TOWARDS A BIOCULTURAL APPROACH: A REVIEW OF FOOD INSECURITY, LOW INCOME AND OBESITY IN THE UNITED STATES

by

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(Under the Direction of Susan Tanner)

ABSTRACT

Currently the United States and other industrialized nations are experiencing the stark impact of a global nutrition transition through a radical and national increase in obesity rates. The increasing presentation of this disease, however, is concentrated in food insecure, and low-income populations. Research that has examined the etiology of this correlation has approached this relationship from either a biological approach, or an economic approach. The literature presented by both camps, however, is unable to fully elucidate the presentation of obesity in these populations. Instead this review argues for the integration of these perspectives in a biocultural approach that addresses how structural inequalities work in conjunction with human biological processes, and cultural coping mechanisms to affect national obesity distributions.

INDEX WORDS: NUTRITION TRANSITION, FOOD INSECURITY, OBESITY, LEPTIN

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DEDICATION

This thesis is dedicated to my family who has always supported me, with special thanks to my grandmother Louisa Mendoza.

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Chapter 1: Introduction

The United States and other industrialized nations are currently experiencing the stark impact of a post-World War II nutrition transition through a national and radical increase in obesity rates (Table 1). In the United States national obesity rates (BMI $\geq$ 30) increased dramatically from 12% in 1978 to 20% in 1990 (Swinburn *et. al.* 2004: 124). This national increase in obesity rates is situated within a larger global nutrition transition and its associated patterns of food production and consumption. Human diet, activity patterns, and nutritional status are often delineated into five shifts (collecting foods, famine, receding famine, nutrition-related noncommunicable diseases, and behavioral changes), each defined by broad periods of food use and corresponding nutrition-related disease. The nutrition transition is synonymous with the transition from the third period, receding famine, to the fourth period, nutrition-related noncommunicable disease.

During the nutrition transition, industrial societies converged on diets high in total fat, cholesterol, sugar, refined carbohydrates, and low in polyunsaturated fatty acids and fiber. These dietary changes were accompanied by a reduction in traditional labor-intensive activities (Drewnowski 2000: 486, Popkin and Gordon-Larsen 2004: S2, Swinburn *et al.* 2004: S3). Overall, the shifts associated with the global nutrition-transition have intensified the presentation of diet-related non-communicable diseases in contemporary industrial communities (Popkin and Gordon-Larsen 2004: S2). "Underlying these trends are the major and rapid shifts in diets of the developing world,

particularly with respect to the greater intake of fat, caloric sweeteners, and added saturated fats" (Popkin and Gordon-Larsen 2004: S8).

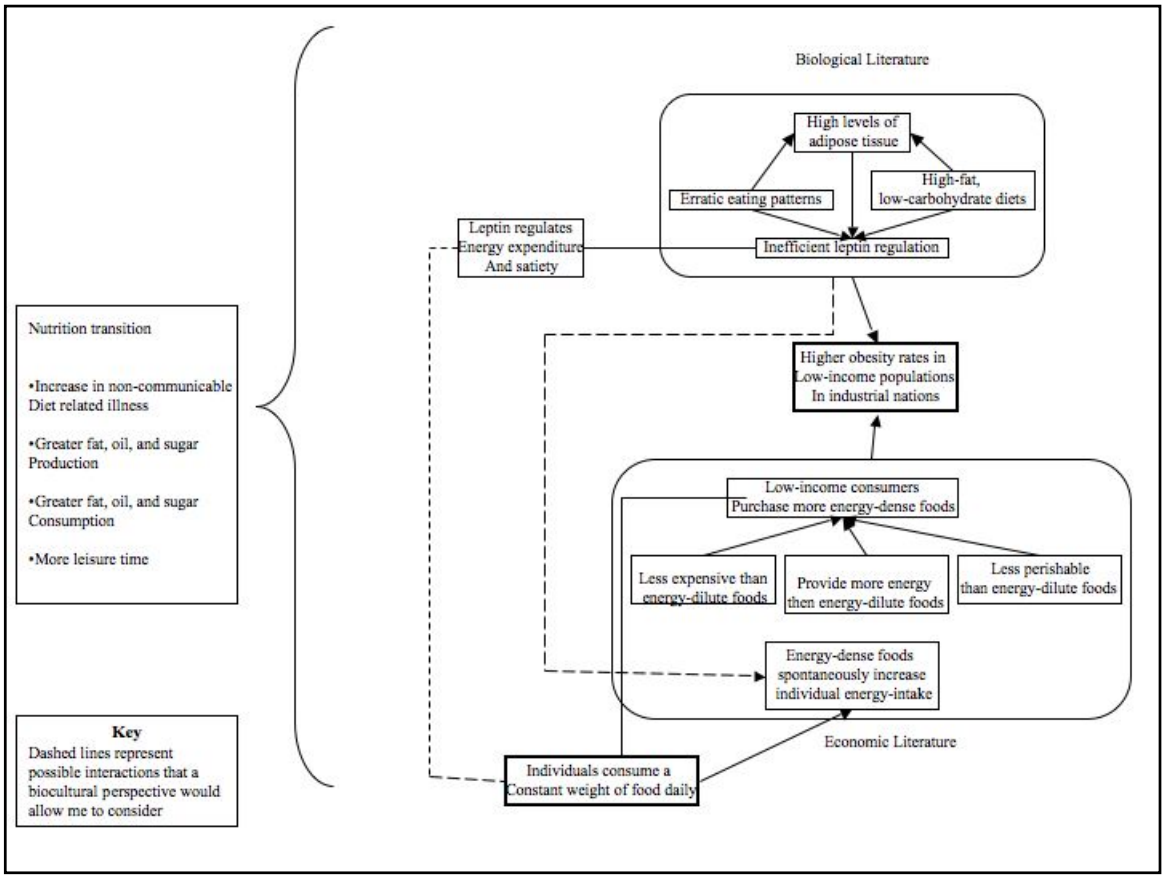
In industrial societies, researchers have been most confounded by the unequal distribution of obesity rates across socioeconomic gradients. In the United States, diet-induced obesity is concentrated in marginalized low-income populations (Alaimo *et al.* 1998, Cole 2001, Drewnowski and Levine 2003, Drewnowski and Spector 2004, Drewnowski and Darmon 2005, Gibson 2003, Gibson 2004, Gibson 2006, Townshead *et al.* 2001, VanEenwyck and Sabel 2003). In addition, researchers have consistently noted a significant, inverse correlation between food security and obesity. Food insecure populations are defined by their inability to acquire nutritionally adequate and safe foods in socially acceptable ways (WHO 1999). Although there is no one-to-one relationship between food insecurity and income measures of poverty, food insecure populations and low-income populations overlap significantly (Alaimo *et al.* 1998, Drewnowski and Spector 2004), as moderate to severe food insecurity appears most often in low-income populations. Due to this overlap, I will assume that low-income populations experience some degree of food insecurity.

Kendall *et al.* (1995), Basiotis and Lino (2002), and Wilde and Peterman (2006) examined the relationship between hunger, food insecurity, and obesity prevalence using National Health and Nutrition Examination Survey (NHANES) Census Bureau Current Population Survey (CPS) Food Security Supplement[1]. These researchers found that mean body mass index (BMI) was higher in food-insecure households. The prevalence of overweight and obesity was also highest in food-insecure households. Because obesity

[1] 1995 NHANES, CPS Food Security Supplement has a core-module questionnaire (Appendix A), which consists of 18 questions. "This core-module addresses specific households conditions, event, behaviors, and subjective reactions" (WHO 1999)

connotes excessive energy intake, and low-income populations traditionally experience some degree of food insecurity (limited or uncertain availability of adequate food supply), the prevalence of both obesity and substandard food supply in the same population appears paradoxical (Alaimo 2001: 1161).

This paradoxical distribution of diet-induced obesity in population subset(s) historically vulnerable to disease, and the elevated risk of coronary heart disease, and type 2 diabetes associated with obesity (Flegal *et al.* 2002: 1726, Kuczmarski *et al.* 1994: 272), have prompted research to determine the causal or underlying variables that structure this relationship. Currently, research that focuses on the distribution of diet-induced obesity in industrial populations can be separated into two distinct camps: economically-structured research and biologically-situated research. Figure 1 illustrates key variables that I will focus on in this thesis.



Biologists study food insecurity and obesity within a purely cellular framework, specifically the impact of hormones such as leptin on satiety, and energy expenditure (Drewnowski and Spector 2004:12). Their literature examines the impact of adipose tissue mass (Ainslie *et al.* 2000, Considine *et al.* 1996, Wisse *et al.* 1999 Zhang and Scarpace 2006), macronutrient composition (low-fat high-carbohydrate diets vs. high-fat low-carbohydrate diets) (Ainslie *et al.*, Wilsey and Scarpace 2004, Wisley *et al.* 2004, Zhang and Scarpace 2005) and erratic eating patterns (Dullo *et al.* 1997, Dullo and Jacquet 1998) on the presentation of a traditional leptin response (reduced appetite, increased energy expenditure). Specifically, they argue that these factors can work independently or in conjunction with one another to alter traditional leptin responses and negatively influence satiety and energy expenditure in favor of fat storage.

In contrast, economic anthropologists approach the relationship between food insecurity and poverty from an economic perspective. They situate the relationship between food insecurity and obesity within food choice literature. Their research examines the economics of food choice (Drewnowksi and Levine 2003, French *et al.* 2001, Glanz *et al.* 1998) and the relationships between dietary energy and socioeconomic status (Daemon *et al.*, 2003, Drewnowksi and Levine 2003, Drewnowski and Spector 2004). They believe that low-income populations adopt energy-dense diets as a coping mechanism to ameliorate the discrepancy between high food costs and individual energetic requirements (Drewnowski *et al.* 2004). Energy-dense diets, however, may contribute to the elevated obesity rates associated with low-income and food insecure populations.

This review of the literature will illustrate that the etiology of food insecurity and obesity cannot be understood exclusively through either of these perspectives. Biological anthropologists have been inattentive to the global and national processes that define individual's environments and shape individual's biologies. Economic approaches have ignored the consequences of a mismatch between a swiftly-changing dietary environment and human biological systems. Instead, I will argue that the interaction between obesity and socioeconomic status demonstrates the necessity of combining both economic and biological theories into a synthetic biocultural approach. Biocultural approaches address the structured inequalities and social relationships that work in conjunction with human biology's to impact illness expression. As such, a biocultural approach is pertinent when addressing the inter-relationships between low socioeconomic status and obesity. Presently, volume-satiety studies provide the best framework for the application of this approach (See Figure 1).

