THE IMPACT OF THE ENVIRONMENT FOR SHAPING FOOD INTAKE

DOES IT CONTRIBUTE TO OBESITY?

By

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America is fast becoming an obesogenic society, defined by environments that promote increased intake of energy-rich foods and physical inactivity from sedentary lifestyles. This energy imbalance has led to a skyrocketing rate of obesity. An energy imbalance, resulting from excessive food intake could certainly be seen as a contributory factor in this obesity crisis. A close examination of food intake habits reveals a propensity for us to let external cues and signals determine the amount of food eaten rather than the internal biological signals of satiety. These external determinants can range from the lighting in the restaurant to the size of the bowl to the number of people with which you dine and most of us are oblivious to this influence.

This paper will examine the role of external cues and signals and the part they play in what we eat, the amount we eat and when we eat it. By recognizing and understanding this dependence on external cues as a guide for fullness, perhaps we can rethink the way we eat and begin to at least slow the growing rate of obesity.
INTRODUCTION

America is fast becoming an obesogenic society, defined by environments that promote increased intake of energy-rich foods and physical inactivity from sedentary lifestyles. Having a greater relative intake of energy compared to expenditure of energy has led to an energy imbalance and a skyrocketing rate of overweight and obesity. Overweight and obesity are defined as abnormal or excessive fat accumulation that presents a risk to health (Centers for Disease Control and Prevention, 2009). One measure of obesity, body mass index (BMI), is calculated by taking a person’s weight (kg) and dividing it by the square of his or her height (m). A person with a BMI equal to or more than 25 kg/m² is considered overweight. A person with a BMI of 30 kg/m² or more is considered obese and a BMI of 40 kg/m² or more has extreme obesity (World Health Organization, 2009).

Between 1980 and 2002, obesity prevalence doubled in adults aged 20 and older and overweight prevalence tripled in children and adolescents aged 6 to 19 years. The 2003-2004 National Health and Nutrition Examination Survey (NHANES) found that approximately 66% of adults and 17.1% of children and adolescents in the United States were overweight as defined by a BMI of 25kg/m² or higher. Additionally, 32.2% of adults were found to be obese (BMI of 30 kg/m² or higher). The obesity rates were even higher for non-Hispanic blacks (45.0%) and Mexican Americans (36.8%) (Ogden et al., 2006).

Overweight and obesity lead to negative metabolic effects on blood pressure, cholesterol, triglycerides and insulin resistance. The non-fatal but crippling health problems associated with obesity include respiratory difficulties, chronic musculoskeletal
problems, skin problems and infertility. The more life-threatening problems include cardiovascular disease, conditions associated with insulin resistance such as Type 2 diabetes, certain types of cancer and gallbladder disease. The likelihood of developing Type 2 diabetes and hypertension rises sharply with increasing body fatness. Approximately 85% of people with diabetes are Type 2, and of these, 90% are overweight or obese. As a result of these complications, an obese person has a 50 to 100% increased risk of premature death from all causes compared to an individual with a healthy weight and severe obesity is associated with a 12-fold increase in mortality in 25 to 35 year olds when compared to lean individuals (World Health Organization, 2009).

As a corollary to this increased incidence of health problems, data analyses have indicated that overweight and obesity-attributable medical spending accounted for 9.1% of the total annual US medical expenditures in 1998 and may have reached as high as $78.5 billion. The average increase in annual medical spending associated with obesity is 37.4 percent ($732) and for the US adult population as a whole, 5.3 percent of medical spending is attributable to obesity (Finkelstein, Fiebelkorn, & Wang, 2003).

Aside from the health and economic toll of overweight and obesity, the psychological consequences can range from lowered self-esteem to clinical depression with the rates of anxiety and depression being three to four times higher among obese individuals. Moreover, obesity creates an enormous social stigma in that negative attitudes toward the obese can lead to discrimination in many areas of their lives, including health care and employment (World Health Organization, 1998).
If this obesity crisis is the result of the energy imbalance, then at least a portion of the problem can be attributed to excessive energy intake. In fact, the average per capita daily calorie intake grew by 603 calories from 1970 to 2007 (USDA, 2009).

A natural assumption would be that we eat when we are hungry and stop when we are full. This is been found to be the case for children three years of age or younger, but around the age of four to five, even young children’s food intake becomes increasingly affected by a variety of social, cultural and environmental factors (Rolls & Engell, 2000).

Most adults conclude the reason they eat is based primarily on hunger, how much they like the food and the mood they are in. While those variables certainly contribute to the energy intake formula, that list is far from complete. Most people also like to believe they are the masters of their eating fate. It is something of which they are aware and can control. Unfortunately, research has demonstrated that often hunger has nothing to do with our propensity to overindulge and more often than not we are not aware of, much less in control of, when, what and how much we eat. Study after study demonstrates that we are influenced by friends and family, music and lighting, labels and packaging, shapes and smells and the list goes on (Wansink, 2006). When we eat may have less to do with the time since our last meal and more to do with the time it takes us to get to the candy dish (Wansink, Painter & Lee, 2006). How much we eat may have less to do with the size of our stomach and more to do with the size of our bowl (Wansink, Painter, & North, 2005). What we eat may have less to do with what we like and more to do with how convenient it is to get to it (Schachter, 1971). The average person makes more than 200 decisions about food every day, and most of them do not reach the conscience state. In one study, subjects estimated they made 15 food choices a day, when in fact they had
made more than 200 (Wansink & Sobal, 2007). This mindless behavior driven by environmental signals and cues culminates in excessive energy intake leading to overweight and obesity. This paper seeks to explore these external signals and cues and the ways in which they can negatively influence our energy intake. By recognizing and understanding this dependence on external cues as a guide for eating behavior, we can become mindful of the way we eat and begin to slow the growing rate of obesity.
CHAPTER ONE

THE PHYSIOLOGY OF FOOD INTAKE

Hunger is a feeling we know well. Each day we oscillate between the extremes of hunger and satiety. Hunger drives us to find food and eat food until we are full. We must, after all, eat to live. But aside from the environmental signals influencing our energy intake, we must also consider the physiological processes associated with hunger and eating. Research has found that energy homeostasis is based on a complex biological feedback system governed by signals that tell us when and how much to eat (Woods, 2005).

Energy intake is controlled by the complex interplay of hunger and satiety signals. These signals are generated in peripheral organs, such as the digestive tract and adipose tissue, as well as in the brain itself. Certain neurons in the hypothalamus are the targets of these signals. The fairly recent discovery of hunger and satiety signals, as well as their receptors, has generated a renewed interest in the physiology of appetite control and how this information can be used to treat eating disorders, including obesity (Erlanson-Albertsson, 2005).

Hunger signals are generated in a time of energy deficiency to stimulate energy intake. Two such identified hunger signals are neuropeptide Y (NPY) and ghrelin. The arcuate nucleus is a collection of nerve cells in the hypothalamus of the brain. Some arcuate neurons contain a substance called NPY which acts as a hunger signal. NPY is released from the hypothalamus in situations of fasting and in situations of increased
energy demand, such as exercise and lactation (Raposinho et al., 2001). Diets high in sugar and saturated fat have been found to stimulate the expression of NPY resulting in high blood sugar, elevated insulin levels and increased body weight (Kaga et al., 2001; Huang et al, 2004). Since NPY stimulates food intake, an increase in the expression of NPY by saturated fat or sugar may be one reason for palatable food inducing overconsumption (Erlanson-Albertsson, 2005).

Ghrelin, another hunger signal, is a meal initiator. Plasma ghrelin levels rise shortly before meals are initiated and fall rapidly when food is consumed. It is produced by endocrine cells in the acid-forming glands of the stomach and secreted into the bloodstream (Dornonville de la Cour et al., 2001). Ghrelin receptors are found on sensory fibers of the vagus nerve and can thus relay a signal to increase eating to the central nervous system via peripheral nerves (Date et al., 2002). The controls for the pre-meal rise in plasma ghrelin are yet to be identified; however, introduction of nutrients into the GI tract causes plasma ghrelin to decrease (Shiiya et al., 2002). While long-term ghrelin treatment has been shown to increase body weight and accumulation of fat, research on the ghrelin levels in the obese Pima Indians found that they have 33% lower fasting plasma ghrelin than lean subjects. These data seem to indicate that the downregulation of the hormone may protect against obesity and there is speculation that leptin and insulin may be involved in this action. It has been postulated that the lowered levels of plasma ghrelin observed in the obese represent a physiological adaptation to the positive energy balance associated with obesity (Tschop, Smiley, & Heiman, 2000).

