

Chocolate & Health

• A SCIENTIFIC OVERVIEW FOR THE HEALTH PROFESSIONAL •



DEVELOPED BY THE

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Summary

The public loves chocolate. A survey of 1,000 American adults found chocolate was ranked as their favorite flavor for desserts and sweet snacks by a three-to-one margin.¹ If asked, some consumers might admit to feeling somewhat guilty about their enjoyment of chocolate although there is no scientific basis for their guilt if chocolate is eaten in moderation as part of a balanced eating plan.

Health professionals have a responsibility to provide consumers with scientifically supported facts to help them make informed dietary decisions.² Despite research to the contrary, chocolate consumption is sometimes associated with deleterious effects on health. Such erroneous beliefs typically are based more on popular folklore than on facts.³ This booklet reviews the scientific literature regarding chocolate and health to help place in perspective the nutritional and medical aspects of this popular food. By communicating current factual information about chocolate, health professionals can help consumers understand how chocolate can be part of a healthy eating plan based on balance, variety and moderation.

This scientific overview addresses the following health issues as they are associated with chocolate.

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Acne

Basic research on the causes of acne vulgaris links its occurrence to rising testosterone levels during puberty. This results in enlargement of the sebaceous glands, which can eventually lead to the development of comedones (blackheads). The American Academy of Dermatology has stated acne is not a dietary disease and following the strictest diet will not by itself clear the skin.⁴

Yet the idea that diet, and specifically chocolate, is implicated in the etiology of acne remained widespread within the medical profession until recently. Among the first to question the link between chocolate and acne were researchers from the University of Missouri in the 1960s. Grant and Anderson⁵ failed in their attempt to induce an acne flare-up in eight individuals with mild to moderate acne by feeding them a large amount of chocolate. The authors discredited the assertion that chocolate causes acne.

A larger study of 65 subjects reported excessive intake of chocolate and fat did not alter the composition or output of sebum from subaceous glands or affect the course of acne. Upon review of studies purporting to show that high carbohydrate or high fat diets aggravate acne, the authors concluded such claims are unproved and that the sebaceous gland has a high degree of autonomy.⁶ In two articles, Shalita^{7/8} has stated there is no evidence that food, including chocolate, has a direct role in the pathogenesis of acne.

In an extensive review of research on chocolate and acne conducted in 1978, Fries³ concluded that the general trend of published reports suggested that chocolate ingestion was unrelated to the cause of acne. As cited in the *Journal of the American Medical Association*: "Diet plays no role in acne treatment in most patients...even



large amounts of chocolate have not clinically exacerbated acne."⁹ The paucity of recent research on chocolate and acne reflects the widespread acceptance of earlier studies acquitting chocolate of any contributing role in acne.

Allergies

A food allergy is a reaction by the body's immune system to a substance or ingredient in food – usually a protein. According to the National Institutes of Health, approximately five million Americans (5-8 percent of children and 1-2 percent of adults) have a true food allergy. Eight major food allergens account for 90 percent of all allergic reactions: milk, eggs, peanuts, tree nuts (such as walnuts or almonds), soy, wheat, fish and shellfish. Children are most commonly affected by reactions to milk and eggs. Chocolate is an uncommon food allergen.¹⁰

Some people, health professionals included, incorrectly confuse the terms "food sensitivity" and "food allergy."¹¹ A *food intolerance or sensitivity* is a reproducible adverse reaction, not psychologically based, which includes enzyme deficiencies, pharmacological effects, non-immunologic histamine-releasing effects and direct irritation. A *food allergy or hypersensitivity* is a form of food intolerance that includes an abnormal immunologic reaction mediated by antibody and/or T lymphocytes.^{12/13}

The only completely objective test for confirming a food allergy is the double-blind placebo-controlled food challenge (DBPCFC).^{14/15/16} The most prevalent food allergens confirmed by DBPCFC are eggs, milk, soy, fish, peanuts, tree nuts, and wheat.¹⁵

Patients' perceptions and physicians' diagnoses of food intolerance are not always accurate; fewer than half of patients with histories of adverse reactions to food can be confirmed by means of objective testing.¹¹ In the case of chocolate, Fries¹⁷ was rarely able to confirm a parent's suspicions of a child's allergies.

According to Fries,³ few scientific publications relate chocolate to allergy. Fries¹⁸ reported positive skin test response to the cocoa bean in about two-thirds of allergy patients. However, test results were not related to clinical symptoms. Maslansky and Wein¹⁹ questioned 500 allergy patients regarding their tolerance of chocolate. Although 81 said they suffered from chocolate allergy, only 10 had the requisite reproducible symptoms for inclusion in the double-blind study. Eight of these patients were challenged and only three had a reaction. Of these three, only one had both symptoms and a positive skin test.

In another study, 20 patients with a history of "allergic" reactions to chocolate were fed either placebo or cocoa in a double-blind manner. The cocoa ingested approximated the amount in an average size chocolate bar. Participants also were fed a chocolate bar at another point in the study. The authors concluded that only one participant probably was allergic to chocolate.²⁰ Likewise, other studies^{21/22/23/24/25} rarely confirmed suspected reactions to chocolate by oral challenge.

Dermal antigen tests to determine allergic response to foods can sometimes be misleading. Problems with dermal

antigen testing include a large number of clinically insignificant positive tests, cross-reactivity among foods from common genetic families, and use of an improper antigen (i.e., cocoa bean instead of processed cocoa). The cocoa bean goes through several procedures (e.g., fermentation, roasting, grinding, conching) before it can be consumed as chocolate.²⁶ Thus, a higher incidence of positive cutaneous tests may result than the incidence of clinical reactions to other ingredients or the products of its digestion.³

Chocolate candies may contain other ingredients that can elicit allergic reactions, including such common foods as milk, soy lecithin, gluten, peanuts and tree nuts. This highlights the importance of label reading by people who may be sensitive to these ingredients.

The diagnosis of chocolate reactions should be based on reliable studies, including food elimination and food challenge tests.²⁷

Antioxidants

Cocoa powder and chocolate are rich sources of high quality polyphenol antioxidants,^{28/29/30} potentially beneficial compounds similar to those found in fruits, vegetables and red wine that may have the potential to reduce the risk of developing heart disease and certain cancers. Cocoa polyphenols were previously thought to only play a role in flavor and aroma development in chocolate, but more recently have been studied for their antioxidant potential and possible health benefits to humans.