Previous volume-satiety research has illustrated that individuals consume a constant volume of food every day (Lissner *et al.* 1987, Rolls and Shide 1994, Saltzman *et al.* 1997). This relationship is couched in gastric and hormonal satiety responses. These biological adaptations, however, impede individuals' ability to identify and compensate for fluctuations in the macronutrient content of food (Drewnowski and Spector 2004, Hayes *et al.* 2002). In industrial populations, global shifts in production have simultaneously impacted food costs and consumption patterns. Economic constraints limit food-insecure and low-income consumers' ability to purchase energy-dilute diets that have low caloric content per unit weight and induce volume-based and hormone-based satiety responses without initiating spontaneous disinhibition (or

excessive calorie intake), and obesity expression (Drewnowski and Spector 2004, Hayes *et al.* 2000).

Biocultural studies are able to incorporate the concurrent effects of biological cues (gastric-volume and hormonal satiety responses) and economic constraints on low-income and food insecure populations. These studies will best address how structural inequalities work in conjunction with human biological processes and cultural coping mechanisms (food cost verses energy-density) to affect national obesity distribution (Leatherman 2005: 51).

Chapter 2: Review of Biologically-situated Literature

Biologically-situated literature addresses the mechanism between food-insecurity and obesity exclusively as a relationship between dietary patterns, adipose tissues stores, and their impact on the expression of the adrenocortical hormone leptin (Drewnowski and Spector 2004: 12). Leptin is an adipocyte-derived hormone that circulates in the blood in proportion to whole body adipose tissue (Ainslie *et al.* 2000, Considine *et al.* 1996, Zhang and Scarpace 2006). Serum leptin levels regulate consumption patterns, energy expenditure, and overall adiposity. Normal serum leptin levels average (12 + 4.4 ng/ml) (Sinha *et. al.* 1995), and fluctuate in a circadian rhythm (physiological rhythm associated with the 24-hr cycle of the earths rotation) (Lazreg *et al.* 2007, Sinha *et al.* 1995, Zhao *et al.* 2002) and in response to consumption patterns (Ainslie *et al.* 2000, Wisee *et al.* 1999,Zhang and Scarpace 2006).

Serum leptin levels function on a loop-like system. During fasting leptin levels decline, this reduces energy expenditure and increases hunger to prompt food consumption. When food is consumed, leptin secretion increases. These elevated leptin levels induce satiation (to reduce hunger) and increase energy expenditure. Body fat composition and dietary consumption patterns, however, can impair leptin signaling. Leptin levels, however, can be influenced by factors other than consumption events. Currently, biological literature stresses three related sources that can impair normal serum leptin levels and leptin signaling: excessive adipose tissue, high-fat, low-carbohydrate

diets, and erratic eating patterns (periods of binge eating followed by periods of energy restriction).

Researchers have found that individuals with high levels of adipose tissue maintain elevated leptin levels in concurrence with high levels of body fat. They believe that obese subjects exhibit a hyperleptin response that reduces cell sensitivity to endogenous leptin. This response helps maintain high fat stores in these individuals. In addition, inefficient leptin responses are associated with high-fat, low-carbohydrate diets. Leptin levels are more responsive to low-fat, high-carbohydrate diets than high-fat, low-carbohydrate diets. This response could contribute to the hyperphagic consumption and obesity associated high-fat diets. Finally, erratic eating patterns such as chronic periods of moderate to severe energy restriction can dramatically reduce leptin levels, independently of changes in whole body adipose tissue. Suppressed leptin levels reduce energy expenditure (to maintain fat stores) and elevate hunger levels (to encourage consumption). Leptin levels return to normal after calorie restriction ends. This leptin response to calorie restriction (reduction of energy expenditure and satiety independent of changes to whole body adipose tissue) encourages the maintenance of high adipose tissue levels. Biologists argue that in low-income populations-which are often characterized by high-levels of whole body adipose tissue, high-fat low-carbohydrate diets, and erratic eating patterns-these factors can work independently or in conjunction with one anther to impair the production and signaling of leptin, in favor of fat storage. I will discuss each of these sources for inefficient leptin regulation in turn.

As previously noted the ob protein leptin directs weight maintenance (through appetite, and energy expenditure) (Ainslie *et al.* 2000, Considine *et al.* 1996, Zhang and

Scarpace 2006). In ob/ob hyperphagic (obese) mice, however, the ob gene is mutated and does not produce leptin. When external leptin is administered to ob/ob mice, they reduce food consumption and increase energy expenditure. Research conducted on obese human subjects has not yielded similar results.

Researchers have found that obese human subjects display elevated leptin levels in concurrence with high levels of body fat (Considine *et al.* 1996). Scientists believe that individuals who display diet-induced obesity are leptin resistant and are unable to produce a traditional leptin response (elevated energy expenditure and reduced hunger levels) in the presence of elevated leptin levels. The presentation of leptin resistance in low-income and food-insecure populations, who (as previously noted) are at a higher risk of obesity, could work to maintain the presentation of obesity of these populations.

Considine *et al.* (1996) examined the relationship between serum leptin concentrations and body weight in humans. Serum leptin levels were taken from lean and obese subjects. The effects of fasting, weight loss, and food consumption were then examined in subsets of these populations. Considine *et al.* (1996) found that serum leptin levels in humans were positively associated with whole body adipose tissue. In addition, Considine *et al.* (1996) also found that despite the presence of elevated leptin levels in obese subjects, these individuals did not manifest a traditional leptin response (reduced food consumption and increased energy expenditure). Instead, these individuals maintained chronically elevated levels of whole body adipose tissue. These data suggest that the maintenance of obesity in humans may be influenced by the central mechanism-leptin that regulates food intakes and energy expenditure. High levels of adipose tissue can induce a hyperleptin response and chronically elevate leptin levels. This condition

can impair the neuroendocrine regulation of the afferent satiety signals that direct appetite, satiety, and energy expenditure in favor of positive energy storage and the overall maintenance of high levels of whole body adipose tissue (Ainslie *et al.* 2000, Wisse *et al.* 1999, Zhang and Scarpace 2006).

Despite the research conducted by Considine *et al.* (1996), traditional research on hyperleptinemia and leptin resistance has focused on rodents. The invasive methods associated with traditional leptin-administration have limited the scope of leptin research on humans. Leptin is traditionally administered through a single intracerebroventricular injection into the third cerebral ventricle. Data gathered on the impact of leptin on patterns of consumption and energy expenditure through non-human subjects (specifically rodents), however, can provide insight into the presence of a similar leptin processes in more complex organism, such as humans.

Conventional rodent-based studies have examined the impact of chronically elevated leptin levels on food intake, energy expenditure, and body weight. Rodents are traditionally separated into two groups: a control group which is administered a control vector and a group receiving recombinant Adeno-associated virus-leptin (rAAV) or synthetic leptin. Rats are administered a single dose of either a control vector or a rAAV-leptin by intracerebroventricular injection into the third cerebral ventricle. Control and leptin rats have access to food ad libitum. The food consumption body weight and whole body oxygen consumption (energy expenditure) are examined.

Scarpace *et al.* (2002) examined the relationship between obesity, elevated leptin levels, and leptin resistance in rats. They hypothesize that the elevated leptin levels traditionally found in obese populations encourage to the presentation of leptin resistance

in these populations. Rats were administered a single dose of either a control vector or rAAV-leptin. Food consumption, body fat, and energy expenditure (as a function of whole body oxygen consumption) was examined over the course of 138 days. At the conclusion of this period, all rats were infused with artificial cerebrospinal fluid or recombinant mouse leptin by mini-pump for seven days. Rats were allowed food ad libitum. Food consumption and body weight were recorded for an additional seven days.

Scarpace *et al.* (2002) found that the conventional leptin response (anorexic consumption and increased energy expenditure) in individuals chronically exposed to leptin wanes over time. Although rAAV administered rats initially consumed significantly less food and had higher rates of whole body oxygen consumption than control rats, this response declined until there was no difference in the food consumption and whole body oxygen consumption of rAAV-leptin rats and the control rats. Oxygen consumption displayed a similar pattern. Additionally, the infusion of leptin by mini-pump at the conclusion of the experiment did not induce a leptin-response in rAAV-leptin rats. In rats pre-treated with the control-vector, food intake and BMI decreased following the infusion. This research indicates that populations chronically exposed to elevated leptin levels can become resistant to leptin and lose their ability to produce a traditional response to high leptin levels (energy expenditure and reduced hunger response). Therefore, these data suggest that the maintenance of high adipose tissue levels and elevated leptin levels in obese populations may be due to leptin resistance and this group's inability to produce a traditional leptin response.

Scarpace *et al.* (2003) examined leptin-induced leptin resistance in young, lean rats. In previous experiments, young, lean rats maintained a normal leptin response

(anorexic behavior and increased energy-expenditure) when exposed to exogenously administered leptin over a seven week period. Scarpace *et al.* (2003), however, argued that even young, lean rats would develop leptin resistance if researchers increased the amount of exogenously administered leptin and extended the length of the exposure. Rodents were separated into two groups: control rats and rAAV-rats. Both groups were then allowed access to food ad libitum. Data on food consumption, body weight, energy expenditure (whole body oxygen consumption) and serum leptin was collected periodically. After 300 days both groups of rats were administered recombinant mouse leptin (rAAV) by a mini-pump for seven days. Rats were allowed access to food ad libitum and food consumption and body weight were recorded daily for an additional seven days.

Scarpace *et al.* (2003) found that rAAV-leptin rats initially demonstrated an anorexic response to food and high levels of whole body oxygen consumption. This response, however, declined over time until there was no difference between the control rats and the rAAV-leptin rats. In addition, the augmented administration of exogenous leptin by mini-pump for seven days did not induce the traditional leptin response in rAAV-rats. In contrast, control rats reduced their food consumption, increased energy expenditure, and lost weight. This study indicates that populations chronically exposed to elevated leptin levels can become resistant to leptin and lose their ability to produce a traditional response to high leptin levels (energy expenditure and reduced hunger response). This research provides good evidence for the possibility of leptin-induced leptin resistance in obese populations (who traditionally display elevated levels of endogenous leptin).

The results from Scarpace *et al.* (2002) and Scarpace *et al.* (2003) indicate that chronically elevated leptin levels can induce leptin resistance in all subjects. This information suggests that populations who maintain elevated leptin levels due to their own endogenous production (such as obese population) could develop leptin resistance. The loss of the traditional biological response to elevate leptin levels (increased energy expenditure and reduced hunger) could contribute to the maintenance of high levels of whole body adipose tissue in these populations.

Despite the exclusive use of rodents, these experiment can provide insight on leptin pathways in humans. Specifically, the results from Scarpace *et al.* (2002) and Scarpace *et al.* (2003), suggest the probability of a similar hyperleptin response and subsequent leptin resistance in more complex organisms, such as humans. The presence of hyperleptin induced leptin resistance in human populations favors positive energy storage and could contribute to the presentation of overweight and obesity in human populations.