Additional hunger hormones that have been identified are the orexins, which are produced in neurons of the lateral and posterior hypothalamus and are activated by
fasting (Sakurai et al, 1998), and agouti-related hormone, a neuropeptide, synthesized and secreted by NPY neurons in the arcuate nucleus, which stimulates appetite (Klebig, Wilkinson, Geisler, & Woychik, 1995).

Satiety signals inhibit the effect of hunger signals and can be mobilized for the purpose of the termination of an ongoing meal. Meal-initiated or acute-acting satiety signals mostly originate in the digestive tract and these gastrointestinal satiety hormones are released from the intestine in response to food. Cholecystokinin (CCK), the first peptide hormone to be associated with meal-induced satiety (Smith & Gibbs, 1975), is released in response to nutrients in the small intestine and acts by triggering nerve impulses which carry CCK-1 receptors to the hindbrain (Moran, Baldessarini, Salorio, Lowery, & Schwartz, 1997). CCK is thought to act as a neurotransmitter in parts of the brain, such as the nucleus of the solitary tract and the hypothalamus (Corp, Curcio, Gibbs, & Smith, 1997). CCK has been found to suppress the NPY (hunger signal) expression in the hypothalamus. Rats lacking the CCK-1 receptor have a greater elevation of NPY expression, which could explain their overeating and obesity; thus, CCK seems to be significant within the brain to regulate hunger and satiety (Bi, Ladenheim, Schwartz, & Moran, 2001). Additionally, research has shown that rats maintained on high-fat diets reduced their food intake less following CCK injection than rats that were maintained on low-fat diets. One explanation is that high-fat diets reduce the sensitivity or responsiveness of CCK-sensitive vagal sensory neurons. Another possibility is that a high-fat diet brings about metabolic or endocrine change that weakens the action of CCK. High fat diets are often accompanied by increased body fat gain and it is possible that elevated body fat brings about reduced CCK sensitivity (Covasa, Grahn, & Ritter, 2000).
Peptide YY, another meal-initiated satiety signal, is released from the gastrointestinal tract to reduce food intake. In contrast to CCK, most peptide YY secretion occurs after a meal rather than during a meal, reaching peak plasma levels 60–90 minutes following meal ingestion. It acts to inhibit NPY neurons in the arcuate nucleus and is released after eating in proportion to the calorie content of a meal. Injection of peptide YY in rats inhibits food intake and reduces weight gain. After a meal, concentrations of peptide YY inhibit food intake in both rodents and man for up to 12 hours, which suggests that peptide YY has a role in longer term regulation of food intake. This contrasts with the short-term satiety signals such as CCK, whose effects are relatively short-lived (Batterham et al., 2002). Also, contrary to CCK, peptide YY is active as a satiety signal with both high-fat and low-fat diets (Challis et al., 2004) and caused a significant decrease in cumulative 24-hour caloric intake in both lean and obese subjects (Batterham et al., 2002).

Between-meal and long-term adiposity signals constitute a complementary regulatory system that most likely acts in coordination with short-term or meal-induced satiety signals. Whereas satiety signals primarily regulate how many calories are eaten during specific meals, adiposity signals are more directly related to how much fat the body carries and maintains (Woods, 2005).

Insulin, one such adiposity signal, originates from pancreatic cells and is released in direct proportion to the amount of fat stored in the body. It was originally thought that insulin was incapable of crossing the blood-brain barrier and was without effects in the brain; however, studies have demonstrated that insulin in the brain is the result of circulation via a specialized compartment through the blood-brain barrier into the central
nervous system (Schwartz et al., 1991) As insulin is transported through the blood-brain barrier, it gains access to neurons in the hypothalamus and elsewhere in the brain to induce satiety (Woods, 2005). The flow of insulin through the blood-brain barrier is reduced after a high-fat meal, compared to a low-fat meal, leading to reduced satiety. In fact, in contrast to rats fed a regular laboratory chow meal, rats fed a lard (fat) meal showed a decreased level of hypothalamic insulin during the meal, with a 60% drop 30 minutes after the meal. Even though the lard meal was much higher in calories than the standard meal, rats would immediately consume additional chow after the lard meal, if more food was offered. The meal of fat produced no general satiation (Gerozissis, Orosco, Rouch, & Nicolaidis, 1997). It might be speculated that a high-fat diet causes the blood-brain barrier to become resistant to the penetration of insulin and the satiating effect of insulin is lost as a result (Erlanson-Albertsson, 2005).

Leptin, another adiposity signal, is a protein produced in white adipose (fatty) tissue and secreted into the blood. Leptin, named from the Greek root, leptos, meaning thin (Speakman, 2004), has emerged as a major appetite suppressor which has its own receptor with signaling properties localized to the arcuate nucleus in the hypothalamus (Tartaglia et al., 1995). The primary role of leptin is to control the expression and activity of other appetite-controlling hormones such as NYP; consequently, leptin deficiency results in elevation of the central NPY levels, leading to excessive energy intake (Erickson, Hollopeter, & Palmiter, 1996). Initial excitement about the potential of leptin as a wonder cure for obesity was short-lived when it was found that, in general, obese people have very high leptin concentrations. This would be as expected since high body fat levels should lead to high circulating leptin if their leptin producing systems were
working correctly (Considine et al., 1996). Certain obese individuals have been identified with lowered leptin production for their body fatness, and treatment with leptin has resulted in a reduction of body weight and reversal of overeating; however, this condition is extremely rare and does not account for the majority of obesity (Montague et al., 1997). Research has found that a high-fat diet brings about elevated levels of circulating leptin. However, despite increased leptin levels, animals fed a high-fat diet became obese without decreasing their caloric intake. This suggests that high levels of dietary fat changes the set point for body weight, at least in part by limiting the action of leptin, a phenomenon call “leptin resistance.” This could indicate that leptin plays no role in the defense against the high-fat diet so commonly encountered today (Frederich, Hamann, Anderson, Lollmann, Lowell, & Flier, 1995).

As has been noted, palatable, energy-dense food increases food intake by altering the expression and activity of the hunger and satiety signals, resulting in prolonged eating. Aside from this alteration of signals, palatable food has also been found to trigger “reward eating,” which is characterized by gratification rather than by energy deficit. Food craving is defined as an intense desire to eat a specific food. There are two critical aspects of this definition. One is that the desire be intense, as in something we could go out of the way for, as opposed to ordinary food choices. The second is that the desire be specific, which differentiates craving from hunger. Cravings in humans can be conditioned such that cravings for food increase in the presence of food-related cues such as the sight or smell of the craved food. Additionally, these cravings occur in the absence of need (Pelchat, 2002). This craving for food has been likened to a craving for addictive drugs, and the neural circuits affected by rewarding drugs that are presumed to be greatly
altered in addiction are the very pathways that control intake of our natural reward: food (Kelley et al., 2002).

Rewarding objects have three basic functions. In their first function, rewards bring forth approach behavior, as the object is labeled with rewarding value through learning. In their second function, rewards increase the frequency and intensity of behavior leading to such objects and they maintain learned behavior by preventing extinction. Rewards are seen as incentives and serve as goals of behavior following associations between behavioral responses and outcomes. In their third function, rewards induce subjective feelings of pleasure (hedonism) and positive emotional states (Schultz, 1998).

The nucleus accumbens (reward center) is a brain region that appears to play a crucial role in behaviors related to natural reinforcers, such as the ingestion of food and sexual behavior. Moreover, the nucleus accumbens is implicated in the rewarding and reinforcing properties of addictive drugs and is well-positioned to participate in neural control of food intake. It receives brainstem information related to taste and gastrointestinal functions through a direct input from the nucleus of the solitary tract. There are also pathways connecting the nucleus accumbens with the hypothalamus, which has been previously discussed as a center for energy homeostasis. Many of the same neurotransmitter systems are implicated in both food cravings and in cravings for drugs of abuse. Three such signals are dopamine, serotonin and opioids (Kelley et al., 2002).

Dopamine, a neurotransmitter that appears to be connected to reward eating, is involved in processing reward information and learning approach behavior. Dopamine
neurons show activation (increase dopamine release in the nucleus accumbens) after food rewards and after conditioned reward-predicting stimuli; however, only few activations follow aversive stimuli. Thus dopamine neurons label environmental stimuli with rewarding value, predict and detect rewards, and signal motivating events. Dopamine neurons are activated by rewarding events that are better than predicted, uninfluenced by events that are as good as predicted and are depressed by events that are worse than predicted. Therefore, dopamine is not only a transmitter involved with activity and reward, but also associated with the prediction of reward (Schultz, 1998). Leptin, the satiety-controlling hormone, has been found to suppress dopamine concentration and reduce the amount and duration of food intake. This suggests that leptin is involved in the dopamine regulation of feeding-induced reward functions (Krugel, Schraft, Kittnre, Kiess, & Illes, 2003).