Science has long held that damage done in the body by free oxygen radicals is linked to heart disease, certain cancers and physical degeneration maladies associated

TOP ANTIOXIDANT FOODS
ORAC* Units per 100 grams

<p>* ORAC (Oxygen Radical Absorbance Capacity) is a measure of the ability of foods to subdue harmful oxygen free radicals that can damage our bodies.</p>	13,120	DARK CHOCOLATE	980	BRUSSELS SPROUTS
	6,740	MILK CHOCOLATE	949	PLUMS
	5,770	PRUNES	930	ALFALFA SPROUTS
	2,830	RAISINS	890	BROCCOLI FLORETS
	2,400	BLUEBERRIES	750	ORANGES
	2,036	BLACKBERRIES	739	GRAPES, RED
	1,770	KALE	710	RED BELL PEPPER
	1,540	STRAWBERRIES	670	CHERRIES
	1,260	SPINACH	450	ONION
	1,220	RASPBERRIES	400	CORN
			390	EGGFPLANT

Source: Data from U.S. Department of Agriculture and the Journal of the American Chemical Society.

with the aging process. Antioxidants in the blood stream can help eliminate free radicals, potentially reducing the risk of developing some serious diseases.

Among products derived from cocoa beans, cocoa powder contains the highest amount of polyphenols, followed by dark chocolate and milk chocolate. Depending upon the measurement methodology used, a 40 g (1.5 ounce) milk chocolate bar contains between 205-300 mg of polyphenols, which compares favorably to a five-ounce glass of red wine which contains 210 mg of polyphenols and has been associated with reducing the risk of developing heart disease.^{28/29} In a Dutch study, chocolate was found to be a rich source of the polyphenol flavonoid catechin, containing four times the amount found in tea.³¹

By all forms of measurement reviewed, cocoa and dark and milk chocolate have high polyphenol antioxidant values compared to fruits and vegetables. A 1999 analysis of chocolate's polyphenol antioxidant levels utilizing the ORAC (Oxygen Radical Absorbance Capacity)

measurement methodology found dark chocolate to contain 13,120 ORAC units per 100 grams, or more than twice the ORAC activity level of prunes (5,770 ORAC units per 100 grams) which contains one of the highest levels of fruits and vegetables tested.^{32/33}

Research focusing on the potential health benefits of polyphenols in cocoa and chocolate has proven that they are absorbed in the bloodstream. In a clinical trial, eight subjects refrained from consuming foods rich in polyphenols from the day before the test until the study was completed. Each subject consumed chocolate with bread and water on two separate occasions: first consuming 40 g of chocolate, and one week later, consuming 80 g of chocolate. The total polyphenol intake from chocolate was 892 mg, and 1783 mg respectively. Blood samples were drawn before chocolate consumption and at intervals of one, two, three and four hours after consumption. Blood analysis found levels of the polyphenol epicatechin rose after consumption of each chocolate sample, reflecting rapid absorption.³⁴ Other studies

have focused on the beneficial effects of the absorbed polyphenols. One study found that feeding a diet of 1 percent cocoa polyphenols to rabbits retarded low density lipoprotein (LDL) cholesterol oxidation in their blood, potentially preventing a first step in the formation of atherosclerotic plaque.³⁵ Feeding 35 g of cocoa powder to 12 male subjects demonstrated a significant increase in LDL cholesterol's resistance to oxidation within two hours of cocoa consumption.³⁶

Ongoing human research continues to explore the health benefits of chocolate's polyphenol content. Preliminary results of a Pennsylvania State University study that fed controlled diets to 23 male and female subjects supports the potential protective effects of polyphenol antioxidants in chocolate and cocoa. Subjects consumed cocoa powder (22g) and dark chocolate (16g) in their daily diets. Blood analysis revealed that the subjects consuming the cocoa and chocolate had improved cholesterol ratios, with a higher ratio of high density lipoproteins (HDL) to LDL. A higher ratio of HDL to LDL is associated with a lower risk for heart disease. The cocoa and chocolate also appeared to have a beneficial effect on the oxidation of LDL cholesterol.³⁷

Behavior

An extensive literature search did not reveal any studies in which attention-deficit hyperactivity disorder (ADHD) was observed as a direct response to chocolate.³⁸ Past studies have observed

the effects of various foods, including chocolate, but none have focused solely on chocolate. However, the relationship between ADHD and ingestion of sugar, a major ingredient in sweetened chocolate, has been explored in depth. The Food and Drug Administration (FDA)³⁹ concluded there is no substantive evidence that the consumption of sugar is responsible for behavior change in children or adults.

Another review of 11 double blind studies conducted between 1984 and 1991 of sugar and ADHD in both "sugar responders" and "non-sugar responders" showed similar findings.³⁸ Nine of the studies reported no significant effect of sucrose on behavior.^{40/41/42/43/44/45/46/47/48} One study found decreased activity following sucrose challenge⁴⁹ and one, the results of which were never replicated, noted inappropriate behavior after sucrose consumption.⁵⁰ The Surgeon General's Report on Nutrition and Health² noted limited data to support the idea that sugar causes uncontrolled behavior in children.

A 1994 study has contributed additional evidence that sugar does not affect a child's behavior.⁵¹ In the double-blind trial, 25 preschoolers and 23 children aged six to 10 years with a subjective history of sugar-induced behavior changes were fed each of three diets over a nine-week period. One diet contained sugar at twice the amount normally consumed by children, the second contained aspartame, and the third contained saccharin. All three diets were free of preservatives, food coloring, chocolate and caffeine. Tests on activity level, concentration and memory showed no effect from any of the sweeteners. The American Dietetic Association has taken the position that the use of nutritive and nonnutritive sweeteners is appropriate when consumed in moderation and within the context of a diet consistent with the U.S. Dietary Guidelines for Americans.⁵²



Caffeine

Although considerable research has been conducted on the effects of caffeine on behavior in children,² the role of caffeine in chocolate is largely a non-issue. According to the Institute of Food Technologists,⁵³ chocolate contributes only 1.5 percent of children's total caffeine intake. Levels of caffeine range from 3 to 10 mg in a typical 1.4 ounce bar of milk chocolate, and from 2 to 7 mg in an 8-ounce glass of chocolate milk. An ounce of bittersweet chocolate contains 5 to 35 mg of caffeine and an ounce of unsweetened baking chocolate has 35 mg of caffeine – all considerably less than the approximately 75 mg of caffeine found in an eight-ounce cup of instant coffee or 135 mg in the same amount of brewed coffee.⁵⁴ People differ greatly in their sensitivity to caffeine, and stimulant effects in adults may result from consumption of 150 to 200 mg.⁵³

Cravings

Food cravings and the effect of diet on mood have been topics of many studies over the past three decades. While research has determined food cravings do exist, there is little scientific support for a link between the consumption of certain foods and behavior or mood changes.