Inefficient leptin responses are also associated with high-fat, low-carbohydrate diets (Ainslie *et al.* 2000, Wilsey and Scarpace 2004, Wilsey *et al.* 2004, Zhang and Scarpace 2005). In mixed fat-carbohydrate diets, carbohydrates are preferentially oxidized (Schutz *et al.* 1989: 307). "The short-term adjustment of oxidation to intake must be more accurate for carbohydrates than fat because the body's glycogen stores (in terms of energy content) are two orders of magnitude smaller than its fat reserves" (Schutz *et al.* 1989: 307). Researchers have associated this preferential oxidation with serum leptin levels.

The consumption of low-fat, high-carbohydrate diets stimulates a swift and elevated leptin response (Jenkins *et al.* 1997, Romon *et al.* 1999, Schutz *et al.* 1989). This reaction induces satiation (which reduces calorie intake) and increases energy expenditure (Havel *et al.* 1999). "Carbohydrate intake elicits a complex metabolic response [leptin] which serves to maintain homeostasis by promoting glucose storage and oxidation" (Schutz *et al.* 1989: 307). In contrast, high-fat, low-carbohydrate diets suppress leptin levels. These reductions in circulating leptin levels lower satiety (encourages consumption) and reduce energy expenditure (Ainslie *et al.* 2000: 441). The blunted response of leptin to high-fat low-carbohydrate diets indicates that the fat content of diets can have a potent impact on individual satiation and fat oxidation. This relationship could encourage hyperphagic consumption and the appearance of obesity in populations with high-fat, low-carbohydrate diets.

Jenkins *et al.* (1997) examined the relationship between macronutrient intakes, leptin concentrations and changes in body composition. They argue that macronutrient intake (low-fat high-carbohydrate diets verses high-fat low-carbohydrate diets) will affect leptin levels. Nine obese individuals matched for age and dietary intake were recruited. Baseline anthropometry, body composition, and habitual dietary intake were determined one week before the initiation of a formula diet, which was instituted for four weeks. During this period weight, body composition, and blood were analyzed. Jenkins *et al.* (1997) found that macronutrient content of diet was related to leptin levels. High leptin levels were produced after high-carbohydrate diets. This response was not duplicated after the consumption of high-fat diets. Overall, Jenkins *et al.* (1997) indicates that leptin levels are linked to carbohydrate consumption and less tightly coupled to fat

consumption. The blunted leptin response to high fat, low-carbohydrate meals can encourage hyperphagic consumption and can induce/maintain overweight and obesity in populations with high-fat, low-carbohydrates diets.

Romon *et al.* (1999) examined the postprandial (post-meal) leptin response to meals differing in macronutrients (high-carbohydrates low-fat, verses low-carbohydrate high-fat meals). They argue that the presentation of a traditional, postprandial leptin response will be affected by the macronutrient composition of the diets that individuals consume. Twenty-two individuals were given isoenergetic meals in a randomized order or remained fasting. Blood samples, and satiety and hunger rating were collected after the consumption of these meals. Romon *et al.* found that high-carbohydrate low-fat meals induced a greater postprandial leptin response than low-carbohydrate high-fat meals in all individuals. These data suggest that high fat and low-carbohydrates diets can impair the expression of a traditional leptin response. This blunted reaction reduces energy expenditure and satiety, and can influence the occurrence of overweight and obesity in individuals.

Havel *et al.* (1999) examined the relationship between the macronutrient composition of individual diets and leptin levels. They believe that leptin levels may be more responsive to low-fat, high-carbohydrate meals than high-fat, low-carbohydrate meals and help contribute to weight gain in populations with high-fat, low-carbohydrate diets. The study was conducted with nineteen normal-weight women. These women were split into two groups. One group was administered three isocaloric (730 kcal) high-fat, low-carbohydrate meals. The other group was administered three isocaloric (730 kcal) low-fat, high-carbohydrate meals. After a ten-day buffer the two groups switched.

Havel *et al.* found that leptin levels were higher after individuals consumed low-fat, high-carbohydrate meals than high-fat, low-carbohydrate meals. This indicates that leptin levels are affected by the macronutrient composition of individual diets. This response could initiate hyperphagic consumption and obesity in populations with low-fat, high-carbohydrate diets.

Jenkins *et al.* (1997), Romon *et al.* (1999), and Havel *et al.* (1999) found that leptin's ability to increase energy expenditure and satiety in favor of fat oxidation is dependant on the macronutrient composition of individual diets. Leptin levels are more responsive to low-fat, high-carbohydrate diets than high-fat, low-carbohydrate diets. Schutz notes that, "The shift towards a preferential metabolic use of carbohydrate rather than of fat results in an increased deposition of body fat, and along with the general decrease in physical activity, and the inability to organism to adjust its metabolism, so as to attenuate the effect of changes in fat intake on the fat balance…" (Schutz *et al.* 1989: 313). This response could contribute to the elevated obesity rates in food-insecure and low-income populations who rely on high-fat, low-carbohydrate diets. Inefficient leptin responses are also associated erratic eating patterns (chronic periods of moderate to severe energy restriction).

Nutritional research on eating behaviors has found that periods of moderate to severe energy restriction predispose individuals to binge eating and weight gain after the cessation of the semi-starvation regime (Dullo *et al.* 1997, Dullo and Jacquet 1998, Frisancho 2003). This relationship has been documented in the United States (Dullo *et al.* 1997, Keyes *et al.* 1950*)*, hunting-gathering populations in Bolivia (Holmberg 1950), and the Ache of Paraguay (Hill and Hurtado 1996, Hurtado and Hill 1991). Results in humans

suggest that post-starvation hyperphagia and reduction in basal metabolic rates (BMR) is determined to a large extent by auto-regulatory feedback control systems from fat and lean tissues that link the state of depleted fat stores to compensatory mechanism that suppress thermogenesis (Dullo *et al.* 1997, Dullo and Jacquet 1998). Researchers have implicated the fat-derived leptin auto-regulatory feedback control systems as the basis of this relationship. They argue that moderate to severe energy restriction dramatically reduces leptin levels independent of changes in whole body adipose tissue. Reduced leptin levels suppress energy expenditure to maintain fat storage and elevate hunger levels. This response encourages consumption and wanes only after calorie restriction ends. This reduced fat mobilization (through reduced energy expenditure), coupled with additional consumption (independent of actual whole body tissue), favors obesity.

Keim *et al.* (1998) examined the relationship between plasma leptin concentrations and hunger, or appetite-related variables in healthy overweight women, during a prolonged energy restriction. The study consisted of a three-week stabilization period in which subjects were given sufficient energy intake to maintain their weight. This was followed by a twelve-week intervention period, in which subjects consumed a diet 2MJ/d less than the diet they consumed for the stabilization period. Diet and activity levels were controlled during the stabilization period and intervention period. Data was gathered on body weight, glucose, leptin, insulin and self-reported appetite. Keim *et al.* (1998) found a significant decrease in weight, percentage of body fat, and plasma leptin levels during the intervention period. The drop in the plasma leptin level was greatest during the first week, after which leptin levels remained low. Hunger and desire to eat increased, doubling in response to the energy deficit. Circulating leptin levels and

changes in leptin correlated to changes in hunger, desire to eat, and prospective consumption. Overall, Keim *et al.* (1998) found an inverse relationship between leptin levels and hunger-related variables independent of body fat and weight. Lower leptin levels were associated with increased hunger and desire to eat, while higher leptin concentrations were related to decreased feelings of hunger and desire to eat.

Wilsey and Scarpace (2004) examined the effect of short-term calorie restriction on leptin expression and function. Brown Norway rats were divided into two groups. One group was administered a standard chow diet, the other group was administered a high-fat diet. Individuals that experienced diet-induced obesity (DIO) on the high-fat diet were separated from the animals whose weight remained constant (DR). After 100 days subgroups of the DIO, DR, and chow fed groups were switched to a calorie-restricted (CR) diet. Animals were given single hypothalamic injections of rAAV-leptin. All groups were then euthanized and blood and adipose tissue was collected for analysis. Wilsey and Scarpace (2004) found that after the injection of artificial leptin DIO and chow fed animals experienced an increase in STAT3 phospoylation. This increase was six-fold in chow fed animals and two-fold in DIO animals. In addition, animals that experienced calorie restriction: DIO animals had an 85% increase in leptin expression and protein; there was a 45% increase in expression in chow fed rats. Overall research indicates that calorie restriction may enhance leptin responsiveness in previously DIO leptin resistant rodents. This reaction could encourage high adipose tissue levels in populations that experience chronic energy-restriction.

Researchers have suggested that the relationship between leptin, satiety, and energy expenditure that is displayed by individuals experiencing mild to moderate

starvation, probably represents a compensatory adaptation to maintain energy reserves in situations where access to food is uncertain. "…humans exposed to moderate energy restriction that can be sustained over years adapt through a reduction in EE [energy expenditure] and preferential accumulation of body fat. This form of energy conservation is a biologically meaningful mechanism, in the face of dangerously and moderately low energy supplies…" (Frisancho 2003: 525). Food insecurity, however, characterizes low-income populations, as low-income populations commonly experience periods of short-term calorie restriction (VanEewych and Sabel 2003: 840). The reduction of leptin levels (reduced energy expenditure, and elevated hunger levels) in response to short-term calorie restriction (independently of changes in adipose tissue) encourages over consumption in low-income and food-insecure populations when economic access to food is resumed. This reaction Frisancho states, "…exposure to under nutrition even during adulthood accelerates the replenishment of fat stores during refeeding and thereby increase[es] the risk of obesity (Frisancho 2003:525). This research indicates that leptin responses to dietary restriction in food-insecure and low-income individuals contribute to the presentation of obesity in these populations.

Overall, leptin provides the missing link for researchers who have long argued that overweight and obesity maintenance is the result of an inefficient system that connects the brain, food ingestion, and adipose-tissue mass (Dullen *et al.* 1997). This body of literature provides good evidence for the role of leptin in regulating satiety, energy expenditure, and fat storage. In addition, scientists have successfully illustrated how high levels of whole body adipose tissue, high-fat, low-carbohydrate diets, and

erratic eating patterns influence the expression of a traditional leptin response and subsequent fat mobilization, maintenance, and storage.

This narrow and increasingly specialized focus, however, ignores the dialectic relationship between biology and culture. It reduces the relationship between food insecurity and obesity to an analysis of biological micro-processes and ignores the larger global and national forces that structure food choice, food access, and individual health. Global transitions, national policy, and individual's placement within these dynamic processes impact personal environments and health. Pearl states, "….economic and social factors and forces are among the most important elements in determining the biologically significant environment of human beings…" (Pearl 1930:540).