Serotonin, a neurotransmitter well known to affect mood, may also contribute to appetite regulation. Serotonin is synthesized in the brain from the amino acid L-tryptophan, and has been implicated in the processes of within-meal satiation and post-meal satiety. Central nervous system serotonin is sensitive to circulating levels of tryptophan, certain macronutrients (fat, carbohydrate and protein) and satiety factors such as CCK. Hypothalamic serotonin receptor systems inhibit neuropeptide Y, an aforementioned stimulator of hunger and food intake (Halford & Blundell, 2000). In addition to the accepted role of serotonin in the control of feeding behavior, it has been shown that serotonin also reduces the intake of other rewarding stimuli, including drugs of abuse (Higgins & Fletcher, 2003). The notion that serotonin is significant in relieving stress has been demonstrated in experiments where carbohydrate and fat diets were fed to
laboratory rats subjected to stress. A high-fat diet was found to reduce some of the behavioral and psychological responses to stressors more effectively than a carbohydrate-based diet. Experiments on serotonin receptor sensitivity before and after social stress showed an absence of a stress-induced serotonin receptor hyposensitivity in high-fat fed rats. While this experiment demonstrates the role of serotonin in relieving stress, it also may explain the drive to eat fat-laden, highly palatable food to relieve stress (Buwalda, Blom, Koolhaas, & Dijk, 2001).

Opioid receptors are located in several neural networks in the brain, including the hypothalamus and the nucleus accumbens. It has been hypothesized that opioids mediate the affective or hedonic response to food, such as liking or food pleasure. Furthermore, the activation of opioids specifically encodes positive affect or emotion brought about by tasty and or calorie-dense foods and promotes behaviors associated with this enhanced palatability (Kelley et al., 2002). Research has demonstrated that the opioids do not impact the perception or recognition of taste, but rather the pleasure derived from tastes (Drewnowski, Krahn, Demitrack, Nairn, & Gosnell, 1992) and that the palatability of the food and not the energy content is what drives this opioid system (Apfelbaum & Mandenoff, 1981). Over the course of evolution, when food was scarce, this adaptive, hedonistic mechanism served to ensure that mammals readily consumed high-energy food when it was available; however, today the relatively unlimited availability of energy-dense, highly palatable food creates a situation that often leads to maladaptive behavior and obesity (Kelley et al, 2002).

Continuing research on the role of dopamine and opioids in the food reward system, has led some to speculate that palatable food may cause dependency, as we see in
drugs of abuse. Drug addiction results from adaptations in specific brain neurons caused by repeated exposure to a drug of abuse (Nestler & Aghajanian, 1997). Fat and sugar-laden palatable food mobilizes opioids and dopamine in the reward system, and opiates and dopamine stimulate food intake, particularly sugar and fat, thus creating a vicious cycle of the intake of palatable food causing the desire to eat more.

Aside from the taste and the content of the food, several factors increase the attractiveness of palatable food (Erlanson-Albertsson, 2005) such as whether the individual is fasted or well fed (Berridge, 1991). Intermittent feeding (Colantuoni et al., 2002) and long-term food restriction (Cabeza de Vaca & Carr, 1998) have both been shown to increase the rewarding effect of food. Therefore, it is not an unexpected finding that binge eating can be provoked by food restriction (Hagan, Holguin, Cabello, Hanscom, & Moss, 1997). Additionally, behavioral and neurochemical signs of opioid withdrawal (teeth chatter, tremors and shakes) were found in rats consuming a repeated, excessive intake of sugar followed by a 24-hour fast. This suggests that highly palatable food may be implicated as eliciting the same addictive responses as drugs of use (Colantuoni et al., 2002).

Based on the research conducted on hunger, satiety and adiposity signals, two models have emerged that have dominated the study of food intake. The depletion-repletion model suggests that the balance of immediately available energy is constantly monitored, with declining amounts triggering food intake. Therefore, a meal is initiated when available energy falls to the threshold level and ends when the levels are properly replenished. The available energy could be blood glucose, fat availability or the total energy derived from these. This model includes the signals generated during the meal that
influence the sensations of satiation or fullness, thus resulting in the decision to end the meal, as well as satiety which functions to prolong the interval until hunger reappears. Therefore, this theory could account for both the initiation and the cessation of a meal (Woods, Seeley, Porte, & Schwartz, 1998).

The second model links food intake to the amount of stored fat in the body. This lipostatic model suggests that signals commensurate with the size of fat stores become integrated with other regulators of food intake. Included in this model are hormones, whose secretions are proportional to the amount of fat in the body, and includes insulin from the pancreas as well as leptin from adipose tissue and stomach. These hormones travel via the circulatory system into the brain through the blood-brain barrier, and once inside the brain they interact with specific receptors on targeted nerve cells. As opposed to satiety signals that are secreted mainly during meals, adiposity signals act as a stimulant, providing an ongoing message to the brain proportional to body fat. When an individual’s body fat changes, the amount of insulin and leptin secreted into the blood changes in response, and this is reflected as an altered adiposity signal reaching the brain. This, in turn, interacts with the neural circuits and the net effect is a change of sensitivity of the brain to satiety signals. A majority of the literature supports the hypothesis that food intake is controlled within the lipostatic system for energy balance (Woods, 2005).

According to this lipostatic model, the onset of eating is not necessarily a factor of immediate energy needs, nor is meal cessation tied to the replenishment of exhausted reserves. Critical to this model and relevant to this paper is the notion that adiposity signals such as insulin and leptin provide, at most, a background influence, and the homeostasis they strive to maintain only subtly influences intake during any given meal.
(Woods et al., 1998). The effectiveness of these satiety signals can also be altered by an environment of readily available high-fat food so frequently encountered today. In addition, reward eating, stress and the addictive quality of some foods can alter the physiology of appetite control rendering ineffective the biological checks and balances necessary to maintain energy homeostasis.
CHAPTER TWO

FOOD ENVIRONMENT AND ENERGY INTAKE
NORM CONSUMPTION, SIZE AND VARIETY

America should probably be renamed “the land of the free and the home of the big.” Compared to the typical kitchen in Europe, the American kitchen is big, probably even huge. The cabinets are huge, the pantry is huge and the refrigerator is huge. The huge cabinets are filled with huge plates and bowls and the huge refrigerator and pantry are filled with huge amounts of food in huge boxes. This hugeness has been perpetuated by the warehouse concept of bulk buying that promotes big savings but most possibly at the expense of big customers. This bigness skews our consumption norms and leads to excessive energy intake that is documented time and time again by research. From the size of the box to the size of the plate to the size of the bag of chips, we are influenced to consume more when we are confronted with bigger (Wansink, 2006).

Research has demonstrated that highly salient, large packages or stockpiles of food encourage increased consumption. Snack intake increased significantly for both men and women as the package size increased and there was no significant reduction of intake at dinner to compensate for the increased energy from the snack (Rolls, Roe, Kral, Meengs & Wall, 2003). Consequently, as the size of the package increases, so does the volume of the product that a person uses. Even when the same amount of product was presented in different sized packages (a full half-gallon bottle of oil vs. a half-full gallon bottle of oil) more was used from the larger-sized package, indicating package size itself has an influence, independent of supply (Wansink, 1996). The salience or visibility of
stockpiled food can mediate the initial consumption of both high (ready-to-eat) and low (requires preparation) convenience foods. A high-convenience food that would be packaged is ready-to-eat popcorn, while a low-convenience food would be a bag of unpopped popcorn kernels. While stockpiling increases consumption quantity per given incidence for both high-and low-convenience foods, it has been found to increase consumption incidence for high-convenience foods. In other words, a low-convenience food once prepared will be consumed just as much, if not more per incidence as a high-convenience food; however, there will be more consumption incidences of high-convenience foods (Chandon & Wansink, 2002). It has also been shown that a larger-sized package increases consumption volume in part, because it is perceived as offering a lower price per unit than a smaller package (Wansink, 1996). Therefore, the big-box warehouse effect of lower-priced, large-size packages of highly-salient, highly-convenient food could certainly be a contributor to excessive energy intake.

Research has continued to demonstrate that salient food increases consumption. When Hershey’s kisses were placed on the desks of secretaries, the candies placed in clear jars were consumed 46% more quickly than those placed in opaque jars. Additionally, the candies in clear containers were rated by the subjects as more difficult to resist and as more attention-attracting (Wansink, Painter & Lee, 2006).