It has been hypothesized that carbohydrate consumption in general may improve mood by increasing tryptophan transport to the brain, resulting in increased serotonin levels.⁵⁵ Studies in this area, however, have produced inconsistent results.^{56/57} It has been suggested that chocolate may elevate mood, due to certain bioactive compounds it contains, such as tyramine and phenylethylamine.^{58/59} Science, however, has

not proven this theory. Other foods contain higher levels of the same bioactive substances and are not associated with affecting mood. Phenylethylamine, for example, is a neurotransmitter-type substance similar to other substances that improve mood when administered to certain depressed patients. The amount of phenylethylamine in chocolate, however, is quite small. Salami sausage contains about nine times the amount of phenylethylamine found in chocolate and cheddar cheese contains more than 10 times the level in chocolate, but neither food is generally associated with mood changes.^{58/59}

Food cravings also have been associated with chocolate. Research on food cravings indicate that chocolate is the most craved food among women while men crave spicy foods, such as pizza.^{60/61/62} There may be several causes for food cravings, including restrictive diets that eliminate or significantly reduce consumption of certain foods. This, in turn, may result in cravings for those foods.⁶³ It also has been suggested that food cravings may be the result of the body's need for certain nutrients, such as the magnesium in chocolate or the calcium in cheese. Scientific studies, however, have not confirmed this hypothesis.^{64/65}

Research conducted by Michener and Rozin studied subjects who reported experiencing chocolate cravings at least once a week.⁶⁶ Their methodology focused on determining which of the two main components of chocolate — cocoa solids (powder) or cocoa butter — actually satisfied cravings. Cocoa powder contains all of the bioactive ingredients found in chocolate, whereas “white chocolate” is made from cocoa butter, milk and flavorings but contains none of the bioactive ingredients.

When the subjects experienced chocolate cravings, they were given one of the following: milk chocolate, white chocolate, cocoa powder in a capsule,

white chocolate along with a capsule of cocoa powder, a placebo capsule or nothing at all.

If chocolate cravings were related to chocolate's bioactive ingredients, then the cocoa powder capsule and the milk chocolate sample should have satisfied the cravings. However, this was not the case. Only the milk chocolate, and to a lesser extent, the white chocolate (which contains no bioactive ingredients) satisfied the cravings. The cocoa powder capsules had a similar effect to consuming nothing. The study concluded that the sensory experience of eating chocolate, not the bioactive components, were the important factor in satisfying the craving for chocolate.⁶⁶

The menstrual cycle also appears to influence dietary intake and food cravings. There is some scientific evidence that women crave sweet foods in the pre-menstrual and menstrual cycle while fiber intake decreases.⁶⁵ There is, however, also an increase in overall appetite in the pre-menstrual period.^{67/68} Claims that women with PMS or depression consume carbohydrate and chocolate to self-medicate have little scientific support. Again, it appears that increased consumption of these foods is linked to their flavor and other sensory properties rather than the bioactive compounds they contain.



Dental Caries

Tooth decay begins when bacteria, particularly *Streptococcus mutans*, accumulate on the teeth in the absence of adequate oral hygiene, forming plaque. Bacteria metabolize fermentable carbohydrates, leading to acid formation and a decrease in plaque pH. Frequent or sustained drops in pH can result in progressive demineralization of the enamel, ultimately leading to caries. The etiology of caries is a combination of elements: susceptible teeth, dental plaque, food and the length of time food remains in contact with the teeth.⁶⁹

Prevention of dental caries, therefore, requires several measures. It is well known that fluoride is a primary factor in the control of dental caries and can significantly reduce caries.⁶⁹ Other intervention methods include control of fermentable carbohydrate intake, proper oral hygiene and the application of plastic sealants.² There may even be a hereditary disposition to caries resistance.⁷⁰

While sugars have been implicated in the development of dental caries,³⁹ it has long been known that any food containing fermentable carbohydrates — including cooked starches such as bread, cereal, crackers, etc. — can promote caries formation.^{71/72} The cariogenicity of food is related to the amount of total fermentable carbohydrates. It is often assumed that starchy foods such as potato chips and bread are not a factor in caries development because oral bacteria cannot break down starches into acids. However, most starchy foods are retained in the mouth long enough to mix with saliva, which contains the enzyme "amylase" that converts starches into sugars, which the oral bacteria then can metabolize to acids.⁷³ In fact, sugars such as sucrose and fructose tend to be less cariogenic than starch because they dissolve in water and saliva,

and are removed from the mouth more rapidly. Processed high-starch snacks, whether gelatinized, baked or fried, produce as much acid in plaque as sucrose alone, but at a much slower rate.^{74/75} Thus, the cariogenicity of food is not necessarily related to its sugar content. Foods that are 50 percent sugar do not necessarily cause more tooth decay than foods that are 10 percent sugar.

It is difficult to categorize foods by degree of cariogenicity. Certain non-carbohydrate characteristics of a food (e.g., fat, protein, minerals, buffering capacity, water content, texture) can modify cariogenicity, as can the sequence of food consumption.^{69/76/77} The frequency of exposure to carbohydrate-containing foods plays an important role. Eating more than five-to-six times per day and continuous nibbling or sipping of foods and beverages promotes tooth decay.^{72/78/79}

The retention time of food on the teeth also impacts cariogenicity. Recommendations to avoid sticky foods may be misleading. A food such as a caramel or jelly bean is initially sticky, but then quickly clears the mouth.⁷⁵ In a study of the perceived stickiness versus actual retention of 21 commercially available foods, Kashket et al.⁸⁰ found that cookies, crackers and potato chips were more retentive than chocolate bars, caramels, jelly beans and raisins.

Cocoa is not intrinsically cariogenic because it does not contain significant fermentable carbohydrate. For that reason, unsweetened chocolate is not considered a contributing factor in the development of caries in animals or humans, or in measures of plaque formation, acidity, or enamel demineralization.³ Chocolate's lack of cariogenicity may hold true even when chocolate is sweetened. Researchers for the classic 1950s Vipeholm study in Sweden reported no statistically significant difference in the incidence of dental caries

among a control group that consumed no sweets and another group that consumed chocolate.⁸¹

The results of several studies counter the common misconception that sweetened chocolate is highly cariogenic. Morrissey et al.⁸² tested the cariogenicity of several common snack foods on rats, and found solid milk chocolate among the lowest in cariogenicity of all the foods evaluated. A study that specifically examined food retention found that chocolate bars cleared the mouth rapidly because they contain fat, despite the fact that subjects rated chocolate bars as fairly sticky.⁸⁰

Moreover, certain naturally occurring substances such as tannins in cocoa may play a role in the inhibition of dental plaque formation.^{83/84} Yankell et al.⁸⁵ found that mixtures of sucrose and chocolate, cocoa, or confectionery coating at ratios commonly used in candy were less cariogenic in human subjects than a 10 percent sucrose solution, as measured by plaque pH and plaque ionized calcium. This may be due to the tannins or other undefined components in chocolate.

Diabetes

The American Diabetes Association has recommended changes in the approach to medical nutrition therapy for people with diabetes mellitus. The new recommendations permit a wider variety of carbohydrate sources than previously allowed. In a move from strict dietary control, the first new treatment goal calls for maintenance of near-normal blood glucose levels by balancing food intake with physical activity plus insulin or oral hypoglycemic agent.⁸⁶

Prior to 1994, people with diabetes were instructed to avoid simple sugars in order to better maintain blood glucose levels.⁸⁷ It was thought that simple sugars were

more rapidly digested and absorbed than complex carbohydrates and therefore would contribute to high blood sugar. However, many complex carbohydrates also elicit a rapid blood glucose response.

