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Comparison of food security status, nutrient intakes, body mass index, and multiple diseases among self-reported depressed and non-depressed female food stamp recipients in Southeast Louisiana

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COMPARISON OF FOOD SECURITY STATUS, NUTRIENT INTAKES, BODY MASS
INDEX, AND MULTIPLE DISEASES AMONG SELF-REPORTED DEPRESSED AND
NON-DEPRESSED FEMALE FOOD STAMP RECIPIENTS IN SOUTHEAST LOUISIANA

A Thesis
Submitted to the Graduate Faculty of the
Louisiana State University and
Agricultural and Mechanical College
in partial fulfillment of the
requirements for the degree
of Master of Science

in

The School of Human Ecology

by
Yifang Bai
B.S., Peking Union Medical College, 1998
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LIST OF ACRONYMS

Ad=Adrenaline

BDI=Beck Depression Inventory

BDNF=Brain-Derived Neurotrophic Factor

BH₄=Tetrahydrobiopterin

BMI=Body Mass Index

BP=Blood Pressure

cAMP=Adenosine 3',5'-Cyclic Monophosphate

CCHIP=Community Childhood Hunger Identification Project

CES-D=Center for Epidemiologic Studies Depression Scale

CHD=Coronary Heart Disease

CREB=cAMP-Response Element-Binding Protein

CSF=Cerebrospinal Fluid

CSFII=Continuing Survey of Food Intake by Individuals

DHA=Docosaehaenoic Acid

DRG=Dorsal Root Ganglion

DSM-IV=Diagnostic and Statistical Manual of Mental Disorders

ER β =Estrogen Receptor β

FS=Food Secure

FIS=Food Insecure

FISH=Food Insecure with Hunger

FSETP=Food Stamp Employment Training Program

FSH=Follicle Stimulating Hormone

FSP=food Stamp Program

GA=General Assistance Program

GNB3=G-Protein β 3-Subunit

HAM-D=Hamilton Rating Scale

HbA_{1C}=Glycohemoglobin

HEI=Healthy Eating Index

5-HIAA=5-hydroxyindoleacetic acid

HOPA=the Human Opposite Paired-Containing Gene

HPG=Hypothalamic-Pituitary-Gonadal

5-HT=5-hydroxytryptophan

5-HT_{1A}=5-Hydroxytryptamine (Serotonin 1A)

5-HTT=Serotonin Transporter

HVA=homovanillic acid

IDO=indoleamine 2, 3-dioxygenase

IL-1 β =Interleukin-1 β

IL-2=Interleukin-2

IL-6=Interleukin-6

IFN- γ =Interferon- γ

IFN- α =Interferon- α

LA=Louisiana

L-DOPA=dihydroxyphenylalanine

LNAA=Large Neutral Amino Acids

MADRS=Montgomery-Åsberg-Depression Rating Scale

MCS=Mental Component Summary

MDD=Major Depressive Disorder

MDE=Major Depressive Episode

MHPG=3-Methoxy-4-Hydroxyphenyl Glycol

MI=Myocardial Infarction

5MTHF=5, 10-methylenetetrahydrofolate

NA=Noradrenaline

NAD⁺=Nicotinamide Adenine Dinucleotide

NADP⁺=Nicotinamide Adenine Dinucleotide Phosphate

NCS=the National Comorbidity Survey

NHANES III=the Third National Health and Nutrition Examination Survey

NMDA=N-methyl-D-aspartate

NMHA=National Mental Health Association

NO=Nitric Oxide

NSDUH=National Survey on Drug Use and Health

PCS=Physical Component Summary

PDE=Phosphodiesterase

PET=Positron Emission Tomography

POMS=Profile of Mood States

PUFA=Polyunsaturated fat

RBC=Red Blood Cell

SAM=S-adenosylmethionine

SERT=Serotonin Reuptake Transporter

SF-12=the Short Form 12-item Health Survey

SFA=saturated fatty acid

SSRI=selective serotonin Transporter Reuptake Inhibitor

TFP=Thrifty Food Plan

THF=Tetrahydrofolate

TDO=tryptophan 2, 3-dioxygenase

TPH=tryptophan hydroxylase

TRP=Tryptophan

USDA=United States Department of Agriculture

WIC=Special Supplemental Nutrition Program for Women, Infants, and Children

ABSTRACT

The objectives of this study were to explore the relationships among depression and food security status, dietary nutrient intakes, sociodemographic characteristics, body mass index (BMI), and the presence of chronic diseases in a female food stamp recipient population (n = 66) in Southeast Louisiana. Women were dichotomized by stated depression and descriptive statistics on socioeconomic characteristics and mean nutrient intakes were presented for each group. Logistic regression models were used to determine the relationship of stated depression with food security status, selected sociodemographic characteristics, nutrient intakes, body mass index, and the number of chronic diseases reported.

The percentage of women with reported depression was 31.8%. A strong relationship was observed between depression and the number of chronic diseases reported ($p = 0.005$). Women with stated depression had more physical chronic diseases reported than those without stated depression. The majority of study participants were unemployed (68.18%), and the odds of stated depression for unemployed women was four times higher than employed women ($p = 0.05$). Food security status was classified into three categories, that is, food secure, food insecure, and food insecure with hunger. For the depressed women, 52.4% were food secure; 38.1% were food insecure; 9.5% were food insecure with hunger. No relationship was found between depression and food security status. Low intake of folate and iron was common in both depressed and non-depressed women. No relationship was found between depression and nutrient intake (e.g., energy, protein, carbohydrate, fat, folate, vitamin B₁₂, or iron). The mean BMI of both depressed and non-depressed groups fell within the obese range. No relationship was found between depression and BMI. We also failed to find relationships between depression and marital status or medical insurance.

CHAPTER 1

INTRODUCTION

Introduction

Depression is an “illness that involves the body, mood, and thought” (1). Depression includes major depressive (MDD), dysthymic, and bipolar disorders (2). The most common type of depression is MDD (3). The annual cost of depression is \$83 billion in the U.S. (4). Results from the Global Burden of Disease Study showed that by 2020 MDD will be a leading cause of disability, second only to ischaemic heart disease (5).

Depression is closely related to sociodemographic characteristics. Depression in females (10.3%) is almost twice as common as in males (5.6%) (6). Poverty increases the risk of depression. Eleven percent of adults (aged 18 or older) from households with a family income less than \$20,000 had a major depressive episode (MDE) in their life time, whereas only 7.0% of adults with a family income above \$50,000 had MDE (6). Unemployment (7), low education level (8), and family fragmentation (9, 10, 11) also compromise mental health.

Food insecurity is common in households with incomes below the federal poverty line, households with children, or households headed by a single woman (12). Inadequate food intake or poor food choices may directly put low-income women at risk for nutrient deficiencies (13-18); food insecurity is a stressful life event (17, 18) and may lead to depression. Nutrient deficiencies and lifetime stress events can undermine mental health. Vitamin and amino acid deficiencies, such as vitamin B₁₂, folate (19-22), and S-adenosylmethionine (SAM) (22, 23), have been reported in depressed patients.

Food insecurity compromises not only mental health but also physical health (24, 25). Individuals from food-insufficient households report a high prevalence of chronic diseases such

as heart disease, diabetes, hypertension, food allergies (25), and obesity (26, 27). Post-chronic disease depression is very common and reduces the quality of life in affected individuals.

Objectives

The objectives of this project were to determine the following in female food stamp recipients in Southeast Louisiana (LA):

1. The association between stated depression and food security status.
2. The independent association between dietary energy and nutrient intakes (protein, carbohydrate, fat, folate, vitamin B₁₂, and iron) and depression.
3. The relationship between depression and BMI.
4. The relationship between depression and the number of chronic diseases.

Hypotheses

It was hypothesized that:

Ho1: Food insecurity does not increase the risk and prevalence of depression.

Ho2: Subjects with depression have low intakes of protein, folate, vitamin B₁₂, carbohydrate, and iron, and high intake of saturated fats.

Ho3: BMI has no difference between stated depressed and non-depressed subjects.

Ho4: An increased self-reported number of chronic diseases is associated with the increased prevalence and the risk of depression.

Assumptions

The assumptions of this study were:

1. The modified United States Department of Agriculture (USDA) short form is a valid instrument to measure food security.
2. Twenty four hour recalls reflected typical intake.

3. Subjects provided honest responses to the household surveys and questions about sociodemographic characteristics.

Limitations

Limitations of this study included:

1. The sample size was small.
2. This cross-sectional study cannot provide causal evidence on the association between nutrient intakes or other variables and depression.
3. All data were self reported; subjects may have had biased responses or they could misunderstand the meaning of depression or that kind of thing.
4. The sample population was limited to female food stamp recipients in Southeast LA, and was almost all black; this limited the ability to generalize the results.
5. The 24-hour food recall depends on memory, and subjects are to under- or over-reporting.
6. No data were available for testing nutrient blood levels to confirm diet intakes.

Justification

Our research seeks to explore the association between diet and depression in a low income population in order to make recommendations about their diet and to improve their overall health status. This study is important for the following reasons. First, Sociodemographic characteristics can undermine mental health in a female food-stamp population. Second, food insecurity compromises physical and mental health (24, 25), so this study will enhance the literature about the relationship between depression and food insecurity, and the relationship between chronic diseases and depression. Third, since poor diet, as is seen in this population, may impact mental health, this study will advance our understanding of the association between

depression and nutrient intake. Fourth, food insecurity is associated with increased risk for obesity in women (26, 27). Most subjects in our study are overweight or obese.

CHAPTER 2

REVIEW OF THE LITERATURE

Depression

Definitions

Depression is “an illness that involves the body, mood, and thought.” The term “depression” is used for a variety of phenomena (1). Based on the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), depressive disorders are categorized into only major depressive disorder (MDD) and dysthymic disorder. The sign of MDD is one or more Major Depressive Episodes (MDE), which last at least 2 weeks in any time frame. During the episode, patients may experience depressed mood; loss of interest; changes in appetite, weight, and sleep; decreased energy; feelings of worthlessness; difficulty thinking, concentrating or making decisions; or increased thoughts of suicide. Dysthymic Disorder is characterized by minor symptoms of depression that last at least 2 years (2).

Depressive symptoms also can be found in other mood disorders. *Bipolar disorder* describes cycling mood changes and is usually accompanied by Major Depressive Episodes. *Adjustment disorder* is characterized by the development of significant emotional or behavioral symptoms in response to an identifiable psychological stressor or stressors. Symptoms must develop within 3 months after the onset of the stressor(s). The depressed mood can be observed in two subtypes of adjustment disorder, that is, adjustment disorder with depressed mood and adjustment disorder with mixed mood. *Mood disorder due to a general medical condition* is a mood disturbance that is due to the direct physiologic effects of a general medical condition. This disturbance must be confirmed by evidence from the history, laboratory findings, or physical examination. *Substance-Induced Mood Disorder* is one in which a substance (a

medication, a toxin) is related to the mood disturbance. *Minor depressive disorder* presents with at least 2 weeks of depressive symptoms but with fewer symptoms compared to MDD (2).

Causes of depression are complex (28). Altered monoamine metabolism (decreased norepinephrine, dopamine, and serotonin levels and activities) (29, 30), altered immune function (increased cytokine secretion) (31-34), an imbalance of cortisol, heredity, and external stressors and significant life changes (28) all contribute to the development of depression.

Prevalence

MDD is the most commonly studied form of depression by investigators. The National Comorbidity Survey (NCS) results (2001-2002 survey results) showed that the prevalence of MDD in general population (adults ≥ 18 years) for a lifetime was 16.2% (3). In 2005, National Survey on Drug Use and Health (NSDUH) results showed that an estimated 14.8% adults had experienced at least one MDE in their lifetime, and 8% had experienced a MDE in the past year (6). The overall risk of a depressive disorder has increased and the age of onset on average has decreased (8). The risk of MDD is fairly low until the early teens (3). By 2020 unipolar major depression is predicted to be the second leading cause of disability after ischemic heart disease (5).

The annual cost of depression in the U.S. remained relatively stable between 1990 (\$77.4 billion) and 2000 (\$83.1 billion). Although the treatment rate of depression increased by over 50%, its annual cost rose by only 17%. For 2000, direct medical costs were \$26.1 billion (31%), suicide-related mortality costs were \$5.4 billion (7%), and workplace costs were \$51.5 billion (62%) (4).