Ironically, container size has been found to influence not only the consumption of tasty foods, but also stale foods which are not particularly palatable. Consistent with similar research, movie goers who were given fresh popcorn ate 45.3% more popcorn from a large container than from a medium container. However, movie goers who were given stale popcorn in large containers ate 33.6% more than those given a medium
container, even though afterwards they rated the popcorn as “stale” or “soggy.” The
distracting environment of a movie theater coupled with a large container size persuaded
individuals to overeat even stale popcorn (Wansink & Kim, 2005).

It is not just a larger package that can encourage us to eat more; this is also the
case with a larger sandwich. While it has been demonstrated that it is especially difficult
for individuals to judge the portion size of foods with an amorphous or formless shape
such as spaghetti or macaroni and cheese (Slawson & Eck, 1997), it would seem easier to
judge and adjust for portion size of food that is served in a discrete unit, such as a deli
sandwich. Research has shown that this is not necessarily the case. Individuals tend to eat
in units. One study showed that of 92 eating opportunities (meals), subjects ate the entire
main food item 58 of the 92 times. In cookie consumption, in only one instance was a
fraction of the cookie eaten. If individuals are offered a pre-portioned or set amount of
food, they are likely to eat the entire portion (Siegel, 1957). One study demonstrated this
to be the case and documented the detrimental effects on energy intake. Male and female
subjects were offered deli sandwiches of four varying lengths (six, eight, ten and twelve
inches) for lunch once a week for four weeks. For both males and females, sandwich
intake increased significantly as the portion size of the sandwich increased. When served
the twelve inch sandwich, compared with the six inch, females consumed 31% more
energy and males consumed 56% more energy. When served the eight inch compared to
the twelve inch sandwich, females consumed 12% more energy and males 23% more.
More disturbing is that despite this increase in intake, ratings of hunger and fullness at the
end of the meal did not differ significantly when subjects were served the eight, ten and
twelve inch sandwiches. Therefore, when presented with bigger portions, the subjects
were able to override or adjust their level of hunger and satiety to accommodate increased energy intakes (Rolls, Roe, Meengs & Wall, 2004).

As evidenced by the research noted above, large packages and portions may increase consumption because they imply larger consumption norms. The size of the package or the amount of food on a plate may intrinsically suggest what might be a normal or appropriate amount to consume. Closely monitoring food intake can help to eliminate disparities between perceived and actual consumption. Unfortunately, the influence that portion size has on consumption is intensified because it can also distort one’s estimate of how much was eaten. The inability to correctly monitor how much they consume can lead people to rely more heavily on visual cues that are related to their consumption expectations. A distracting environment can also lessen a person’s ability to correctly monitor how much they consume and may lead to an over-dependence on visual cues. The convenience of such a visual cue reduces the person’s need to constantly monitor their level of satiation. Unfortunately, if that cue is inaccurate, it could inadvertently lead one to overeat. When given inaccurate visual cues, a person’s consumption estimation and perception of satiety will be more influenced by the biased visual cue than by how much they actually consume. What the eyes perceive is more influential than the physiological cues of satiety. From the size of the plate and spoon to the bottomless soup bowl, research has consistently demonstrated this phenomenon (Wansink, Painter & North, 2005).

The “Bottomless Bowl” research illustrates clearly this problem with reliance on visual cues. This study recruited subjects to eat a soup-only lunch with one of two variable conditions. While both groups were given a standard 18 ounce bowl, one group
had altered bowls that were designed to imperceptibly refill as they ate. Basically, with the help of plastic tubing under the table, the bowl would never empty. The other group ate from regular unaltered bowls. The subjects who were in the self-refilling condition ate 73% (113 calories) more soup than those eating from the normal soup bowls. Despite consuming 73% more, those subjects eating from the self-refilling bowl did not believe they consumed any more than those in the normal condition. Those eating from the normal bowls underestimated consumption by 32.3 calories; however, those eating from the self-refilling bowls believed they had eaten 140.5 calories less than they had actually eaten. When subjects were asked how they made their estimates, the common reply was they estimated how much the bowl held and then used it as the upper limit of how much they consumed. This could certainly account for the underestimation for those in the self-refilling condition. Although they ate 73% more, those eating from the bottomless bowls did not perceive themselves as feeling any more satiated then those who had eaten from the normal bowls. This study demonstrates how portion size can influence intake in two ways. First, the amount of food in the bowl provides a visual cue or consumption norm that can influence how much one expects to eat and how much one eventually consumes. Second, the amount of food in the bowl can influence consumption when its visibility lessens the extent one self-monitors consumption (Wansink, Painter & North, 2005).

While the bottomless bowl experiment is not an event that would occur in everyday life, there are many everyday objects that can alter our consumption in much the same way: by giving us inaccurate visual cues. It has been estimated that 72% of our calories come from food that we eat from bowls, plates, and glasses. This tableware can create convincing visual illusions that cause us to misjudge the amount of food they
contain. Just as the illusion of the same size black dot looking smaller when surrounded by large circles than when surrounded by small circles, the same serving of food would look smaller on a large plate than on a small one. We use background objects to judge the size of objects (Wansink, 2006). In fact, an entire diet book has been written based on this premise. Alex Bogusky (2008), the author of *The 9-inch “Diet”, Exposing the Big Conspiracy in America*, bought an older home and realized the dinner plates he owned would not fit into the cabinets. And it was not the fault of the cabinet maker. He discovered that plates have gone from an average of 8.5 inches to an average of 12 inches in the span of about 60 years and theorizes that as the plates got bigger we put more food on them. The amount of food that would have normally been served on a smaller plate would look skimpy and unfulfilling on a larger plate so we increased our serving size to fill the plate. Our mind expects to see a plate filled to a certain capacity and only that mental view would seem right regardless of the size of the plate. Bigger plates, more food and a compulsion to complete are a sure ticket to excessive energy intake. Unfortunately, it is not just plates, but also spoons, bowls and glasses that have undergone this size transformation.

Sometimes an environmental factor like a spoon or a bowl can have an impact on consumption even when the subjects are educated and informed of such nutritional pitfalls. One study set out to determine if the size of the bowl or the size of the serving spoon influenced ice-cream consumption in nutritionally-conscious grad students and professors. The subjects were invited to attend a celebration for a colleague where ice cream was served. Each subject was given either a small (17 oz.) or large (34 oz.) bowl and a small (2 oz.) or large (3 oz) serving spoon with which to dish out their ice cream.
The subjects who received the larger bowl served and ate 31% more ice cream than those
given smaller bowls. Those who were given the larger size serving spoon increased the
amount they served themselves by 14.5%. When used in combination, a large bowl and a
large serving spoon led people to serve themselves 56.8% more ice cream than those
given a smaller bowl and a smaller serving spoon. Most significant was that in a survey
taken after having scooped their ice cream, the subjects did not perceive themselves as
having served more. The larger bowls tricked the eye and gave an illusion that more ice
cream was required to make it look like a “normal” sized portion. This attests to the
pervasive nature of these environmental cues (Wansink, Van Ittersum, & Painter, 2006).

Larger serving bowls, just like the larger individual ice cream bowls, have been
shown to contribute to energy overconsumption. Graduate students at a Super Bowl party
were invited to serve themselves energy-dense snacks (nuts and pretzel/chip mix) out of
either a medium (approximately one-half gallon) or large (approximately one gallon)
bowl. Each student served themselves the snack mix onto a 10-inch plate using a 1 cup
serving scoop. Participants serving from large bowls took 53% more (146 calories) and
consumed 56% more (142 calories) than those who served themselves from medium
bowls. The size of the serving bowl may have provided an environmental cue that
essentially implied an appropriate amount to eat. Larger bowls, like larger packages or
larger portions, may suggest that a larger amount is appropriate to consume (Wansink &
Cheney, 2005). An interesting aside to this research is that these graduate students had
been intentionally exposed to a 90-minute class, consisting of a lecture and videos, a live
demonstration and small group discussion, about size-bias and how to avoid being tricked
by it, approximately a month before this experiment was conducted (Wansink, 2006).
Just as the size of the plate and the bowl encourage overconsumption, drinking glasses can create a problematic visual illusion as well. Our brains tend to focus on the height of objects at the expense of width, and tall, thin glasses tend to create an illusion very different from short, wide glasses. Research has demonstrated that the elongation (tall, thin) of glasses negatively influences how much people pour. Teenagers at a health camp were given either a tall, thin glass or a short, wide glass with the same capacity into which to pour their juice. The campers who had been given the tall, thin glasses poured about 5.5 ounces while campers who had been given the short, wide glasses poured an average of 9.6 ounces or about 74% more. More importantly, the campers with the short, wide glasses only estimated they poured about 7 ounces. This research was replicated with adults at a weekend jazz camp. Adults who were given short, wide glasses poured 19.44% more juice than those given tall, slender glasses. Just as with the children, the adults who poured into the short, wide glasses perceived themselves to have poured less than those pouring into the tall, slender glasses. The difference in over-pouring between children and adults indicate that age might reduce the elongation effect; however age does not eliminate this effect. Additionally, there was surprise shown during the debriefings (“You’re kidding!” or “Can you weigh it and show me?”) indicating the lack of awareness of this effect. Because 97% of the children and 98% of the adults in the study finished their self-poured drink, it can be concluded that short, wide glasses positively influence consumption volume (Wansink & Van Ittersum, 2003). This research could have important implications for children pouring and drinking sugary, sweet drinks. Additional research demonstrating this effect has shown that despite an average of 6 years of experience, bartenders poured 20.5 % more alcohol into short, wide glasses than
tall, slender glasses. Being told to take their time reduced, but did not eliminate the effect, indicating this illusion is a problem for even the most experienced of pourers (Wansink & Van Ittersum, 2005).