The new recommendations permit limited amounts of sucrose and other nutritive sweeteners, as long as they are factored into the total carbohydrate consumed in a day. These liberalized guidelines allow people with diabetes to enjoy a wider variety of foods, including desserts, chocolate and sweets that contain sugar, to the extent that individual dietary goals continue to be met and that blood sugar control is maintained.⁸⁸

A second goal emphasizes the importance of maintaining optimal serum lipid levels through diet, activity and weight management. Specific diet recommendations should be individualized. A person with normal serum cholesterol and triglyceride levels should consume less than 30 percent of calories from fat, another person with elevated low-density lipoproteins may require a more stringent fat restriction. Elevated serum triglycerides may necessitate more liberal use of monounsaturated fats and a moderate restriction of carbohydrates to control triglycerides.⁸⁶

People with diabetes can eat small amounts of chocolate as part of their total food plan and still adhere to the new nutrition guidelines. A study of 14 adolescents with diabetes found that an occasional snack of milk chocolate as part of a regularly planned snack did not have a negative effect on postprandial blood sugar.⁸⁹

Individuals with high serum triglycerides may need to limit their chocolate intake. In a study of six men with non-insulin-dependent diabetes, researchers found that test meals of 75 g (2.6 ounces) of milk chocolate that



was sweetened with sucrose, fructose or a disaccharide alcohol elevated serum triglyceride levels.⁹⁰ This study suggests that chocolate should be consumed in moderation, especially by those individuals with high serum triglyceride levels. Chocolate sweetened with non-nutritive sweeteners was not tested.

In diabetes management as in general health promotion, maintenance of a healthy weight is a primary goal. Moderate amounts of chocolate and other foods at the tip of the Food Guide Pyramid can and should be included to the extent that they add flavor and enjoyment without pushing the diet beyond desirable calorie, fat, and cholesterol guidelines.⁹¹

Migraines

Some individuals report reactions to chocolate that are not classified as allergies. These reactions, including migraine headaches, may be exacerbated by genetics, lifestyle, medications and hormones.⁹²

Migraines, in particular, have long been associated with chocolate consumption, particularly among women. One study of 490 persons with migraines found that 19 percent reported they thought chocolate caused their headaches.⁹³ Other research demonstrates a lack of relationship between chocolate and headache. Moffet, Swash and Scott⁹⁴ studied a group of 25 migraine sufferers, giving them a chocolate

sample and a placebo (carob) two weeks apart. The subjects completed questionnaires regarding their reactions within 48 hours of sample consumption. There was no difference in headache occurrence after either sample. In a second study, the researchers repeated the same procedure with 15 of the 25 subjects and again found no difference in reported headaches after either sample.

In a recent study, Pittsburgh State University researchers demonstrated a lack of relationship between chocolate and headache in a large sample of women with migraine or tension-type headaches.⁹⁵ After following a vasoactive-amine elimination diet, 63 subjects ingested two 60 g chocolate samples and two 60 g carob samples in random order on four different occasions. Results indicated that chocolate was no more likely to trigger a headache than carob, even in subjects who strongly believed chocolate was a trigger food. The subjects also were unsuccessful at guessing which samples were chocolate during the trials. The researchers conducted a separate evaluation on 17 percent of the study's subjects who identified themselves as sensitive to chocolate as a headache trigger. Ingestion of chocolate samples did not result in headaches.⁹⁵

The reason that chocolate is frequently cited as a food trigger by migraine sufferers despite evidence to the contrary may lie in other aspects of chocolate. Women are three times more likely than men to suffer from migraines⁹⁶ and women also crave chocolate more frequently than men,⁹⁷ but this does not prove a cause-effect relationship. Sweet craving itself has been reported as a prodromal symptom of migraine,⁹⁸ suggesting that chocolate craving and consumption may be a symptom rather than a cause of migraine. Other factors, including fluctuating estrogen levels associated with the onset of menses^{99/100/101} have been clearly associated with the onset of migraines.

Obesity

Obesity is a condition which occurs when a person's body is unable to balance energy intake with energy expenditure. The National Health and Nutrition Examination Survey (NHANES III) states that 22 percent of the U.S. adult population is obese and 55 percent of American adults are obese or overweight.¹⁰² Health risks related to obesity include coronary heart disease, hypertension, diabetes, certain cancers, gall stones and others. Medical conditions related to obesity are the second leading cause of death in the United States, following diseases related to the habit of smoking.^{102/103}

According to the American Dietetic Association, successful weight management requires lifestyle modification that includes increased consumption of fruits, vegetables and grains; a nonrestrictive approach to eating based on hunger and satiety; and a gradual increase in physical activity to at least 30 minutes a day.¹⁰⁴

The dietary debate continues on the role of diet composition and weight gain, particularly fat and carbohydrate. Most nutritionists agree that excess calories, regardless of the source, are the cause of weight gain. Others point to excess calories from fat or carbohydrate for causing weight gain. While few studies actually have analyzed different kinds of carbohydrates in relation to weight gain, research studies do point to a relationship between fat intake and obesity.¹⁰⁵ Regardless of the measure of diet composition, physical activity is a critical factor in the energy balance equation for both weight loss regimens and weight maintenance.¹⁰²

Proponents of certain fad diets suggest that the growing prevalence of obesity in the United States is due to high carbohydrate diets—particularly diets rich in foods with a high glycemic index, such as white bread, white rice and even certain

vegetables. Consumption of these foods leads to a rapid blood sugar response and elevated blood insulin levels, especially among the estimated 25 percent of the U.S. population believed to be insulin resistant. This, according to the theory, is believed to lead to excess carbohydrates being stored as body fat. Research studies, however, do not support this theory.¹⁰⁵

In fact, research on carbohydrate consumption and obesity indicates that diets high in carbohydrates, including sugar, do not result in weight gain when they are consumed in amounts that do not exceed energy expenditure.^{106/107/108} Studies even have found that groups of people consuming the most sugar have the lowest levels of obesity,¹⁰⁹ which some nutritionists believe may be the result of a decrease in fat intake as sugar intake increases. In another recent study, 60 overweight women consumed a low-fat, reduced-calorie diet that was either high or low in sucrose. After the six-week long diet, the high and low sucrose groups demonstrated equally significant reduction of body weight and percentage of body fat.¹¹⁰

While a “sweet tooth” appears to have little or no connection to obesity, preference for foods containing fat may have a role in weight gain. Studies suggest that obese individuals prefer the same concentrations of sugar in their diets as normal weight individuals, but they prefer higher concentrations of fat.¹¹¹ A fat, whether saturated, mono or polyunsaturated, provides 9 kcal/g, or more than twice the calories in carbohydrate (4 kcal/g). A diet made up of predominately fat-rich foods increases the possibility of excess calorie consumption.¹¹³ Studies of fat preferences among obese adults reveal a close relationship between preferences for fat and body fat.^{111/113}

There is no scientific evidence that chocolate consumption is associated with obesity. On average, chocolate has been found to contribute only 0.7-1.4 percent of total daily energy intake.¹¹⁴ The pleasurable sensory properties of chocolate make it a desirable food which may generate feelings of guilt and associations with weight gain in some people. However, no single food causes obesity or weight gain. The overall diet, coupled with a lack of appropriate energy expenditure, is the underlying cause of weight gain.