Depression and Sociodemographic Characteristics

Gender and Depression

The overall prevalence of psychiatric disorders is the same between men and women (35). However, depression in females (10.3%) was almost twice as likely as in males (5.6%) (6). Gender differences in rates of depression can be partially explained by biology (35). Studies show that low brain serotonin levels were associated with depression (29, 36), and the mean rate of serotonin synthesis in healthy males was 53% higher than in healthy females. This statistically significant difference may contribute to the lower prevalence of depression in males (37). The increased rate of depression in females emerges at puberty and declines after menopause, suggesting that sex hormones play an important role in the onset of depression (38). The prevalence of postpartum depression is 19.1%, and low economic status is a key element in the development of postpartum depression (39). Moreover, depressed patients were reported to have elevated plasma follicle stimulating hormone (FSH) levels during perimenopause suggesting a potential link between depression and endocrine events of perimenopause (40).

One hypothesis of increased prevalence of depression in women is that dysregulation between the hypothalamic-pituitary-gonadal (HPG) axis and serotonin during the premenstrual period, the postpartum period, and at the initiation of menopause is a key contributor to depression in women (38), because pregnancy, delivery, and perimenopause can result in dramatic changes in estrogen. Serotonin neurons expressed estrogen receptor β (ER β) in the central nervous system of murine and macaques (41, 42), and estrogen can increase tryptophan hydroxylase (TPH) mRNA expression via ER β (42). TPH is a rate-limiting enzyme for the synthesis of serotonin, suggesting that the serotonin neural system can transduce the action of estrogen via ER β on aspects of mood (41, 42).

Estrogen can increase the serotonin level in several ways. Using an ovariectomized rat model, it was found that ovariectomy led to a state of serotonin 1A (5-hydroxytryptamine, 5-HT_{1A}) inhibitory autoreceptor hyperactivity, which resulted in decreased serotonin release and neuronal firing. However, treatment with estradiol can correct the 5-HT_{1A} autoreceptor hyperactivity (43). Estrogen may increase the capacity for serotonin synthesis by selectively increasing tryptophan hydroxylase-2 mRNA expression. The higher the tryptophan hydroxylase mRNA expression observed, the lower the anxiety behavior expressed (44). Estrogen also decreases serotonin reuptake transporter (SERT) gene expression in all serotonin neurons (41). SERT inactivates the synaptic action of serotonin by reuptake of serotonin into the nerve terminals. So, this function will increase the quantity of serotonin in the neuron gaps because of a decreased number of SERTs. Impaired cognition is common in depression. Estrogen replacement can improve mood, have a positive effect on letter-cued verbal fluency, and protect verbal memory (45). Overall, biological variables play an important role in the development of depression in women. However, these factors do not adequately explain all causes of depression. In fact, biological, sociocultural, and personal contexts are all involved in the development of depression in females (33).

Race and Depression

Previous study results about the rates of depression in different racial populations have been inconsistent. Some researchers found that blacks had a higher prevalence of depressive symptoms than whites (46), whereas others found a higher prevalence in whites compared to blacks (3). Still others found no relationship between race and depression (47). Several investigators examined the relationship between race and depression while controlling for gender, age, and physical functioning, and found that blacks had a higher level of depressive symptoms than whites. However, when socioeconomic variables were included in analysis, the relationship

between race and depression reversed (48). Moreover, Dunlop et al. reported that among blacks, adjustment for sociodemographics, health needs, and economic differences sequentially reduced depression prevalence 17% (from 88.5 to 73.1/1000), 17% (to 58.1/1000), and 6% (to 52.3/1000), respectively (9). After controlling socioeconomic variables, some studies showed blacks had a low rate of depression (9, 48), but others revealed no relationship between race and depression (49, 50). At this time, we cannot say if different racial groups are at higher risk of depression.

Income and Depression

Women compose 70% of the world's 1.2 billion people who live in poverty. Working women are paid on average 30-50% less than men doing the same job and often work without minimum wage protection or benefits (access to pension plans, regular schedules, and health insurance) (35). In the U.S., the poverty rate has increased between 2000 and 2004. Particularly, African Americans were over-represented in severe poverty (51). In 2005, 38.2 million people in the U.S. (12.6%) were living in poverty, for blacks, it was 24.9% (52). Moreover, women have a greater risk of living in poverty than men, since women are paid on average less than men. For instance, Median income of men in the U.S. in 2005 was \$41,965, while for women; it was \$32,168, or 76.7% of men's earnings (53). The rate of poverty in women in the U.S. in 2005 was 14.1%, while it was 11.1% in men (54).

Women's mental health can be undermined by poverty (35). The rate of major depression among low-income women was about twice that of women in the general population (55). Absolute income level is related to depression. The 2005 NSDUH report revealed that lower income levels were associated with increased the rates of MDE. Adults from households with a family income of less than \$20,000 experienced the highest rate of MDE (11.0%) in the year

prior to the study, while adults from households with a family income of \$50,000 or more had lowest rate (7.0%) (6).

Income inequality or distribution of individual income is also associated with depression (56, 57). The underlying mechanism is thought to be that the direct and indirect effects of income inequality on individuals make them aware of their relative standings in the society and produce negative emotions (56, 57). Individuals living in counties with higher income inequality are more depressed compared with those in counties with lower income inequality (56, 57).

Unemployment and Depression

Loss of a job, which is an external stressor, can result in major depressive disorder or a form of depression known as adjustment disorder (2). The duration of unemployment is significantly linked with depressive mood. Results from a cross-sectional survey results showed that long-term unemployed individuals had more episodes of a depression than individuals with short-term unemployment. Being female or elderly, and having increased number of episodes of unemployment had significant effect on the development of depression while having a higher level of education and income buffer the risk of depression. Moreover, the duration of unemployment is also related to the severity of depressive symptoms, that is, long-term unemployed individuals showed more depressive symptoms than did short-term unemployed individuals (7).

Following job loss, a cascade of other secondary stressors and changes may be triggered and subsequently impact mental health (58). Inventory job loss and unemployment increase financial strain, which increases depression. Financial strain and depression directly affect personal control, which in turn has adverse impacts on health and role and emotional functioning (58). At the same time, depression reduces the likelihood of reemployment and increases

financial strain (58, 59). One population based study comprehensively assessed the work outcomes of employees with depression. At the six-month follow-up, individuals with depression had lost their jobs significantly more times than those without depression. The rates of new unemployment in the dysthymia group, major depression group, the group with both dysthymia and major depression, and control group were 14%, 12%, 15%, and 2%, respectively. Among individuals who were still employed at the six-month follow up, those with depression had increased job turnover (13% for the dysthymia group, 20% for the major depression group, and 13% for the group with both dysthymia and major depression) in comparison to the control group (5%). Among participants who experienced job turnover, 33% of participants with depression took a lower paying job for health reasons, compared with 20% of participants in the comparison group (59).

Education and Depression

A National Mental Health Association (NMHA) survey showed that lower educational level was associated with a higher rate of depression (8). Low-income women with 13 or more years of education had a prevalence of depression of 11% compared to more than 40% of women in the National Comorbidity Survey (this survey excluded homeless persons) (55). “Education is the key to power, health, better nutrition, higher economic well being, education of the whole family participation in development and social status among women”, although high educational achievement does not protect against depression (35).

Family Structure and Depression

The NMHA survey also revealed that respondents with depression had higher rates of divorce than respondents without depression (8). Other studies demonstrated that being widowed, divorced, and unmarried increased the likelihood of developing depression (9-11).

Single-parent families headed by females were more likely to experience chronic (low income, low levels of social support) and acute stress (major life events) compared with two-parent families headed by males. Some stress associated with the event of marital disruption was a short-term phenomenon. Females who were recently divorced, separated, and widowed were more likely to experience major life events (income changes, residential relocations, and household-composition changes) than those who had been single more than three years. Three years after marital disruption, the difference of life events between single and two-parent families was considerably smaller. That events continue to a lesser extent during the second and the third year, indicated that they were consequences of disruption not alternative indicators. In fact, two-parent families with an absence of marital disruption also experienced these events suggesting that life events were not necessarily associated with marital status (60). Female head of single-parent families were less likely to receive social supports than male heads in two-parent families (60). These findings may partially explain why family fragmentation is correlated with the development of depression, and having a partner, high self-esteem, mastery, self-efficacy and feeling less lonely can buffer depressive symptoms (61).

Food Insecurity and Depression

Food Security in the United States: Definitions and Prevalence

“Food secure means they had access, at all times, to enough food for an active, healthy life for all household members.” A recent food security survey revealed that 88.1% of American households were food secure during the calendar year 2004 (12).

Food insecure refers to “uncertain of having, or unable to acquire, enough food for all household members because they had insufficient money and other resources for food.” In 2004, 11.9% of U.S. households (13.5 million households) were food insecure; 38.2 million people

lived in food insecure households, including 13.9 million children. Households with incomes below the official poverty line (36.8%), households with children, headed by a single woman (33%), black households (23.7%), and Hispanic households (21.7%) had higher rates of food insecurity than the national average (11.9%). Regionally, food insecurity was more common in principal cities of metropolitan areas (15.4%) and nonmetro areas (13.1%). The South and West had higher rates of food insecurity (13.3% and 12.8%, respectively) than the Northeast and Midwest (9.7% and 10.9%, respectively). The food insecure with or without hunger in Louisiana (11.8%) was lower than the Southern and national averages (12).

“Food insufficiency refers to restricted household food stores or insufficient food intake.” (12) “The distinction between food insufficiency and food insecurity can best be understood from a temporal frame of reference: food insecurity can be experienced prior to the onset of food insufficiency, and may or may not result in food insufficiency” (12). The term “food insufficiency” was used in the late 1980s and early 1990s in national food surveys by the USDA. Now, food insecurity substitutes the term of food insufficiency, and is widely considered a core indicator of food hardship (62).

Hunger means “uneasy or painful sensation caused by lack of food” (62). Food insecurity with hunger was seen in approximately 3.9% of all U.S. households (one-third of food-insecure households). About 7.4 million adults and 3.3 million children experienced hunger during 2004 (12). The prevalence of food insecurity with hunger was much higher in families headed by single women (9.2%), black and Hispanic households (8.1% and 5.95%, respectively), and households below the poverty line (13.6%) than the national average (3.9%). Geographically, food insecurity with hunger was also more common in principal cities of metropolitan areas (4.7%) and in the South and West (4.5% and 4.2%, respectively) (12). Recent trends showed that

the prevalence of food insecurity (from 11.2% to 11.9%) and food insecurity with hunger (from 3.5% to 3.9%) increased from 2003 to 2004 (12).

Low-Income Household Food Security and Food Expenditure

Previous research highlights the positive association between food insecurity and sociodemographic characteristics of households and individuals including low income, single parenthood, race (African ancestry and Hispanic ethnicity), low education, lack of home ownership, lack of savings, recent changes in income, unemployment, poor health status, and social isolation (63). The prevalence of food insecurity among poor households (35.1%) is much higher than that among households with incomes above 1.85 times the federal poverty line (4.9%) (63). Low-income single mothers with children are often affected by food insecurity and hunger (47.9%) (12). However, “more than half of poor households are not considered food insecure, and equally important, more than half of food-insecure households are not poor.” These findings indicate that the determinants of food insecurity are complex (62, 63).

Usually, food-secure households spend more on food compared to food-insecure households. In 2004, median per person weekly food expenditure in the typical U.S. household was \$40. “The typical household usually spent 25 percent more on food than the cost of the Thrifty Food Plan (TFP) for its household type.” Among food-secure, food-insecure without hunger, and food-insecure with hunger households, median food spending relative to the TFP was 1.28, 0.99, and 0.96, respectively. This was true even for households below the poverty line. Among households with income below the poverty line, median food expenditures relative to the TFP were 0.85 for food-insecure households compared to 0.96 for food-secure households (12).

In order to meet spending constraints, low-income shoppers work to increase the amount of food purchased while paying less. They may purchase more discounted products, private-label

products (generic or store brand), promotional items, larger packages, and low-priced items within a product class. Low-income households may keep their food expenditures down by purchasing low quality items. These households purchased 7.6% more meat and poultry (by weight) than middle-income households and 6.7% more than high-income households, but paid less (6.2% less for meat and 5.5% less for the poultry) than did middle-income households. Moreover, low-income households purchased 3.3% less fruit and vegetables (by weight) per person than high-income households, but paid 13% less, because they select less expensive fruits and vegetables (13).

In addition to income, educational level and employment status also modify food purchasing behavior. The least educated and those employed in blue-collar occupation households purchased few fruits and vegetables (64). Households headed by someone with four or more years of college increased per capita spending on fruits and vegetables from \$5.55/week in 1991 to \$5.99/week in 2000. However, households headed by someone with or without a high school diploma, or with less than 4 years of college decreased expenditure on fruits and vegetables during this time. For households headed by someone with a college education, per person expenditure on fruits and vegetables was about 27% higher than the total U.S. average (65).

