

SCIENTIFIC REVIEW OF *FAT CHANCE*

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The goal of this review is to critically analyze concepts presented in the book *Fat Chance* by Robert H. Lustig, MD. Overall, the author tells an interesting and compelling story to the reader. The major problem with this book is that the story is incomplete and misleading. It also demonstrates the author's lack of understanding of many of the concepts that are presented. Outlined below are several issues that call into question the veracity of the information provided within the book. First, however, it is important to consider the apparent author bias, conflicts of interest and inaccurate conclusions that are evident throughout the book.

Although Dr. Lustig makes a case in the introduction of the book that he is free of any biases or conflicts of interest, this is not entirely true. This book itself represents a potential conflict of interest. If Dr. Lustig is accepting any form of payment for sales of this book, it is in itself a conflict of interest and could introduce a potential for author bias that is either purposeful or unintentional. Even if all proceeds are being donated to charity, a bias could be induced since the charity of his choice would stand to gain from the book's sales. Furthermore, the potential notoriety that could be gained from publishing the book could result in subsequent financial gain for the author through speaking engagements, future book deals, etc.

The author also states in the introduction that "there is not one statement made in this entire book that can't be backed up by hard science." As you read through this review, you will find that there are many statements that are indeed inaccurate. In fact, the author even states in the introduction that "in four places in the book, I let my imagination run wild." Such a declaration does not inspire confidence that all of the statements in the book are backed by solid research. Furthermore, the omission of some information that may influence the readers' opinions is often just as egregious as making statements that are not accurate. **An incomplete story can lead readers to draw inaccurate conclusions.**

Important facts are misrepresented and omitted throughout Dr. Lustig's book, leading the reader to think his arguments are science-based, when in reality many are not.

I. POOR EXPLANATION OF THE DEVELOPMENTS OF METABOLIC SYNDROME AND OBESITY

The author provides readers with a poor explanation of the developments of metabolic syndrome and obesity, as well as the factors that are responsible. This stage is set by the author defining “metabolic syndrome” in multiple ways rather than using a common, standardized definition (as on page 94). While he seems to attempt to provide the most generally accepted definition, he fails to do so clearly. The definition used by most researchers, as established by the Adult Treatment Panel (ATP) III, is that metabolic syndrome consists of 3 of the following 5 conditions (National Cholesterol Education Program 2002):

- waist circumference greater than 102 cm in men and 88 cm in women
- serum triglycerides concentration of at least 150 mg/dL
- serum high-density lipoprotein cholesterol concentration of less than 40 mg/dL in men and 50 mg/dL in women
- blood pressure of at least 130/85 mm Hg
- serum glucose concentration of at least 110 mg/dL

In that report, the ATP III panel also clearly delineates the root causes of metabolic syndrome as obesity, physical inactivity and genetics; whereas, Dr. Lustig states on page 7 that “obesity is not the cause of chronic metabolic disease.” While the author is clear that he does not believe that obesity is a root cause, he is equally clear that he believes that fructose is. On page 118, he states that “it’s the fructose that causes chronic metabolic disease.” These sentiments are reiterated elsewhere in the book as well (e.g., page 125, etc.). **Since negative effects of fructose on metabolism are almost universally not evident unless intake is at a level that approximates the 95 %ile of intake or higher (Benado et al. 2004), support for this assertion at typical intakes is unavailable.** On page 118, he also muddies the water by stating that fructose is both a carbohydrate and a fat “because that’s how fructose is metabolized in the liver.” This is blatantly false. The misrepresentations and ambiguities of the author only serve to confuse the book’s readers.

To the author’s credit, at one point in the book he admits that the effects of fructose are dose-dependent (pages 125-127). There, he states that the threshold for negative effects of fructose is likely 50 grams per day, but he only relates the value to a reference related to alcohol intake and does not cite a source about fructose to support his statement. **In reality, reports on the metabolic implications of fructose have indicated that negative effects of fructose (e.g., elevated fasting triglyceride concentrations,**

There is no scientific evidence to support the idea that fructose causes obesity or metabolic syndrome when consumed in typical amounts.

