ferraro, A.M. Fontvielle, R. Rising, C. Bogardus, and E. Ravussin. Spontaneous physical activity and obesity: Cross-sectional and longitudinal studies in Pima Indians. *Am. J. Physiol.* 263:E296–E300. 1992.

Schoenfeld

Brad Schoenfeld is President of Global Fitness Services in Scarsdale, NY.