Serum Cholesterol

The National Cholesterol Education Program has increased public awareness of the need to reduce saturated fat intake.¹¹⁵ However, the relative effects of specific saturated fatty acids on blood lipids is not fully understood by most consumers.

Cocoa butter is the fat that occurs naturally in cocoa beans. The U.S. government standards of identity for chocolate specify that cocoa butter is the only fat that is allowed in all types of chocolate except milk chocolate, which contains approximately 80 percent fat from cocoa butter and 20 percent from milk fat. Cocoa butter, like all fats, is composed of several fatty acids.

The percentage composition of major fatty acids in cocoa butter is as follows:

STEARIC ACID	35%	
OLEIC ACID	35%	
PALMITIC ACID	25%	
LINOLEIC ACID	3%	
OTHER	2%	

Stearic and palmitic acids are saturated fatty acids, oleic acid is a monounsaturated fatty acid, and linoleic acid is a polyunsaturated fatty acid.

Early studies showed that cocoa butter did not raise cholesterol as might be predicted by its saturated fatty acid content because of the neutral effects of stearic acid on blood cholesterol levels. Unlike palmitic acid or other saturated fatty acids, studies have shown that stearic acid does not raise blood cholesterol. As discussed in a review of nearly 40 years of scientific research, cocoa butter has been shown to have a neutral effect on blood cholesterol levels, possibly because of its high stearic acid content.¹¹⁶ Two classic metabolic ward studies in 1965 reported that diets high in cocoa butter had a neutral cholesterolemic effect.^{117/118} For this reason, cocoa butter has long been used as a neutral control in studies designed to measure increases in serum cholesterol caused by other fats high in saturated fatty acids.¹¹⁷

Researchers have hypothesized that cocoa butter's anomalous effect on serum cholesterol may be due to more than one factor. One theory suggests cocoa butter is not as well absorbed by the body as other saturated fatty acids.^{119/120} Another is that absorbed stearic acid is rapidly converted by the body to oleic acid, a monounsaturated fatty acid.¹²¹

Kris-Etherton and colleagues¹²² at The Pennsylvania State University have studied not only cocoa butter, but also milk chocolate. In their study, subjects consumed 10 ounces of milk chocolate per day in pudding and brownies, supplying 80 percent of the approximately 37 percent of their dietary calories contributed by fat. The total diet was controlled in all other ways. Despite the fact that the chocolate enriched diet was high in saturated fatty acids (approximately 20 percent of total calories), subjects experienced a neutral cholesterolemic response compared to their usual diet that did not include chocolate and which contained about 14 percent

of total calories from saturated fatty acids. When participants consumed diets rich in dairy butterfat on an equal fat and saturated fat calorie basis, their mean cholesterol levels were higher than when they consumed the milk chocolate enriched diet. The study further demonstrated that stearic acid did not produce hypercholesterolemic effects compared to other long-chain saturated fatty acids such as myristic and lauric acid.¹²³

In another study, the same researchers¹²⁴ had subjects consume a National Cholesterol Education Program/American Heart Association Step-One Diet, with the daily substitution of a 1.6-ounce milk chocolate bar (a typical candy bar weighs 1.4 ounces) in place of a high carbohydrate snack. Consuming the chocolate bar did not adversely affect their low-density lipoproteins, or LDL cholesterol levels. Including the chocolate bar resulted in an increase in the total fat content of the diet from 30 to 34 percent of total calories but did not alter the mean LDL-cholesterol levels. At the same time, the study demonstrated an increase in high-density lipoproteins, or HDL-cholesterol levels. The study suggests, strategies to reduce dietary fat should emphasize reduction of the atherogenic saturated fatty acids rather than stearic acid.

Placing fewer restrictions on certain foods, thereby providing greater flexibility in diet planning, enhances palatability and promotes adherence to diets for the health-conscious individual.¹²⁵ This approach allows moderate intake of foods containing fat, with greater emphasis on those higher in stearic acid or unsaturated fats. However, caloric consumption in excess of expenditure contributes to obesity and can have a blood cholesterol raising effect. Therefore, chocolate can be included in small amounts, to the extent that the overall diet falls within total calorie and saturated fat goals.

A SUMMARY OF CHOCOLATE NUTRITION FACTS

	Weight g	Calories	Calories from Fat	Total Fat g (% DV)	Saturated Fat g (% DV)	Cholesterol mg (% DV)	Sodium mg (% DV)	Carbohydrate g (% DV)	Protein g	Calcium % DV
Dark Chocolate Bar (1.4 oz)	40	200	100	11(17)	7(35)	0(0)	0(0)	25(8)	1	0
Milk Chocolate Almond Bar (1.4 oz)	40	210	130	14(22)	7(35)	7(2)	30(2)	21(7)	4	8
Milk Chocolate Bar (1.4 oz)	40	210	120	13(20)	7(35)	11(4)	35(2)	23(8)	3	10
Milk Chocolate Covered Peanuts (16 pieces)	40	210	120	13(20)	4(20)	2(1)	15(1)	21(7)	6	6
Milk Chocolate Covered Raisins (35 pieces)	40	160	50	6(9)	3.5(18)	2(1)	15(1)	27(9)	1	4
Milk Chocolate Malted Milk Balls (17 pieces)	40	180	50	6(9)	3(15)	2(1)	55(2)	28(9)	0	6
Semi-Sweet Chocolate Chips (30 pieces)	15	70	35	4(6)	2.5(12)	0(0)	0(0)	27(9)	1	0