Dr. Lustig’s understanding of fructose metabolism and the published literature on fructose and health is limited.

impaired insulin sensitivity, elevated uric acid, development of obesity and metabolic syndrome, etc.) are not likely to occur unless intake exceeds at least 100 grams per day (Livesey and Taylor 2008, Taylor 2009). In fact, positive effects are typically detected on glycosylated hemoglobin (HbA1c) as fructose consumption increases to at least 100 grams per day. Also, according to that research, the only negative effect that is typically demonstrated with intakes above the threshold of 50 grams per day is a transient increase in plasma triglycerides that occurs following consumption of a single high dose of fructose. It is important to keep in mind that a 50 gram dose of fructose would typically be accompanied by at least that amount of other carbohydrates. Regardless, his recognition that negative effects likely occur only at high doses does not fit with the gist of the book, wherein he repeatedly suggests that sugar is a toxin to be avoided. The author suggests that his own research demonstrates that sugar is a toxin (pages 126-127), yet he provides no details on his methodologies, and since this research is apparently unpublished, it has not undergone the rigors of the peer-review process. **The key for preventing obesity and metabolic syndrome is to avoid consuming excessive energy (Calories)** and reducing sugar and fructose intakes is just one way that individuals can improve their health if they consume excessive amounts of fructose.

The author also diminishes the notion that genetics are important in predisposing individuals to obesity; however, multiple gene candidates have been implicated in this predisposition (Walley et al. 2009, Loos 2012). It is clear that our genetics are linked to all the components of energy expenditure (i.e., basal metabolism, thermic effect of exercise, thermic effect of food), our propensity to engage in physical activity and our dietary intake; therefore, genes may predispose some individuals to higher energy intake and/or lower daily energy expenditure (Loos and Bouchard 2003, Loos and Ranikinen 2005). Surprisingly, the author places little emphasis on the role of genetics in the development of obesity and metabolic syndrome, yet places an unwarranted amount of emphasis on the role of branched chain amino acid consumption in enhancing risk of metabolic syndrome. On page 99 he includes branched chain amino acids as among “The Four Foodstuffs of the Apocalypse” and cites one study in which plasma concentrations of branched chain amino acids were different in obese versus lean individuals and in which rats that were fed a high fat diet rich in branched chain developed obesity. The difference in valuing this research above the overwhelming roles of factors such as genetics in predisposition to obesity and metabolic syndrome indicates a major disconnect in logic or a significant scientific bias. Furthermore, the author demonstrates a weak understanding of branched chain amino acid content of food by stating on page 20 that “we have high-quality protein (such as egg protein)...and we have low-quality protein (hamburger meat), which is full of branched-chain amino acids.” The branched chain amino

Obesity is a multifactorial disease that is impacted by a wide array of genetic and lifestyle factors. Attributing obesity and chronic disease to the excessive intake of a particular food or nutrient (i.e., sugar or fructose) is misguided.

acid contents of eggs (~2.6 grams per 100 grams) and hamburger meat (~2.9 grams per 100 grams) are very similar and both are considered to be rich sources. He also refers to corn as a high in branched chain amino acids (page 99); however, the branched chain amino acid content of corn is similar to other protein-rich plant foods such as beans, nuts and other grains, so distinguishing corn in this regard is misleading.

II. A LACK OF ACCURACY IN DESCRIBING METABOLISM

There are many instances in which the author appears to have a weak understanding of metabolism or ignores research on important metabolic processes. The use of scientific terms to describe these processes can be extremely convincing to the typical reader even when the information presented is inaccurate.

