

Chocolate: Food or drug?

KRISTEN BRUINSMA, MS; DOUGLAS L. TAREN, PhD

ABSTRACT

Although addictive behavior is generally associated with drug and alcohol abuse or compulsive sexual activity, chocolate may evoke similar psychopharmacologic and behavioral reactions in susceptible persons. A review of the literature on chocolate cravings indicates that the hedonic appeal of chocolate (fat, sugar, texture, and aroma) is likely to be a predominant factor in such cravings. Other characteristics of chocolate, however, may be equally as important contributors to the phenomena of chocolate cravings. Chocolate may be used by some as a form of self-medication for dietary deficiencies (eg, magnesium) or to balance low levels of neurotransmitters involved in the regulation of mood, food intake, and compulsive behaviors (eg, serotonin and dopamine). Chocolate cravings are often episodic and fluctuate with hormonal changes just before and during the menses, which suggests a hormonal link and confirms the assumed gender-specific nature of chocolate cravings. Chocolate contains several biologically active constituents (methylxanthines, biogenic amines, and cannabinoid-like fatty acids), all of which potentially cause abnormal behaviors and psychological sensations that parallel those of other addictive substances. Most likely, a combination of chocolate's sensory characteristics, nutrient composition, and psychoactive ingredients, compounded with monthly hormonal fluctuations and mood swings among women, will ultimately form the model of chocolate cravings. Dietetics professionals must be aware that chocolate cravings are real. The psychopharmacologic and chemosensory effects of chocolate must be considered when formulating recommendations for overall healthful eating and for treatment of nutritionally related health issues. *J Am Diet Assoc.* 1999;99:1249-1256.

Gotta have it' is the driving thought of an addict—a drink, a drag, a hit, a line, a pill, another piece of chocolate.... This urgent inner demand overrides all others, undermines reason, resolve, and will... It does not stop until it is satisfied. And then, it starts again" (1, p 1). Although addictive behavior is generally associated with drug, alcohol, or sexual behavior, it is becoming apparent that certain food substances, most notably chocolate, may effect similar physiological and psychological reactions in susceptible people. Many foods typically viewed simply as sources of nutrients or pleasure contain constituents that are biologically active, thus blurring the once distinct line between foods and drugs. Although chocolate is not clearly established as an addictive substance, it is, by a large margin, the most commonly craved food in North America, especially among women (2-4). In fact, one classic study documented chocolate-specific cravings as constituting almost half of all food cravings (5). The study results are presented in Table 1.

Does chocolate simply have exceptional orosensory properties or are its effects more complex? Several explanations have been proposed, but no solid evidence has pinpointed exactly how chocolate induces its drug-like effects. Can chocolate be classified as a drug? Can one be addicted to a food? Many have argued that chocolate contains biologically active compounds that may have addictive properties; yet why aren't other pharmacologically related foods craved with the same intensity? What about nutritional deficiencies? Could chocolate intake be a form of self-medication to compensate for nutrients lacking in the diet, similar to the practice of ingestion of unusual dietary substances (known as pica)? Then other foods containing those nutrients should easily be substituted, but they are not. Clearly, controversy surrounds the question of whether motivations for chocolate are physiological, psychological, or pharmacologic, and no unambiguous answer has been proposed. Thus, scientists continue to tackle the issue in hopes of illuminating the basis for the potent, ubiquitous effects of chocolate.

D. L. Taren (corresponding author) is an associate professor and K. Bruinsma is a research assistant with the Arizona Prevention Center, University of Arizona, College of Medicine, 1612 E Mabel St, Tucson, AZ 85719.

Table 1
Targets of food craving^a

Food type	No. of cravings	% of total
Chocolate and chocolate-containing foods (eg, confections, cakes, puddings)	107	49
Baked goods (eg, biscuits, cakes, puddings)	24	11
"Something sweet"	35	16
Other confections	4	2
Fruit	9	4
Savory food (eg, chips, pizza, cheese toast)	26	12
Drinks (nonalcoholic)	4	2
"Anything"	10	4
Total	219	100

^aAdapted from Hill and Heaton-Brown (3) with permission.

Table 2
Main distinctive characteristics of Criollo, Forastero, and Trinitario varieties of chocolate^a

	Criollo	Forastero	Trinitario
Pod Husk			
Texture	Soft	Hard	Mostly hard
Color	Red	Green	Variable
Beans			
Average no. per pod	20-30	30 or more	30 or more
Color of cotyledons	White, ivory, or very pale purple	Pale to deep purple	Variable (white beans occur rarely)

^aAdapted from Pertwee (33).

HISTORY

Chocolate originates from Mexico where the Mayas, Incas, and Aztecs cultivated the cacao tree (*Theobroma cacao*) and touted it "a gift of the gods." Chocolate, considered an aphrodisiac, was available only for special occasions and for those with wealth and power. In 1520, chocolate, mixed with vanilla and sugar, was introduced to Europe by the early Spanish explorers. There, too, chocolate was reserved for the nobility. Eventually, because of its high cost, chocolate was displaced by coffee and tea as the predominant beverage, although it did ultimately become a favorite confection in Europe, and some time later, in North America. Although today cocoa is harvested primarily in West Africa, Indonesia, and Sri Lanka, chocolate consumption as a confection is widespread in most developed countries; Europe and the United States are the leading consumers (6).

Cocoa is produced through a process of fermenting the seeds from the pods of the cacao tree. The beans are dried, roasted, and crushed; the result is high-fat, "unsweetened chocolate." This intermediate is pressed into cakes and alkalized to form cocoa powder, which is then homogenized with sugar and cocoa butter, and sometimes milk, to ultimately form chocolate.

Although many different types of cocoa beans grow throughout the world, 3 varieties of cocoa beans are mainly used to make chocolate products; (a) Criollo (meaning "native"), distributed to the north and west of the Andes; (b) Forastero (meaning "foreign"), found mainly in the Amazon basin; and (c) Trinitario (meaning "sent from heaven") (7). These types of cocoa are separated by their distinct flavors and colors, which arise during manufacturing. Table 2 shows the unique characteristics of Criollo, Forastero, and Trinitario cocoa beans.