Recipes may also create problems with visual illusions and consumption norms as even the cookbook, *Joy of Cooking*, did not escape the lure of bigger. An investigation of the classic cookbook revealed that since 1936, the mean average calorie density in 18 of its recipes has increased 35.2% per serving over the past 70 years. This was found to be due mostly to the use of higher-calorie ingredients and partly to serving sizes that showed small increases in the late 1940s and early 1960s and then a 33.2% increase since 1996. While these calories and portion sizes may reflect popular tastes and norms, they may also contribute to the problem of excessive energy intake (Wansink & Payne, 2009).

Further compounding this problem with visual cues is that the same amount of food can vary greatly in caloric content. Energy density is the amount of energy (calories or joules) in a particular weight of food. It is usually expressed as the number of calories per gram of food (kcal/g). Energy density values range from 0 kcal/g to 9 kcal/g. Fat (9 kcal/g), with its high energy content, influences energy density values more than carbohydrate or protein (4 kcal/g). A food that is high in energy density (calories) provides a large amount of calories in a small weight, while a food that is low in energy density has few calories for the same amount or weight. Water lowers the energy density of foods because it adds weight but not the energy (Rolls, 2005).

A person will typically eat the same weight or volume of food over a span of days. While there will be some fluctuation, generally the weight of food that a person eats is more similar from day to day than the number of calories eaten (Rolls, 2005). Therefore,
if water is added to food, a person could eat the same weight of food with a much lower energy density and feel just as sated. To test this theory, one study served a group of women different meals consisting of either a chicken rice casserole, a chicken rice casserole with a glass of water or chicken rice soup. The casserole and the soup meals contained the same ingredients and each weighed the same. The addition of the water to the ingredients in the chicken and rice casserole to make soup increased the volume of the food and lowered the energy density. In one meal, the casserole was served with a glass of water equal to the amount added to the soup. The study found that the participants consumed 26% less energy at lunch and rated themselves fuller after consuming the soup than after either the casserole alone or the casserole served with water. Water can have a substantial impact on satiety when incorporated into a food by reducing its energy density. However, it was noted that the same amount of water consumed with a meal did not have the same impact on satiety. What looks like the same amount of food can vary greatly depending on the energy density of that food (Rolls, 1999).

This was replicated in men who were served vanilla-flavored, milk-based drinks that were identical in total energy content but varied in volume (300, 450 or 600 mL.). The volume was increased by whipping air into the drinks to give them an illusion of more without changing their energy content. The subjects were given one of the three volumes of the milk-based drinks followed by a self-selected lunch and four hours later by a self-selected dinner. Subjects ate significantly more at lunch and dinner combined after the 300 mL drink than after the 600 mL drink. Subjects also reported greater reductions in hunger and future consumption and greater increases in fullness after
consumption of the 600 mL drink than after consumption of the 300 mL drink. The eyes perceived more drink in the 600 mL glass causing the visual cues to override the energy cues (Rolls et. al., 1998).

Not just visual cues, but also cognitive factors have been found to affect satiety. Subjects were given milk-shake drinks of equal volume that were either high calorie or low calorie; however, in half of the instances, the subjects were told the opposite of what the drinks actually were. A subject was given a high-calorie drink and told it was low-calorie or a subject was given a low-calorie drink and told it was high-calorie. Twenty minutes after the drink, the subjects were given a lunch of unlimited sandwich quarters. Subjects ate significantly less and reported feeling fuller after consuming the drink designed to appear high-calorie. It appears that the belief that one has eaten more calories causes a person to feel fuller and eat less (Wooley, 1972).

Not only can visual and cognitive cues be problematic for the eater, but the effects of variety can also influence overconsumption. Satiety can be specific to certain foods which can play an important part in influencing food selection and the amount of food eaten. As a food is eaten, its taste and appearance decrease in pleasantness, but the taste of other foods remains basically unchanged. Therefore, more food is eaten during a meal consisting of a variety of foods than during a meal with just one of the foods, even if that food is a favorite (Rolls, 1985). If people are offered an assortment of 3 different flavors of yogurt, they consume an average of 23 % more than if they are offered only one flavor (Rolls, et.al., 1981). This sensory-specific satiety has been demonstrated in research and it has been found that we not only eat more when there is more variety, we also eat more if we perceive there is more variety. Just as with the biased visual cues we have seen
before, if our eyes lead us to believe we have more choices, we serve ourselves more.
Unfortunately, it does not require a meal for these effects to be seen (Wansink, 2006). 
Utilizing jelly beans that were offered either sorted (organized) or unsorted (disorganized) researchers were able to demonstrate how perceived variety can influence consumption. When 6 colors of jelly beans were offered in a segregated tray either separated by color (organized) or all mixed together (disorganized), both children and adults consumed almost twice as much from the disorganized tray. In both cases the number and flavors of jelly beans are identical; however mixing them up (more perceived variety) nearly doubled the amount a person selected and ate. An additional study increased the variety from 6 to 24 colors and found that as actual variety increased; the amount consumed more than doubled with the organized assortment but remained constant in the disorganized assortment. In further experimentation, the tray was expanded from 6 cells to 12 cells. The number of colors was held constant while doubling the size of the assortment in the tray. In this study, consumption from the small and large tray was virtually identical in the disorganized condition, while in the organized condition, consumption from the large tray was triple that of consumption from the small tray. These last two studies indicate that given a larger variety, assortment structures may cause consumers to apply consumption rules such as salient size or salient organization as a guide to consumption quantities. For organized assortments, the number of discrete categories or replications may serve as a benchmark that subjects use to gauge how many items should be taken. So when either the size of the assortment or the number of options offered were large, participants choosing from organized assortments appeared to use size as a cue to consume more. However, if the assortments were not organized, these
variables had no influence on consumption quantity (Kahn & Wansink, 2004). This research could have disastrous implications for the 100-item all-you-can-eat buffet.

Many diets requiring food restriction such as the Cabbage Soup Diet or the Grapefruit Diet have basically employed this variety theory. The success of the Atkins Diet may have been partially influenced by this notion because it eliminated most foods (particularly carbohydrates) from the diet except for meats and vegetables, and after awhile it becomes monotonous to eat just meats and vegetables. Sadly, the food industry realized there was a huge market for low-carbohydrate foods and began marketing them, providing more variety and calories for these dieters. More recently, this may account for some unsuccessful weight loss attempts on the Atkins Diet (Wansink, 2006).

Compounded by the environmental cues of big packages and inaccurate visual cues confronting us daily, it is difficult for us to keep track of how much we eat. Our bodies lack the empty/full gauges that are so conveniently located on our cars. Our satiety signals have been shown to be easily overridden. Our stomach cannot count, and our memory and attention are of little help. Many food-related decisions occur in distracting everyday environments. Once we are past the candy dish, we have no idea if we ate five pieces or ten. Once we leave the party, we have no recollection of the number of hors d’oeuvres we ate (Wansink, 2006). Exit interviews conducted with diners who were asked to estimate how much bread they had eaten underestimated their consumption by 28% (Wansink & Linder, 2003) and 12% of bread-eaters denied having eaten any bread at all (Wansink, 2006). Environmental cues can prompt us to eat but are no help in keeping track of what we have eaten.
One intrinsic problem with eating is that once the food is eaten, there is no trace of what has been consumed. We have no visual reminder as to what we have already eaten to prompt us to say “that is enough.” One group of researchers determined that there was one food, chicken wings, which left behind a visual cue, and decided to see if that visual reminder of the leftover bones could alter how much was consumed, especially in a distracting eating environment. Graduate students were invited to a Super Bowl party at a public sports bar where big screen televisions were provided for viewing. Chicken wings and soft drinks were provided free of charge. There were 21 tables and randomly assigned to each table was the condition that the table would either be cleared of the chicken bones as they were generated or that the bones would be left to pile up on the plate. In both cases the subjects were encouraged to help themselves to the chicken wings. As expected, subjects whose leftover chicken bones were not cleared from their table consumed significantly less than those whose chicken wing bones were cleared away. Those eating from the cleared table consumed 27.3% more pieces than those eating from an uncleared table. This suggests that people limit their consumption when an environmental cue, in this case evidence of food consumed, signaled how much food was eaten. Knowing how much they had eaten might have helped people better judge how much more they cared to eat (Wansink & Payne, 2007).