Henry et al. explored salient factors influencing fruit and vegetable choices among a group of low-income African American women (18 to 45 years of age). The majority of them were single mothers with children, and high school graduates. All received public assistance through the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC), welfare, food stamps, or free or reduced-price school meals for their children. Researchers found that preference, cost, specific needs, availability, seasonality, appearance, convenience,

nutrition/health, advice from others, and spoilage concerns were all involved in decision making about fruit and vegetable purchasing and preparation (66). Further, women in the later stages of this study had more skills and positive experiences with meal planning including fruits and vegetables, and had more concerns about nutrition and specific health issues than those in earlier stages (66).

Food Insufficiency and Low Nutrient Intakes

Inadequate food intake may directly put low-income women at risk of nutrient deficiencies (14-16). In 1997, Rose et al. analyzed the diets of adult women (aged 19-50 y) using 24-hour recall data from the 1989 through 1991 Continuing Survey of Food Intake by Individuals (CSFII). In that study, adult women with food insufficiency had significantly lower intakes of eight nutrients, including magnesium; calcium; protein; phosphorus; and vitamins A, E, C, and B₆ compared with food sufficient women (15). The Third National Health and Nutrition Examination Survey (NHANES III) analyzed both dietary intakes and serum nutrients in adults from food-insufficient and food-sufficient families. Findings revealed that adults (aged 20-59y) from food-insufficient families had lower intakes of calcium and vitamin E; lower one month frequency of consumption of milk/milk products, fruits/fruit juices and vegetables; and lower serum concentrations of total cholesterol, vitamin A and three carotenoids (α -carotene, β -cryptoxanthin and lutein/zeaxanthin). Older adults (aged ≥ 60 y) from food-insufficient families had lower intakes of energy, vitamin B₆, magnesium, iron, and zinc; and lower serum concentrations of high-density lipoprotein cholesterol, vitamin A, β -cryptoxanthin, and vitamin E. Both of these two groups of adults were more likely to have very low serum albumin ($< 35\text{mg/dL}$). Although low intakes and low serum concentrations of many nutrients and lower intakes of related food groups were common in adults from food-insufficient families, the dietary

intakes were weakly correlated with the serum nutrient levels except those observed for calcium and milk/milk products and for vitamin C and fruits/fruit juices (16).

Regardless of food-sufficiency status, most American diets need improvement (67). The 1999-2000 Healthy Eating Index (HEI) indicated that only 10% of the population had a good diet; 16% had a poor diet, and 74% needed improvement. Moreover, fewer than 50% of the population met the dietary recommendation for the grains, vegetables, fruits, milk, meat, total fat, saturated fat, and sodium. Fruits and milk were the two lowest components of the mean HEI scores (3.8 and 5.9 respectively). “The HEI, consisting of 10 components (each representing different aspects of a healthful diet), provides an overall picture of the type and quantity of foods people eat, their compliance with specific dietary recommendations, and the variety in their diets.” “Only 17 percent of the people consumed the recommended number of servings of fruit per day, and only 30 percent met the dietary recommendation for milk” (67).

The Impact of Food Insecurity on Depression

Food insecurity is a significant predictor of major depression (17, 24). A series of surveys from nine states and the District of Columbia by the Community Childhood Hunger Identification Project (CCHIP) reported that hungry children were more likely to receive special education services, and have a history of mental health counseling, fighting and stealing, and academic failure (68). Alaimo et al. analyzed data for adolescents (15 and 16 years) from NHANES III to explore the relationship between food insufficiency and depressive disorders. Results showed that food-insufficient individuals were four times more likely to have had dysthymia, and two times more likely to have had thoughts of death (18). These findings indicated that hunger may impair human’s psychosocial function.

There are two common explanations for the impact of food insufficiency on depression. First, food-insufficiency is a stressful life event which may be a potential pathway to depression (17, 18). Stressful life events are correlated to sustained circulating levels of cortisol (17), which alter mood, cognition, and behavior (17, 28). Second, limited food availability and poor diet quality contribute to depression (17, 18). Nutrient deficiency is very common in diets and low serum concentrations of nutrients are common in individuals with food-insufficiency (14-16). Some nutrient (essential amino acids, vitamin B₁₂, folate, iron, and zinc) deficiency may compromise mental health (19-21, 69-71, 136, 137). This will be discussed in detail in the following sections.

Nutrients and Depression

Metabolism of Nutrients and the Biosynthesis of Neurotransmitters

The initial step in the biosynthesis of serotonin is from tryptophan (TRP) to form 5-hydroxytryptophan (5-HT) through the indoleamine pathway. This action is catalyzed by tryptophan hydroxylase and a cofactor, tetrahydrobiopterin (BH₄) is needed. The 5-HT is then decarboxylated to form serotonin. The decarboxylation reaction is catalyzed by aromatic L-amino acid decarboxylase and a coenzyme, vitamin B₆ (72). During catabolism, serotonin is deaminated and oxidized to 5-hydroxyindoleacetic acid (5-HIAA) (72). Although the exact pathway is not clear, folate is involved in the synthesis of BH₄, at least in cerebrospinal fluid (CSF). The deficiency of 5, 10-methylenetetrahydrofolate (5MTHF) can decrease the regeneration of BH₄ from q-dihydrobiopterin (73). The main pathway for tryptophan metabolism is the kynurenine pathway which provides precursors for the biosynthesis of nicotinamide adenine dinucleotide (NAD⁺) and adenine dinucleotide phosphate (NADP⁺). The regulatory enzymes for this pathway are tryptophan 2, 3-dioxygenase (TDO) and indoleamine 2, 3-

dioxygenase (IDO) (72). Several neurotoxic metabolites (3-hydroxykynurenine, quinolinate) are generated during this pathway, and may contribute to the development of psychiatric diseases (anxiety, depression, and schizophrenia). The 3-hydroxykynurenine compound can induce oxidative stress and apoptosis, and quinolinate can lead to hippocampal damage and oxidative stress through stimulation of N-methyl-D-aspartate (NMDA) receptors (74). Activation of NMDA receptors can increase intracellular calcium and lead to cellular protein release and cell death (75). Tryptophan hydroxylase, TDO and IDO are all regulatory enzymes and catalyze the rate-limiting steps in the pathway. Tryptophan hydroxylase has to compete for TRP with TDO and IDO. It is possible that insufficient tryptophan hydroxylase can decrease brain concentrations of serotonin and result in depression (72).

The initial step in the biosynthesis of catecholamine is from tyrosine to form dihydroxyphenylalanine (L-DOPA) which is catalyzed by tyrosine 3-monooxygenase and the cofactors BH_4 . Tyrosine can be made from phenylalanine or obtained from the diet. The synthesis of dopamine is from L-DOPA catalyzed by L-DOPA decarboxylase (72). Some brain neurons use dopamine as their neurotransmitter; in others, containing the enzyme dopamine β -monooxygenase, dopamine can be converted to another neurotransmitter, norepinephrine (76).

SAM is the major donor of methyl groups for biosynthetic reactions. Adenosyl transferase catalyzes methionine to form SAM. Following transfer of the methyl group, SAM forms homocysteine. Methionine can be regenerated from homocysteine by methylation using N^5 -methyl tetrahydrofolate in a salvage pathway. This reaction is catalyzed by homocysteine methyltransferase and requires vitamin B_{12} . "In vitamin B_{12} deficiency, tetrahydrofolate (THF) cannot be released and remains trapped as N^5 -methyl THF." So, vitamin B_{12} deficiency can result

in secondary folate deficiency (72). Homocysteine can be either a partial antagonist or an agonist on the NMDA receptor (77).

In addition, neurotransmitters, which are released from nerves, can be inactivated by reuptake into the nerve terminals where they originate, or by uptake into adjacent cells by neurotransmitter transporters (noradrenaline and serotonin neurotransmitter transporters). Neurotransmitter transporters are key targets for antidepressant drugs (e.g. tricyclic antidepressants, serotonin-selective reuptake inhibitors [SSRI]) (78).

Cytokines and Depression

Altered cytokine secretion as a mechanism in the etiology of depression has been reported previously (31-34). The mRNA expressions in leukocytes of some cytokines (interleukin-1 β [IL-1 β], interleukin-6 [IL-6], interferon- γ [IFN- γ], interferon- α [IFN- α]) were higher in depressed patients than in healthy controls. Elevated serum levels of these four cytokines in the depressed patients were also observed (31). Administration of fluoxetine can cause a marked decrease in IFN- γ mRNA expression, but no significant differences were seen in IL-1 β , tumor necrosis factor- α [TNF- α], and IL-6 levels compared with healthy controls (31). Cytokine therapy (interleukin-2 [IL-2] or IFN- α) can significantly increase the proportion of patients with cancer or viral infection to develop depressive symptoms, such as: anorexia, pessimistic thoughts, suicidal ideation, and loss of concentration (33).

Cytokines regulate 5-HT brain neurotransmission in the several ways: The first way is by regulating the enzyme IDO expression in cells from the hypothalamic-pituitary-adrenal (HPA) (32). IFN- γ can induce IDO expression in hypothalamic GT₁₋₇ and pituitary T₋₂₀ cells, but IL-10 (anti-inflammatory cytokine) inhibits this effect of IFN- γ (7). As described earlier, IDO competes with tryptophan hydroxylase for TRP, and increased IDO activity will reduce the

availability of TRP for serotonin synthesis (34). The second way is that cytokines (IL-1 β , IL-6, IFN- γ , and TNF- α) up-regulate serotonin transporter (5-HTT) mRNA expression, and cause decreased extra-cellular 5-HT and the onset of depression (31). Antidepressant therapy can reduce 5-HTT mRNA expression in the patients with major depression, but it cannot achieve levels similar to those in healthy controls (31). The third way is that cytokines (IL-2 and or IFN α) reduce appetite and result in decreased serum concentrations of TRP (33). The sum of the large neutral amino acids (LNAA) and the TRP/LNAA ratio (TRP competes with LNAA for crossing the brain blood barrier) is also significantly decreased during cytokine therapy (33). The overall effects of increased cytokine levels result in TRP depletion and reduced 5-HT synthesis, which significantly increases the development of depressive symptoms (31, 33).

Brain-Derived Neurotrophic Factor (BDNF) and Depression

BDNF, a member of the neurotrophin family, is synthesized by several populations of sensory neurons. BDNF may act in an autocrine or paracrine fashion to support dorsal root ganglion (DRG) sensory neurons within the brain (79).

BDNF is involved in many aspects of neural development and function, including cell fate decisions, axon growth, dendrite pruning, synaptic function, and plasticity (79). Lyons et al. generated BDNF^{+/-} mice and reported that a partial impairment in BDNF expression induced physiological disturbances in central 5-HT neurons in young adult mice and led to a structural deterioration of these neurons in advanced age. Further, BDNF^{+/-} mice also showed the early functional deficits in the 5-HT system of forebrain areas such as the frontal cortex, hippocampus, and hypothalamus (80).

Serum BDNF levels in patients with depression were significantly lower than in controls, and showed a gender difference, that is, depressed females released less BDNF than depressed

males (81). Serum BDNF levels were negatively correlated with the severity of depression. The lower BDNF levels observed, the higher depression score obtained (Montgomery-Åsberg-Depression Rating Scale [MADRS] or Hamilton Rating Scale [HAM-D]) (81, 82). In addition, patients with depression had a dysregulation of BDNF homeostasis in the face of a serotonergic perturbation. During tryptophan depletion, BDNF levels increased in healthy volunteers but remained low in patients with depression (83).

On the other hand, antidepressants may increase BDNF in depressed patients (82). Shimizu et al. measured serum BDNF in three different groups: antidepressant-naïve patients with MDD, antidepressant-treated patients with MDD (patients received antidepressants for a minimum of 3 weeks.), and healthy control subjects (subjects in this group had no medical or psychiatric diagnosis.). Mean serum BDNF level in the antidepressant-naïve group was significantly lower than that in the control group or the treated group (82).

Tryptophan and Depression

The precursor of 5-HT is TRP (29), an essential amino acid (84) and the least common amino acid in many proteins (85). Cerebral TRP concentration depends on both absolute TRP level and the ratio of TRP/LNAA (tyrosine, phenylalanine, leucine, isoleucine, and valine) in serum, because LNAAs compete with TRP for crossing into the brain barrier (29).

Animal and human studies suggest that dietary TRP variations affect brain TRP concentration and serotonin synthesis (86-88). Diets low in TRP significantly decrease cerebrospinal fluid TRP levels, plasma TRP levels (88), and 5-HT levels in the hippocampus. Diets high in TRP significantly increase extracellular 5-HT levels in the brains (86).