One example of Dr. Lustig's misleading depictions of metabolism is related to his descriptions of impact of carbohydrate-containing foods on blood sugar and insulin responses. On multiple occasions, he leads readers to believe that all refined carbohydrates are notorious for raising blood sugar and insulin (e.g., pages 82, 115, etc.). These statements are misleading from two perspectives. Some readers may interpret this as meaning that refined carbohydrates but not unrefined carbohydrates can exert these effects; however, unrefined foods rich in carbohydrates often produce greater glycemic and insulinemic responses than refined foods (Atkinson FS et al. 2008). Additionally, some highly refined carbohydrates such as pure fructose produce minimal effects on blood glucose and insulin (Atkinson FS et al. 2008, Chong et al. 2007). The author also gives the readers the idea that when blood glucose is elevated, the glucose is destined for our adipose tissue for storage as fat. On page 35 he says, "where does the glucose go? To the fat," and on page 82 he remarks that "insulin shunts sugar to fat." The truth is that only a very small portion would be used for this purpose; therefore, it is extremely misleading to suggest this. We are very ineffective at making fat from carbohydrate (Hellerstein 2001) and when we do overeat sugar and consume excess energy, fat accumulation through fat synthesis from the sugar accounts for only a very small fraction of the positive fat balance (McDevitt et al. 2001).

Dr. Lustig's perspectives on glucose and lipid metabolism are also incorrect elsewhere in the book. On page 97, he states that "glucose is the preferred energy source of all organisms on the planet." At rest, however, the major source of energy production for humans is actually fat (Kuo et al. 2005). This false statement and other inaccurate depictions of how carbohydrate and fat are used for energy metabolism are troubling. On that same page, the author walks the reader through glucose metabolism describing metabolic pathways such glycolysis and Krebs cycle. He says that when glucose

is metabolized we first encounter glycolysis and then Krebs cycle, which he describes as being the process in which pyruvate is broken down to carbon dioxide and water, “second”. In truth, the pyruvate produced during glycolysis must be converted into acetyl coA through a series of reactions known as the pyruvate dehydrogenase complex; therefore, it is acetyl coA that can be broken down to carbon dioxide in Krebs cycle, which follows the pyruvate dehydrogenase complex. Furthermore, the water produced in metabolism is actually made when products of Krebs cycle are oxidized through a metabolic pathway known as the electron transport chain. **It is possible that the author’s intent was to simplify these processes for the reader, but doing so inaccurately calls into question his knowledge of metabolism, upon which he bases much of his book.** To add to concerns about his lack of metabolism knowledge or forthrightness, the author makes a blatantly false statement on page 99 in saying that “trans fats...can’t be broken down.” Actually, we break them down for energy at a higher rate than most fatty acids (DeLany et al. 2000). His description of lipid metabolism is also erroneous when he states on pages 106 and 112 that the liver produces LDL particles. The liver actually makes VLDL particles, which it exports to the circulation where LDL particles can be formed. He further says that LDL particles would “take up residence in fat cells,” which is not a primary target of LDL particles. He goes on to describe triglycerides being released into the blood stream, but it is free fatty acids that are released into the bloodstream from triglycerides that are stored in the fat cells.

Furthermore, many of the metabolic events described throughout the book likely only occur at high doses of the food component he discusses; yet, the author describes these as occurring at relatively low levels of intake. For example, on pages 120-121, he describes the implications of glucose on metabolism, but at the dose he selected (120 Calories) these negative effects are highly unlikely. Furthermore, even at higher doses, these effects would be dependent on variables such as the source of the glucose, fitness and metabolic state of the individual, etc. Likewise, on page 122, the author describes the negative effects of alcohol at high doses, but does so using an example of a small dose (96 Calories). Additionally, Dr. Lustig does not accurately describe metabolism of alcohol when consumed at high doses, completely ignoring the microsomal ethanol oxidizing system, which is critical during consumption of excess alcohol.