DEFINING CHOCOLATE INTAKE PATTERNS

This seemingly harmless substance, chocolate, has over the years maintained its noble status as a true passion and a divine substance. Not until recently did scientists begin asking why. Now scientists distinguish between chocolate use, preference, liking, and craving and have even coined the terms *chocoholic* and *chocolohism* to express the helplessness of some in avoiding this "addictive" substance (4). The term *use* refers simply to the amount consumed; the terms *preference*, *liking*, and *craving* represent different measurements of attitude toward a food.

Craving is an intense, periodic motivation aimed at gaining the craved substance. Defined in this way, chocolate cravings appear to exist in 40% of females and 15% of males, three fourths of whom claim that no other substance will appease their desire (4,5). The term *addiction* is frequently used to describe such cravings. Addiction, however, is a complex concept that includes several components, and some have questioned its applicability to chocolate cravings. Figure 1 presents the classic components of drug addiction as well as those included in the more modern definition. Although there are many definitions of addiction, 2 characteristics are consistently included: a compulsion to use a substance that results in excessive and uncontrolled consumption, and existence of withdrawal symptoms when the substance is withheld (8).

Self-identified "chocolate addicts" are not a homogeneous group in their patterns of consumption and attitudes toward chocolate. Nevertheless, the majority do tend to experience cravings, although consumption of chocolate is not always preceded by these intense urges (9). Many people eat chocolate in secret or abuse or binge on it as others might abuse other substances such as drugs or alcohol. These chocolate addicts exhibit a heightened sense of well-being during consumption;

however, they report that their drive for chocolate interferes in their lives by interrupting thoughts or actions and influencing mood state (9). For example, one subject stated, "I get irritable if I don't have chocolate. It's on my mind all the time. I can't think about other things properly" (9, p 243). Many chocoholics report that attempts at avoiding chocolate intake result in episodes of overeating with disregard for any adverse consequences, which is consistent with the relapse phase of terminating drug addiction (9). In this regard, craving can actually be identified as a component of the response to withdrawal from addictive drugs. Thus, chocolate addiction parallels other forms of addictions in several ways, namely, the experiences of salience, conflict, relapse, and relief.

Several theories have been proposed to elucidate chocolate's mechanism of action, yet few researchers agree or can prove a given hypothesis (4,9-11). Table 3 shows the pattern of results predicted by 3 of the most prominent theories explaining chocolate cravings (12). Next we review the theories proposed and conclusions drawn from these studies as well as discuss potential hormonal and neurochemical mechanisms underlying chocolate cravings.

FAT AND SUGAR

Chocolate's high hedonic ratings and unique orosensory characteristics (taste, smell, and texture) are at the heart of the most widely accepted explanation for chocolate cravings. Chocolate contains large amounts of fat (cocoa butter) and sugar; cocoa butter melts at body temperature, thereby contributing to the overall pleasurable, mouthwatering experience of chocolate ingestion (4). Because chocolate is generally consumed in an extremely sweet form, it has been suggested that chocolate preference may be confounded with preference for sugar. In fact, some studies report a high association between chocolate and sweet preference. However, 75% of all self-titled chocolate cravers claim there is no substitute when they crave chocolate (5,11,13). In a retrospective study, Rozin et al (4) demonstrated that although chocolate craving was related to craving for sweet foods, it showed partial independence of craving for sweet foods. Hill and Heaton-Brown (3) found that although chocolate cravings were moderately associated with cravings for other sweet foods, the chocolate cravings took far longer to abate. Sweet and high-fat foods are highly palatable and a preference for them appears to exist from birth (13,14). As it appears that chocolate has innate appeal, perhaps chocolate cravings need not be explained in any other way. Moreover, research has shown that animals physically dependent on ethanol prefer sweet-tasting fluids over alcohol; this finding suggests that consumption of sweets may in itself be an addictive behavior (15,16).

If sensory experience is the ultimate object of chocolate craving, then only the chocolate should satisfy the craving. Investigations involving white chocolate have been useful in delineating the extent to which the sensory qualities of chocolate contribute to cravings. White chocolate has the texture and sweetness of chocolate because it also contains cocoa butter and sugar; hence, it might be expected to effect a similar experience as chocolate. However, it does not emit chocolate's distinctive aroma and lacks substantial amounts of chocolate's many pharmacologic constituents; consequently, it would not completely satisfy a chocolate craving if the driving force of the craving is purely sensory. This has, in fact, been demonstrated by Michener and Rozin (12) in an experiment focused on isolating the sensory effects of chocolate from the potential pharmacologic effects. White chocolate produced only an intermediate effect in reducing craving—significantly less than milk chocolate, which largely alleviated craving. This finding

Classical description

- Compulsion to continue drug use. An overwhelming involvement with securing and taking a drug.
- Tendency to increase the dose as tolerance develops.
- Physical dependence on the effects of a drug.
- Development of an abstinence syndrome when the drug is no longer available.
- Occurrence of detrimental effects on the individual and on society with prolonged drug taking.

Modern description

- One extreme on a continuum of involvement with drug usage (ie, compulsion).
- Quantitative rather than qualitative measure of the degree to which drug use affects total life activity and the spectrum of circumstances under which drug use controls the user's behavior (ie, social consequences).
- Physical dependence, tolerance, and compulsive drug use, although commonly seen together, are not necessarily interrelated.

FIG 1. Components of drug addiction. Adapted from Jaffe (66).

lends credence to either a sensory effect of chocolate's aroma or a pharmacologic effect of biologically active compounds in chocolate (12).