TRP depletion can cause impairment of cognition in humans (89, 90). TRP-free amino acid mixtures consumed by healthy volunteers resulted in decreased plasma total and free TRP

concentrations, increased response times for happy but not sad targets in an affective go/no-go task (89), and impaired long-term memory in this population (90). A go/no-go task examines inhibitory control over different components of cognitive processing and emotional processing (91). In this task, happy and sad words were rapidly presented in the center of the screen, one by one. Half of words were targets and half were distractors (89). Further, the mood response to TRP depletion showed gender differences (92). One study investigated the mood response of healthy volunteers to acute TRP depletion. After ingesting a TRP deficient amino acid mixture, females showed a significant lowering of mood compared with the control group (92). However, similar results were not found in a comparable sample in a study with males (92). In addition, women with a history of depression showed impaired regulation of brain 5-HT function in response to diet. A low TRP diet in healthy females can increase endocrine response (increased prolactin response is probably mediated via brain 5-HT) to 5-HT challenge. However, this phenomenon cannot be observed in individuals with a history of depression (93).

TRP deficiency results in low levels of the 5-HT product. Because TRP competes with LNAAs to cross the blood brain barrier, the decreased ratio of TRP/LNAA in plasma results in less TRP available in brain. An animal study revealed that TRP-free diet can decrease the ratio of TRP/LNAA, central TRP, and 5-HT concentrations by 71-78%, 50%, 50%, respectively (94). When protein is less than 2% of the total energy, there was more TRP available. A meal with 5% of energy from protein is enough to ensure that this did not occur. However, protein intake has been found to be relatively constant ($13 \pm 2\%$ of daily energy) in human diets (95). Murtman et al. reported that a usual American breakfast rich in protein can cause a decrease in the ratio of TRP/LNAA. After eight subjects consumed the protein-rich breakfast, plasma TRP/LNAA decreased about 17%, 23%, 28%, and 35% below baseline at 40, 80, 120, and 240

minutes, respectively. The median nadir was -37% , and the greatest drop was 240 min after baseline in five subjects (96).

However, protein with a high TRP content can increase the plasma TRP/LNAA ratio (97, 98) and improve cognitive performance in high stress-vulnerable subjects (97). In a double-blind, placebo-controlled, crossover study, plasma TRP/LNAA ratio was 43% higher after the whey protein rich in α -lactalbumin diet (high TRP diet) than after the control diet (97). Consistent with above study design and results, Beulens et al. studied the effect of α -lactalbumin supplement combined with regular diet on plasma TRP/LNAA ratio. They found that plasma TRP/LNAA ratio increased by 16% after α -lactalbumin and carbohydrates supplement, but decreased by 17% after carbohydrate supplement only (98).

Dietary TRP regulated BDNF in the brain in ddY mice. In a study, the mice were divided in three groups, that is, TRP deficient group (0.15%), TRP adequate group (0.25%), and TRP excess group (1.25%). After feeding the mice a TRP deficient diet for two weeks, the levels of BDNF in the cerebral cortex of mice significantly were lower than the other two groups. However, feeding a TRP excess diet had no further increase in the brain levels of BDNF. These findings indicated that the brain level of BDNF was dependent on dietary tryptophan intake (99).

TRP supplementation can improve mood status in healthy female volunteers. Females with TRP supplementation showed increased recognition of happy facial expression, decreased recognition of disgusted facial expression, reduced attentional vigilance towards negative words, and decreased baseline startle responsivity (100). TRP also can enhance the effects of the antidepressant fenfluramine. Local administration of fenfluramine into the dorsal hippocampus of rats caused 5-HT release which was significantly diminished in rats on TRP low diet and increased in rats on a TRP high diet (86).

S-adenosylmethionine (SAM) and Depression

SAM is formed from methionine, which is another essential amino acid (72, 84). Low CSF SAM levels have been observed in depressed patients (23, 24), and a SAM supplement can improve symptoms of depression (101, 102). Administration of SAM showed a significant acute reduction in depressive symptoms which were measured by both the HAM-D and the Beck Depression Inventory (BDI). The rapid antidepressant effect of SAM showed as soon as the first week of treatment (101). In addition, SAM treatment was accompanied a low number of adverse events. The efficacy and safety of SAM in the treatment of major depression have been studied in two multicenters. In the first, 1600 mg SAM/d was given orally. In the second multicenter, 400mg SAM/d was given intramuscularly. In both studies, the effects of SAM were compared with those of 150 mg imipramine/d given orally in a double-blind design. The results of both studies showed that SAM and imipramine were not significantly different, but SAM was significantly better tolerated (102). Both intravenous and oral administration of SAM can raise CSF SAM levels, indicating that SAM can cross the blood-brain barrier in humans (23).

SAM has quick efficacy in the first week and speeds the onset of the antidepressant effect of imipramine via modifying 5-HT_{1A} receptors. Both SAM and imipramine increase norepinephrine and serotonin levels, but imipramine shows a delayed therapeutic effect of anti-depression compared to the rapid effect of SAM. Acute imipramine up-regulated the frontal cortex of 5-HT_{1A} receptors and did not show an antidepressant effect. However, SAM inhibits the up-regulation in the frontal of 5-HT_{1A} receptors and then speeds the onset of the antidepressant effect of imipramine. Further, all the chronic treatments of these two drugs showed antidepressant effects and up-regulated the hippocampus 5-HT_{1A} receptors. So, SAM not

only showed antidepressant effects, but also increased the effect of the antidepressant drug through preventing the 5-HT_{1A}receptor up-regulation in the frontal cortex (103).

Tyrosine and Depression

Alterations in noradrenergic function have been found to compromise symptoms of depression. After taking α -methylparatyrosine (AMPT), which can result in depletion of norepinephrine and dopamine, subjects showed a return of depressive symptoms (91, 104); decreased brain metabolism scanned by multiple positron emission tomography (PET) (91); and reduced serum homovanillic acid (HVA: a dopamine metabolite) and 3-methoxy-4-hydroxyphenyl glycol (MHPG: a norepinephrine metabolite) (104).

However, the dietary effects on the noradrenergic function are not clear. McLean et al. examined the physiologic, subjective and cognitive effects of acute tyrosine depletion in healthy volunteers. Results showed that brain tyrosine availability was more reduced in the tyrosine and phenylalanine-free (TYR-free) group compared to the balanced amino acid mixture (BAL) group. For the affective go/no-go task, the TYR-free group showed a sad latency bias, but the BAL group exhibited a happy latency bias (105). Other studies got the opposite results (106, 107). In a double-blind, placebo-controlled study, acute phenylalanine and tyrosine depletion did not induce the relapse of depressive symptoms in subjects who recovered from depression, although acute phenylalanine and tyrosine depletion induced a significant reduction in the ratio of plasma tyrosine and phenylalanine to LNAA (106). Another double-blinded controlled partially randomized sequence multiple crossover study assessed the effects of the acute depletion of the catecholamine precursor phenylalanine and tyrosine on mood in healthy males. The three test conditions were: a nutritionally balanced amino acid mixture, followed by injection of normal saline placebo (AA-S); or injection of pentagastrin (AA-PG), which can induce a negative effect

on mood; and an amino acid mixture deficient in phenylalanine and tyrosine followed by pentagastrin (APTD-PG). The results showed that the amino acid mixture type had no significant effects on scores on the Profile of Mood States (POMS) Elation-Depression Scale. As expected, pentagastrin induced significant increased anxiety ratings compared with placebo. “However, there were no significant differences between the APTD-PG and the AA-PG treatment groups on the anxiety or mood ratings.” (107)

Carbohydrate and Depression

Previous studies revealed that carbohydrate intake was associated with good mood (95, 108). In a study of college students, “participants were classified as being carbohydrate, protein, or fat cravers based on their response to the question which asked them to identify the type of food that they most craved.” Sixty-seven percent of the carbohydrate cravers felt anxiety, fatigue, and depression before they experienced craving. After consuming the craved food, 79% of students felt satisfied, happy/good, relaxed, or energetic (108). Another population based study also revealed that carbohydrate-rich and protein-poor food (CR/PP) can prevent a deterioration of mood and performance under uncontrollable laboratory stress conditions in stress-prone subjects. Twenty-four subjects with a high stress-proneness and 24 subjects with a low stress-proneness attended an uncontrollable stress situation under both a CR/PP and a protein-rich, carbohydrate-poor (PR/CP) diet condition. During the PR/CP diet, high stressed subjects showed stress-induced rise in depression, decline in vigor, and cortisol elevation. However, this did not occur during the CR/PP diet (109).

Cereal and caffeinated coffee are a common breakfast items in American diets. Smith et al. compared the effects of breakfast cereal and caffeinated coffee on working memory, attention, mood, and cardiovascular function. The results showed that subjects ($n = 36$) who ate a breakfast

cereal and a cup of coffee had a more positive mood, performed better on a spatial memory task, and felt calmer in comparison to those ($n = 36$) who only ate a cup of coffee. Ingestion of caffeine had no effect on mood and memory but did improve encoding of new information and against fatigue. These results suggested that carbohydrate affected on mood (110).

The underlying mechanism of carbohydrate on mood is that carbohydrates increase brain TRP concentrations and serotonin synthesis (87). The normal carbohydrate-rich and protein-rich breakfasts had significantly different effects on both the plasma insulin and TRP/LNAA ratio. Plasma insulin levels rose significantly after carbohydrate but not after the protein meal (96). Increased insulin release will increase LNAAs uptake into muscle (111). Since LNAAs compete with TRP for transport into the brain, LNAAs removal from the blood enhances the ratio of TRP/LNAA, increasing brain TRP concentration and serotonin synthesis (96, 109, 112). Different carbohydrate meals showed different affects on the ratio of plasma TRP/LNAA. Maximum differences among the meal responses occurred at 180 minutes after the test meal. The ratio rose higher after a sucrose meal (34%) than a starch meal (20%). whereas, the ratio declined after the fat + protein meal (45%) (112).

Fat and Depression

Fats in the diet are monounsaturated, polyunsaturated, saturated, and trans fatty acids. Monounsaturated fats come from canola oil, olive oil, nuts, and avocado. The polyunsaturated fats (PUFAs) are found in marine (fish) sources and vegetable sources (corn oil, soybean oil, sunflower oil). Omega-3 and omega-6 fatty acids, types of PUFAs, are all essential fatty acids and must be obtained from diet. Saturated fats (SFA) are found in bacon, butter, cream, coconut, and shortening. Trans fatty acids, which have been hydrogenated, are found in baked “dessert-type” foods and in hard margarine (113, 114).

During the past, human have increased total fat intake. Intake of some types of fat has increased, for example SFA, omega-6 fatty acids, and trans fatty acids, while omega-3 fatty acids intake have decreased in human diets (115). Western diets are deficient in omega-3 fatty acids but rich in omega-6 fatty acids. Trans fatty acids interfere with the desaturation and elongation of both omega-6 and omega-3 fatty acids and this results in decreased amount of arachidonic acid, eicosapentaenoic acid, and docosahexaenoic acid (DHA) availability for human metabolism. A high omega-6/omega-3 ratio is found in today's Western diets (15/1-16.7/1) (115). The ideal dietary ratio of omega-6 to omega-3 has been recommended by international lipid experts to be approximately 2:1 (116). Increased omega-6 fatty acid intake and decreased omega-3 fatty acid intake can increase the production of phosphodiesterase (PDE), leukotriene B₄, IL-1 β , IL-6, and TNF, which are associated with depression (115). Significant depletions of total omega-3 fatty acids and particularly DHA in red blood cell (RBC) membranes have been found in depressed patients, suggesting that RBC membranes in depressed patients have oxidative damage (117).

In animal models, a high SFA intake in the diet reduces hippocampal BDNF, neuronal plasticity, learning, and cognition (118-120). After rats were fed a diet, similar in composition to the typical diet of most industrialized Western societies (rich in saturated fat and refined sugar [HFS]), hippocampal BDNF mRNA and protein decreased, and neuronal plasticity was impaired. The HFS diet reduced levels of adenosine 3',5'-cyclic monophosphate (cAMP)-response element-binding protein (CREB) mRNA and CERB protein, and this reduction was positively correlated with BDNF levels. "CREB is required for various forms of memory and is under regulatory control of BDNF" (118).

In addition, different diets with different types of fat (high SFA, omega-6 fatty acid, and omega-3 fatty acid diet) show different effects on serotonin receptor and transporter binding in

different brain areas. Specifically, a diet rich in omega-6 fatty acid exerted the most influence on serotonin receptor and transporter binding in the mammillary nucleus, prefrontal cortex, and hippocampus of male Sprague-Dawley rats' brain. These findings may be important in relation to the development of depression (121).

An ecological analysis about international variations in the outcomes of schizophrenia and the prevalence of depression in relation to national dietary practices showed that reduced prevalence of depression was strongly related to a high intake of fish and seafood (rich in omega-3 fatty acids) (122). Omega-3 fatty acids had therapeutic effects on depression. After one month treatment of omega-3 fatty acids, symptoms of depression were significantly reduced in the patient group compared with the control group (123).