To fully understand the elevated presentation of obesity in food insecure industrialized populations, we need to incorporate food choice literature and data from hormone-derived satiety response into a synthetic biocultural approach. This approach will provide an effective framework for analysis of how the processes of inequality and social change interact with human biologies.

Chapter 3: Review of Economically-Situated Literature

Traditionally, social science research on the relationship between low income and high obesity prevalence has addressed this paradox as a function of education (Gorbach *et al.* 1990, McCann *et al.* 1990). Researchers argued that increasing individuals' nutritional knowledge through educational programs would elevate the number of healthy foods that individuals purchased and consumed and help lower obesity rates (McCann *et al.* 1990). Economic anthropologists, however, hypothesize that the unequal concentration of obesity in low-income and food insecure populations is not due to insufficient knowledge of healthy eating practices. Instead, these researchers believe that individuals have sufficient knowledge of nutrition and that individual and household-level access to economic resources impact food choice (Drewnowski and Darmon 2005: 903).

Economic anthropologists argue that families of low socioeconomic status preferentially purchase and consume energy-dense foods as a coping mechanism to ameliorate the discrepancy between high food costs and individual energy requirements (Kearney and McElhone 1999, Lloyd *et al.* 1995). Energy-dense diets are inexpensive, less perishable, and highly palatable. Drewnowski and Spector state, "[F]ats and oils, sugar, refined grains, potatoes, and beans [represent] some of the lowest-cost options and provided dietary energy, at minimal cost." (Drewnowski and Spector 2004: 9). The ability to purchase diets low in energy-density, however, can impact individuals ability to

maintain healthy weight (Drewnowski and Darmon 2005: 903, Poppitt and Prentice 1996: 154).

Research that has examined the relationship between socioeconomic characteristics and food choices, (purchase and consumption patterns) has found that economic status impacts which types of food individuals purchase (Glanz *et al.* 1998: 1120). Economic resources, therefore, affect overall dietary quality (Drewnowski and Specter 2004:11). Wealthier households buy higher-quality meats, fish, seafood, fruits and vegetables (Drewnowski and Specter 2004:10). In contrast, individuals of low socioeconomic status place an increased emphasis on food cost (Glanz *et al.* 1998: 1120) and buy more energy-dense foods, which are high in added sugars and fats.

The Healthy Eating Index (HEI) has been used to evaluate the relationship between household dietary quality and socioeconomic factors. It is a statistic that refers to an overall picture of household diet and nutrition and includes factors such as preparation, style and specific dietary requirements. Relying on a portion of the 1992-2002 National Health and Nutrition Examination Survey (NHANES) [2] set Basiotis *et al.* (2004) examined the dietary status of Americans. They found HEI score to vary across demographic and socioeconomic lines, increasing with both education and income. People in higher income households had a higher HEI scores (better scores on grains, vegetables, fruits, milk, meat, and variety components of the HEI) than people with household incomes below the poverty line. Overall, this study indicates that consumption patterns are influenced by socioeconomic status. Individuals of lower socioeconomic status consume diets further away from the American nutritional ideal than individuals of

[2] NHANES provides information on individuals' food and nutrient consumption (via 24hr. dietary recall), health-related data, demographic and socioeconomic data.

higher socioeconomic status. The consumption patterns associated with socioeconomic status (low-income consumers purchase diets further from the American ideal) have been linked to food cost. Low-income consumers place a greater emphasis on food costs than consumers with greater access to resources. Accordingly, low-income consumers differentially purchase energy-dense, low cost foods.

French *et al.* (2001) examined the association between individual food selection and food cost. This CHIPS (Changing Individuals' Purchase of Snacks) study investigated the association between targeted price reductions and point-of-purchase promotions on low-fat vending machine snacks and the sales of these products. The prices on lower fat snacks in twelve vending machines at twelve work sites and twelve secondary schools in Minnesota were reduced relative to the higher fat snacks by 10%, 25%, and 50%. The percentage of lower fat snacks sold under each price reduction was then examined. French *et al.* (2001) found that food selection was strongly correlated with food cost. Price reductions of 10%, 25%, and 50% on lower fat snacks were associated with a 9%, 39%, and 93% respective increase in the percentage of these snacks sales. Overall French *et al.* (2001) found that food price had a strong and consistent impact of food purchase patterns, such that decreasing the price of low-fat foods increased the sales of those foods.

Glanz *et al.* (1998) examined the relationship between taste, nutrition, cost, convenience, and weight control on individual food selection and consumption. Health lifestyles, dietary data, and food choice determinants were assessed through a nationwide lifestyle and "health-styles" survey. Glanz *et al.* (1998) found that taste was the most important consideration for respondents when determining food choice. This was

followed by cost, nutrition, convenience, and weight control. In addition, demographic factors were significant predictors of the relative importance of these variables for consumers. Cost and convenience were the most important factors to individuals of low socioeconomic status. “Income predicts the importance of cost and convenience, both of which matter most to individuals of low socioeconomic status” (Glanz *et al.* 1998: 1120). These individuals consumed high quantities of fast foods, which are less expensive and perceived as more convenient. Overall Glanz *et al.* (1998) found that socioeconomic status influences the importance of food cost in determining patterns of food selection and consumption.

The results of Basiotis *et al.* (2004), French *et al.* (2001), and Glanz *et al.* (1998) suggest that the reason for rising obesity rates in low-income, food-insecure populations is not insufficient nutritional information. Instead, the literature suggests that socioeconomic status impacts dietary quality through individuals and households food purchase and consumption patterns. The uneven distribution of obesity across a socioeconomic gradient may be affected by the ability of an individual or a household to purchase and consume healthy food products. French states, “In general people may possess knowledge about healthful food choices, but when considered in tandem with the choice dimensions of price and taste, they may choose the tastier and cheaper, but less nutritious food” (French 2003: 842S).

Dietary energy-density could provide the link to understanding the concurrent expression of food insecurity and obesity. The energy density of foods is defined by that items caloric content per unit weight. Energy-dense foods have a high caloric content and are characterized by refined sugars, oils, and fats. Energy-dense foods also

traditionally have low nutrient value (low in essential vitamins and nutrients). Currently, economic models indicate that household economic resources influence the purchase of energy-dense food items. Wealthier U.S. households (mean annual income $77, 311) spend more money on food ($748 more per person per year) than low-income households (mean annual income $6,669) (Drewnowski and Spector 2004: 9). These additional funds are invested in the purchase of energy-dilute products such as fruits and vegetables, which are traditionally high in essential nutrients and minerals (Drewnowski and Spector 2004:10).

In contrast, low-income households have energy-dense diets and preferentially purchase energy-dense items in place of energy-dilute foods. Low-income diets are characterized by products such as refined grains, processed meats, and soft drinks (Drewnowski and Levine 2003, Drewnowski and Spector 2004: 7). These items have high levels of refined sugars, fats and oils, and low quantities of beneficial vitamins and minerals.

Therefore, the increased purchase of energy-dense foods by low-income consumers is problematic in two ways. First, energy-dense items are high in calories and low in nutritional value. Second, populations that invest in energy-dense items consume these foods in place of energy-dilute foods such as fruits and vegetables, which are low-calorie and traditionally have high nutritional values. The purchase patterns associated with socioeconomic status, therefore, could significantly impact the obesity rates in low-income populations.

Darmon *et al.* (2003) examined the role of economic constraints in the selection of energy dense diets. They believe that economic constraints encourage the selection of

energy-dense diets. Darmon *et al.* (2003) found that the introduction of progressively stronger energy constraints correlated to a direct increase in dietary energy density. These diets contained smaller quantities of fruits, vegetables, seafood and fiber but were high in fats, sugars and cereals. Overall, Darmon *et al.* (2003) found that populations endeavoring to maintain a traditional French diet at a low cost will necessarily increase energy density (Darmon *et al*. 2003: 320). This study indicates that economic constraints impact the percentage of energy-dense foods in individual's diets. This relationship between economic access and food selection may impact obesity trends and encourage the presentation of elevated obesity rates in low-income populations.

Drewnowski *et al.* (2004) argues that the relationship between food supply trends (dietary quality, dietary energy density) in freely-chosen French diets is structured by the economics of food choice. Consumers with limited resources may select energy-dense diets high in refined grains, added sugars and fats as an effective way to save money. Nutrient and cost analysis was collected on 527 families. Multivariate analysis was then used to determine the relationship between diet composition and diet costs. Drewnowski *et al.* (2004) found that higher consumption of fats, refined grains and sugars were associated not only with reduced energy costs, but also with lower absolute diet costs (Drewnowski *et al.* 2004: 1558). In contrast, higher intakes of energy dilute foods such as fruits, and vegetables were associated with increased diet costs. This research illustrates that fats and sweets offer high dietary energy at low costs. Drewnowski *et al.* (2004) argues that limited economic resources and food costs pose a barrier to the acquisition of healthy diets for low-income and minority populations. These populations may consume less prudent, energy-dense diets to maintain energy at low costs.

Previous research has illustrated that reduced access to economic resources is a barrier to individuals and households who want to maintain an energy-dilute diet and a healthy weight. The data provided by Darmon *et al.* (2003), and Drewnowski *et al.* (2004) support these conclusions. Their research illustrates that energy-dense food may function as a coping mechanism for low-income consumers by resolving the conflict between high food costs and low income. Energy-dense foods that are high in fats and refined sugars are economically savvy. These diets provide low-income consumers with high concentrations of energy at low-cost (Drewnowski and Darmon 2005: 901). Energy-dense diets, however, are associated with obesity, and this decision may affect obesity expression in these populations.

The National Institutes of Health strategies for obesity research have argued that one of the reasons for rising obesity rates in low-income and food insecure populations may be the abundance of relatively inexpensive, calorically-dense foods that are convenient and taste good (Drewnowski and Darmon 2005: 900). Economically-situated literature that has explored the juxtaposition of obesity across a socioeconomic gradient has provided excellent support for this hypothesis and for the impact of economic restrictions on food choice and obesity. This research indicates that lower-income and food insecure consumers make sound food choice decisions when confronted with the disparity between their economic limitation and energetic needs. These individuals differentially purchase and consume energy-dense foods that are inexpensive and taste good. These foods ameliorate the disparity between economic limitations and energy requirements because energy-dense foods are high calorie and inexpensive. The purchase of these food items, however, despite its economic validity, may impact individual health.