On pages 123 and 124, the author describes an example of how a 60 Calorie dose of fructose can deplete ATP, promote insulin resistance, raise blood concentrations of insulin, etc. No research is available to support the idea that such a low dose can exert the negative effects described on these pages and, even at high doses, many of these effects do not occur or are unlikely. **On these and many other pages (i.e., pages 20, 97), the author indicates that “fructose...is inevitably metabolized to**

Dr. Lustig misrepresents the available scientific evidence by making sweeping conclusions based on studies that do not examine real-life consumption patterns.

fat”; yet, synthesis of fructose to fat represents only a small fraction of fructose metabolism and that fraction remains small even during consumption of excessive doses of fructose (Chong et al. 2007, Parks et al 2008, Tappy and Le 2010). Interestingly, on page 174 the author acknowledges that only a fraction of fructose is converted to fat, but provides an inaccurate estimation of 25% in obese, insulin resistant individuals with no valid reference to support it. On page 191, he contends that 19 % of Latinos possess a gene defect promoting full conversion of fructose to fat, but provides no reference and the subsequent citation (reference 19 for that chapter) is not from a peer-reviewed study. Dr. Lustig’s contention that “there is not one statement made in this entire book that can’t be backed up by hard science” is clearly not supported with regard to the impact of fructose consumption on fat synthesis. Relatedly, the author also states that “fructose does not go to glycogen.” In reality, researchers have suggested that glycogen production from an oral load of fructose is likely approximately 15% of the fructose ingested and that about half of the fructose ingested is converted to glucose that is secreted into the circulation (Tappy and Le 2010). **Overall, the misleading depiction of fructose metabolism at either low or high doses does not foster the ability of the reader to make adequately informed decisions regarding their consumption of fructose-containing foods.**

Dr. Lustig’s assertion that excessive fructose is converted to fat by the body, thereby uniquely contributing to obesity and metabolic syndrome, is unsubstantiated.

III. DISTORTIONS OF BIOENERGETICS

Dr. Lustig provides inaccurate or misleading information regarding energy homeostasis or factors that influence bioenergetics in many cases. These issues are found throughout the book and provide the reader with a distorted understanding of the issues.

A theme that the author brings up on many occasions is that “a calorie is not a calorie.” While he qualifies this statement on occasion, the author is not clear in recognizing that, in reality, energy balance is determined by caloric intake versus caloric expenditure. If energy intake exceeds total energy expenditure, weight gain will occur. If energy intake is lower than total energy expenditure, weight loss will occur. This concept does not preclude the idea that changes in intake may positively or negatively impact one or more of the components of energy expenditure (and potentially total energy expenditure); however, the author is misleading on this concept on multiple occasions.

Dr. Lustig’s point of view on energy balance is opposed by leading government and scientific sources on health and nutrition, including the National Institutes of Health and the Academy of Nutrition and Dietetics.

Government health authorities and registered dietitians agree that a Calorie is a Calorie in terms of weight management.

In an example of inaccurate bioenergetic information presented on page 20, the author states that “working off a Big Mac would require four hours of biking.” A more accurate estimate is that an average-sized person of about 150 pounds would expend enough energy during one hour of moderate intensity cycling (12-13.9 mph) to expend the energy provided by one Big Mac (Ainsworth et al.2000). Four hours is a gross underestimation even

for average individuals completing only moderate exercise. Another key misunderstanding that the author has on bioenergetics is expressed on page 140, where he states that “Burning a pound of fat liberates 2,500 calories.” It should be noted that the unit of energy he is trying to describe is a Calorie rather than a calorie, since a Calorie is the equivalent of a kilocalorie (1000 calories). More importantly, one pound of fat really possesses about 4086 Calories. Historically, the number of Calories that have been equated to a pound of weight loss (which would include not only fat, but other body components as well such as water, carbohydrate, protein, etc.) is 3500 Calories rather than 2500 calories. It is actually very consistent with our scientific understanding of bioenergetics that, as the author cites on page 141, “On average, obese people had to eat 3,977 calories (Calories) less to burn off that one pound of fat.” This demonstrates a lack of basic understanding of nutritional energetics.

In that same chapter, Dr. Lustig describes physical activity as the minority of energy expenditure. For the average person, the thermic effect of food is in reality the most minor component of energy expenditure. Interestingly, the author also suggests that increasing the thermic effect of food by eating protein can promote higher rates of energy expenditure and that fat only raises the thermic effect of food to a small extent, but he fails to mention that carbohydrate is also a key nutrient that elevates the thermic effect of food. This omission appears to be a disingenuous way to strengthen his anti-carbohydrate arguments.