There are several possible mechanisms of action for the positive effects of the omega-3 fatty acid treatment. First is the omega-3 fatty acids effect on brain structure and function. Fatty acids are major components in brain structure (50% of the neuronal membrane and 70% of the myelin sheath are composed of lipids). Different ratios of various fatty acids can change neuronal membrane fluidity and physiologic function (124). PUFAs form kinks and make the membrane less stiff and rigid; however, trans-fatty acids do not kink, and increase the membrane stiffness and rigidity as if they were fully saturated. "Because proteins embedded in a biomembrane float or sink depending on the membrane's fluidity, biomembrane viscosity is important for membrane protein (membrane proteins act as transporters and receptors) function"(113, 125). Essential fatty acid deficiency can induce myelin impairment which disturbs proper functions of axons in the nervous system, and impairs cognitive function (124). Second, omega-3 fatty acids can modulate levels of cytokines in major depression. Omega-3 fatty acids partially prevented the elevation of TNF- α and IL-1 β , and increased the level of IL-2

(126). Third, omega-3 fatty acids may enhance the cAMP signal-transduction cascade, increase expression of CREB, and increase BDNF (127).

Vitamins and Depression

Vitamin B₁₂ and folate are involved in the methionine salvage pathway for the de novo synthesis of methionine and SAM (72). Both vitamin B₁₂ and folate deficiency can cause SAM deficiency and elevated homocysteine (72). Previous studies suggested that the relationship among folate and vitamin B₁₂ deficiencies and depression was mediated by high plasma homocysteine levels and low concentrations of SAM in CSF (19-21, 102, 128). Increased homocysteine levels were significantly related to low red cell folate suggesting a failure of methylation of homocysteine to methionine due to a shortage of folate or vitamin B₁₂ (22). Folate deficiency can occur by a dietary deficiency, malabsorption, or secondary to vitamin B₁₂ deficiency (methyl-folate trap) (72). Depressed people with high levels of homocysteine had significantly lower levels of CSF folate, CSF SAM concentration, and CSF monoamine metabolites (5-HIAA, HVA, and MHPG) than controls (22).

Although the US government mandated food fortification with folate to lower the incidence of neural tube birth defects (129), folate deficiency still can occur in elderly women (128). This population not only had high Center for Epidemiologic Studies Depression Scale (CES-D) scores, but also had low education levels (128). Folate deficiency also can occur in alcoholics (130). Both male and female alcoholics had decreased platelet 5-HT concentrations compared with healthy controls (131). Alcoholics reported the highest BDI scores compared with problematic/heavy and light social drinkers. Moreover, female alcoholics reported significantly more of depression than male alcoholics (132).

Vitamin B₁₂ and folate supplementation improved depression (133,134). Daily folic acid supplements enhance the action of antidepressants. Female patients in an antidepressant plus folic acid group showed significantly decreased homocysteine levels and a lower depressive score (HAM-D score) compared to females in the antidepressant plus placebo group (134). Daily vitamin B₁₂ supplements had effects similar to folate. Although patients with major depressive disorder did not have vitamin B₁₂ deficiency, increased vitamin B₁₂ levels were still beneficial for decreasing the HAM-D scores (133). Coppen et al. recommended oral doses of both folic acid (800µg daily) and vitamin B₁₂ (1mg daily) to improve treatment in depression (135).

Iron and Depression

Few previous studies reported the relationship between iron deficiency and depressive symptoms. Further, human studies showed inconsistent results previously. Lozoff et al. reevaluated a group of 11 to 14 years children who had been tested and treated for iron deficiency as infants. They found that iron deficiency in infancy still had a more than 10 years behavioral risk, although iron deficiency has been corrected at that time. The behavior of children with chronic iron deficiency in infancy was rated as more problematic and included anxiety/depression, and social and attention problems (136). Similarly, Bears et al reported that postpartum depression and cognitive impairment in low socioeconomic females may be associated with iron deficiency anemia. Moreover, iron treatment can significantly improve iron-deficient mothers' depressive symptoms (137). However, Hunt et al. reported opposite results. They found that non-pregnant pre-menopausal women (aged 20 to 45 years) with low hemoglobin, low ferritin and low transferrin were less likely to describe themselves as depressed (138).

In rats, iron-deficiency can alter dopamine metabolism in the brain of rats with hemolytic anemia. Compared with controls, iron-deficient rats had higher extracellular dopamine levels in the caudate-putamen. These findings may provide a causal relationship of iron status with depression via the dopaminergic system (139).

Chronic Diseases and Depression

Food insecurity is closely associated with not only mental health but also with physical health (24, 25). Significantly more individuals from food-insufficient households reported poor/fair health, and were more likely to report diabetes, heart diseases, high blood pressure, food allergies (25), and obesity (26, 27) than those from food-sufficient households. Chronic diseases may compromise mental health, because patients may confront numerous threats and challenges such as: pain, disfigurement, impaired physical functioning, life-threat, and changes in future perspectives (61). So, understanding the relationship among chronic diseases and depression is important for improving the quality of life of these people.

Diabetes and Depression

The global prevalence of diabetes was estimated at 2.8% in 2000, and expected to be 4.4% in 2030 (140). Although the prevalence of diabetes is higher in men than in women, more women have diabetes than men (140). The number of people with diabetes in the US was estimated at 17.7 million in 2000 and is expected to be 30.3 million in 2030 (140). Since diabetes increases the risk of depression (141) and low-income patients are less likely to receive routine diabetes care (142), the prevalence of depression in this population is of concern. One meta-analysis of 42 studies demonstrated that diabetes doubled the odds of having a comorbid depression (143). Similar to the general population, women with diabetes had an increased

prevalence of depression compared with men in the diabetic group (141, 143, 144). There was no difference in the prevalence of depression between type 1 and type 2 diabetes (143).

Depression is associated with hyperglycemia in patients with diabetes (145).

Hyperglycemia is very common in patients with diabetes because of relative or absolute insulin deficiency (146). Glycemic control over the past 120 days is determined by hemoglobin A_{1C} (HbA_{1C}) (147). A population-based study showed that for subjects younger 65 y, 54.4% of those with depression and 37.3% of those without depression had HbA_{1C} \geq 8.0% (normal range: 5.0% \sim 8.0%) respectively. For subjects aged \geq 65 y, 36.4% of those with depression had HbA_{1C} \geq 8.0% in comparison to 32.3% of those without depression (10). A meta-analysis used HbA_{1C} as a physiologic measure and confirmed that depression was associated with higher HbA_{1C} levels (145). Hyperglycemia can alter mood state in subjects with diabetes (148). These changes included “increased feeling of agitation and anxiety, increased feelings of tiredness and lethargy, and decreased feelings of happiness” (148), which are very common symptoms in depression.

Depressed mood is also associated with hypoglycemia. Hypoglycemia is a common side effect of treatment when diabetic patients cannot follow the correct instructions for diet and medication (146), delay in eating, or engage in exercise. Acute hypoglycemia can induce an acute and persistent effect on mood 1.5 days, 8.9 days, and 30 days (149, 150). After acute hypoglycemia episode, subjects had significant elevated Hospital Anxiety and Depression Scale (HAD Depression scale) scores at all three time points, and increases of HAD Anxiety scale scores at 1.5 days was significant (149).

Depression is associated with insulin resistance (151). Insulin resistance is an important mechanism in the development of type 2 diabetes mellitus (146). A population based study defined insulin resistance with the qualitative insulin sensitivity check index, and evaluated the

severity of symptoms of depression with BDI 21 scores. Insulin resistance was related positively to the severity of symptoms of depression. (151).

Depression is associated with complications from diabetes (152). Poor control of blood glucose results in multiple system and organ damage (146). A meta-analysis of 27 studies in patients with diabetes reported that depression was positively associated with a variety of diabetes complications, including diabetic retinopathy, nephropathy, neuropathy, macrovascular complications, and sexual dysfunction (152).

Comorbid depression and diabetes had more negative quality-of-life effects than that of depression or diabetes alone (153, 154). About 78%, 58%, 51%, and 25% of individuals with diabetes and depression, diabetes, depression, and no diabetes and depression respectively reported having overall functional disability (154). Individuals with comorbid depression and diabetes had higher ambulatory care use and filled more prescriptions compared with those without depression (11). The total health care expenditures for patients with diabetes and depression were 4.5 times higher than those with diabetes but without depression (11).

Heart Disease and Depression

Based on the report of the global and regional burden of disease and risk factors, 2001, ischemic heart disease is the leading cause of death in high-income countries (155). In the United States, coronary heart disease (CHD) has ranked as the first cause of death for more than 50 years. Although CHD mortality has declined since the 1960s in the US as a whole, the decreasing trend among black women has been slower than that among white women. (156).

Depression is not uncommon among CHD patients (157), particularly after myocardial infarction (MI). The prevalence of depression in the cardiac population is three to four times that of the general population (158). Similar to the general population, depression is far more

prevalent in females with CHD than males (159, 160). The risks of post-CHD (or post-MI) depression are related with the severity of the MI, personality, and psychological and social consequences of MI (161, 162). Spijkerman et al. pointed out that the first-ever post-MI depression may be triggered by a severe infarction or its consequences (such as pain or disability) (161). Post-MI depression had a strong relationship with poor left ventricular ejection fraction, revascularization and the occurrence of arrhythmic events during hospitalization (161). Personality played an important role in the development of ongoing and recurrent depression because post-MI depression was strongly related to neuroticism, which is associated with a “type D” personality (161). “Type D is a taxonomy based on the two stable personality traits of negative affectivity and social inhibition and denotes those individuals who experience increased negative distress and who do not express these negative emotions in social interaction” (163). Type D personality is positively related to increased risk of depression in patients with heart disease (164). In another population based study, it was suggested that psychological and social consequences of MI may drive post-MI depression in patients vulnerable to depression (162). There was a greater chance for post-MI depression if patients had a history of depressive disorders in the month before MI, lack of social support, or a MI with complications (162). Once post-MI depression was present, the symptoms of depression were persistent during the first year following MI (159). A meta-analysis study reported that post-MI depression was associated with 2 to 2.5-fold increased risk of impaired cardiovascular outcome, and all-cause and cardiac mortality (165).

Some pathophysiologic factors may link depression and CHD. Patients with combined CHD and post-CHD depression had higher rates of cardiac complications including recurrent ischemia, infarction or congestive heart failure, recurrent acute MI, and arrhythmia compared

with non depressed patients (166). In addition, patients with combined depression and CHD have been shown to have alterations of the sympathetic nervous system, and depressive symptoms are associated with elevated levels of norepinephrine excretion (167) leading to elevated heart rate at rest (168) and reduced heart rate variability (169). Low heart rate variability is believed to be a powerful predictor of sudden heart death (170). Depressed patients have also been reported to have endothelial dysfunction. In one controlled study, 15 patients with treated depression but no other risk factors for CHD and 12 controls were recruited; and 12 patients and 10 controls finished the study. Results showed that arterial endothelial function, as measured by flow mediated dilatation of the brachial artery, was significantly impaired in the patients' group (171). Because these two groups had no other risk factors for CHD, this endothelial dysfunction may be a mechanism by which depression increased disease risk (171).

Depressed patients also exhibit alterations in platelet reactivity (172-174). Decreased platelet nitric oxide (NO) synthase activity and plasma NO metabolites in patients with major depression have been reported previously (172). Platelet activation is important to the formation of atherosclerosis and acute coronary syndromes. NO, which is synthesized from L-arginine by NO synthase, inhibits platelet adhesion to the vascular wall and platelet aggregation (173). Moreover, depressive patients showed greater procoagulant activity as detected by increased plasma concentrations of platelet factor 4 compared with controls (174). So, increased platelet reactivity may contribute to an increased CHD risk in patients with depression.

Health-care costs increased significantly among patients with post-MI depression during the first year following MI. Total costs were about 41% higher for patients with combined MI and depression than those without depression. Increased health expenditure was related to out-

patient and emergency room visits and readmission costs linked with longer stays in hospital wards but not to mental health services (175).

Hypertension and Depression

Hypertension prevalence in blacks is among the highest in the world. Among Americans aged 20 years and older, the prevalence of hypertension among black women, black men, white women, Mexican-African women, white men, and Mexican-American men is 43.4%, 40.4%, 28.4% , 27.8%, 27.5%, and 26.7%, respectively. In the southeastern US, the prevalence of hypertension in Blacks and Whites is greater and death rates from stroke are higher compared those in other regions of the country. Within the black community, those middle aged or older, less educated, overweight or obese, physically inactive, and with diabetes have the highest rates of hypertension (176). The prevalence of depression in women with hypertension is higher (20.6%-27.1%) than for those without hypertension (177).