PHARMACOLOGIC COMPONENTS

Chocolate contains many pharmacologic agents, any or all of which may evoke physiological or psychological sensations and may be the driving force behind chocolate cravings. In the past several years, the psychopharmacologic effects of chocolate have been a topic of increasing interest among nutrition neuroscientists as evidence continues to build for the localization of chocolate's actions and the precise biomolecules involved.

Biogenic Amines

Several endogenous biogenic amines, which act as sympathomimetic compounds, are found in chocolate, most notably, tyramine and phenylethylamine (PEA) (17). PEA, a neuro-modulator of brain synapses, is structurally and pharmacologically similar to catecholamines and amphetamine (12,18). On a normal basis, PEA is heterogeneously distributed within the central nervous system (CNS), and at physiological doses it may act to potentiate dopaminergic and noradrenergic neurotransmission (19). PEA is produced by brain tissue and is rapidly metabolized by monoamine oxidase- β and aldehyde dehydrogenase to phenylacetic acid, the major metabolite of PEA in the brain (18). Studies have demonstrated that PEA is pharmacologically active and that it is stimulatory when administered (18,20).

Several studies have suggested that PEA is an important modulator of mood and that a deficit may contribute to the pathogenesis of depression (18). Both PEA and its metabolite, phenylacetic acid, have been shown to be reduced in the biological tissues and fluids of depressed subjects, and replacement with PEA and/or its amino acid precursor, L-phenylalanine, appears to ameliorate some types of depression (19). Although PEA is highly lipid soluble and crosses the blood-brain barrier against a concentration gradient, biochemical evidence for high affinity and saturable binding sites for PEA in the brain has yet to be solidified (18).

PEA is found in substantial concentrations in chocolate (0.4 to 6.6 $\mu\text{g/g}$), and some experts have contended that craving for chocolate may be an attempt to self-regulate brain PEA level

Table 3
Predicted effect of various forms of chocolate on satisfying chocolate cravings^{ab}

Theory	Milk chocolate	White chocolate	Cocoa capsule	Placebo capsule	White chocolate plus cocoa capsules
Sensory^c	++	+	0	0	+
Nutritive^d	++	++	0	0	++
Energy	++	0	++	0	++
Magnesium	++	0	++	0	++
Pharmacologic^e	++	+	0	0	++

^aAdapted from Schuman et al (11) with permission.

^bExpected outcomes if the individual theory holds true. ++=full effect, +=partial effect, 0=no effect.

^cSensory: craving may simply be due to the positive chemosensory characteristics of chocolate (high fat and sugar content, attractive aroma, pleasant oral sensation).

^dNutritive: energy—dieting or energy deprivation may lead to craving; magnesium—magnesium deficiency may stimulate consumption because of chocolate's high magnesium content.

^ePharmacologic: chocolate contains many biologically active substances, any or all of which could potentially contribute to the addictive nature of chocolate.

and mood (11,12). However, several other foods, such as some cheeses and sausage, contain substantially greater quantities of PEA and tyramine, yet they are not sought after as chocolate is (12). In addition, because PEA is rapidly metabolized within the body (half-life=5 to 10 minutes), consumption of PEA does not ensure entry into systemic circulation (21,22). In fact, studies have shown that ingestion of 200 g milk chocolate containing 1 g PEA has no noticeable effect on urinary concentrations of PEA or its metabolites (11).

Relevant to PEA involvement in chocolate cravings is a study by Schifano and Magni (23), which focused on 7 persons who abused 3,4-methylenedioxymethamphetamine (MDMA, "ecstasy") (23). The authors found that each of the addicts displayed a novel psychopathologic disturbance: an intense craving for chocolate joined with episodic chocolate binges. Although few conclusions could be drawn from this retrospective study as it was merely an observation of an unexplained correlation, it is worth noting that PEA is structurally related to MDMA: both are amphetamine analogs; hence, they may have similar actions in the brain (23). This association of chocolate cravings with certain drug-induced psychoses suggests that the psychopharmacologic effects of chocolate deserve further attention.

Methylxanthines

Another group of compounds present in chocolate are the alkaloid methylxanthines, most prominently caffeine (1,3,7-trimethylxanthine) and theobromine (3,7-dimethylxanthine), both of which are stimulants and, in the case of caffeine, often cause noticeable behavioral effects. Although the methylxanthines are bases, they have a very low pK_a (0.5); hence, they are highly lipid soluble and are absorbed from the stomach as well as through the walls of the intestines, easily crossing both the blood-brain and placental barriers (23). Nehlig et al (25) have suggested that, in the brain, the methylxanthines compete with adenosine, a presynaptic inhibitory neuromodulator, and block its receptor, thereby eliminating its inhibitory action and causing arousal. Moreover, caffeine catalyzes the release of epinephrine and other catecholamines from the adrenal gland, thus contributing to its stimulatory effects (24).

Although extensive research has been conducted to establish the sympathomimetic effects of caffeine, the behavioral effects of theobromine have been more elusive. In animals, theobromine appears to have effects congruent to those of caffeine, although theobromine causes less stimulation and takes longer to induce a peak pharmacologic effect (26). It has

been postulated that because theobromine is chemically similar to caffeine, and because caffeine may have addictive properties, theobromine may be potentially addictive as well (4). A related study has shown, however, that theobromine administered in capsule form (to eliminate possible sensory effects) exerts no noticeable effects on craving symptoms (27). The lack of substantial physiological and psychological effects has led some to conclude that theobromine is behaviorally inert (27). Likewise, Rozin et al (4) have discounted the link between chocolate addiction and caffeine content as chocolate contributes relatively little to the typical daily intake of caffeine.

The quantities of methylxanthines in chocolate are variable even within a brand. A typical 1.65-oz milk chocolate bar contains only 10 mg caffeine (22 mg/200 g) and 92 mg theobromine (197 mg/100 g), a minimal amount considering that a cup of coffee usually contains 80 to 100 mg caffeine and no theobromine (10,28). Mumford et al (27) have speculated that although theobromine in the dose typically found in chocolate appears to exert only modest caffeine-like symptoms, the combined effects of theobromine and caffeine in cocoa products may differ from those of either compound alone.