Dr. Lustig also uses bioenergetics in misleading ways to emphasize his points of view. For example, on page 57 he states that “the most popular combination at McDonald’s is a Big Mac, medium french fries and medium regular soda, providing 1,130 calories (Calories) for \$5.99.” However, to support his argument he chooses to “make it a large” and describes the potential negative impact if people were eating more than what he has specifically said is typical.

Lastly, on page 273, the author suggests that “we should rationally eat 1800-2000 kcals.” With that statement he does not identify who he is referring to as “we”; however, this is a gross underestimation of the total energy requirements for the average adult. According to the Dietary Reference Intake standards established by the Food and Nutrition Board, the estimated energy requirement for men who are 5’5” with a BMI of 24 that are within the low activity classification is 2566 Calories. For similar men who are 6’1” with a BMI of 24, the requirement is 2999 Calories. For women who are 5’1” that are of low activity status with a BMI of 24, the estimate is 1956 Calories. For similar women who are 5’9” the estimate is 2372 Calories. Requirements are much higher for people who are active or very active and are lower for those who are even less active. Overall, it is clear that the energy range provided by the author might better apply to individuals desiring weight loss or for those who are small and highly

Dr. Lustig’s Calorie recommendations ignore the complexity of determining individual Calorie requirements, which depend on a variety of factors, including but not limited to age, gender, and physical activity level. Readers should use the Dietary Guidelines for Americans as an authoritative source for energy intake recommendations.

inactive and that the author is either misleading the readers or is poorly informed about energy requirements.

IV. MISREPRESENTATION OF THE CURRENT FOOD ENVIRONMENT

Dr. Lustig demonstrates a general lack of understanding of food regulations in the United States. His portrayal of the current food environment is misleading to the readers. The author seems to have promoted these inaccuracies in an attempt persuade the readers to agree with his arguments. On page 195 he states that “real food doesn’t have or need a Nutrition Facts Label.” While it is true that labels do not need to appear on unpackaged foods such as a produce, the Code of Federal Regulations is clear regarding nutrient labeling for these types of foods. The regulation from the Federal government is that nutrition facts must voluntarily be posted for at least 90% of fresh food items in a conspicuous place by at least 60% of companies that sell food. While each individual apple or pear may not have a label, the expectation is that the information must be accessible in the store for consumers. On page 198 the author states that “I would also add that if the food has a company logo you’ve heard of, it’s processed”; however, many consumers can recognize logos of common fresh foods such as oranges or bananas. The author makes other sweeping statements on pages 205 and 206, stating myths such as “no good can come of” fast food restaurants or that “you have no control over what goes into the food” there. **Such generalizations do not recognize the potential for selection of healthful foods in these establishments or the fact that the individual can consume the foods chosen in moderation.** It is also disingenuous of the author to state on page 234 that “the food industry currently has carte blanche over what can be put in a food and how it can be processed, packaged and marketed.” **This statement ignores important regulations that restrict ingredients (i.e., GRAS list), nutrition labeling laws, etc.**

While many nutrition professionals recommend including whole foods as the basis of a diet, it is very clear that processed foods and foods eaten outside of the home can fit within a healthy diet (Academy of Nutrition and Dietetics 2013) and can contribute significantly to the nutrient intake of an individual (Eicher-Miller et al. 2012); therefore, an attitude of recommending avoidance of all processed foods is unwarranted. Claiming otherwise is disingenuous and only serves to limit the dietary selections of consumers. By introducing artificial limitations, it is possible that some consumers would become exceedingly frustrated and give up on their goal for eating a healthful diet. In a society where overconsumption of energy and low levels of physical activity are rampant, we must be careful in introducing artificial limitations that will only act as a disservice to the public.