Previous studies have shown that blood pressure (BP) is closely associated with depression. High diastolic BP is related positively to depression. Women with higher levels of diastolic BP (≥ 90 mmHg) had significantly higher levels of depression, ate fewer fruits and vegetables, and had more stress and less social support (178). Systolic hypertension (≥ 140 mmHg) is also associated with depression (179). A longitudinal study revealed that subjects with systolic hypertension were more likely to report a significant number of depressive symptoms and exhibit cognitive impairment (reduced verbal learning and memory function) (180). Poor blood pressure regulation was also related to increased depressive symptoms and poor prospective memory. One study examined neuropsychological function and mood in relation to blood pressure regulation following orthostatic challenge in black children, and calculated the difference between supine and 1-minute standing systolic blood pressure and pulse pressure

scores. Findings showed that “smaller increases in systolic blood pressure was predictive of decreased verbal memory whereas smaller increases in pulse pressure were associated with increased depressive symptoms and poor prospective memory” (181). Kario et al. reported that the association between depression and disrupted diurnal blood pressure variation had a gender difference. In men, depression was positively related with the sleep/awake systolic blood pressure ratio, whereas associations of anxiety with systolic BP and pulse rate were found in women (182).

The neural system and genetic background were involved in the underlying mechanism of the relationship between hypertension and depression (146, 183-186). Major depressive patients showed a nonresponsive adrenal sympathetic activity while presenting a high neural sympathetic activity. The resting levels of noradrenaline (NA) + NA/Ad (adrenaline) were higher in major depressed patients than in non-depressed controls (183, 184). Increased neural sympathetic activity can also contribute to hypertension (146). Parental history of hypertension also enhanced the impact of depressed mood. Subjects with parental history had significantly higher BDI scores than those without this history. These findings suggest genetic background may play an important role in this correlation (184). One example is the G-protein $\beta 3$ -subunit (GNB3) which has been found related to both hypertension and major depressive disorders (185, 186). The polymorphism C825T of the gene encoding the GNB3 was found to be linked to an increased prevalence of hypertension (185), and the C825T allele was present in more patients with MDD (186). MDD patients with this T allele had severe symptoms and a better response to antidepressant treatment compared with patients without the T allele (186).

The combination of hypertension and depression increased the rates of disability, angina, past MI, stroke, and digitalis use. Specifically, the rates of stroke in individuals with depression

were 2.3 to 2.7 times higher than those without depression. Since the high rate of stroke in individuals with depression, further evaluation of the role of depression in the progression of hypertension seems warranted (177).

Obesity and Depression

Data from NHANES III showed that the prevalence of obesity and overweight increased dramatically in the U.S. over the past 30 years (187), specifically in the last 2 decades (188). A recent report among adults aged at least 20 years in 1999-2002 revealed that the prevalence of overweight or obesity was 65.1%, obesity was 30.4%, and extremely obese was 4.9% (188).

Food insecurity is associated with increased risk of obesity (26, 27). Data from the 1998 and 1999 California Women's Health Survey showed that obesity was more prevalent in food insecure than that in food secure women. "Food insecurity is associated with increased likelihood of obesity and risk is greatest in non whites" (26). Among low income women, but not men, food stamp program (FSP) participation was positively linked to obesity. Low income women who were currently participating in the FSP had a 9.1% increase in the predicted probability of obesity. "Participation in the FSP in each of the previous five years compared to no participation over that time period was associated with approximately a 20.5% increase in the predicted probability of current obesity" (189). Previous studies have suggested that obesity has been associated with major depression. Onyike et al. analyzed data from the NHANES III (1988-1994) and reported that obesity (defined as a BMI ≥ 30) was related to past-month depression in women but not in men (190). Increasing evidence suggested that central abdominal obesity is also associated with depression (191, 192).

Genetics may link obesity and depression. The human opposite paired-containing gene (HOPA) polymorphisms increased the risk for both major depression and obesity. Normal

thyroid hormone receptor function is important to mammalian central nervous system development and homeostasis. HOPA is one of the thyroid receptor-associated proteins. Researchers performed standard polymerase chain reaction for radioisotopic genotyping of the HOPA exon 42 locus and found that 100% HOPA variant (n = 5) had a lifetime diagnosis of major depression compared with only 30% of the HOPA wild type subjects (n = 63). Among HOPA variant females, four of them were with a BMI over 30, and the fifth female had a BMI well above the mean BMI for the 68 females tested for HOPA” (193). Another example of genetics is the 5-HT_{2A} receptor gene promoter polymorphism which is related to abdominal obesity. Rosmond et al. reported that the observed genotype frequencies of polymorphisms of the 5-HT_{2A} gene were 35.6%, 51.6%, and 12.9% for -1438A/A, -1438A/G, and -1438G/G, respectively. Homozygotes for the -1438G allele had higher BMI, waist-to-hip ratio, and abdominal sagittal diameter than the -1438A allele homozygotes (194).

Sustained weight loss in obese people improves depression. Dixon et al. “conducted a comparison of two weight-stable groups with BMI in the 30 to 35 kg/m² range.” The weight loss subjects (n = 79) were selected obese patients 3 years after laparoscopic adjustable gastric band surgery. The control subjects (n = 79) were obese patients seeking weight loss therapy. Weight loss subjects, who maintained a mean weight loss of 32.8 ± 18 kg after surgery, reported fewer symptoms of depression compared to controls. These results suggested that sustained weight loss conveys benefits regardless of BMI (195).

CHAPTER 3

SUBJECTS AND METHODS

Participants

The analyses presented here used data collected from a previous study: “Effects of Weight History, Resource Cycling, and Fast Food on Overall diet Quality and Health in Low-Income Louisiana Women.” In that study, two 24-hour dietary recalls, food security status, and a household survey conducted on 72 female food stamp recipients in southeast Louisiana (196). Of the 72 women who provided data, five women were excluded because they were pregnant (there was no attempt to quantify their BMI) and one was excluded because of a reported energy intake of greater than 13,000 kcals. Therefore, the final sample used data from 66 (91.67%) women in the original study.

The original project was approved by the Institutional Review Board by Louisiana State University Agricultural Center (approval number: H03-05 [Appendix A]) (196).

Data Collection

1. Sociodemographic characteristics such as age, education level, employment, marital status, medical insurance, household members, and health related characteristics were obtained from household survey questions (Appendix B).
2. Food security status was determined using a modified version of the USDA Food Security Module Short Form (Appendix C), which evaluated food security status during the past 30 days.
3. Weight and waist circumference were measured using a Tanita scale (model number: Body Composition Analyzer BF-350; Tanita Corp of America; Arlington Heights,

Illinois). An average of 3 measurements was used to delineate weight and waist circumference. Height was stated by subjects (Appendix D).

4. Two 24 hour dietary records were collected from all subjects. The multiple pass approach using an in-person 24-hour dietary recall was conducted when food stamp benefits were first received (Day 1). A telephone administered 24-hour dietary recall was obtained approximately 3½ weeks after the initial interview (Day 2). Food, brand names, and amount were documented. Food models and pictures of portion sizes and measurements were used to estimate the actual intake.

Data Analysis

Food Security Status

Three mutually exclusive categories of food security status were defined food secure (FS), food insecure (FIS), and food insecure with hunger (FISH) (12). Each condition was coded categorically (FS = 1, FIS = 2, and FISH = 3).

Body Mass Index (BMI) and Waist Circumference

BMI was calculated as weight in kilograms divided by height in meters squared. Classification of weight by BMI was: underweight (< 18.5), normal (18.5 - 24.9), overweight (25.0 - 29.9), obesity (≥ 30) (197). Categorical variables (from 1 to 4) were used to reflect weight classifications. Waist circumference was used as a continuous variable for analysis.

Sociodemographic and Health-Related Characteristics

Questionnaires were administered during the in-home interview. Self-reports included age (continuous variable), marital status (coded as categorical variable: single = 1, separated/divorced/widowed = 2, married = 3); number of household members (continuous variable); education level (coded as categorical variable: below high school = 1, high school

graduate = 2, and above high school = 3); employment (coded as dummy variable: yes = 1, no = 0); medical insurance (coded as dummy variable: yes = 1, no = 0), and the number of diseases except depression (continuous variable). The number of diseases a person reported was obtained by self-reporting the presence of a specific disease. From a list of 11 diseases, subjects were first asked whether a doctor had ever told them that they had: heart disease, high cholesterol, high blood pressure, diabetes, fluid retention, a problem weighing too much, anemia, cancer, arthritis, osteoporosis, or depression. Each disease condition was coded as a dummy variable (not present or present and not present = 0; present = 1). Depression status was also coded as a dummy variable (present = 1, not present = 0).

Dietary Intakes

Food intakes for two single day recalls were inputted into Nutritionist Pro (Single Version 2.2, San Bruno, CA) to determine nutrient intake. Mean intakes of energy, macronutrients (protein, carbohydrate, total fat, cholesterol, SFA, PUFA, and MUFA), vitamins (folate, vitamin B₁₂), and iron for individuals were determined for Day 1 and Day 2. Intake of each nutrient was used as a continuous variable for analysis.

Individual intakes of protein, carbohydrate, total fat, folate, vitamin B₁₂, and iron for Day 1 and Day 2 were all compared with the Dietary Reference Intakes (DRI) (125). A series of 2 × 2 contingency tables were made according to two categorical variables (met DRI or not, depressed or not), and differences of meeting DRI for Day 1 and Day 2 intakes of protein, carbohydrate, total fat, folate, vitamin B₁₂, and iron were compared between depressed and non-depressed groups.

Statistical Analysis

All statistical analyses were performed using SPSS 12.0 (SPSS Inc.) for Windows. Descriptive statistics on selected socioeconomic characteristics and mean nutrient intakes were presented for depressed and non-depressed individuals. Chi-square tests (Pearson chi-square test or Fisher's exact test) were conducted for categorical variables. Mann-Whitney tests, which were applied for metric continuous variables when the data distribution violated parametric assumptions, were used to compare median intakes of energy, protein, carbohydrate, total fat, SFA, PUFA, MUFA, folate, vitamin B₁₂, and iron between depressed and non-depressed groups. Chi-square and Mann-Whitney tests were two-tailed, and the level of significance was set at $p < 0.05$.

Four logistic regression models were run to assess the associations of depression with food security status, dietary nutrient intakes, BMI, and the number of diseases.

1. The first model assessed the relationship between depression and food security status while controlling for employment, marital status, education level, number of household members, and medical insurance.
2. The second model assessed the association of depression and multiple nutrient intakes while controlling for food security status and education level.
3. The third model assessed the association between depression and BMI; while controlling food security status and nutrient intakes.
4. The fourth model assessed the relationship between depression and the number of diseases while controlling food security status, medical insurance, BMI, and nutrient intakes.

In each model, the dependent variable was depression status, and the independent variables were food security status, sociodemographic variables, nutrient intakes, BMI, and the number of diseases reported. In our initial analyses, we looked at the unadjusted effects of each explanatory variable, so a single explanatory variable in the model at a time was included. Stepwise selection procedure was used for each model. We used a conventional criterion for selecting a model in logistic regression analysis, that is, a variable had to be significant at the 0.05 level before it was allowed to enter into the model and that a variable in the model had to be significant at the 0.1 for it to remain in the model. The fit of each model was measured by Hosmer-Lemeshow goodness-of-fit test. Results from logistic regressions are shown as odds ratios with associated 95% confidence intervals.

CHAPTER 4

RESULTS

Individual sociodemographic characteristics of depressed and non-depressed subjects are presented in the Table 1. Of the 66 participants, 21 (31.82%) reported depression, and 45 (68.18%) did not. The mean ages of the depressed and non-depressed groups were 45.10 years and 40.58 years, respectively. Most participants in this study were black. Of participants reporting depression, 90.48% were black, and in those not reporting depression, 95.56% were black. The majority of our study participants were unemployed (68.18%).