Despite the breadth of this literature, however, researchers have ignored the biological relationships (hormonal satiety based responses) that structure food consumption and weight gain. A comprehensive understanding of the relationship between food insecurity and obesity is best structured through a biocultural analysis that situates this relationship within the larger framework of the biology of poverty. This will enable researchers to explicitly link larger global processes, such as the nutrition transition to smaller-scale economic choices and their impact on individual biological processes, to examine how these interactions work to create and maintain the relationship between food insecurity, low-income and obesity.

Chapter 4: A Biocultural Synthesis: Volume-Satiety Studies

Currently volume-satiety studies provide the best framework for exploring the relationship between low-income, food-insecurity, and obesity within a biocultural perspective. Volume-satiety studies explore the biological relationships that structure the expression of traditional satiety responses to consumption events. These studies, however, also addresses the economic and cultural factors influence the types of foods we eat at meals and where, when, and how we eat our meals (Marshall and Bell 2002: 53). Consumption patterns (the foods that we eat and our eating behaviors) vary greatly based on socioeconomic status. As previously noted in the United States wealthier households consume diets closer to the American ideal, while low-income households preferentially purchase and consume energy-dense diets. Consumption patterns are also impacted by cultural variables such as time of day (Kramer Rock &Engall 1992), social environment (Clendenen Herman & Polivy 1994, DeCastro 1990), location (Marshal 2000, Stroebele and DeCastro 2006) and occasion (Marshall and Bell 2003). Volume-satiety studies, therefore, are able to examine the concurrent effects of biological cues (gastric-volume and hormonal satiety responses) and economic constraints on low-income and food insecure populations. The integrative effects of volume-satiety studies make them the best framework for the application of a biocultural approach

Although research indicates that cultural variables (time of day, social environment, location and occasion) impact **discrete consumption events.** Results from satiety studies (Saltzman *et al.*, 1997, Stubbs *et al.*, 1996, Van Stratum *et al.*, 1978) have

consistently found that sensory-specific satiety-or the decline in the perceived pleasantness of food as it is consumed that contributes to the cessation of eating (Bell *et al.* 2003: 593) and gastric satiety are linked to food volume. These biological satiety responses cause individuals to consume a **fixed volume of food daily** (Lissner *et al.*1987, Rolls and Shide 1994, Saltzman *et al*. 1997 Stubbs *et al.*, 1996)**.**

These volume-based (visual and gastrointestinal) satiation cues induce disinhibition, or the passive over-consumption of fats and energy in the presence of energy-dense food (Poppitt and Prentice 1996: 154). This occurs because individuals do not alter their consumption volume to compensate for changes in energy density (Lissner *et al.*1987, Rolls and Shide 1994, Saltzman *et al*. 1997 Stubbs *et al.*, 1996). The portion of energy dense foods in individuals' diets, therefore, spontaneously impacts daily energy intake and the ability to maintain a health body weight (Drewnowski and Spector 2004: 8, Hayes *et al.* 2002). Poppitt and Prentice state, "When selecting an energy-dense diet individuals readily consume a sufficient weight of food to maintain a high energy intake. However, when selecting a low energy-dense diet, individuals appear unable to eat sufficient weight of food to maintain a high intake and consequently energy intake falls" (Poppitt and Prentice 1996: 170).

In addition, individuals who consume diets high in energy-dense foods have an increased risk of marginal nutrient intake. This occurs because energy-dense foods are traditionally low in essential vitamins and minerals. Energy-dense foods are also often consumed at the expense of energy-dilute foods such as fruits and vegetables, which provide high micronutrient loads (Kant 2000: 934). Overall, the higher caloric load per unit weight and marginal nutrient intake associated with energy-dense diets can initiate

(Drewnowski and Spector 2004: 8, Hayes *et al.* 2002) or help maintain high BMI's in populations that consume diets high in energy-dense foods (Drewnowski and Spector 2004: 8, Hayes *et al*. 2002: 479).

Studies that investigate the effect of macronutrient composition and volume on satiety have utilized the common method of preloading. During preloading, individuals are provided with a preload (fixed amount of food) that differs in macronutrient composition, volume, etc. Researchers then assess the effects of these preloads on subsequent food consumption by individuals. Results from preloading satiety studies (Saltzman *et al.* 1997, Stubbs *et al.*1996, Van Stratum *et al.* 1978) have consistently found that macronutrient variation does not influence satiety when palatability and energy density are controlled. Instead, sensory-specific satiety and gastric satiety are consistently linked to food volume.

Bell *et. al.* (2003) examined the independent influences of volume and energy content on individual perceptions of sensory specific satiety. They argue that short-term consumption is affected by the volume of the food they consume and not the energy content. By having women assess the palatability of milk-based foods that varied in both volume and energy-density, Bell *et al.* (2003) found that the volume of liquid consumed significantly affected satiety. There were greater declines in ratings of the pleasantness of odor, taste, texture and prospective consumption after the high-volume energy-dilute preload, than after the low-volume energy-dense preload. Overall, the data collected by Bell *et al.* (2003) supports the hypothesis that the volume of food that individuals consume has a greater impact on satiety than energy content.

Rolls and Roe (2002) examined the relationship between food volume and energy content on the gastric and postgastric factors that influence satiety. Previous research has noted a strong relationship between the energy density of food and energy regulations. The mechanism that structures this relationship, however, is poorly understood. This experiment manipulates two factors that influence energy density, food volume and energy content, and examines their independent impact on satiety when sensory clues are bypassed by infusing foods intragastrically.

This study was conducted with fifty-four women: twenty-five lean and twenty-nine obese. The women were of a similar age, health, and had similar scores of dietary restraints. Physical information (BMI, height, and weight) was collected on all individuals through a physical examination. In addition, psychosocial information on food behaviors (eating patterns and perceptions) was collected. Preloads were administered intragastrically and varied in volume and/or energy content: 200ml/200 kcal, 400ml/200kcal and 400ml/200kcal and 400ml/400kcal. Analysis of food intake and ratings of hunger were conducted using the mixed linear model. The outcomes for energy density and food weight were analyzed. In addition, a summary measure of hunger and satiety was evaluated throughout the day.

Rolls and Roe (2002) found that lean and obese subjects exhibited similar satiety responses to variations in energy and food volume. Volume significantly affected energy intake. Mean energy intake decreased after the 400-ml preload when compared with the iso-energetic 200-ml preload. Variations in the energetic content of the preload, however, did not impact food consumption. Overall, this study indicates that humans

satiety responses are dependant on gastric and postgastric process. These processes respond to changes in food volume and not the energy content of those foods.

Rolls *et al.* (1998) examined the impact of food volume on satiation. Rolls *et al.* (1998) hypothesize that fluctuations in food volume will impact the appearance of a satiety response in subjects independent of energy, macronutrient content, and sensory properties. The study was conducted with twenty normal-weight young men. Physical information (BMI, height, and weight) was collected on all individuals through a physical examination. In addition, psychosocial information on food behaviors (eating patterns and food perceptions) was collected. On four separate days, all men consumed all of their meals in the laboratory. On three of the four days, subjects were served a milk-based drink, followed by a self-selected lunch and a self-selected dinner. On one day, no pre-load was administered (control). The milk-based preloads were similar in energy content (2088kJ), macronutrient composition, and sensory properties (palatability). They varied, however, in volume (300, 450, or 600-mL). Subjects also rated their hunger, thirst, nausea, fullness, and prospective consumption on visual analog scales (VAS) before and after each meal.

Rolls *et al.* (1998) found that preload volume impacted consumption patterns such that greater preload volume was associated with reduced consumption. Higher preload volume also reduced individual ratings of hunger, fullness, and the amount that individuals believed they could comfortably consume. Overall, these results indicate that food volume can impact satiety independent of that foods sensory properties, energy, and macronutrient content.

The results of Bell *et al.* (2003), Rolls and Roe (2002), and Rolls *et al.* (1998) indicate that individuals consume a fixed volume of food daily and do not adjust the food consumption (volume) in response to changes in energy density and macronutrient composition. Instead, these data indicate that individual satiety is linked to food volume.

Although energy density does not impact individual satiety responses, changes in energy density can spontaneously impact energy intake. Poppit and Prentice state, "Increasing the energy density of a diet, which normally occurs when [the] fat content [of said food] increase[s], may result in a passive increase in energy intake, simply because; people who maintain a constant body weight are habituated to eating a relatively constant weight of food" (Poppitt and Prentice 1996: 157).

Bell and Rolls (2001) examined the relationship between energy density food consumption (volume) and energy intake in lean and obese individuals. They believe that individual satiety is linked to the volume of a diet and not to the macronutrient content of a diet. They argue that this volume-specific satiety response can lead individuals to spontaneously increase or decrease their energy intake in response to diets that maintain constant volume but vary in macronutrient composition. Subjects participated in six experimental sessions. During these sessions, all individuals consumed all meals in the laboratory. Meals included a manipulated entrée that was consumed ad libitum. These entrees varied by percentage of fat (low-fat, medium-fat, and high-fat) and energy density (low energy density and high energy density). Food and beverages were weighed before and after each meal to determine the amount of food consumed. Subjects also rated their hunger, thirst, nausea, fullness, and prospective consumption on visual analog scales (VAS) before and after each meal.

Bell and Rolls (2001) found that all individuals (lean and obese) consumed a constant volume of food (mL) across all manipulations. They did not adjust their consumption volume (mL) in response to changes in energy density and fat content of the diet. All women consumed less energy in low-energy dense diets than in high-energy dense diets. These differences are due to the differences in energy intake of high-energy dense diets and low-energy dense diets. These results indicate that manipulation of dietary energy density spontaneously impacts the amount of energy individuals consume.

Bell *et al.* (1998b) examined the relationship between energy density and energy intake. They hypothesize that individual satiation is not sensitive to energy density, but is sensitive to food volume. Therefore, they argue that changes in the energy density of foods will result in a corresponding and spontaneous change in energy intake. Eighteen normal-weight women participated in this study. Physical information (BMI, height, and weight) was collected on all individuals through a physical examination. In addition, psychosocial information on food behaviors (eating patterns and eating perceptions) was collected. Food volume (mL) (energy and macronutrient intake) and satiety were calculated.

Bell *et al.* (1998b) found that individual cumulative intake (by meals) did not fluctuate to accommodate changes in dietary energy density. Energy intake was larger under high-energy density conditions than under medium conditions and low energy density conditions. This study indicates that the energy density of individual diets can significantly impact energy intake and weight.

Using data gathered by Bell and Rolls (2001), Bell *et al.* (1998b) indicates that energy density directly affects energy intake. The relationship of sensory-specific and

gastric satiety to food volume (and not to the macronutrient composition of food) links individual energy intake to dietary energy density. In populations that consume diets high in energy-dense foods (such as low-income and food-insecure populations) this relationship encourages high-energy intake and could contribute to the elevated presentation of obesity in these populations.