While an investigation of the research has shown that it is difficult for us to keep track of how much we eat, some studies have concluded that obese people are particularly poor estimators of how much they eat, with some of them underestimating the amount they eat by as much as one-half (Lansky & Brownell, 1982). Based on a principle of psychophysics that says estimations increase at a slower rate than do actual
sizes and people become more likely to underestimate objective sizes as they increase, a more recent study has shown that it may not be the size of the person, but the size of the meal that leads to calorie underestimation. The first part of this study revealed that the calorie estimations of the same meals by overweight (BMI>25) and normal weight (BMI<25) subjects are indistinguishable and similarly influenced by the size of the meal. The estimations of small meals tend to be on average accurate while those of medium and large meals are well below the actual number. In a field study to further document this, interviewers found that respondents in a food court estimated that, on average, larger meals contained 254 more calories than smaller meals, when in reality, they contained 660 more calories. The second part of this study helped to clarify the results that had been reported in previous studies of obese eaters. When the subjects were allowed to self-select their meal and then estimate the calories in that meal, participants with a high BMI consistently chose larger meals than participants with a low BMI. This, as would be expected, led to an underestimation of the calories by the high BMI subjects, not because they were obese, but because they chose larger meals. And unfortunately, practice does not make perfect as even professional dieticians underestimated calories in larger meals. While more accurate than the other consumers (for a 1000 calorie meal, the dieticians’ mean estimate was 857 calories, whereas regular consumers’ was only 660 calories) this does demonstrate that even with training, these biases are difficult to eliminate. Also disturbing is that it was found that dieticians expected high-BMI people to underestimate meal size compared with low-BMI people. This could hold important implications for the clinical treatment of obesity (Chandon & Wansink, 2007).
This research, as all the other research we have investigated, indicates that at every turn bigger is causing problems for the American diet. Consumption norms are skewed by bigger packages, more food is filling larger plates and bowls, the variety of food available is at an all-time high and our estimation of bigger meals is difficult at best. We seem to be engulfed in an environmental perfect storm of energy overconsumption.
CHAPTER THREE

EATING ENVIRONMENT AND ENERGY INTAKE
EFFORT, AMBIANCE AND OTHERS

While it is apparent there is some physiology that prompts us to begin and end eating, if you asked someone to tell you why they did so, their answers might have less to do with biology and more to do with their surroundings. They might say they decided to eat because they happen to spot the Golden Arches or they smelled the Cinnabons as they walked through the food court, or they ordered a dessert because the ambiance in the restaurant was so pleasant. They might say they stopped eating because their favorite TV show ended or the friends they were dining with finished eating. We are surrounded every day by people, places and things that influence our eating decisions and often, just as we were not conscious of the influence of size and variety, we are not conscious of the influence of this ubiquitous eating environment that surrounds us (Wansink, 2004).

In a country where a popular retailer can market an “easy-button” to demonstrate the convenience of its products, it is not difficult to imagine that mind-set might spill over into other aspects of our lives. And when it comes to food, we make no exception. The ease, access or convenience with which a food can be consumed has a strong influence on its consumption. The more effort it takes to eat a food, the less we eat. While this has been shown to be true for most people, it may be more pronounced for people who are obese (Wansink, 2004). One such study, under the guise of a personality test, explored this theory. Each subject was shown to an exam room where the examiner explained he had to leave the room for a few minutes, but told the subject to help themselves to some
nuts (almonds) while he was gone. In one condition, the almonds were shelled; in the other, they were unshelled (A nutcracker was available on the desk.) For normal weight subjects, when the nuts had shells, 50% of them ate; when the nuts had no shells, 55% ate nuts. The results for the obese were very different. When the nuts had shells and required the use of a nutcracker, only one of the 20 subjects ate. However, when the nuts had no shells, 19 of the 20 ate nuts. The amount of effort required made a huge difference in the amount of nuts eaten by the obese subjects (Schachter & Friedman, 1974). In a related study, researchers surmised that eating with chopsticks requires more effort for most Western eaters; therefore, they decided to observe patrons at a Chinese restaurant to see who was eating with chopsticks. They categorized each patron as normal, chubby or obese and then observed whether they ate with chopsticks or silverware. Observing patrons in 16 different Asian restaurants, the researchers found that approximately 30% of normal-sized Western eaters used chopsticks to eat their meal while only about 2.5% of those considered chubby or obese used the chopsticks. When it comes to effort and food, the obese will choose the quickest and most efficient means of eating (Schachter, Friedman, & Handler, 1974).

The effect of effort on consumption by the obese was further edified in a study which required subjects to pull on a weighted ring with their index finger for a specified length of time in order to earn portions of a sandwich. This “work” was done either in the presence of a sample sandwich wrapped in transparent wrap or a sandwich wrapped in white non-transparent paper. Food salience had a marked effect on the obese; they worked harder to obtain food when it was prominent than when it was remote. In contrast, the performance of normal subjects was relatively unaffected by this food prominence.
While the effort required to obtain food may stop the obese in some cases, the salience of food cues may override this effect (Johnson, 1974).

Other studies have shown that the effort to obtain food influences all of us to some degree. High-calorie and low-calorie desserts were rotated to either the front (more accessible) or back (less accessible) of the display in a cafeteria. Subjects were more likely to select the dessert in the front and the percentages did not differ for obese, overweight or normal-weight subjects (Meyers, Stunkard, & Coll, 1980). Another study placed a candy dish in one of three locations in an office setting to measure convenience on the consumption of chocolate candy. In the first condition, the candy dish was placed on the top of the worker’s desk, where it was visible and convenient. In the second condition, the dish was placed in the worker’s desk drawer, where it was convenient but not visible. In the third condition, the container was visible but inconveniently placed on a shelf approximately six feet from the desk so that the worker had to leave the desk to get to the candy. Consistent with other research, the visibility and the convenience of the chocolates significantly contributed to how many chocolates were consumed. Additionally, convenience contributed more to overeating than visibility; having to walk six feet to get to the visible candy led the subjects to eat fewer chocolates than if they were conveniently, but not visibly located in the desk drawer. But an interesting result emerged; there was an underestimation of consumption for candy that was inconveniently located six feet away from the desk by 63%. People appear to take the visibility and convenience of a food into account when they estimate their prior consumption of it. Therefore, a food that is inconvenient to consume may be eaten in greater amounts than is thought or recalled. So while high-convenience foods cause us to eat more, low-
convenience foods cause us to underestimate our consumption of this food which could once again lead to an excessive energy intake (Painter, Wansink & Hieggelke, 2002). Another study on the convenience of food and energy consumption employed a food most everyone loves, ice cream, and an inconvenience as trivial as opening a glass lid. Ice cream was stored in a container with a glass lid in a cafeteria. In the experiment, part of the time the lid was open and part of the time the lid was closed. True to the other research, it was found that if the lid was closed, only 14% of the patrons decided it was worth the effort to open it. However, if the lid was open, 30% decided to have ice cream. The convenience of having the lid open was one less obstacle to keep the patrons from taking the time to decide if they really needed the ice cream or not (Meyers et al., 1980).

Is it any wonder there is a preponderance of highly convenient, pre-packaged energy-dense food on every aisle of the grocery store and a fast-food restaurant on every corner?

While we may frequent a convenient restaurant more often, it may be that once inside there are other things that cause us to linger and consume more. The temperature, lighting, odor and noise are all part of the environment of the restaurant and it may be that they are deliberately designed to keep us there and eating and drinking more while we are there (Wansink, 2004).

Food intake bears a relationship to environmental temperature. At a high temperature where loss of heat is difficult, food intake should be low to prevent the body from acquiring more heat than it can dispose of during the eating and digestion process. At a low temperature, food intake should be high to help the body generate extra heat to prevent hypothermia. Rats exposed to an environmental temperature of 72° F. ate 30% more than those exposed to an environmental temperature of 94° F. During exposure to
cold where heat was needed, food intake was high, while during exposure to heat food intake was low (Brobeck, 1948). This information might certainly make it more profitable for the restaurant to keep it cool and keep us eating.