Table 1: Individual sociodemographic characteristics of the study subjects

Characteristic	Depressed subjects (n = 21)	Non-depressed subjects (n = 45)
Age, year (mean \pm SD)	45.10 \pm 13.51	40.58 \pm 14.81
Race, %		
Black	90.48 (n = 19)	95.56 (n = 43)
White	4.76 (n = 1)	0 (n = 0)
Other	4.76 (n = 1)	4.44 (n = 2)
Education, %		
Below high school	52.38 (n = 11)	37.78 (n = 17)
High school	42.86 (n = 9)	35.56 (n = 16)
Above high school	4.76 (n = 1)	26.67 (n = 12)
Marital status, %		
Single	47.62 (n = 10)	53.33 (n = 24)
Separate/divorce/widow	38.1 (n = 8)	31.11 (n = 14)
Married	14.29 (n = 3)	15.56 (n = 7)
Employment, %		
Yes	14.29 (n = 3)	40 (n = 18)
No	85.71 (n = 18)	60 (n = 27)
Number in household (mean \pm SD)	2.95 \pm 1.50	3.76 \pm 1.79

Table 2: Individual health-related characteristics and food security status of study subjects

Characteristic	Depressed subjects (n = 21)	Non-depressed subjects (n = 45)
Number of diseases (mean \pm SD)	3.81 \pm 2.71	2.04 \pm 1.64
Medical insurance, %		
Yes	57.14 (n = 12)	55.56 (n = 25)
No	42.86 (n = 9)	44.44 (n = 20)
Waist circumference (inches)(mean \pm SD)	41.31 \pm 8.24	40.05 \pm 8.43
BMI (kg/m ²) (mean \pm SD)	35.50 \pm 11.88	34.19 \pm 10.42
BMI (kg/m ²), %		
<18.5	4.76 (n = 1)	4.44 (n = 2)
18.5-24.9	14.29 (n = 3)	11.11 (n = 5)
25.0-29.9	23.81 (n = 5)	20 (n = 9)
\geq 30	57.1 (n = 12)	64.4 (n = 29)
Food security status, %		
Food secure	52.38 (n = 11)	44.44 (n = 20)
Food insecure	38.1 (n = 8)	40 (n = 18)
Food insecure with hunger	9.52 (n = 2)	15.56 (n = 7)

Table 2 shows the individual health-related characteristics and food security status of study participants reporting or not reporting depression. Depressed women reported more chronic diseases than non-depressed women. For the overall study sample, 44% participants had no medical insurance. For the entire population, the prevalence of overweight and obesity in the total sample was 83.3%. The depressed women tended to have higher mean waist circumferences and BMI, and a lower percentage of overweight and obesity than the non-depressed women. For the entire population, the prevalence of combined food insecure and food insecure with hunger in our study population was 53%. In depressed women, 52.4% were food secure; 38.1% were food insecure; 9.5% were food insecure with hunger. In non-depressed women, 44.44% were food secure; 40% were food insecure; 15.56% were food insecure with hunger.

Table 3: Day 1, Day 2, and mean intakes of energy, protein, and carbohydrate are compared between depressed and non-depressed subjects; data are presented as mean \pm standard deviation

Nutrient	Depressed subjects (mean \pm SD)	Non-depressed subjects (mean \pm SD)	Mann-Whitney test P-value
Energy (kcal)			
Day 1	1733.2 \pm 819.2	2015.6 \pm 1296.3	0.79
Day 2	1702.8 \pm 664.1	1700.5 \pm 1091.2	0.55
Mean	1718.0 \pm 606.9	1858.0 \pm 1021.3	0.89
Protein (g)			
Day 1	78.5 \pm 52.5	84.7 \pm 72.8	0.95
Day 2	71.0 \pm 25.1	65.3 \pm 43.4	0.19
Mean	74.8 \pm 32.7	75.0 \pm 48.2	0.45
Carbohydrate (g)			
Day 1	196.6 \pm 90.8	243.8 \pm 130.2	0.21
Day 2	225.3 \pm 106.5	228.9 \pm 151.3	0.76
Mean	210.9 \pm 76.7	236.3 \pm 118.4	0.84

Table 3 shows Day 1 and Day 2 intakes of energy, protein, and carbohydrate for depressed and non-depressed participants. Although none of the intakes were significantly different, depressed women tended to have lower intakes of energy (Day 1), protein (Day 1), and carbohydrate (Day 1, Day 2) than the non-depressed women. Day 2 energy and protein intakes in depressed group tended to be higher than those in non-depressed group. Overall, the mean energy, protein, and carbohydrate intakes in depressed participants were lower than those in non-depressed participants. Median intakes of energy, protein, and carbohydrate between these two groups were not statistically different for Day 1 and Day 2. The mean intake of protein (Day 1, Day 2) and carbohydrate (Day 1, Day 2) met DRIs in both depressed and non-depressed groups.

Table 4 shows Day 1 and Day 2 intakes of cholesterol, total fat, SFA, MUFA, and PUFA for depressed and non-depressed women. The median intakes of cholesterol, total fat, SFA, MUFA, and PUFA between these two groups were not statistically different for Day 1 and Day 2.

The mean intake of total fat (Day 1, Day 2) met DRIs in both depressed and non-depressed groups.

Table 4: Day 1, Day 2, and mean intakes of cholesterol, total fat, saturated fatty acid (SFA), monounsaturated fatty acid (MUFA), and polyunsaturated fatty acid (PUFA) are compared between depressed and non-depressed groups; data are presented as mean \pm standard deviation

Nutrient	Depressed subjects (mean \pm SD)	Non-depressed subjects (mean \pm SD)	Mann-Whitney test P-value
Cholesterol (mg)			
Day 1	379 \pm 427.8	422.0 \pm 507.0	0.47
Day 2	288.2 \pm 151.1	316.9 \pm 288.7	0.6
Mean	333.6 \pm 242.6	369.4 \pm 308.1	0.78
Total Fat (g)			
Day 1	72.4 \pm 47.7	80.4 \pm 76.1	0.84
Day 2	58.7 \pm 24.1	59.3 \pm 49.2	0.2
Mean	65.5 \pm 29.6	69.8 \pm 54.2	0.53
SFA (g)			
Day 1	22.5 \pm 17.7	27.6 \pm 31.0	0.92
Day 2	20.0 \pm 9.8	19.6 \pm 17.1	0.19
Mean	21.2 \pm 11.2	23.6 \pm 21.1	0.73
MUFA (g)			
Day 1	21.6 \pm 14.5	25.6 \pm 34.2	0.48
Day 2	16.9 \pm 7.1	17.6 \pm 18.8	0.12
Mean	19.3 \pm 9.0	21.6 \pm 23.7	0.44
PUFA (g)			
Day 1	12.9 \pm 10.6	10.3 \pm 10.9	0.26
Day 2	8.4 \pm 4.6	7.9 \pm 7.2	0.17
Mean	10.6 \pm 5.8	9.1 \pm 7.1	0.18

Table 5 shows the comparison for Days 1 and 2 for mean intakes of folate, vitamin B₁₂, and iron by depressed and non-depressed subjects. The median intake of vitamin B₁₂ on Day 2 was significantly different between these two groups ($p = 0.004$).

The mean intake of vitamin B₁₂ (Day 1, Day 2) and iron (Day 2) met DRIs in both depressed and non-depressed groups. The mean intake of folate (Day 1, Day 2) in both depressed and non-depressed groups and iron (Day 1) in the depressed group did not meet the DRIs.

Table 5: Comparison for Days 1 and 2 for mean folate, vitamin B12, and iron are compared between depressed and non-depressed subjects; data presented as mean \pm standard deviation

Nutrient	Depressed subjects (mean \pm SD)	Non-depressed subjects (mean \pm SD)	Mann-Whitney test P-value
Folate (μg)			
Day 1	218.7 \pm 90.5	342.0 \pm 354.0	0.5
Day 2	327.2 \pm 280.9	253.6 \pm 323.4	0.07
Mean	273.0 \pm 153.4	297.8 \pm 253.2	0.71
Vitamin B 12 (μg)			
Day 1	7.9 \pm 15.8	5.9 \pm 9.5	0.57
Day 2	3.6 \pm 1.9	2.9 \pm 5.2	0.004
Mean	5.8 \pm 8.0	4.4 \pm 6.0	0.11
Iron (mg)			
Day 1	12.1 \pm 8.8	20.4 \pm 22.3	0.25
Day 2	24.0 \pm 25.6	22.4 \pm 27.4	0.29
Mean	18.0 \pm 14.2	21.4 \pm 21.6	0.94

Table 6 showed the number and percent of subjects reporting or not reporting depression who met the Dietary Reference Intakes (DRI) for protein, carbohydrate, total fat, folate, vitamin B₁₂, and iron for Day 1 and Day 2. Compared to women who were not depressed, depressed women had a lower percentage to meet the DRIs for protein (Day 1), carbohydrate (Day 1), total fat (Day 1), folate (Days 1 and 2), and iron (Day 1) intakes; and also had a higher percentage to meet DRIs for protein (Day 2), carbohydrate (Day 2), total fat (Day 2), vitamin B₁₂ (Day 1, Day 2), and iron (Day 2) intakes. The difference between these two groups met the DRIs on Day 1 folate intakes approached statistical significant. No statistically significant differences between these two groups for the intakes of protein, carbohydrate, total fat, vitamin B₁₂, and iron met DRIs on both days.

Tables 7, 8, and 9 present the results of logistic regression analyses. Table 7 shows unadjusted effects of food security status, age, education level, employment, household members, and marital status on depression status. The odds of depression were four times higher for

unemployed participants than for employed ones. No relationships were found among depression and food security, age, and marital status. The relationship between the number in household or education and depression fell just short of significance.

Table 6: Number and % of subjects who were depressed or non-depressed meeting The Dietary Reference Intakes for protein, carbohydrate, total fat, folate, vitamin B12, and iron for Day 1 and Day 2

	Depressed subjects	Non-depressed subjects	Chi-square test (p-value)
Protein			
Day 1	14 (66.67%)	35 (77.78%)	0.34*
Day 2	16 (76.19%)	25 (55.56%)	0.11*
Carbohydrate			
Day 1	16 (76.19%)	37 (82.22%)	0.74^
Day 2	16 (76.19%)	30 (66.67%)	0.43*
Total fat			
Day 1	19 (90.5%)	43 (95.56%)	0.59^
Day 2	20 (95.2%)	39 (86.7%)	0.42^
Folate			
Day 1	1 (4.76%)	11 (24.44%)	0.09^
Day 2	4 (19.05%)	10 (22.22%)	1^
Vitamin B12			
Day 1	13 (61.90%)	23 (51.11%)	0.41*
Day 2	12 (57.14%)	16 (35.56%)	0.1*
Iron			
Day 1	6 (28.57%)	13 (28.89%)	0.98*
Day 2	11 (52.38%)	18 (40%)	0.56*

* Pearson chi-square test; ^ Fisher's exact test

Table 8 shows the unadjusted effects of energy and nutrients (protein, carbohydrate, cholesterol, total fat, SFA, PUFA, MUFA, vitamin B₁₂, folate, and iron) on depression. No relationships were found.

Table 9 shows unadjusted effects of health related characteristics on depression. There was a strong relationship between the number of chronic diseases and depression. No relationships were found among depression status with waist circumference, BMI (continuous or categorical variable), and medical insurance.

Table 7: Unadjusted effects of food security status, age, education level, employment household members, and marital status on depression obtained from logistic regressions

Predictor	P-value	Odds ratio	95% CI
Food security status	0.75		
Food secure	0.46	1.93	0.34-10.92
Food insecure	0.63	1.56	0.26-9.21
Food insecure with hunger*		1	
Age	0.24	1.02	0.99-1.06
Education	0.18		
Below high school	0.07	7.77	0.88-68.44
High school	0.09	6.75	0.75-60.76
Above high school*		1	
Employment			
No	0.05	4	1.03-15.59
Yes*		1	
Number in household	0.08	0.74	0.53-1.04
Marital Status	0.85		
Single	0.97	0.97	0.21-4.54
Separate/Divorce/Widow	0.73	1.33	0.27-6.65
Married*		1	

* Reference category (by default k-1 indicator variables are generated for k categories with the largest category code representing the reference category)

Table 8: Unadjusted effects of nutrients on depression obtained from logistic regression

Predictor	P-value	Odds ratio	95% CI
Energy			
Day 1	0.36	1	0.999-1.00
Day 2	0.99	1	0.999-1.001
Mean	0.56	1	0.999-1.00
Protein			
Day 1	0.73	0.999	0.99-1.01
Day 2	0.57	1.004	0.99-1.02
Mean	0.99	1	0.99-1.01
Carbohydrate			
Day 1	0.14	0.996	0.99-1.001
Day 2	0.92	1	0.996-1.004
Mean	0.37	0.998	0.99-1.003
Cholesterol			
Day 1	0.73	1	0.999-1.001
Day 2	0.67	1	0.997-1.002
Mean	0.64	1	0.998-1.001
Total Fat			
Day 1	0.65	0.998	0.990-1.006
Day 2	0.96	1	0.99-1.01
Mean	0.73	0.998	0.99-1.01
SFA			
Day 1	0.49	0.99	0.97-1.02
Day 2	0.92	1.002	0.97-1.04
Mean	0.63	0.99	0.96-1.02
PUFA			
Day 1	0.37	1.02	0.98-1.07
Day 2	0.78	1.01	0.93-1.1
Mean	0.39	1.03	0.96-1.12
MUFA			
Day 1	0.61	0.995	0.98-1.02
Day 2	0.88	0.997	0.97-1.03
Mean	0.66	0.99	0.97-1.02
Vitamin B12			
Day 1	0.51	1.01	0.97-1.06
Day 2	0.56	1.03	0.93-1.16
Mean	0.44	1.03	0.97-1.11
Folate			
Day 1	0.15	0.998	0.995-1.001
Day 2	0.38	1.001	0.999-1.002
Mean	0.68	0.999	0.997-1.002
Iron			
Day 1	0.14	0.966	0.92-1.01
Day 2	0.82	1.002	0.98-1.02
Mean	0.51	0.99	0.96-1.02