Cannabinoid-like Fatty Acids

In a study enacted at the Neurosciences Institute in San Diego, Calif, a group of biologically active constituents in chocolate that appear to target the endogenous cannabinoid system of the brain were identified (29,30). Anandamide, which literally means "internal bliss," is the endogenous brain lipoprotein that binds to and activates cannabinoid receptors within the brain, mimicking the psychoactive effects of cannabinoid drugs such as heightened sensitivity and euphoria (31). Anandamide is produced and released by brain neurons and is catabolized by selective enzyme activity, which suggests that it may function as an endogenous cannabinoid neuromodulator (32). Such neuromodulators, which act primarily in the nucleus accumbens septi where the majority of cannabinoid receptors reside, increase the activity of the mesolimbic dopamine reward system by potentiating the actions of endogenous opioid peptides and possibly altering the functioning of other neurochemicals such as norepinephrine, dopamine, serotonin, acetylcholine, histamine, and prostaglandins (33). It is likely that the reinforcing effects of cannabis, and possibly chocolate, are due to alterations in this system.

Chocolate and cocoa are thought to contain the unsaturated *N*-acylethanolamines (*N*-oleoylethanolamine, *N*-linoleoylethanolamine), which are chemically and pharmacologically

related to anandamide (29). These lipids could mimic cannabinoid ligands either directly (by activating cannabinoid receptors) or indirectly (by increasing anandamide levels). Elevated brain anandamide levels could magnify the sensory properties of chocolate that are fundamental to craving. Elevated anandamide levels could also interact with other biologically active constituents of chocolate (ie, caffeine, theobromine) to induce a noticeable sense of well-being (28). Tomaso et al (29) did not detect unsaturated *N*-acylethanolamines in white chocolate or in brewed espresso (29). The researchers also found that the 2 *N*-acylethanolamines appear to interfere with the brain's ability to hydrolyze anandamide; hence, they may extend the consequent sense of well-being (29). It is possible, however, that the concentration of anandamide analogs in chocolate is insufficient to induce these neurochemical effects.

CHOCOLATE AND BEHAVIOR

Self-Medication

Some persons may use chocolate as a form of self-medication to compensate for insufficient food intake or specific nutrient deficiencies. Scientists have speculated that chocolate consumption may be motivated in some part by magnesium deficiency (10). Chocolate and cocoa powder both contain exceptionally high concentrations of this nutrient (100 mg/100 g and 520 mg/100 g, respectively), and in some cases magnesium supplements may have subdued symptoms of chocolate craving (7). Moreover, magnesium deficiency may contribute to the symptomology of premenstrual syndrome (PMS) (34). Stress stimulates secretion of mineralocorticoids and glucocorticoids, which together increase renal excretion and decrease intestinal absorption of magnesium (35,36). Magnesium deficiency results in selective depletion of CNS levels of dopamine, a neurotransmitter that transmits signals of euphoria and satisfaction and that may be the master molecule of addiction. Elevated CNS levels of serotonin unopposed by dopamine are believed to play a primary role in several PMS symptoms (37). Therefore, it is possible that stress-induced magnesium deficiency contributes to the increase in chocolate cravings associated with PMS. Nuts contain comparable amounts of this nutrient, however, and should be craved in a similar way (11). Complete energy and nutrient profiles of chocolate and cocoa powder are provided in Table 4.

Alternatively, chocolate consumption may be a behavioral mechanism for homeostatic regulation of certain neurotransmitters involved in the regulation of appetite, hunger, mood, and/or addictive behaviors. Several studies have found that negative mood states are particularly prominent among cravers and that cravers have a high tendency toward "emotional eating" (38). Low CNS levels of serotonin have been associated with depression, addiction, and obsessive-compulsive disorder, and ingestion of carbohydrates, especially chocolate, has been purported to selectively increase tryptophan uptake and serotonin production by the brain (39,40). Several animal studies have found that drugs that increase serotonin levels at postsynaptic receptors cause a reduction in carbohydrate intake (41). However, studies have also demonstrated that ingestion of even a small amount of protein with the carbohydrate will negate any neurochemical effect (42). A typical chocolate bar contains about 10% protein by weight, an amount that may be sufficient to block a rise in serum level of tryptophan and the resultant increase in CNS levels of serotonin (42). In addition, according to this hypothesis, all carbohydrates with a high glycemic index should be equally effective in promoting serotonin synthesis and elevating mood and should be targets of food cravings (43).

Table 4
Energy and nutrient composition of cocoa powder, plain chocolate, and milk chocolate^a

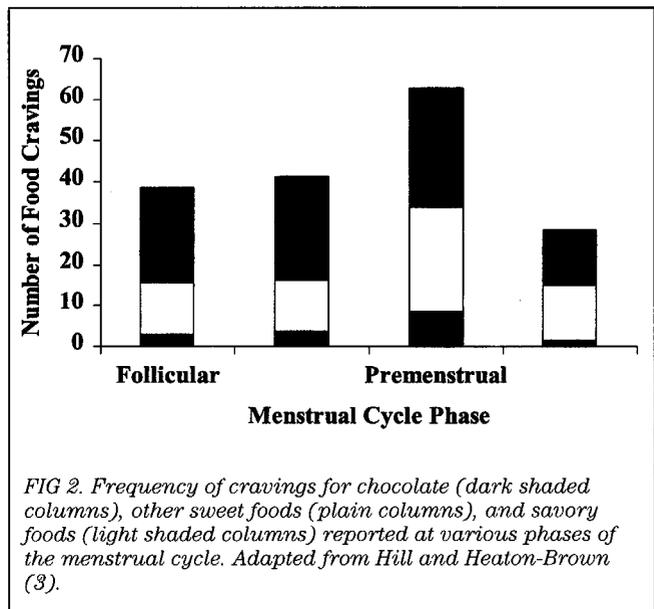
Variable	Cocoa powder ^b	Milk chocolate ^c	Plain chocolate ^c
Energy and nutrient (g per 100 g)			
Protein	18.5	8.4	4.7
Fat	21.7	30.3	29.2
Carbohydrate	11.5	59.4	64.8
Energy (kcal)	312	529	525
Element (mg per 100 g)			
Sodium	950	120	11
Potassium	1,500	420	300
Calcium	130	220	38
Magnesium	520	55	100
Iron	10.5	1.6	2.4
Copper	3.9	0.3	0.7
Phosphorus	660	240	140
Chlorine	460	270	100
Antioxidant (mg per 1 g)^d			
Total phenol gallic acid equivalents	20	5.0	...