The addition of the Nutrition Facts Panel to packaged foods was a major step forward in public nutrition education and transparency on behalf of food manufacturers. Registered dietitians use the Nutrition Facts Panel as a tool to help patients and clients make healthier purchasing decisions for themselves and their families

Healthy diets include processed foods, like pasteurized milk and whole wheat bread. It is the role of registered dietitians to help consumers understand how to build healthy diets that are compatible with the current food environment.

V. LACK OF RECOGNITION OF SUGAR INTAKE RECOMMENDATIONS

Many organizations, including those of the Federal government, recommend limiting the consumption of sugar, yet the author leads the readers to believe that most health experts and organizations suggest that consumers may eat as much sugar as they desire without negative consequences. On page 245, he says there is “no sugar limit”; however, the Dietary Guidelines for Americans clearly recommend that less than 25% of energy intake be from added sugars. This is also consistent with the recommendations from the Food and Nutrition Board through the Dietary Reference Intake recommendations (Institute of Medicine 2005). Multiple other Federal agencies and publications recommend limiting sugar intake. For instance, the Centers for Disease Control recommends that “Youth should drink fewer sugar-sweetened beverages and more water and low-fat or fat-free milk, or limited amounts of 100% fruit juices. Families, schools and other institutions need to provide healthy beverage choices” (<http://www.cdc.gov/Features/HealthyBeverages/>). For many years messages for limiting the consumption of sugar have been common.

International and national governments, as well as professional and scientific organizations, have published sugar consumption recommendations.

The following inaccuracies suggest that the author is not well-versed in the sciences of foods and nutrition or is misleading the readers in a way to promote their agreement with his views.

VI. GENERALLY INACCURATE STATEMENTS

Dr. Lustig makes several blatantly inaccurate statements that are either directly related to his narrative or that are meant to more tangentially support some of his arguments. Among these statements, are the following:

- “Grains, roots and tubers...have no fat.” On page 109 the author is trying to make a point that naturally occurring foods do not have combinations of fat and carbohydrate. However, the truth is that all grain foods include some naturally occurring fat and smaller amounts are present in roots and tubers. For a few examples, as %age of Calories, wheat is typically ~6% fat, oats are ~16% fat, barley is ~4% fat and corn is ~13% fat.
- “In fact, calorie for calorie, 100 % orange juice is worse for you than soda, because the orange juice contains 1.8 grams of fructose per ounce, while the soda contains 1.7 grams of fructose per ounce.” This statement, found on page 119, is not based on any scientific evidence and the much greater contents of nutrients in orange juice along with the extremely minor difference in fructose content provided by the author; it is obviously false. In 8-ounce servings of the two beverages described, the total difference in fructose content would amount to 0.8 grams. There is no logical reason to believe that such a small quantity could produce any negative effects on health.

- "...this is the reason bananas turn brown with time." On pages 122 and 123, the author is describing the browning of a banana as a process that occurs due to its fructose content through the Maillard browning reaction. This type of browning is actually an enzymatic browning process that is not related to the Maillard reaction.
- "More has been written about this compound than all other vitamins, minerals and supplements combined." In this statement (page 153) the author is referring to vitamin D. While vitamin D has become a topic of greater amounts of research in recent years, this statement is entirely baseless. As a simple test of this notion, a search of the term "vitamin D" on the Google search engine resulted in less than one-third of the hits that a search of the term "calcium" yielded.
- **"White food...means the fiber is gone (or, in the case of potatoes, was never there in the first place.)"** This statement is found on page 212 of the book. The reality is that many white foods, including potatoes, are rich in fiber. Examples besides potatoes include white beans, turnips, celery root, cauliflower and mushrooms. A medium-sized baked potato typically has approximately 4 grams of fiber and a large % age of the fiber is located in the white flesh rather than the skin.

In summary, these types of inaccuracies suggest that the author is not well-versed in the sciences of foods and nutrition or is misleading the readers in a way to promote their agreement with his views.