1. Chocolate Manufacturers Association, 1999.
2. *The Surgeon General's Report on Nutrition and Health*. Washington DC: Public Health Service: 1988. U.S. Department of Health and Human Services. Public Health Service Publication No. 88-50210.
3. Fries JH. Chocolate: A review of published reports of allergic and other deleterious effects real or presumed. *Annals of Allergy*. 1978;41:195-207.
4. *Acne*. Schaumburg, IL: American Academy of Dermatology; 1986, revised 1991. Form No. TPAMO2-4/92.
5. Grant JD, Anderson PC. Chocolate as a cause of acne: A dissenting view. *Missouri Med*. 1965;62:459-460.
6. Fulton JE Jr, Plewig AM. Effect of chocolate on acne vulgaris. *JAMA*. 1969;210:2071-2074.
7. Shalita AR. Acne vulgaris: not curable but treatable. *Mod Med*. August 1, 1975;66-76.
8. Acne patients fact. *JAMA*. 1977;237:622-628. Medical News.
9. Kaminester LH. Acne, When friends or patients ask about...*JAMA*. 1978.239:2171.
10. Food & Agricultural Organization of the United Nations. 1995. Report of the FAO Technical Consultation on Food Allergies, Rome, Italy, Nov. 13-14.
11. Ferguson A. Definitions and diagnosis of food intolerance and food allergy: consensus and controversy. *J Pediatr*. 1992;121:S7-S11.
12. American Academy of Allergy and Immunology Committee on Adverse Reactions to Foods and National Institutes of Allergy and Infectious Diseases. Adverse Reactions to Foods. July 1984. NIH Publication No. 84-2442.
13. Anderson J. In: Perkin J, ed. Food Allergies and Adverse Reactions. Gaithersburg: ASPEN Publishers, Inc. 1990.
14. Metcalfe D, Sampson H. Workshop on experimental methodology for clinical studies of adverse reactions to foods and food additives. *J Allergy Clin Immunol*. 1990;86:421-442.
15. Bock SA, Atkins FM. Patterns of food hypersensitivity during 16 years of double-blind, placebo-controlled food challenges. *J Pediatr*. 1990;117:561-567.
16. Van Arsdel P, Larson E. Diagnostic tests for patients with suspected allergic disease. *Ann Inter Med*. 1989;110:304-312.
17. Freis JH. Food allergy: current concerns. *Ann Allergy*. 1981;46:260-263.
18. Freis JH. The cocoa bean and the allergic child. *Ann Allergy*. 1966;24:484.
19. Maslansky L, Wein G. Chocolate allergy: a double-blind study. *Conn Med*. 1971;35:5-9.
20. Drelich JM, Anderson JA, Sears-Ewald DA. Chocolate allergy evaluated by double-blind, placebo-controlled food challenge (DBPCFC). *J Allergy Clin Immunol*. 1993;91:340.
21. Bock SA, Buckley J, Holst A, May CD. Proper use of skin tests with food extracts in diagnosis of hypersensitivity to food in children. *Clin Allergy*. 1977;7:375-383.
22. Bock, SA, Lee W-Y, Remigio L, Holst A, May CD. Appraisal of skin tests with food extracts for diagnosis of food hypersensitivity. *Clin Allergy*. 1978;8:559-564.
23. Bock SA, Lee W-Y, Remigio L, May CA. Studies of hypersensitivity to foods in infants and children. *J Allergy Clin Immunol*. 1978;62:327-334.
24. Bernstein M, Day J, Welsh A. Double-blind food challenges in the diagnosis of food sensitivity in the adult. *J Allergy Clin Immunol*. 1982;70(3):205-210.

25. Zeitz H, Cato M, Lekach R, Thomas L, Jurmuszuk I, Samter M. Reactions to specific foods in adults (I) chocolate (C). *J Allergy Clin Immunol.* 1986;77:238.
26. Krummel D. Chocolate and food allergies: fact or fiction. *Immunology & Allergy Practice.* 1992;14(8):306/33-312/39.
27. Cantani A, Ferrara M, Vazzoler C. Case in point: allergy, intolerance or pseudoallergy to chocolate? *Riv Eur Sci Med Farmacol.* 1989;11(3):247-9.
28. Waterhouse AL, Shirley JR, Donovan, JL. Antioxidants in Chocolate. *Lancet.* 1996;348: 834.
29. Vinson JA Presentation to the American Chemical Society, March, 1999.
30. Sanbongi C, Osakabe N, Natsume M, Takizawa T, Gomi S, Osawa T. Antioxidative polyphenols isolated from theobroma cacao. *J Ag Food Chem.* 1998;46:454-457.
31. Arts IC, Hollman PC, Kromhout D. Chocolate as a source of tea flavonoids. *Lancet.* 1999;354:488.
32. Adamson G, Lazarus S, Amitchell A, Prior R, et al. HPLC method for the quantification of procyanidins in cocoa and chocolate samples and correlation to total antioxidant capacity. *J Am Chem Soc.* 1999.
33. U.S. Dept. of Agriculture, Can foods forestall aging? *Ag Research.* Feb. 1999;47:15-17.
34. Richelle M, Tavazzi I, Enslin M, and Offord, EA. Plasma kinetics in man of epicatechin from black chocolate. *European J of Clin Nut.* 1999;53: 22-26.
35. Natsume, et al. Annual meeting of the Japan society of biotechnology and agrochemistry, 1997.
36. Kondo K, Hirano R, Matsumoto A, et al. Inhibition of LDL oxidation by cocoa. *Lancet.* 1996;348:1514.
37. Kris-Etherton P and Wan Y. Fifth international symposium on chocolate and cocoa nutrition. Tokyo, Japan; Sept. 1999.
38. Krummel DA, Seligson FA, Guthrie DA. Hyperactivity: Is Candy Causal? *Critical Reviews in Food Science and Nutrition.* 1996;36(1 and 2):31-47.
39. Glinsmann WH, Irausquin H, Park YK. *Evaluation of Health Aspects of Sugars Contained in Carbohydrate Sweeteners. Report of Sugars Task Force, 1986. Executive Summary.* Washington, DC: Division of Nutrition and Toxicology, Center for Food Safety and Applied Nutrition, Food and Drug Administration: 1986.
40. Gross MD. Effect of sucrose on hyperkinetic children. *Pediatrics.* 1984;74:876-878.
41. Wolraich M, Milich R, Stumbo P, Schultz F. Effects of sucrose ingestion on the behavior of hyperactive boys. *J Pediatr.* 1985;106:675-682.
42. Milich R, Pelham WE. Effects of sugar ingestion on the classroom and play group behavior of attention-deficit disordered boys. *J Consult Clin Psychol.* 1986;54:714-718.
43. Wender EH, Solanto MV. Effects of sugar on aggressive and inattention behavior in children with attention-deficit disorder with hyperactivity and normal children. *Pediatrics.* 1991;88:960-966.
44. Ferguson HB, Stoddart C, Simeon JG. Double-blind challenge studies of behavioral and cognitive effects of sucrose-aspartame ingestion in normal children. *Nutr Rev.* 1986;44:144-150.
45. Mahan LK, Chase M, Furukawa CT, et al. Sugar "allergy" and children's behavior. *Ann Allergy.* 1988;61:453-458.
46. Rosen LA, Bender ME, Sorrell S, Booth SR, McGrath ML, Drabman RS. Effects of sugar (sucrose) on children's behavior. *J Consult Clin Psychol.* 1988;56:583-589.