^aAdapted from Pertwee (33) with permission.

^bMean of 10 samples of 2 brands.

^cMean of 10 samples of 1 brand. Plain chocolate is unsweetened or dark chocolate.

^dAdapted from Waterhouse et al (67).



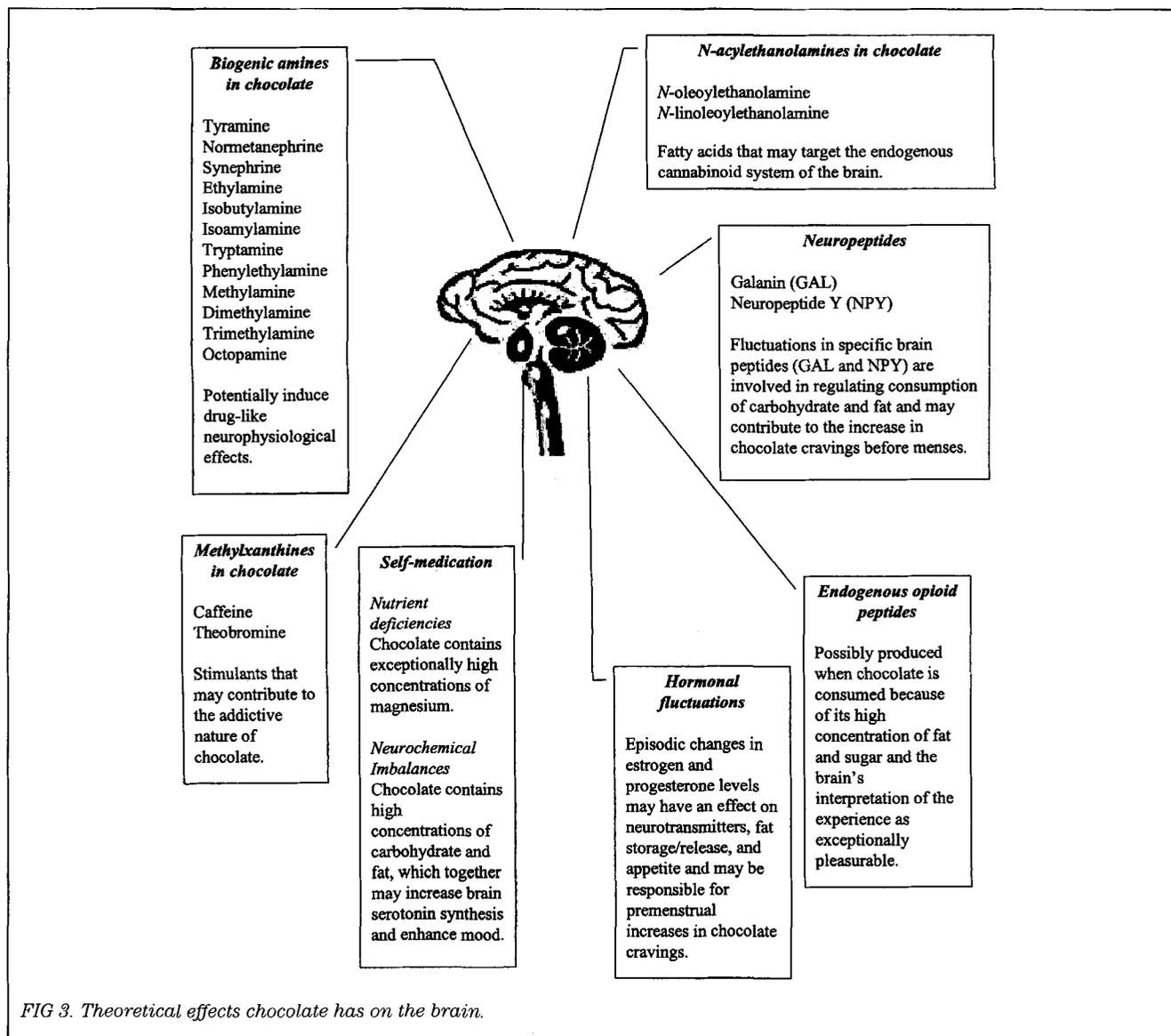


FIG 3. Theoretical effects chocolate has on the brain.

Although a few human studies have observed that self-reported cravings for sweets are, indeed, modestly correlated with carbohydrates such as bread and potatoes, most experiments report that specific chocolate cravings cannot be appeased with other high-carbohydrate foods, as might be expected with a serotonin deficiency (10). A study that focused on chocolate's role in modulating mood state in self-described chocolate addicts found no improvement in mood; rather, the chocolate appeared to induce a noticeably negative affect among the addicts compared with the control subjects, possibly because for most of these people intake of chocolate was typically an uncontrolled, guilt-ridden behavior (44). Although this finding certainly does not support the controversial serotonin hypothesis of craving of Wurtman and Wurtman (40), it is possible that among this sampling, the psychological factors of food addiction simply outweighed the neurochemical effects in the brain (43).