Volume-satiety studies explore the effects of biological satiety responses within the larger cultural and economic frameworks that define meal events. This unique feature makes these studies the best avenue to combine economic and biological approaches in order to explore the seemingly paradoxical relationship between low-income and obesity.

Chapter 5: Conclusion

Currently literature that addresses the high presentation of obesity in low-income and food insecure populations is divided into separate economic and biological perspectives. Biological perspectives address this relationship as a function of individual hormonal responses (serum leptin levels). Biological anthropologists argue that the high-fat, low-carbohydrate diets, erratic eating patterns, and high percentages of whole body adipose tissue that characterize food insecure populations impair leptin's ability to impact satiety, energy expenditure, and fat storage. Economic perspectives, in contrast attribute this association to the economic underpinnings of individual food choice. They believe that the energy-dense diets that characterize low-income populations function as coping mechanisms to alleviate the incongruence between individual energetic requirements and food costs. Energy-dense diets are inexpensive and contain more energy per unit weight than healthier alternatives. Energy-dense diets, however, could encourage the presentation of obesity in these populations.

The data presented by both camps does not fully explain the high presentation of obesity in food insecure and low-income populations. Biological approaches have not addressed the larger national and local processes that structure human consumption patterns. Economic approaches have ignored the biological adaptations that exacerbate the obesiogenic effects of energy-dense diets. Instead analysis of these camps indicates that both areas would benefit from the integration of their hypotheses into a single and synthetic biocultural perspective. "Biocultural approaches address the dynamic

interactions between human beings as biological beings and the social, cultural, and physical environments they inhabit. They focus on understanding variability in human biology, as a function of responsiveness to the large (social and physical) environment" (Dufour 2006: 1).

Further development of existing volume-satiety studies provides the best option to elucidate the expression of obesity prevalence in the United States. As previously acknowledged, volume-satiety studies indicate that individuals eat a constant weight of food daily. This consumption pattern is independent of macronutrient content; therefore, changes in the macronutrient content of an individual's diet can spontaneously impact **individual** energetic consumption.

A larger-scale application of this information suggests that **populations** whose diets are high in energy-dense foods, (such as low-income food-insecure populations), are consuming more calories per gram of food than they would if they replaced these items with energy-dilute foods. This economic-biological correlation could affect the appearance and/or maintenance of obesity in these populations. An expansion of existing volume-satiety studies, however, is needed to support this inference. Currently, volume-satiety studies are conducted exclusively within the confines of controlled laboratory conditions. Observational studies of consumption patterns (volume, energy density) across socioeconomic status are needed to see if data produced under controlled laboratory conditions are mimicked in non-controlled environments. In addition, long-term studies are needed that observe the effect of manipulations of energy density and the maintenance and/or appearance of whole body adipose tissue.

Finally researchers need to explore the relationship between volume-derived satiety responses and leptin levels. Does leptin influence the maintenance of constant daily volume intakes in human individuals? If so, constant volume consumption and high energy diets could lead to obesity in low-income and food insecure populations, not only because of increases in energy, (cal/g) but also due to the decreased leptin levels (and reduced energetic mobilization) associated with the blunted leptin response to high-fat diets vs. high-carbohydrate diets.

Overall, a biocultural approach that investigates consumption patterns (volume and energy density) and leptin levels across a socioeconomic status within a traditional volume-satiety framework is better able to explain the relationship between food insecurity and obesity. It can explore the multidimensional social and economic phenomenon of poverty and how disease prevalence (the relationship between food insecurity and obesity) is a function of human action shaped and maintained through intuitions and policies that structure access to power and resources.

Table 1

Table 1. Trends in the Age-Adjusted and Age-Specific Prevalence of Obesity for Adults Aged 20-74 Years, 1960-2000*

		Prevalence, %					Change, % (95% CI)‡	
Sex	Age, y†	NHES I, 1960-1962 (n = 6126)	NHANES I, 1971-1974 (n = 12 911)	NHANES II, 1976-1980 (n = 11 765)	NHANES III, 1988-1994 (n = 14 468)	NHANES Continuous, 1999-2000 (n = 3601)	NHANES II to NHANES III	NHANES III to NHANES 1999-2000
Both sexes	20-74	13.4	14.5	15.0	23.3	30.9	8.3 (6.6-10.0)	7.6 (4.2-11.0)
Men	20-74	10.7	12.1	12.7	20.6	27.7	7.9 (6.0-9.8)	7.1 (3.4-10.8)
	20-39	9.8	10.2	9.8	14.9	23.7	5.1 (2.9-7.2)	8.8 (4.8-12.8)
	40-59	12.6	14.7	15.4	25.4	28.8	10.0 (6.9-13.0)	3.4 (–2.8-9.6)
	60-74	8.4	10.5	13.5	23.8	35.8	10.3 (6.3-14.3)	12.0 (5.0-19.0)
Women	20-74	15.8	16.6	17.0	25.9	34.0	8.9 (6.5-11.3)	8.1 (3.7-12.5)
	20-39	9.3	11.2	12.3	20.6	28.4	8.3 (5.2-11.4)	7.8 (2.5-13.1)
	40-59	18.5	19.7	20.4	30.4	37.8	10.0 (6.1-13.9)	7.4 (0.5-14.3)
	60-74	26.2	23.4	21.3	28.6	39.6	7.3 (3.9-10.6)	11.0 (4.6-17.4)

*NHES indicates National Health Examination Survey; NHANES, National Health and Nutrition Examination Survey; and CI, confidence interval.
†Estimated prevalences for ages 20-74 years were age-standardized by the direct method to the 2000 Census population using age groups 20-39, 40-59, and 60-74 years.
‡Overall and within each age-sex group, the changes between 1988-1994 and 1999-2000 are not significantly different from the changes between 1976-1980 and 1988-1994.

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Bibliography

Ainslie Deborah, Joseph Proietto, Barbara C. Fam, and Ann W. Thorburn
1997 Short-term, High-fat Diets Lower Circulating Leptin Concentrations in Rats. American Journal of Clinical Nutrition 71: 438-442.

Alaimo Katherine, Christine M. Olson and Edward A. Frongillo
1999 Low Family Income and Food Insufficiency in Relation to Overweight in US Children. Arch. Pediatr. Adolec Med. 155: 1161-1166.

Basiotis Peter P., Andrea Carlson, Shirley A. Gerrior, WenYen Juan, and Mark Lino
2004 The Healthy Eating Index, 1999-2000: Charting Dietary Patterns of Americans. Family Economics and Nutrition Review 16(1): 39-48.

Basiotis, P. Peter and Mark Lino
2003 Food Insufficiency and Prevalence of Overweight Among Adult Women. Family Economics and Nutrition Review 15(2): 55-57.

Bell Elizabeth A., Liane S. Roe, Barbara J. Rolls
2003 Sensory-specific Satiety is Affected More by Volume than by Energy Content of a Liquid Food. Physiology & Behavior 78: 593-600.

Bell Elizabeth A. and Barbara J. Rolls
2001 Energy density of foods affects energy intake across multiple levels of fat content in lean and obese women. American Journal of Clinical Nutrition 73:1010–1018.

Bell Elizabeth A., Victoria H Castellanos, Christine L Pelkman, Michelle L Thorwart, and Barbara J Rolls
1998 Energy density of foods affects energy intake in normal-weight Women. American Journal of Clinical Nutrition 67: 412-420.

Besharov D.
2003 Growing Overweight and Obesity in America: the potential role of federal nutrition programs. Testimony before the Senate Committee on Agriculture, Nutrition and Forestry, Apr 3, 2003.

Center for Disease Control (CDC)
1996 Nutritional status of children participating in the Special Supplemental Nutrition Program for Women, Infants, and Children United States, 1988-1991 Morbidity and, Mortality Weekly Rep, 45(3): 65-9

Centers for Disease Control and Prevention pediatric nutrition surveillance, 1983 to 1995 Pediatrics, 101(1): E12 Center for Disease Control (CDC)

1996 Nutritional status of children participating in the Special Supplemental Nutrition Program for Women, Infants, and Children--United States, 1988-1991. Morbidity and, Mortality Weekly Rep, 45(3): 65-9.

Center on Hunger & Poverty

2000 Child hunger, child obesity: an examination of the paradox. Tufts University: Bedford, MA.

Chan Jean L., Giuseppe Matarese, Greeshman K. Shetty, Patricia Raciti, Iosif Kelesidis, Daniela Aufiero, Veronica De Rosa, Francesco Perna, Silvia Fontana, and Christos S. Mantxoros.

2005 Differential Regulation of Metabolic, Neuroendiocrine, and Immune Function by Leptin in Humans. Proceedings of the National Academy of Sciences of the United States of America 103(22): 8481-8486.

Cole N.

2001 The prevalence of overweight among WIC children WIC-010PCOM US Department of Agriculture, Food and Nutrition Services, Office of Analysis, Nutrition and Evaluation.

Condrasky, Margaret and Janet Marsh

2003 Food Stamps and Dietary Intake of Low-income Women in the Rural South in the Time of Welfare Reform. Topics in Clinical Nutrition 20(4): 366-373.

Considine Robert V., Madhur K. Sinia, Mark L. Heiman, Aidas Kriauciunas, Thomas W. Stephens, Mark R. Nyce, Joanna P. Ohannesian, Cheryl C. Marco, Linda J. McKee, Thomas L. Baur, and José F. Caro.

1996 Serum Immunoreactive-Leptin Concentrations in Normal-Weight and Obese Human. The New England Journal of Medicine 334: 292-295.

Crooks, Deborah L.

1998 Poverty and Nutrition in Eastern Kentucky: The Political Economy of Childhood Growth. In Building a New Biocultural Synthesis : Political-Economic Perspectives on Human Biology. Alan H. Goodman and

Cucó G., V. Arija, C. Marti-Henneberg, and J. Ferdández-Ballart

2001 Food and Nutritional Profile of High Energy Density Consumers in an Adult Mediterranean Population. European Journal of Clinical Nutrition 55: 192-199.

Darfour, Darna L.
2002 Biocultural Approaches in Human Biology. American Journal of Human Biology 18: 1-9.

Darmon Nicole, Elaine Ferguson, and André Briend
2003 Do Economic Constraints Encourage the Selection of Energy Dense Diets. Appetite 41: 315-322

Darmon Nicole, Elaine L. Ferguson, and André Briend.
2002 A Cost Constraint Alone Has Adverse Effects on Food Selection and Nutrient Density: An Analysis of Human Diets by Linear Programming. Journal of Nutrition 132: 3764–3771.

DeCastro John M.
1990 Social Facilitation of Duration and Size but not Rate of Spontaneous Meal Intake in Human. Physiology & Behavior 47: 1129-1135.