The influence of odors on food intake is complex. Odors can be in the environment or directly related to the presence of food. Odor can act as a potent retrieval cue for recent events or even situations that happened in the past. Olfactory experiences are remembered in the form of highly distinctive traces and remain cogent cues that are fairly impervious to forgetting (Richardson & Zucco, 1989). Perhaps that is why the smell of the cookies your grandmother always made is a cue to eat that can satisfy not only a physical hunger, but also revive positive memories of a more simple, unencumbered time. While pleasant food odors have not been directly associated with increased consumption volume, unpleasant ambient food odors are likely to shorten the duration of a meal and diminish food consumption (Wansink, 2004). Olfactory sensory-specific satiety has been demonstrated in humans. Subjects were asked to smell a food (banana) for 5 minutes and then rate the pleasantness of the smell. The pleasantness of the smell of banana decreased significantly when it was smelled for 5 minutes but other foods did not show decreases in pleasantness. Smelling one food to satiety can decrease its pleasantness, as well as decrease the pleasantness of similar foods; however this can also increase the pleasantness of dissimilar food. This would indicate that a variety of odors in a meal or in a restaurant could perhaps contribute to increased food intake (Rolls & Rolls, 1997).

The lighting in a restaurant can influence consumption. In bright illumination people tend to eat food more quickly, whereas with soft, dim lighting, they are more
likely to take their time eating. Dimmed or muted lighting appears to influence intake in two different ways: by increasing eating time and by increasing comfort and lowering inhibition. Restaurant designers try to take advantage of this. In fast-food restaurants where the profit on each item is small and a quick turnover is desirable, bright light and basic furnishings are used. An atmosphere of soft illumination is often used where the margin of profit is greater and a quick turnover is not desirable (Lyman, 1989). Both instances can promote excessive energy intake. When the lights are bright and we eat more quickly, our mouths get ahead of our satiety signals and we can often overeat before we feel full. When the lights are soft and we are more comfortable, we may linger longer and order an unplanned dessert or an extra drink (Wansink, 2004).

One highly controllable environmental factor in a restaurant is music, and different variations in music have been shown to have varying influences on consumption. In a study to examine the effect of the tempo of background music on the behavior of restaurant customers, it was found that once the food was served, customers exposed to a slow-music condition took significantly more time to complete their dinners (56 minutes) and leave than those exposed to a fast-music condition (45 minutes). The tempo of the background music modified the dining speed of the patrons. Most significant was that the slower background music encouraged customers to drink an average of 3.04 more drinks per customer group. The average gross margin per itemized statement was $7.20 more for the slow-music group (Milliman, 1986). This study was replicated using not only tempo as a variable, but also music preference. In this study, the customers in the slow-music condition spent, on average, 15.03 minutes longer and spent 23% more in the restaurant than individual dining in the fast-music condition. Additionally, respondents were asked
to rate how much they like the music played in the restaurant and it was found that musical preference was also highly correlated with total spending (Caldwell & Hibbert, 2002). A diary intake study of college students reported the mean meal size increased 447 calories (17%) in meals eaten with music versus meals eaten without music. There was also an increase in the mean amount of beverage consumed with music (27%) and also in the mean amount of fat consumed with music (24%). While subjects in this study reported they listened to music while eating at home, in the car and in restaurants, there was a tendency to consume more calories, and fat in particular, when music was played in a restaurant (Stroebele & de Castro, 2006).

Most all chain restaurants maintain consistency in the above mentioned atmospherics, and the façade and menu are generally identical regardless of their location. Perhaps this consistency is intentional and has the potential to influence consumption. Research has shown that rats who are fed a specific food in a conditioned environment over a period of time, will, when placed in the conditioned environment, consume more of that food than control rats (non-conditioned) even when sated. After a few pairings with food consumption, the feeding environment acquired conditioned motivational properties that allowed it to stimulate eating, even when the rats were full. The uniform look and consistent menu selections of chain restaurants, particularly fast-food restaurants, could provide an opportunity for specific food context pairings leading to overconsumption even when hunger is not really the issue (Petrovich, Ross, Gallagher, & Holland, 2007). Perhaps, similar to Pavlov’s dogs, we have been conditioned to salivate when we see the Taco Bell even when it is not dinner time.
The presence of others is an environmental factor that has been found to influence consumption in unique ways. The complexities of human psychology seem to alter one’s eating in the presence of others. The size of the group, the size of the person and the relationship and sex of the persons eating can impact consumption in varied and sometimes even opposite ways (Wansink, 2004).

Bandura’s (1986) social cognitive theory postulates that the overt behaviors of others represent important sources of social influence. Most human behavior is learned through modeling and observing others, and from this one forms an idea of how one should behave. Research has demonstrated that when it comes to social eating, this may clearly be the case. One laboratory study explored this by varying the amount of crackers an unknown social model ate in the presence of a subject. The variables were sex of the model, sex of the subject, weight of the subject and amount eaten by the model. There was also a model absent condition. The subject was brought into a room with another subject (the model) where they were instructed they would be filling out a questionnaire about taste perception. There was a heaping bowl of crackers on the table and they were told they could eat as many as they wanted but they needed to eat at least one cracker so they could answer the questionnaire. The model was instructed to eat either a high or low amount or no crackers at all. For all groups, it was found that cracker consumption of the subject increased with increases in the model’s consumption. Both males and females ate more when the model ate than when the model did not eat. Subjects ate more when the model was of the same sex than when the model was of the opposite sex. Obese females ate less than normal weight females, but overweight males did not differ. Normal weight females ate more than in the low-eat and no-eat model conditions with little difference in
the no model condition; however overweight women ate significantly less in all conditions of the model present condition and more in the model absent condition. While all subjects clearly ate more when the model was eating, the presence of a nonconsuming social model in an eating context inhibited the eating responses of both overweight and normal weight subjects and it seems to be most inhibiting for overweight women (Conger, Conger, Costanzo, Wright, & Matter, 1980).

In the modeling situation above, the intake of the unknown model is predetermined by the researcher; there is no opportunity for the mutual influence of eating that may occur among freely eating individuals. Often our social events involve the sharing of food with family and friends. When we are in the presence of family and friends we are generally more relaxed, we may lose track of how much we are eating or as we saw above, we can let the amount others eat influence our consumption (Wansink, 2006). Several studies have sought to document this social facilitation of eating through observation, laboratory experiments and food diaries. The effect of group size and acquaintance on consumption was investigated in a laboratory setting by serving dinner to female subjects alone, in pairs or in groups of four. The subjects in both pairs and groups of four ate more than did subjects alone. Subjects who ate with friends ate more dessert than subjects with strangers, indicating that the relationship of dining companions is an important factor contributing to the social facilitation of consumption. We eat more with others and are less inhibited when eating with our friends (Clendenen, Herman, & Polivy, 1994).

Another study observed customers in three different types of lunch settings: a workplace cafeteria, a fast-food restaurant and a moderately priced restaurant. In each of
these settings, observers noted the time of the initiation of the meal, the time at which the
table members left and the number of people per table. There was a significant
correlation between group size and meal duration for all settings indicating that an
increase in the number of people present for an eating occasion causes an increase in its
duration. There is evidence that a longer meal is a larger meal (Bell & Pliner, 2003).

Another study asked subjects to keep a 7-day food diary of everything they ate or
drank, the time of the occurrence, self-rated hunger, anxiety and elation, the number of
other people present and their gender and relationship to the subject. This study found
that meals eaten with other people were larger and longer in duration compared to meals
eaten alone regardless of the relationship of the eating partner to the subject. However,
meals eaten with spouse, family or friend were significantly larger (22%, 23% and 14%,
respectively) than meals eaten with co-workers (16%). This disinhibition model predicts
that the better known the companion, the greater the relaxation and thus the greater
likelihood of increased intake. Additionally, meals eaten with spouse and family were
larger and eaten faster, while meals eaten with friends were larger and of longer duration.
This time extension model predicts that the better known the companion, the greater the
likelihood of socialization and conversation, thus lengthening the meal and promoting
when eating with others is an orderly phenomenon that can be measured with the
correlation between the number of people present and the amount eaten in a meal. Meals
eaten with one other person present were 33% larger than meals eaten alone and 47%,
58%, 69%, 70%, 72% and 96 % increases were associated with two, three, four, five, six,
and seven or more people present, respectively.
Just as the ambiance of the restaurant and social facilitation of others can influence our energy intake, distractions such as reading, listening to the radio or watching television can increase consumption by initiating, obscuring and prolonging consumption. Distractions can set in motion script-related patterns of food consumption that are unrelated to hunger. They can cloud one’s ability to monitor consumption and they can extend the time of a meal (Wansink, 2004).