Hormone Mechanisms

Chocolate consumption and specific cravings among women may also be influenced by fluctuating hormone levels. Studies

of women with and without self-designated PMS have consistently demonstrated increases in the frequency and severity of cravings for pleasant-tasting, sweet, high-fat foods premenstrually (45-49), and some have suggested a link between chocolate and the menstrual cycle (48-52). In fact, an open-ended survey about food cravings administered to undergraduates documented chocolate as the most frequently mentioned craved substance among women, 32% of whom reported an association with the menstrual cycle (5). Figure 2 presents the total number of food cravings reported by menstrual phase from a related study (3).

The episodic nature of chocolate cravings supports the involvement of some internal state, most likely hormonal fluctuations, because cravings tend to be exaggerated just before the menses when estrogen levels are moderate and progesterone levels are high (4,53). Buffenstine et al (53) have suggested that the ratio of estrogen to progesterone may directly and indirectly modulate food intake (53). Progesterone induces fat storage, leading to a drop in plasma triacylglycerol level, which may trigger a concomitant increase in cravings for fatty foods (53). Estrogen, on the other hand, promotes lipolysis, which

raises plasma concentrations of free fatty acids, thereby making them available for use as a metabolic fuel and possibly inhibiting exogenous fat intake (53). Estrogen also has an influence on the central neurotransmitter systems, namely, the biogenic amine neurotransmitters norepinephrine, dopamine, and serotonin, which collectively control eating behavior and appetite regulation (54). Noradrenergic neurons induce eating, serotonergic innervation terminates eating, and dopamine modulates eating responses. Estrogen blocks the activity of dopamine β -hydroxylase, the enzyme necessary for conversion of dopamine to norepinephrine (53). Some evidence suggests that serotonin levels are low premenstrually, and it is possible that premenstrual carbohydrate cravings are the body's attempt to raise CNS concentrations of serotonin (53).

The sensory reward of chocolate, shaped by physiological state and cognitive factors, is an exceptionally potent force and appears to be the predominant factor in the phenomenon of chocolate cravings

Neurochemical Mechanisms

A separate body of research focused on brain peptides has presented a supplementary explanation for the premenstrual increase in food cravings. Two brain peptides, galanin and neuropeptide Y, are integrally involved in eating behavior, food preferences, and cyclic food cravings (55,56). Studies have shown that hypothalamic injections of neuropeptide Y in female rats stimulates daily consumption of carbohydrates and fats (57,58). Galanin also appears to increase the intake of fat and carbohydrate (59). Moreover, female animals showing a preference for fat-rich diets exhibit higher levels of galanin and neuropeptide Y in the areas of the brain responsible for stimulating feeding (60). Similar to monthly hormonal fluctuations, levels of galanin and neuropeptide vary over the menstrual cycle, both sharply increasing premenstrually (60). Likewise, there are positive correlations between progesterone and both neuropeptide Y and galanin, which suggests that progesterone may mediate the rise in these 2 peptides (60). It is likely that the findings from these studies could explain, in part, the ubiquitous premenstrual escalation of chocolate-specific cravings.

Preliminary evidence suggests that addiction for chocolate may be mediated partly by endogenous opiates, which are involved in drug addictions and are responsible for the body's response to pleasure, stress, and pain (61). A study investigating this hypothesis found that infusions into rats of the opiate antagonist naloxone diminished taste preferences for high-fat and sweet foods and selectively suppressed consumption of these foods; in contrast, rats infused with morphine, an opiate agonist, increased their fat intake (62). Likewise, several animal studies have demonstrated that CNS β -endorphins are released after ingestion of palatable foods such as chocolate, as well as cakes, pastries, and ice cream (62,63). However,

Yeomans and Gray (64) found that endogenous opioid peptides had a limited role in the determination of food pleasantness and were unrelated to the regulation of fat and protein intake, although a role in appetite was evident.

Although the evidence that food selection is based on nutritional or neurochemical imbalances is minimal and contradictory, and few studies have investigated this phenomenon in human beings, the consumption of high-fat sweet foods may fulfill a drive for some neurochemical response (61). Thus, it has not been established whether craving for sweets, specifically chocolate, represents a desire for sensory gratification or whether it involves an attempt to balance the chemistry of the brain, although an association between self-medication with sweets and dysphoric mood is apparent (10).

APPLICATIONS

The literature on chocolate is dominated by 4 general issues: the motivating chemosensory characteristics of chocolate, the desired psychopharmacologic effects of chocolate or its biologically active constituents, the theorized self-medication of nutritional or neurochemical deficiencies, and the association of chocolate cravings with hormonal variations in women (see Figure 3). Although any or all of these explanations may contribute to the occurrence of cravings, the sensory reward of chocolate, shaped by physiological state and cognitive factors, is an exceptionally potent force and appears to be the predominant factor in the phenomenon of chocolate cravings. As Roach (65) has stated, "Caviar is exquisite, but people don't declare their love with ten-pound heart-shaped boxes of it...No one makes 3:00 AM runs to the 7-Eleven for butterscotch. But chocolate...chocolate inspires a passion normally reserved for things grander than food" (p 135).

The knowledge that fat and sugar may be hazardous to health and counterproductive to weight maintenance/loss has caused chocolate consumption to become an intense conflict of interest for many. Clinicians and dietetics practitioners should be aware that chocolate cravings affect a large percentage of Americans, particularly women. Following are some specific approaches:

- Although the exact mechanism or mechanisms by which these cravings are manifested may not yet be elucidated, the cravings are real and must be evaluated in terms of overall healthful eating when assessing a client's diet and making clinical recommendations.
- Moreover, it is important to probe clients, particularly women, about chocolate intake in a nonjudgmental manner.
- Being attentive to chocolate cravings and consumption patterns is of clinical relevance when formulating recommendations designed as general guidelines and as specific guidelines for weight loss, diabetes, and mental health problems and for other individualized care/intervention plans.

References

1. Rudin RA. *The Craving Brain: The Biobalance Approach to Controlling Addictions*. New York, NY: Harper Collins; 1997.
2. Weingarten HP, Elston D. The phenomenology of food cravings. *Appetite*. 1991;15:231-246.