Dimitriou Triantafillia, Christiane Maser-Gluth, and Thomas Remer
2003 Adrenocortical Activity in Health Children is Associated with Fat Mass. American Journal of Clinical Nutrition 77: 731-736.

Drewnowski Adam, and Ann Hoisington
2005 Poverty Linked to Obesity, Say New Research. Diverse: Issues in Higher Education 22(15): 16-16.

Drewnowski, Adam and Nicole Darmon
2005 Food Choices and Diet Costs: an Economic Analysis Journal of Nutrition 135: 900–904.

Drewnowski Adam and SE Spector
2004 Poverty and Obesity: the Role of Energy Density and Energy Costs. American Journal of Clinical Nutrition 79: 6-16.

Drewnowski Adam, Nicole Darmon, and Andre Briend
2004 Replacing Fats and Sweets With Vegetables and Fruits—A Question of Cost. American Journal of Public Health 94:1555-1559.

Drewnowski, Adam
2003 Fat and Sugar: An Economic Analysis. American Society for Nutritional Sciences 133: 838S-840S.

Drewnowski Adam, and Allen S. Levine
2003 Sugar and Fat-From Genes to Culture. American Society for Nutrition Sciences 133: 829S-830S.

Drewnowski, Adam
1998 Energy Density, Palatibility, and Satiety: Implications for Weight Control. Nutrition Reviews 56(12): 347-351.

Dulloo Abdul G, and Jean Jacquet
1998 Adaptive Reaction in Basal Metabolic Rate in Response to Food Deprivation in Humans: A role for Feedback Signals from Fat Storage. American Society for Clinical Nutrition 68: 599-606.

Dulloo Abdul G., Jean Jacquet, and Lucien Girardier
1997 Poststarvation Hyperphagia and Body Fat Overshooting in Humans: A Role for Feedback Signals from Lean and Fat Tissues. American Journal of Clinical Nutrition 65: 717-723.

Dulloo Adbul G., and Lucien Girardier
1990 Adaptive Changes in Energy Expenditure During Refeeding Following Low-calorie Intake: Evidence for a Specific Metabolic Component Favoring Fat Storage. American Journal of Clinical Nutrition 52: 415-420.

Flegal Katherine M., Margaret D. Carrol, Cynthia L. Odgen, Clifford L. Johnson
2002 Prevelance and Trends in Obesity Among U.S. Audlts 1999-2000. The Journal of American Medical Association 288(14): 1723-1727.

Flegal KM, MD Carroll, RJ Kuczmarski, and CL Johnson
1998 Overweight and Obesity in the United States: Prevelance and Trends, 1960-1994. Journal of American Medical Association 22(1): 39-47.

French, Simone
2003 Pricing Effects on Food Choices. American Society for Nutrition Sciences 133: 841S-843S

Frisancho, Roberto A.
2003 Reduced Rate of Fat Oxidation: A Metabolic Pathway to Obesity in the Developing Nations. American Journal of Human Biology 15: 522-532.

Froidevaux Françoise, Yves Schutz, Laurent Christin, and Eric Jéquier
1993 Energy Expenditure in Obese Women Before and During Weight Loss, After Refeeding, and in the Weight-Relapse Period. American Journal of Clinical Nutrition 57: 35-42.

Gibson, Diane
2006 Long-term Food Stamp Program Participation is Positively Related to Simultaneous Overweight in Young Daughters and Obesity in Mothers Journal of Nutrition 136:1081-1085.

Gibson, Diane
2004 Long-term food stamp program participation is differentially related to overweight in young girls and boys. Journal of the American Society for Nutritional Sciences 134(2): 372-379

Gibson, Diane
2003 Food Stamp Program Participation is Positively Related to Obesity in Low Income Women Journal of Nutrition 133: 2225-2231.

Gibson, Diane
2001 Food Stamp Program Participation and Health: Estimates from the NLSY97 in Social Awakening: Adolescent Behavior as Adulthood Approaches, edited by Robert T. Michael. Russell Sage Foundation, New York, 2001. 258-295.

Glanz Karne, Michael Basil, Edward Maibach, Jeanne Goldberg, and Dan Snyder
1998 Why American Eat What They Do: Taste, Nutrition, Cost, Convenience, and Weight Control Concerns as Influences on Food Consumption. Journal of the American Dietetic Association 98(10): 1118-1126.

Goodman Alan H, and Thomas L. Leatherman
1998 Traversing the Chasm between Biology and Culture: An Introduction. In Building a New Biocultural Synthesis : Political-Economic Perspectives on Human Biology. Alan H. Goodman and Thomas L. Leatherman, eds. Pp. 3-33. Ann Arbor : University of Michigan Press.

Gorbach Sl, A. Morrill-Labrode, MN Woods, JT Dwyer, WD Selles, M. Henderson, W. Insull, S. Goldman, D. Thompson, C. Cliffors, L. Sheppard.
1980 Changes in Patterns During a Low-fat Dietary Intervention in Women. Journal of American Dietic Association 77: 140-148. S

Gotch Pamela Miller
1981 Teaching Patients About Adrenal Corticosteroids. American Journal of Nursing 78-81.

Hamilton WL. and Rossi, PH
2002 Effect of Food Assistance and Nutrition Programs on Nutrition and Health: Volume 1, Research Design. Food Assistance and Nutrition Research Report No. 19-1. U.S. Department of Agriculture, Economic Research Service.

Havel Peter J., Raymond Townsend, Leslie Chaump, and Karen Teff
1999 High-fat Meals Reduced 24-h Circulating Leptin Concentrations in Women. Diabetes 48: 334-341.

Hays Niicolas, Gaston P. Bathalon, Megan McCrory, Ronenn Roubenoff, Ruth Lipman, and Susan B. Roberts
2002 Eating Behavior Corrlates of Adult Weight Gain and Obesity in Health Women Aged 55-65y. American Journal of Clinical Nutrition 75:476-483.

Horowitz Jeffrey, Simon W. Coppack, and Samuel Klein
2001 Whole-body and Adipose Tissue Glucose Metabolism in Response to Short-term Fasting in Lean and Obese Women. American Journal of Clinical Nutrition 73: 517-522.

Jenkins A.B., T.P. Markovic, A. Fleury, and L.V. Campbell
1997 Carbohydrate Intake and Short-term Regulation of Leptin in Human. Diabetologia 40: 348-351.

Jones S, Jahns L, Laraia B, and B Haughton
2003 Lower risk of overweight in school-aged food insecure girls who participate in food assistance: results from the panel study of income dynamics child development

Kant, Ashima K.
2000 Consumption of Energy-Dense, Nutrient-Poor Foods by Adult Americans: Nutritional and Health Implication: The Third National Health and Nutrition Examination Survey, 1988-1994. American Journal of Clinical Nutrition 72: 929-936.

Keatney, J.M. and S. McElhone
1999 Perceived Barriers in Trying to Eat Healthier- Results of a pan-EU Consumer Attitudinal Survey. British Journal of Nutrition 81 (Supp 2): S133-S137.

Keim Nancy L., Judith S. Stern, and Peter J. Havel
1998 Relation Between Circulating Leptin Concentrations and Appetite During a a Prolonged, Moderate Energy Deficit in Women. American Journal of Clinical Nutrition 68: 794-801.

Kendall Anne, David A. Levitsky, Barbara J. Strupp, and Lauren Lissner
1991 Weight loss on a low-fat diet: consequence of the imprecision of the control of food intake in humans. American Journal of Clinical Nutrition 53: ll24-1129.

Kennedy, Eileen T.
2005 The Global Face of Nutrition: What can Governments and Industry Do? American Society for Nutrition Sciences 135: 913-915.

Kennedy, Eileen
2004 Dietary Diversity, Diet Quality, and Body Weight Regulation. Nutrition Reviews 62(7): S78-S81.

Kuczmarski RJ, Flegal KM, Campbell SM, Johnson CL.
1994 Increasing prevalence of overweight among US adults: the National Health and Nutrition Examination Surveys, 1960 to 1991. Journal of American Medical Aassociation 272:205-211.

Lang, Susan S.
1998 Food Insecurity Can Lead to Binge Eating, Poor Diet. Human Ecology 26(1): 24-24.

Lazreg Taheni Ben, Monia Zaouali, Molka Chaieb, Nada Dallel, Larbi Chaieb, Zouhaier Tabka, and Abdelkarim Zbidi
2007 Circadian Leptin, Insulin and Cortisol Rhythms in Obese Subjects. Biological Rhythms Research 38(1): 9-17.

Leatherman Thomas and Alan H. Goodman
1999 Expanding the Biocultural Synthesis Toward a Biology of Poverty. American Journal of Physical Anthropology 102: 1-3.

Leatherman, Thomas
2005 A Space of Vulnerability in Poverty and Health: Political-Ecology and Biocultural Analysis. Ethos 33(1): 46-70.

Ledikwe Jenny H., Heidi M. Blanck, Laura Kettel Khan, Mary K. Serdula, Jennifer D. Seymour, Beth C. Tohill, and Barbara J. Rolls
2006 Dietary Energy Density is Associated with Energy Intake and Weight Status in US Adults. American Journal of Clinical Nutrition 83: 1362-1368.

Levine Allen S., Catherine M. Knotz, and Blake A. Gosnell
2003 Sugars and Fats: The Neurobiology of Preference. American Society for Nutrition Sciences 133: 831S-834S.

Lloyd Helen, C.M. Paisley, David J. Mela
1995 Barriers to the Adoption of Reduced-Fat Diets in UK Populations. Journal of the American Dietetic Association 95(3): 316-322.

Marshall David, and Rick Bell
2003 Meal Construction: Exploring the Relationship Between Eating, Occasion and Location. Food Quality and Preference 14: 53-64.

Matkovic V., J.Z. Ilich, E. Badenhop, M. Skugor, A. Clairmont, D. Klisovic, and J.D. Landoll.
1997 Gain in Body Fat is Inversly Related to the Noctunal Rise in Serum Leptin Levels in Young Females. Journal of Clinical Endocrinology 82(5): 1368-1372.

McCann BS, BM Retzlaf, AA Dowdy, CE Walden, RH Knopp.
1990 Promoting adherence to Low-fat, Low-cholesterol Diets: Review and Recommendations. Journal of American Diet. Association 90: 1408-1414.

Mei Z, Scanlon K, Grummer-Strawn L, Freedman D, Yip R, Trowbridge F.
1998 Increasing prevalence of overweight among US low-income preschool children: the Dietary-effects of the national school lunch program and the school breakfast program American Journal of Clinical Nutrition, 61(1): S221-S231

Melnik T, Rhoades S, Wales K, Cowell C, and W Wolfe
1998 Overweight schoolchildren in New York City: prevalence estimates and characteristics International Journal of Obesity and Related Metabolic Disorders, 22(1): 7-13

Milligan Christine, Amanda Bingley, and Anthony Gatrell
2005 Digging Deep: Using Dietary Techniqures to Explore the Place of Health and Well-Being Amongst Older People. Social Science & Medicine 61: 1882-1892.