47. Roshon MS, Hagen RL. Sugar consumption, locomotion, task orientation, and learning in preschool children. *J Abnorm Child Psychol.* 1989;17:349-357.
48. Kruesi MJ, Rapoport JL, Cummings M, et al. Effects of sugar and aspartame on aggression and activity in children. *Am J Psychiatry.* 1987;144:1487-1490.
49. Behar D, Rapoport JL, Adams AJ, Berg CJ, Cornblath M. Sugar challenge testing with children considered behaviorally "sugar reactive." *Nutr and Behav.* 1984;1:277-288.
50. Goldman JA, Lerman RH, Contois JH, Udall JN. Behavioral effects of sucrose on preschool children. *J Abnorm Child Psychol.* 1986;14:565-577.
51. Wolraich JL, Lindgren SD, Stumbo PJ, et al. Effects of diets high in sucrose or aspartame on the behavior and cognitive performance of children. *N Engl J Med.* 1994;330:301-307.
52. Position of The American Dietetic Association: Use of nutritive and nonnutritive sweeteners. *J Am Diet Assoc.* 1993;93:816-821.
53. A Scientific Status Summary by the Institute of Food Technologists' Expert Panel on Food Safety & Nutrition. Chicago, IL: *Institute of Food Technologists*; 1987.
54. U.S. Department of Agriculture Handbook #8.
55. Wurtman RJ, Wurtman JJ. Carbohydrate cravings, obesity and brain serotonin. *Appetite.* 1986;7:99-103.
56. Christensen L, Redig C. Effect of meal composition on mood. *Behav Neurosci.* 1993;107:346-353.
57. Reid, M, Hammersley R. Effects of carbohydrate intake on subsequent food intake and mood state. *Physiol Behav.* 1995;58:421-7.
58. Hurst WJ, Martin RA, Zoumas, BL. Biogenic amines in chocolate: a review. *Nutr Rep Intl.* 1982;26:1081-6.
59. Koehler PE, Eitenmiller RR. High pressure liquid chromatographic analysis of tyramine, phenylethylamine and tryptamine in sausage, cheese and chocolate. *J of Food Sci.* 1978;43:1245-7.
60. Weingarten HP, Elston D. Food cravings in a college population. *Appetite.* 1991;17:167-75.
61. Rozin P, Levine E, Stoess C. Chocolate preference and craving. *Appetite.* 1991;17:199-212.
62. Hill AJ, Heaton-Brown L. The experience of food craving: a prospective investigation in healthy women. *J Psychosom Res.* 1994;38:801-14.
63. Polivy J. Psychological consequences of food restriction. *J Am Diet Assoc.* 1996;96:589-92.
64. Weingarten HP, Elston D. The phenomenology of food cravings. *Appetite.* 1990;15:231-46.
65. Vlitos ALP, Davies GJ. Bowel function, food intake and the menstrual cycle. *Nut Res Rev.* 1996; 9:111-134.
66. Michener W and Rozin P. 1994. Pharmacological versus sensory factors in the satiation of chocolate craving. *Physiol. Behav.* 56,419-422.
67. Dalvit SP. The effect of the menstrual cycle on patterns of food intake. *Am J Clin Nutr.* 1981;34:1811-5.

68. Lissner L, Stevens J, Levitsky DA, et al. Variation in energy intake during the menstrual cycle: implications for food intake research. *Am J Clin Nutr.* 1988;48:956-62.
69. Greene JC, Louie R, Wycoff SJ. Preventive dentistry: Dental caries. *JAMA.* 1989;262:3459-63.
70. Mandel ID. Resistance to caries; The case for heredity. In: *Changing Perspectives in Nutrition and Caries Research.* New York, NY; Medcom, Inc; 1979;28-29.
71. Bibby BG, Goldberg HJV, Chen E. Evaluation of caries-producing potentialities of various foodstuffs. *J Amer Dent Assoc.* 1951;42:491-509.
72. Kandelman D. Sugar, alternative sweeteners and meal frequency in relation to caries prevention: New perspectives. *Brit J Nutr.* 1997;77:S121-S128.
73. Carbohydrates and tooth decay. *Tufts University Diet & Nutrition Letter.* 1986;4:1.
74. Mormann JE, Muhlemann HR. Oral starch degradation and its influence on acid production in human dental plaque. *Caries Res.* 1981;15:166-175.
75. Grenby TH. Snack foods and dental caries. Investigations using laboratory animals. *Brit Dent J.* 1991;353-361.
76. Alfno MC. Understanding the role of diet and nutrition in dental caries. In: *Changing Perspectives in Nutrition and Caries Research.* New York, NY: Medcom, Inc; 1979;6-12.
77. Tinanoff N. Dental plaque in the carious process. In: Stewart RE, Barber TK, Troutman KC, Wei SHY, eds. *Pediatric Dentistry, Scientific Foundations and Clinical Practice.* St. Louis, MO: CV Mosby co; 1982;548.
78. Firestone AR, Schmid R, Muhlemann HR. Effect of the length and number of intervals between meals on caries in rats. *Caries Res.* 1984;18:128-133.
79. Gatenby SJ. Eating frequency: methodological and dietary aspects. *Brit J Nutr.* 1997;77:S7-S10
80. Kashket S, Van Houte J, Lopez, LR, Stocks S. Lack of correlation between food retention on the human dentition and consumer perception of food stickiness. *J Dent Res.* 1991;70:1314-1319.
81. Gustafson B, Quensel CE, Lanke L, et al. The Vipeholm dental caries study: The effect of different levels of carbohydrate intake on dental caries activity in 436 individuals observed for five years. *Acta Odontol Scand.* 1954;11:232.
82. Morrissey RB, Burkholder BD, Tarka SM Jr. The cariogenic potential of several snack foods. *J Amer Dent Assoc* 1984;109:589-591.
83. Paolino VJ, Kashket, S. Inhibition by cocoa extracts of biosynthesis of extracellular polysaccharide by human oral bacteria. *Arch Oral Biol.* 1985;30:359-363.
84. Kashket, S, Paolino VJ, Lewis DA, Van Houte J. In-vitro inhibition of glucosyltransferase from the dental plaque bacterium streptococcus mutan by common beverages and food extracts. *Arch Oral Biol.* 1985;30:821-826.
85. Yankell SL, Emling RC, Shi X, Greco MR. Low cariogenic potential of mixtures of sucrose and chocolate, cocoa or confectionery coatings. *J Clin Dent.* 1988;1:28-30.
86. American Diabetes Association. Nutrition recommendations and principles for people with diabetes mellitus. *Diabetes Care.* 1994;17:519-522.