3. Hill AJ, Heaton-Brown L. The experience of food craving: a prospective investigation in healthy women. *J Psychosom Res*. 1994;38:801-814.
4. Rozin P, Levine E, Stoess C. Chocolate craving and liking. *Appetite*. 1991;17:199-212.
5. Weingarten HP, Elston D. Food cravings in a college population. *Appetite*. 1991;17:167-175.
6. Chocolate Manufacturers Association National Confectioners Association. US 1996 statistics. Available at: <http://www.candyusa.org>. Accessed July 30, 1998.
7. Wood GAR, Lass RA. *Cocoa*. 4th ed. New York, NY: Longman; 1985:28-35,596.
8. Roberts AJ, Koob GF. The neurobiology of addiction: an overview. *Alcohol Health Res World*. 1997;21:101-106.
9. Hetherington MM, Macdiarmid JI. "Chocolate addiction": a preliminary study of its description and its relationship to problem eating. *Appetite*. 1993;21:233-246.
10. Rodin J, Mancuso J, Granger J, Nelbach E. Food cravings in relation to body mass index, restraint and estradiol levels: a repeated measures study in healthy women. *Appetite*. 1991;17:177-185.
11. Schuman M, Gitlin MJ, Fairbanks L. Sweets, chocolate, and atypical depressive traits. *J Nerv Ment Dis*. 1987;175:491-495.
12. Michener W, Rozin P. Pharmacological versus sensory factors in the satiation of chocolate craving. *Physiol Behav*. 1994;56:419-422.
13. Drewnowski A. Energy intake and sensory properties of food. *Am J Clin Nutr*. 1995;62(suppl):1081S-1085S.
14. Desor JA, Maller O, Greene LS. Preference for sweet in humans: infants, children, and adults. In: Weiffenbach J, ed. *Taste and Development: The Genesis of Sweet Preference*. Washington, DC: US Dept of Health, Education, and Welfare; 1977. Publication No. NIH 77-1068.
15. Colombo G, Agabio R, Diaz G, Fa M, Lobina C, Reali R, Gessa GL. Sardinian alcohol-preferring rats prefer chocolate and sucrose over ethanol. *Alcohol*. 1997;14:611-615.
16. Falk J. Sweet abuse and the addiction model. In: Weiffenbach J, ed. *Taste and Development: The Genesis of Sweet Preference*. Washington, DC: US Dept of Health, Education, and Welfare; 1977. Publication No. NIH 77-1068.
17. Hurst WJ, Marin RA, Zoumas BL. Biogenic amines in chocolate—a review. *Nutr Rep Int*. 1982;26:1081-1086.
18. Sabelli HC, Javadi JI. Phenylethylamine modulation of affect: therapeutic and diagnostic implications. *J Neuropsychol*. 1995;7:6-14.
19. Paterson IA, Juorio AV, Boulton AA. 2-Phenylethylamine: a modulator of catecholamine transmission in the mammalian central nervous system. *J Neurochem*. 1990;55:1827-1837.
20. Philips SF, Robson AM. In vivo release of endogenous dopamine from the rat caudate nucleus by phenylethylamine. *Neuropharmacology*. 1983;22:1297-1301.
21. Shannon HE, Cone EJ, Yousefnejad D. Physiologic effects and plasma kinetics of β -phenyl-ethylamine and its N-methyl homolog in the dog. *J Pharmacol Exp Ther*. 1982;223:190-196.
22. Marley B, Blackwell B. Interactions of monoamine oxidase inhibitors, amines and foodstuffs. *Adv Pharmacol Chemother*. 1970;8:185-239.
23. Schifano F, Magni G. MDMA ("ecstasy") abuse: psychopathological features and craving for chocolate: a case series. *Biol Psychiatry*. 1994;36:763-767.
24. McKim WA. *Drugs and Behavior: An Introduction to Behavioral Pharmacology*. NJ: Prentice Hall; 1997.
25. Nehlig A, Daval JL, Debry G. Caffeine and the central nervous system: mechanisms of action, biochemical, metabolic and psychostimulant effects. *Brain Res Rev*. 1992;17:139-170.
26. Tarka SM. The toxicology of cocoa and methylxanthines: a review of the literature. *Crit Rev Toxicol*. 1982;9:275-312.
27. Mumford GK, Evans SM, Kaminski BJ, Preston KL, Sannerud CA, Silverman K, Griffiths RR. Discriminative stimulus and subjective effects of theobromine and caffeine in humans. *Psychopharmacology (Berl)*. 1994;115:1-8.
28. Barone JJ, Roberts HR. Caffeine consumption. *Food Chem Toxicol*. 1996;34:119-129.
29. di Tomaso E, Beltramo M, Piomelli D. Brain cannabinoids in chocolate. *Nature*. 1996;382:677-678.
30. Devane WA, Lumir H, Aviva B, Pertwee RG, Stevenson LA, Griffin G, Gibson D, Mandelbaum A, Etinger A, Mechoulam R. Isolation and structure of a brain constituent that binds to the cannabinoid receptor. *Science*. 1992;258:1946-1949.
31. Restak R. Brain by design. *The Sciences*. September-October 1993:27-33.
32. Di Marzo V, Fontana A, Cadas H, Schinelli S, Cimino G, Schwartz JC, Piomelli D. Formation and inactivation of endogenous cannabinoid anandamide in central neurons. *Nature*. 1994;372:686-691.
33. Pertwee RG. In vivo interactions between psychotropic cannabinoids and other drugs involving central and peripheral neurochemical mediators. In: Murphy L, Bartke A, eds. *Marijuana/Cannabinoids: Neurobiology and Neurophysiology*. Boca Raton, Fla: CRC Press; 1992:165-218.
34. Abraham GE, Lubran MM. Serum and red cell magnesium levels in patients with premenstrual tension. *Am J Clin Nutr*. 1981;34:2364-2366.