Neumark-Sztainer Dianne, Peter J. Hannan, Jillian Croll
2002 Overweight Status and Eating Patterns Among Adolescents: Where Do Youths Stand in Comparison with the Healthy People 2010 Objectives? American Journal of Public Health 92(5): 844-851.

Olson, Christine
2000 Nutrition and Health Outcomes Associated with Food Insecurity and Hunger. American Society for Nutritional Sciences 129: 521S-524S.

Olson C.
1999 Nutrition and health outcomes associated with food insecurity and hunger. Journal of Nutrition, 129(2S Suppl): 521S-524S.

Popkin, Barry M.
2006 Technology, Transport, Globalization, and the Nutrition Transition Food Policy. Food Policy 31: 554-569.

Popkin BM, and P. Gordon-Larsen
2004 The Nutrition Transition: Wordwide Obesity Dynamics and their Determinants. International Journal of Obesity 28: S2-S9.

Popkin, Barry M.
2004 The Nutrition Transition: An Overview of World Patterns of Changes. Nutrition Review 62(7):S140-S143

Popkin Barry M.
2003 An Overview on the Nutrition Transition and its Health Implication: The Bellagio Meeting. Public Health Nutrition 5(1A): 93-103.

Popkin Barry M. and Samara Joy Nielson
2003 The Sweetening of the World's Diet. Obesity Research 11(11): 1325-1332.

Popkin, Barry M
2001 Nutrition in Transition: The Changing Global Nutrition Challenge. American Journal of Clinical Nutrition 10(Supplement): S13-S18.

Poppitt S.D. and A.M. Prentice
1996 Energy Density and its Role in the Control of Food Intake: Evidence from Metabolic and Community Studies. Appetite 26: 153-174.

Reinehr Thomas and Werner Andler
2004 Cortisol and its Relation to Insulin Resistance Before and After Weight Loss in Obese Children. Horm. Res. 62: 107-112.

Rohner-Jeanrenaud, Françoise
1996 Obesity, Leptin, and the Brain. The New England Journal of Medicine 334(5): 324-325.

Rolls Barbara J., Julia A. Ello-Martin, and Beth Carlton Tohill
2004 What Can Intervention Studies Tell Us About the Relationship between Fruit and Vegetable Consumption and Weight Management. Nutrition Reviews 62(1): 1-17.

Rolls BJ, and EA Bell
1999 Intake of Fat and Carbohydrate: Role of Energy Density. European Journal of Clinical Nutrition 53 (Suppl 1): S166-S173.

Rolls Barbara J., Victoria H. Castellanos, Jason C. Halford, Arun Kilara, Dinakar Panyam, Christine L. Pelkman, Gerard P. Smith, and Michelle L. Thorwart
1998 Volume of Food Consumed Affects Satiety in Men. American Journal of Clinical Nutrition 67: 1170-1177.

Roman M, P. Lebel, C. Velly, N. Mareczux, J.C. Fruchart, and J. Dallongeville
2000 Leptin Response to Carbohydrate or Fat Meal and Association with Subsequent Satiety and Energy Intake. American Physiological Society-Endocrinology and Metabolism 277(40): E855-E861.

Romon M., P. Lebel, C. Velly, N. Marecaux, J.C. Fruchart and J. Dallongeville
1999 Leptin Response to Carbohydrate or Fat Meal and Association with Subsequent Satiety and Energy Intake. American Journal of Physiology-Endocrinology and Metabolism 277: 855-861.

Rosch, Paul J.
2004 All Obesity is not Created Equal. Science 301(5638): 1325-1326.

Row, K, Rettammel, A.
2003 Obesity and Food Insecurity-Is There a Relationship? Research Summary. August 2003.

Saltzman, Gerard E. Dallal, and Susuan B. Roberts
1997 Effect of High-fat and Low-fat diets on Voluntary Energy Intake and Substrate Oxidation: Studies in Identical Twins Consuming Diets Matched for Energy Density, Fiber, and Palatability. American Journal of Clinical Nutrition 66: 1332-1339.

Sarlio-Lahteenkorva S, Lahelma, E.
2001 Food insecurity is associated with past and present economic disadvantage and body mass index. Jorunal of Nutrition, 131(11): 2880-4.

Scarpace Philip J, Michael Matheny, Sergei Zolotukhin, Nihal Tümer, and Yi Zhang
2003 Leptin-Induced Leptin Resistant Rats Exhibit Enhanced Responses to the Melanocortin Agonist MT II. Neuropharmacology 45: 211-219.

Scarpace Philip J., Michael Matheny, Yi Zhang, Eugene W. Shek, Victor Prima, Segei Zolotukhin, and Nihal Tümer
2002 Leptin-Induced Leptin Resistance Reveals Separate Roles for the Anorexic and Thermogenic Responses in Weight Maintenance. Endocrinology 143(8): 3026-3035.

Sinha Madhur K., Joanna P. Ohannesian, Mark L. Heimanm, Aidas Kriauciunas, Thonas W. Stephens, Susan Magosin, Cheryl Marco, and Jose F. Caro.
1996 Noctural Rise of Leptin in Lean, Obese, and Non-Insulin Dependant Diabetes Mellitus Subjects. Journal of Clinical Investigation 97(5): 1344-1347.

Stookey, JD
2001 Energy Density, Energy Intake and Weight Status in a Large Free-Living Sample of Chinese Adults: Exploring the Underlying Role of Fat, Protein, Carbohydrate, Fiber, and Water Intakes. European Journal of Clinical Nutrition 55: 349-359.

Stricker-Krongrad A., F. Cumin, C. Burlet, and B. Beck.
1998 Hypothalamic Neuropeptide Y and Plasma Leptin After Long-term High-fat Feeding in the Rat. Neuroscience 254: 157-160

Stroebele Nanette, and John M. de Castro
2006 Influence of Physiological and Subjective Arousal on Food Intake in Humans. Nutrition 22: 996-1004.

Stubbs James R., Christopher G. Harbron, Peter R. Murgatroyd, and Andrew M. Prentice
1995 Covert Manipulation of Dietary Fat and Energy Density: Effect on Substrate Flux and Food Intake in Men Eating ad libitum. American Journal of Clinical Nutrition 62: 316-329.

Stuff Janice, Patrick H. Casey, Kitty L. Szeto, Jeffrey M. Gossett, James M. Robbins,
2005 Pippa M. Simpson, Carol Connell and Margaret L. Bogle. American Society for Nutritional Sciences 134: 2330-2335.

Swinburn BA, I Caterson, JC Seidell, and WPT James
2004 Diet, Nutrition, and the Prevention of Excess Weight Gain and Obesity. Public Health Nutrition 7(1A0: 123-146.

Thomas, R. Brooke
1998 The Evolution of Human Adaptatbility Paradigms: Toward a Biology of Poverty. In Building a New Biocultural Synthesis : Political-Economic Perspectives on Human Biology. Alan H. Goodman and Thomas L. Leatherman, eds. Pp. 43-71. Ann Arbor : University of Michigan Press.

Townsend M, Peerson J, Love B, Achterberg C, and Murphy S.
2001 Food insecurity is positively related to overweight in women. Journal of Nutrition, 131(6): 173-145.

Townsend Marilyn, Janet Peerson, Bradley Love, Cheryl Acterberg, and Suzanne P. Murphy.
2001 Food Insecurity is Positively Related to Overweight in Women. Top. Clin. Nutr. 20(4): 366-373.

VanEenwyck J, and Sabel J.
2003 Self-reported concern about food security associated with obesity—Washington 1995-1999. Morbid Mortal Weekly Rep, 52(35): 840-842.

Whitten, Patricia.
1999 Diet, Hormones, and Health: An Evolutionary-Ecological Perspective. In Hormones, Health, and Behavior: A Socio-Ecological and Lifespan Perspective. C. Panter-Brick and C.M. Worthman, eds. Pp 210-235. New York: Cambridge University Press.

Wilsey J., and PJ Scarpace
2004 Caloric Restriction Reverses the Deficits in Leptin Receptor Protein and Leptin Signaling Capacity Associated with Diet-Induced Obesity: Role of Leptin in the Regulation of Hypothalamic Long-form Leptin Receptor Expression. Journal of Endocrinology 181: 297-306.

Wilsey Jared, Sergei Zolotukhin, Victor Prima, and Phillip Scarpace
2003 Central Leptin Gene Therapy Fails to Overcome Leptin Resistance Associated with Diet-Induced Obesity. American Journal of Physiol. Regul. Integer. Comp. Physiol. 285: R1011-1020.

Wisse, Brent E, L. Arthur Campfield, Errol B. Marliss, José A Morais, Renata Tenenbaum, and Réjeanne Gougeon
1999 Effect of Prolonged Moderate and Sever Energy Restriction and Refeeding on Plasma Leptin Concentrations in Obese Women. American Journal of Clinical Nutrition 70: 321-330.

Wilde, P, McNamara, P. and. Ranney C.
2000 The effect on dietary quality of participation in the food stamp program and WIC programs. Food Assistance and Nutrition Research Report Number Prepared for the Food and Rural Economics Division, Economic Research Service, U.S. Department of Agriculture.

Webb Patrick, Jennifer Coates, Edward A. Frongillo, Beatrice Lorge Rodgers, Anne Swindels, and Paula Bilinsky.
2006 Measuring Household Food Insecurity: Why It's So Important and Yet So Difficult to Do Journal of Nutrition 136:1404S-1408.

Wolfe W, Campbell C, Frongillo E, Haas J, and T Melnik
1994 Overweight schoolchildren in New York State: prevalence and characteristics American Journal of Public Health, 84(5): 807-

World Health Organization.
1990 Diet, Nutrition, and the Presentation of Chronic Disease: Report of a WHO study group. Technical Report Series No. 797. Pp.1-203. Geneva: WHO Press.

Yanovski, Susan
2003 Sugar and Fat: Cravings and Aversions. American Society for Nutrition Sciences 133: 835S-837S.

Zhang Yi and Philip J. Scarpace
2005 The Role of Leptin in Leptin Resistance and Obesity. Physiology and Behavior 88(3): 249-256.

Zhao Zi-Yan, Yue-Rong Fu, Xin-Hua Li, Yu-Yang Li, Andre Bogdan, Yvan Touitou.
2002 Age-Realted Modifications of Circadian Rhythm of Serum Leptin in Healthy Men. Gerontology 48: 309-314.