CONCLUSION

Fat Chance: Beating the Odds Against Sugar, Processed Food, Obesity and Disease is the product of one individual's point of view – a perspective that is not supported by the vast majority of scientific research on nutrition and metabolism. This review highlights the critical need for the increased communication of weight management strategies that are science-based and realistic. As we've witnessed in the past several decades, blaming one particular food or ingredient for the obesity and chronic disease rates in America is unsubstantiated; restriction and avoidance of particular foods (especially those that are most enjoyed by many consumers) is not a sustainable healthy eating strategy. **Numerous scientific authorities, including the Academy for Nutrition and Dietetics, have acknowledged that the most effective way to achieve and sustain a healthy weight is to exercise regularly and eat a balanced, nutrient-dense diet that allows for the enjoyment of all foods within individual calorie limits.** Books like *Fat Chance* are regressive and only serve to increase consumer confusion about nutrition, rather than help create smarter consumers who are able to make informed decisions about their health.

REFERENCES

Academy of Nutrition and Dietetics. *Position of the Academy of Nutrition and Dietetics: Total Diet Approach to Healthy Eating*. J Acad Nutr Diet. 2013;113:307-317.

Ainsworth BE et al. *Compendium of physical activities: an update of activity codes and MET intensities*. Med Sci Sports Exerc. 2000;32:498-504.

Atkinson FS et al. *International tables of glycemic index and glycemic load values: 2008*. Diabetes Care. 2008;31:2281-2283.

Benado M et al. *Effects of various levels of dietary fructose on blood lipids of rats*. Nutrition Research. 2004;24:565-71.

Chong MFF et al. *Mechanisms for the acute effect of fructose on postprandial lipemia*. Am J Clin Nutr. 2007;85:1511-1520.

DeLany JP et al. *Differential oxidation of individual dietary fatty acids in humans*. Am J Clin Nutr. 2000;72:905-11.

Eicher-Miller HA et al. *Contributions of processed foods to dietary intake in the US from 2003-2008: A report of the Food and Nutrition Science Solutions Joint Task Force of the Academy of Nutrition and Dietetics, American Society for Nutrition, Institute of Food Technologists, and International Food Information Council*. J Nutr. 2012;142:2065S-2072S.

Hellerstein MK. *No common energy currency: de novo lipogenesis as the road less traveled*. Am J Clin Nutr. 2001;74:707-708.

Institute of Medicine (US). *Panel on Macronutrients, and Institute of Medicine (US). Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids*. National Academy Press, 2005.

Kuo CC et al. *Lipid oxidation in fit young adults during postexercise recovery*. J Appl Physiol 2005;99: 349–356.

Livesey G and Taylor R. *Fructose consumption and consequences for glycation, plasma triacylglycerol, and body weight: meta-analyses and meta-regression models of intervention studies*. Am J Clin Nutr. 2008;88:1419-1437.

Livesey G. *Fructose ingestion: dose-dependent responses in health researcher*. J Nutr. 2009;139:1246S-1252S.

Loos RJF. *Genetic determinants of common obesity and their value in prediction*. Best Practice & Research Clinical Endocrinology & Metabolism. 2012;26:211–226.

Loos RJF, Bouchard C. *Obesity – is it a genetic disorder?* J Intern Med. 2003; 254: 401–425.

Loos RJF, Ranikinen T. *Gene-diet interactions on body weight changes*. J Am Diet Assoc. 2005;105:S29-S34.

McDevitt RM. *De novo lipogenesis during controlled overfeeding with sucrose or glucose in lean and obese women*. Am J Clin Nutr. 2001;74:737-746.

National Cholesterol Education Program. *Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (ATP III Final Report)*. Circulation. 2002;106:3143.

Parks EJ et al. *Dietary sugars stimulate fatty acid synthesis in adults*. J Nutr. 2008;138:1039-1046.

Tappy L, Le K. *Metabolic effects of fructose and the worldwide increase in obesity*. Physiol Rev. 2010;90:23-46.

Walley AJ et al. *The genetic contribution to non-syndromic human obesity*. Nature Reviews Genetics. 2009;10: 431-442.