35. Abraham GE. The normal menstrual cycle. In: Givens JR, ed. *Endocrine Causes of Menstrual Disorders*. Chicago, Ill: Year Book Medical Publishers; 1978:15-44.
36. Rude RK, Behune JE. Renal tubular maximum for magnesium in normal, hyperparathyroid, and hypoparathyroid man. *J Clin Endocrinol Metab*. 1980;51:1425-1431.
37. Abraham GE. Premenstrual tension. In: Levanthal M, ed. *Current Problems in Obstetrics and Gynecology*. Chicago, Ill: Year Book Medical Publishers; 1980:1-48.
38. Hill AJ, Weaver CFL. Food craving, dietary restraint and mood. *Appetite*. 1991;17:187-197.
39. Wurtman RJ, Wurtman JJ. Do carbohydrates affect food intake via neurotransmitter activity? *Appetite*. 1988;11(suppl):42-47.
40. Wurtman RJ, Wurtman JJ. Carbohydrate craving, obesity and brain serotonin. *Appetite*. 1986;7(suppl):99-103.
41. Curzon G. Serotonin and appetite. *Ann N Y Acad Sci*. 1990;600:521-529.
42. Fernstrom JD. Dietary amino acids and brain function. *J Am Diet Assoc*. 1994;94:71-77.
43. Wurtman JJ. Neurotransmitter control of carbohydrate consumption. *Ann N Y Acad Sci*. 1985;443:145-151.
44. Macdiarmid JI, Hetherington MM. Mood modulation by food: an exploration of affect and cravings in "chocolate addicts". *Br J Clin Psychol*. 1995;34:129-138.
45. Bancroft J, Beckstrum T. Premenstrual syndrome. *Clin Endocrinol*. 1985;22:313-336.
46. Bancroft J, Cook A, Williamson L. Food craving, mood and the menstrual cycle. *Psychol Med*. 1988;18:855-860.
47. Abraham GE. Nutritional factors in the etiology of the premenstrual tension syndromes. *J Reprod Med*. 1993;28:446-464.
48. Cohen T, Sherwin BB, Fleming AS. Food cravings, mood and the menstrual cycle. *Horm Behav*. 1987;21:457-470.
49. Dye I, Blundell JE. Menstrual cycle and appetite control: implications for weight regulation. *Hum Reprod*. 1997;12:1142-1151.
50. Bancroft J, Rennie D. The impact of oral contraceptives on the experience of perimenstrual mood, clumsiness, food craving and other symptoms. *J Psychosom Res*. 1993;37:195-202.
51. Bowen DJ, Grunberg NE. Variations in food preference and consumption across the menstrual cycle. *Physiol Behav*. 1990;47:287-291.
52. Tomerelli R, Grunewald KK. Menstrual cycle and food cravings in young college women. *J Am Diet Assoc*. 1987;87:311-316.
53. Buffensteine R, Poppitt SD, McDevitt RM, Prentice AM. Food intake and the menstrual cycle: a retrospective analysis, with implication for appetite research. *Physiol Behav*. 1995;58:1067-1077.
54. Blundell JE. Serotonin and the biology of feeding. *Am J Clin Nutr*. 1992;55:155S-159S.
55. Leibowitz SF. Brain peptides and obesity: pharmacologic treatment. *Obes Res*. 1995;3:573S-589S.
56. Tempel DL, Leibowitz SF. Adrenal steroid receptors: interactions with brain neuropeptide systems in relation to nutrient intake and metabolism. *J Neuroendocrinol*. 1994;6:479-501.
57. Stanley BG, Daniel DR, Chin AS, Leibowitz SF. Paraventricular nucleus injections of peptide YY and neuropeptide Y preferentially enhance carbohydrate ingestion. *Peptides*. 1985;6:1205-1211.
58. Chae HJ, Hoebel BG, Tempel DL, Paredes M, Leibowitz SF. Neuropeptide Y, galanin and opiate agonists have differential effects on nutrient ingestion [abstract]. *Soc Neurosci Abstracts*. 1995;21:696.
59. Tempel DL, Leibowitz KJ, Leibowitz SF. Effects of PVN galanin on macronutrient selection. *Peptides*. 1988;9:309-314.
60. Leibowitz SF, Akabayashi A, Alexander JT, Wang J. Gonadal steroids and hypothalamic galanin and neuropeptide Y: role in eating behavior and body weight control in female rats. *Endocrinology*. 1998;139:1771-1780.
61. Drewnowski A. Fats and food acceptance. In: Gaul FE, Kosonis FN, Mackey MA, eds. *Nutrition in the 90's*. New York, NY: Marcel Dekker; 1991:25-39.
62. Drewnowski A, Krahn DD, Demitrac MA, Nairn K, Gosnell BA. Taste responses and preferences for sweet high-fat foods: evidence for opioid involvement. *Physiol Behav*. 1992;51:371-379.
63. Dum J, Gransch CH, Herz A. Activation of hypothalamic β -endorphin pools by reward induced by highly palatable food. *Pharmacol Biochem Behav*. 1983;33:119-126.
64. Yeomans MR, Gray RW. Selective effects of naltrexone on food pleasantness and intake. *Physiol Behav*. 1996;60:439-446.
65. Roach M. More reasons to love chocolate. *New Women*. February 1989;135-236.
66. Jaffe JH. Drug addiction and drug abuse. In: Spiller GA. *The Methylxanthine Beverages and Foods: Chemistry, Consumption and Health Effects*. New York, NY: Alan R. Liss; 1984:149-178.
67. Waterhouse AL, Shirley JR, Donovan JL. Antioxidants in chocolate. *Lancet*. 1996;348:834.

The authors thank Gary Wenk and Iris Bell, who provided valuable consultation throughout the development of this review study, and Cyndi Thompson, PhD, RD, for reviewing and commenting on the manuscript.