In a self-monitoring diary study, obese subjects were asked to indicate the main reason for starting and stopping an eating episode. While hunger was rarely chosen as a reason to start eating (the environmental cue of mealtime was the main reason), some of the subjects indicated they stopped eating simply because a television program had ended or because they finished reading a magazine (Toumisto, Toumisto, Hetherington, & Lappalainen, 1998). While a portion of the overconsumption associated with distractions such as television or magazines can be a function of longer meals, this overconsumption may also be attributed to the fact that the distraction can obscure one’s ability to accurately monitor how much one has eaten. In one controlled study, female subjects ate in one of four conditions: condition 1, subjects ate alone; condition 2, subjects ate alone while listening to recorded instructions focusing on the sensory characteristics of the food creating an attention condition; condition 3, subjects ate alone while listening to a recorded detective story creating a distraction condition; and condition 4, a group of 4 subjects ate lunch together. The subjects who ate lunch in the distracted condition ate 301 (15%) more calories than the baseline condition. The attention and the group condition ate 54 (2%) and 63 (3%) more calories, respectively, than the baseline condition (Bellisle & Dalix, 2001). Another study of lean women indicated that distractions may redirect
attention to the point where oral sensory satiety signals are ignored (Poothullil, 2002). This has been corroborated in a study of free popcorn eaten at a movie theater. When the subjects reported to paying more attention to the movie and less attention to the amount of popcorn eaten, they ate more popcorn (Wansink & Park, 2001). To further implicate distractions and their role in overconsumption, it may be the case that distractions initiate consumption because they lead people to associate the distraction with food, such as popcorn at the movies, peanuts at the baseball game or chips in front of the TV. Regardless of hunger, we eat during a particular distraction simply because we have been conditioned to eat in that environment (Wansink, 2004). In fact, one study in which the participants kept a 7-day food diary of what they ate, the location of the meal, the time it was eaten, how hungry they were and whether the television was on, found that the participants ate more on the days in which the television was on, to the tune of almost one additional meal. The amount eaten was related to the time spent with the television on. Additionally, the participants recorded less hunger before meals on the eating-with-television days. People may choose to snack in these distracting environments because eating is part of a habitual consumption script and not because they are actually hungry (Stroebele & de Castro, 2004). Even something as simple as a clock can be a distraction. Time manipulation studies have demonstrated that even if they are not physically hungry, just thinking it is time to eat is enough to cause some people to eat. The altered time on a clock is a more powerful motivator than the internal cues to eat (Schachter & Gross, 1968).

The problem of distraction is but one of the perils of television viewing. Television programming is inundated with advertisements and depending on the time of
day and the audience, many of them are food advertisements. Food advertising seems to be particularly aimed at children. Every day, children view on average 15 food ads, and the leading categories of food advertising seen by children include fast food, sweetened cereal, desserts and sweets, snacks and sweetened drinks. Unfortunately, these products are generally high in fat, sugar and/or sodium (Federal Trade Commission, 2007) and the products in children’s food advertising were most often associated with fun and good times, being hip or cool and feelings of happiness (Folta, Goldberg, Economos, Bell, & Meltzer, 2006). Adults are also victims of this massive food advertising campaign. It has been suggested that the effect of food advertising on eating may occur outside of the person’s awareness. One experimental study proposed that television food advertising embedded within a television program will activate an automatic increase in snack food consumption in both children and adults. True to the hypothesis, children who saw a cartoon with food advertising ate 45% more goldfish crackers while watching than did children who saw nonfood advertising. (Goldfish crackers were not advertised during the viewing) These children ate 8.8 grams more during the 14 minutes they watched television and at that rate, snacking while watching commercial television with food advertisements for only 30 minutes per day would lead to 94 additional calories consumed for a weight gain of almost 10 pounds per year. A variation of this study examined the effect of exposure to food advertising on adults who were not provided with a snack while watching television, but afterwards. During the course of a television show, subjects saw either food ads with a snacking message that emphasized fun and excitement or a food ad with a nutrition message or a nonfood ad. Afterward they were asked to participate in a second experiment to test consumer products where they would
taste and rate snack foods that varied in perceived nutritional value. In this second part of
the experiment, subjects who saw snack ads during the television show consumed
significantly more than those who viewed the nutrition ads or the control ads. Additionally, consumers who saw snack ads ate for the longest amount of time. These experiments provide evidence of a causal link between food advertising and snack consumption. Additionally, the adult study revealed that it may not be possible to avoid influence simply by not snacking while watching television; television viewing could also lead to increased consumption during a subsequent snack or meal (Harris, Bargh, & Brownell, 2009).

The eating environment can create for us an atmosphere of consumption perils of
which we may be totally unaware. Using some of the same research investigated here, the restaurants know what brings us there, keeps us there and keeps us eating more. They know what we like to see when we come in and what might prompt us to stay longer. They adjust the temperature, the lighting and the music so that we are primed for the best possible consumption experience, for after all they want us to come back. And all of this has nothing at all to do with the food. The presence of others can have conflicting effects on consumption; however, the general consensus is that we eat more when we eat with others, especially others with whom we are most comfortable like friends and family. And that is exactly who we eat with the most. The distractions provided by television, radio, books and magazines may lead us to eat or keep us eating long past the point of satiety and the food advertisements on television may subtly influence us to snack even when we are no longer watching. We are surrounded at every turn by an eating environment that encourages energy overconsumption.
CONCLUSION

We are becoming a nation of obese people; not just obese, but in many cases morbidly obese. Our body has physiologic and metabolic mechanisms in place to regulate energy overconsumption, but they seem to be easily overridden by the energy-dense, highly palatable food we encounter daily. Compounding the problem is a food and eating environment that tricks and deludes us into eating more without our being conscious of doing so. We have mindlessly become a nation of overeaters, resulting in obesity of epidemic proportions.

In *The Tipping Point*, Malcolm Gladwell (2002) proposes a theory he describes as the “biography of an idea” and uses this theory to explain such phenomena as the emergence of a fashion trend, the rise and fall of crime in certain cities and spread of messages in our society. He postulates that certain ideas, products and messages spread just like viruses and are best understood as epidemics. He further postulates that there is one moment when an idea, trend or social behavior tips and begins to spread like wildfire and he identifies certain factors that contribute to this “tipping point.” While his theory can be utilized to successfully market products and manage social groups, it seems tailor-made to explain this obesity epidemic and the research we have examined.

Gladwell (2002) mentions several characteristics that can cause something to “tip.” One characteristic is that little causes can have big effects. We saw this repeatedly in the obesity research. Placing the candy bowl on the worker’s desk, the shift from 9-inch to 12-inch plates or the addition of another person while we are eating, each seems small in and of itself, but increased energy intake as a result of each of these can add up.
A few calories here and there add up to pounds over the course of a year. Gladwell mentions another characteristic: contagiousness. The eating of highly palatable, energy-dense food has shown that it can be physically “contagious,” but more importantly, poor eating habits can be spread to friends and family, as a form of social contagiousness. “Eating out” and “snacking” are also examples of contagious eating behavior that can contribute to energy overconsumption. The two characteristics above lead to the third, which is that epidemics can tip in one “dramatic moment.” And perhaps that is the biggest problem for energy overconsumption; the small environmental changes coupled with contagiousness tipped the scales, literally, to an obesity epidemic. We awoke one day to find an environment chock-full of eating perils. Increased variety, larger consumption norms, more convenient highly palatable food, bigger plates, cups and bowls, more and more food advertising, and more eating distractions have caused a “tipping point” of huge proportions.

Gladwell (2002), in his theory, points out the “power of context” which says that human beings are much more sensitive to their environment than they may seem, and much of our behavior can be explained by the environment in which we find ourselves. This corroborates the research we have seen. Time and time again the environment controls when, what and how much we eat without our conscious attention. This, however, is the good news. Environmental tipping points are things we can change. Once we can see and identify the specific and relatively small elements in the environment that can serve as tipping points for each of us, we can begin to change: we can move the candy dish, we can buy smaller packages, we can choose the smaller plates and we can begin to conquer our problem with energy overconsumption.
REFERENCES


VITA

Sharon Edwards Payne was born September 23, 1957 in High Point, North Carolina. She graduated from the University of North Carolina at Chapel Hill, Phi Beta Kappa with a Bachelor of Arts in Psychology in 1979. After college she moved to Asheboro, North Carolina and began working for Union Carbide Corporation until the birth of her first child in 1984. She spent nineteen years enjoying her children and volunteering in various school, church and civic activities. In 2003, she returned to work full-time at Wake Forest University. She began work on her Master of Arts in Liberal Studies in 2006 where she was able to pursue her interest in diet and health.