87. American Diabetes Association. Nutritional recommendations and principles for individuals with diabetes mellitus: 1986. ADA Position Statement *Diabetes Care*. 1987;10:126-132.
88. Diabetes Care and Education, Tinker LF, Heins JM, Holler, HJ. Commentary and translation: 1994 nutrition recommendations for diabetes. *JADA*. 1994;94(5):507-11.
89. Cedermark G, Selenius M, Tullus K. Glycaemic effect and satiating capacity of potato chips and milk chocolate bar as snacks in teenagers with diabetes. *Eur J Pediatrics*. 1993;152:635-9.
90. Gee JM, Cooke D., Gorick S, Worthley GM, et al. Effects of conventional sucrose-based, fructose-based and isomalt-based chocolates on postprandial metabolism in non-insulin-dependent diabetics. *Eur J Clin Nutr*. 1991;45(11) 561-6.
91. The Food Guide Pyramid. Washington, DC: U.S. Department of Agriculture, Human Nutrition Information Service, 1992. *Home and Garden Bulletin No. 252*.
92. American Academy of Allergy and Immunology Committee on Adverse Reactions to Foods and National Institute of Allergy and Infectious Diseases. *Adverse Reactions to Foods*. July 1984. NIH Publication No. 84-2442.
93. Peatfield RC, Glover V, Littlewood JT, Sandler M, Clifford RF. The prevalence of diet-induced migraine. *Cephalgia*. 1984;4(4):179-183.
94. Moffet AM, Swash M, Scott DF. Effect of chocolate in migraine: a double-blind study. *J Neurol, Neurosurg & Psychiat*. 1974;37:445-448.
95. Marcus DA, Schraff L, Turk DC. A double-blind provocative study of chocolate as a trigger of headache. *Cephalgia*. 1997;17:855-862.
96. Stewart W, Lipton R. Migraine epidemiology in the United States. In Olsen, J. (ed.), *Headache Classification and Epidemiology*. Raven Press, Ltd., New York, NY. 1994;239-246.
97. Rozin P, Levine E, Stoess C. Chocolate craving and liking. *Appetite*. 1991;17:199-212.
98. Blau JN, Diamond S. Dietary factors in migraine precipitation: the physician's view. *Headache*. 1984;25:184-187.
99. Nattero G. Menstrual headache. In Critchley, M. (ed.), *Advances in Neurology*. 1982. Vol. 33. Raven Press, New York.
100. Schraff L, Turk DC, Marcus DA. Triggers of headache episode and coping responses of headache diagnostic groups. *Headache*. 1995;35:397-403.
101. Kohler T, Haimerl C. Daily stress as a trigger of migraine attacks: results of thirteen single-subject studies. *J Consult Clin Psychol*. 1990;58:870-872.
102. National Institutes of Health, National Heart, Lung and Blood Institute, 1998. *Clinical Guidelines on the Identification, Evaluation and Treatment of Overweight and Obesity in Adults*.
103. Flegal KM, et al. 1998. Overweight and obesity in the United States: prevalence and trends. 1960-1994. *Int. J. Obesity*. 22:39-47.
104. ADA reports: Position of the American Dietetic Association: Weight management. *J Am Diet Assoc*. 1997;Vol. 7: No. 1:71-74.
105. Hill JO and Peters, JC. Environmental contributions to the obesity epidemic. *Science*. 1998;280 (5368):1371-1374.
106. Willet WC. Dietary fat and obesity: an unconvincing relationship. *Am J Clin Nutr*. 1998;68:1149-1150.

107. Rolls, BJ. And Hill JO. Carbohydrates and weight management. 1998; International Life Sciences Institute, Washington, D.C.
108. Nutall, F. Q. and Gannon, M. C. Carbohydrates and diabetes. IN: Franz, M. J. and Battle, J. P., eds. American Diabetes Assoc. Guide to Medical Nutrition Therapy. Am. Diabetes Assoc. 1999; 85-106.
109. Bolton, Smith C. and Woodward M. Patterns of food and nutrient intake in adults consuming high and low levels of table sugar in Dublin suburb of chronically high employment. Proceedings of the Nutrition Society 1994;48-132A.
110. Surwit RS. Et al. Metabolic and behavioral effects of a high-sucrose diet during weight loss. *Am. J Clin Nutr.* 1997;65:908-915.
111. Drewnowski A, et al. Sweet tooth reconsidered; taste preferences in human obesity. *Physiol Behav* 1995; 35:617-622.
112. Mela, DJ and Sacchetti, DS. Sensory preferences for fats in foods: relationships to diet and body composition. *Am J Clin Nutr.* 1991; 53:908-915.
113. Green SM, Delargy HJ, Joanes D and Blundell JE A satiety quotient: a formulation to assess the satiating effect of food. *Appetite.* 1997; 29:291-304.
114. Seligson FH, Krummel DA and Apgar JR. Patterns of chocolate consumption. *Am J Clin Nutr.* 1994;60:S1060-S1067.
115. *Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults.* U.S. Department of Health and Human Services, Public Health Service. National Institutes of Health; 1989. NIH Publication No. 89-2925.
116. Kritchevsky D. Effects of Triglyceride Structure on Lipid Metabolism. *Nutrition Reviews.* 1988;46:177-181.
117. Keys A, Anderson JT, Grande F. Serum cholesterol response to changes in the diet. IV. Particular saturated fatty acids in the diet. *Metabolism.* 1965;14:776-787.
118. Hegsted, DM, McGandy RB, Myers, ML, et al. Quantitative effects of dietary fat on serum cholesterol in man. *Am J Clin Nutr.* 1965;7281-295.
119. Apgar JL, Shively CA, Tarka SM. Digestibility of cocoa butter and corn oil and their influence on fatty acids distribution in rats. *J Nutr.* 1987;117:660-664.
120. Mitchell DC, McMahan KE, Shively, CA, Apgar JL, Kris-Etherton PM. Digestibility of cocoa butter and corn oil in human subjects: a preliminary study. *Amer J Clin Nutr.* 1989;50:983.
121. Bonanome, A, Grundy, SM, Effect of dietary stearic acid on plasma cholesterol and lipoprotein levels. *N Engl J Med.* 1988;318:1244-1248.
122. Kris-Etherton PM, Derr JA, Mitchell DC, et al. The role of fatty acid saturation on plasma lipids, lipoproteins and apolipoproteins. I. Effects of whole food diets high in cocoa butter, olive oil, soybean oil, dairy butter and milk chocolate on the plasma lipids of young men. *Metabolism.* 1993;42:121-129.
123. Derr JA, Kris-Etherton PM, Pearson TA, Seligson FH. The role of fatty acid saturation on plasma lipids, lipoproteins and apolipoproteins. II. The plasma total and LDL-cholesterol response of individual fatty acids. *Metabolism.* 1993;42:130-134.
124. Kris-Etherton PM, Derr JA, Mustad VA, Seligson FH, Pearson TA. A milk chocolate bar/day substituted for a high carbohydrate snack increases high density lipoprotein cholesterol in young men on an NCEP/AHA Step One diet. *Am J Clin Nutr supplement.* December 1994.
125. Cobb TK. Effects of dietary stearic acid on plasma cholesterol levels. *South Med J.* 1992;85:25-27.

A collection of chocolate bars, blueberries, and a small cup of chocolate-covered blueberries. The chocolate bars are broken into pieces, and the blueberries are scattered around them. A small white paper cup on the right contains several chocolate-covered blueberries. The entire scene is set against a light, neutral background.

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