Table 9: Unadjusted effects of health related characteristics on depression obtained from logistic regressions

Predictor	P-value	Odds ratio	95% CI
Medical insurance			
No	0.9	0.94	0.33-2.67
Yes*		1	
Waist circumference	0.57	1.02	0.96-1.08
BMI	0.65	1.01	0.96-1.06
Categorical BMI	0.95		
<18.5	0.88	1.21	0.1-14.62
18.5-24.9	0.65	1.45	0.3-7.05
25.0-29.9	0.65	1.34	0.37-4.85
≥30*		1	
Number of diseases	0.005	1.47	1.13-1.93

* reference category

CHAPTER 5

DISCUSSION

The major findings of this study are: 1) depression is positively related to the number of chronic diseases reported, 2) the majority of our study participants are unemployed, and the odds of depression in unemployed women was four times higher than for employed women, and 3) low intake of folate and iron is common in both depressed and non-depressed women. Overall, the percentage women reporting depression was 31.8%. For the entire population, the prevalence of combined food insecurity and food insecurity with hunger was 53%, and no relationship was found between stated depression and food security status. Mean intakes of protein, carbohydrate, total fat, and vitamin B₁₂ for Day 1 and Day 2 and iron for Day 2 all met the DRIs in both groups. No relationship was found between depression and nutrient intake. The mean BMI of both depressed and non-depressed groups fell within the obese range. There was no relationship between depression and BMI or waist circumference. A large percentage of our study individuals lacked health insurance, but this was not related to depression. There was no relationship between depression and sociodemographic variables (education, marital status, and number in household).

Depression in Low Income Female Blacks

Almost all study participants in our sample are black. The percentage of depression in our population is much higher than the national average for blacks (7.5%) (198). However, our results are similar to two previous studies of low income black females. Jesse et al. used BDI to evaluate symptoms of depression in 128 low income blacks and whites, aged 14 ~ 44 years and enrolled in an urban Midwestern pre-natal clinic, and found that 24% of blacks reported symptoms of depression at levels indicating risk for clinical depression (199). Similarly, Coiro

used CES-D, a self-report measure of depressive symptomatology developed for use in non-psychiatric populations, to evaluate symptoms of depression in 173 low-income, single, black women who participated in the Child Outcomes Study of the National Evaluation of Welfare to Work Strategies. In that study, all of the women (average age 29 years) lived in Fulton county, GA, and received Aid to Families with Dependent Children Program. Results showed that 40% reported symptom levels indicative of a diagnosis of clinical depression (200).

There are several differences between our study and the other studies that have looked at depression in low income populations. These including differences in the population sampled (e.g., different age ranges, different geographic regions, different marital status, participation in different assistance program) and differences in the way depression was assessed (e.g., using BDI/CES-D to evaluate depressive symptoms compared with “has a doctor ever told you that you have depression?”). Nonetheless, results from our study and previous studies suggest that low-income black women are significantly affected by depression and this could result in physical and other mental sequella.

Relationship between Depression and Food Security Status

Although the food stamp program is designed to help alleviate hunger, food insecurity is common in food stamp recipients (201, 202). Cohen et al. analyzed data from national household surveys conducted between June 1996 and January 1997, and found that FSP participants in the survey experienced more frequent food insecurity (50%) than eligible or near-eligible non-participants (34%, 25%, respectively) in the previous year. Characteristics of FSP participants in that study were more likely to be black, between 20 and 49 years of age, unmarried/separated/divorced, and less educated (201). Smaller regional studies have also examined the prevalence of food insecurity in FSP participants. Oberholser et al. assessed the

household food security among 245 food stamp recipients in Maryland. In that study, the majority of the participants (aged 19 to 70 years) were single (79%), female (97%), and blacks (64%). Researchers used a modified version of the US Food Security Survey Module to evaluate food security, and found that 66% of the sample experienced food insecurity with or without hunger (202). In contrast, our study used a modified version of the USDA Food Security Module Short Form and evaluated food security status in a female food stamp recipient population in the past 30 days. Our study sample shared similar characteristics with the above studies, that is, female, black, single/separated/divorced, and less educated. Although the methods used to evaluate food security were different between our study and these two previous studies, our results also showed high proportion of combined food insecure and food insecure with hunger (53%) in our study population. This suggests that food stamps are insufficient to meet the needs of the population or that food stamp recipients need nutrition education to help them understand how to maximize their food stamp benefits to attain a healthy diet.

A positive relationship between food insecurity and depression has been documented previously (17, 24, 203). One study was conducted in the Lower Mississippi Delta region to examine the association between household food insecurity and self-reported physical and mental health status in adults (203). The study sample lived in 36 counties in the Delta region of Arkansas, Louisiana, and Mississippi. More than half of the sample was female, black, and between the ages of 18 and 44 years. In that study, food security status was evaluated using the 18-question U.S. Food Security Survey Module, and mental health status was evaluated using the Short Form 12-item Health Survey (SF-12). Results showed that household food insecurity was associated with poorer self-reported mental health in this area (203). Further, Siefert et al. analyzed information from single women (aged 18 to 54 years) who received cash assistance in

an Urban Michigan county from the first wave of the Women's Employment Study (24). Food security status in that study was determined by asking the question: "which of the following describes the amount of food your household has to eat-enough to eat, sometimes not enough to eat, or often not enough to eat?"; depression was assessed by DSM-IV. Results showed that food insufficiency was a significant predictor of major depression (24). Moreover, a follow up study conducted by the same research group confirmed their previous results (17). However, we did not find a positive relationship between food insecurity and depression in our study.

Unexpectedly, both depressed and non-depressed women had high prevalence of food insecurity; and, although not significant, there was a lower percentage of food insecurity in depressed women (47.6%) compared with non-depressed women (55.6%).

Our study was considerably different than the other studies discussed above. For example, the study populations were different (e.g., both genders versus women only, different age ranges, all-income versus low-income only, different regional samples and sample size, different assistance types received), and assessment methods to determine food security and depression were different. Since food stamp recipients in our population had high prevalence of food insecurity and share similar characteristics, a larger sample size is probably needed to determine if there is a relationship between depression and food security status. Another reason is that we asked if the women had ever been diagnosed with depression, not if they currently suffered from depressive illness; thus, the temporal relationship may not have been optimal for observing a relationship. Finally, the women may accept that they are food insecure and not consider it a hopeless situation, and are thus not depressed. We accept our hypothesis that there is no relationship between depression and food security status.

Relationship between Depression and Nutrient Intakes

Food insecurity may result in low nutrient intakes (e.g., folate, iron) (14-16) which, in turn, may contribute to depression (20, 136, 137). Folate deficiency which occurs from a low dietary intake or failure to supplement adequately can, in turn, result in a SAM deficiency and elevated homocysteine levels (72); both have been found in depressed individuals (22). We compared nutrient intakes between depressed and non-depressed women, and also compared nutrient intakes of both groups with the DRIs. Results showed that low folate and iron intakes were common in both depressed and non-depressed women, and less than 50% participants in both groups met the DRIs for folate and iron. These findings are the result of a single 24 hour dietary recall at the beginning of the month and another at the end of the month; although the women stated this was typical of their usual intake, it is impossible to determine whether they actually had a folate or iron deficiency without looking at blood levels of these nutrients.

Almost all previous studies have used blood folate levels to look at the relationship with depression (20, 21); few studies have examined the association between dietary folate intake and symptoms of depression. To our knowledge, only Tolmunen et al. investigated the relationship between dietary folate intake and symptoms of depression in a cross-sectional study of Finnish men (204). In that study, symptoms of depression were assessed using the 18-item Human Population Laboratory Depression Scale. The dietary intake was assessed by 4-day food recalls, and nutrient intake was calculated using Nutrica software. Subjects (mean age 53 years) were grouped into tertiles according to their daily intakes of folate. Results showed that participants in the lowest tertile of folate intake had a 67% higher risk of being depressed than those in highest tertile (204). One year later, the same investigators published another prospective follow-up study. In that study, subjects were classified into two groups according to the median intake of

folate. Results showed that participants with folate intake below the median had a 3-fold higher risk of getting a discharge diagnosis of a depressive disorder during the follow-up period than those with higher folate intakes (205).

Dietary folate intake in our study population was low. However, we did not find an association between folate intake and depression. In our study sample, the mean age was 42.5 years, which was lower than the studies described above. Our participants were female, but the previous study showed an association between folate intake and depression in men. We did not use psychological instrument to quantify depressive symptoms and just recorded 2-day dietary recalls but not 4-day, which is a limitation of our study. It should be noted that every assessment of diet in free-living individuals has limitations. It is worth repeating that our study subjects were asked if they had ever had depression, so they may not have been depressed at the time the dietary recalls were obtained.

The diets of the women in our study are very poor. Poor folate intake in our study population is probably due to their low intakes of fruits/vegetables/fortified grain products. Low reported folate intake may result from under-reporting, which is common in 24-hour dietary recalls. Under-reporting is common in females and persons who were overweight or obese (206, 207); however, fruits, vegetables, and grains are generally not the foods under-reported. The majority of the population was obese; therefore, under-reporting may have occurred. A follow-up study should take three day dietary recalls at the beginning of the month, three day dietary recalls at the end of the month, and with weekend days included in each recalls. This should provide better overview of dietary intake in this population.

It is of concern that the women in this study, the majority of which were of child bearing potential, did not consume adequate folate. Folate reduces the risk of neural tube defects in the

fetuses of pregnant women, and women 18 years and older should have 400 ug/day. Clearly our participants fell far short of this mark. Nutrition education may help women improve their intake.

Anemia may develop from the low dietary intake of folate and iron (146). The relationship between anemia and depression has been reported previously (208, 209). Corwin et al. recruited women from two hospitals in central Pennsylvania, and used hemoglobin concentration and CES-D to evaluate anemia and symptoms of depression, respectively. Women who suffered from anemia were at increased risk for depression (208). Another cross-sectional study in Greece analyzed the data of a population of elderly, and used hematocrit and the Mini Mental State Examination to evaluate anemia and cognitive impairment, respectively. Results showed statistically significant association between anemia and possible cognitive impairment (209). It should be noted that cross sectional studies cannot be used to prove a cause and effect relationship, and intervention studies are needed to confirm these relationships. In our study, we examined the relationship of women who had ever been diagnosed with anemia with women who had ever been diagnosed with depression; therefore, it's possible that the temporal relationship in our cross-sectional study was missing.

Future studies with larger sample sizes and more detailed instruments need to be performed to better assess this question. However, based on our current results, we reject our original hypothesis, since we did not find a relationship between depression and nutrient intakes.

Relationship between Depression and BMI

The relationship between depression and BMI is not clear and study results have been inconsistent. McIntyre et al. analyzed data from Canada's Canadian Community Health Survey and examined the relationship between depression and obesity (210). In that study, individuals aged ≥ 15 years with a diagnosis of depression in their lifetime (based on the DSM-IV) were

studied. Results showed that individuals with a lifetime history of depression were more likely to be obese (as defined as $BMI \geq 30$) compared with individuals without this history (210). Simon et al. also showed in a cross-sectional epidemiologic survey of nationally representative adults that obesity was related to a lifetime diagnosis of major depression (211). As with all cross-sectional studies, the study by Simon et al cannot be used to show cause and effect.

Other investigators have failed to show a relationship between depression and BMI. In one community study, researchers analyzed data from two large, school-based, studies of adolescent health and well-being. Results showed no relationship between obesity and symptoms of depression (212). Another cross-sectional study analyzed data from the Centers for Disease Control and Prevention's 1999 Youth Risk Behavioral Surveillance system, and results also showed no relationship between self-reported depressive symptoms and BMI in adolescents' population (213). The difference seen in the results of the previous four papers may have been the result of the age of the participants or that different methods were use to assess depression. However, our study did not show this relationship between depression and BMI. This may be because of the high prevalence of obesity in both of our groups—this may have precluded our seeing a different between the populations. It also may be that blacks appear to have fewer weight-related concerns than other ethnic groups (213). In a cross-sectional study, Daniels reported that overweight blacks (52%) were more likely to see their weight as “OK” than overweight whites (38%), and obese blacks (22%) viewed their weight as “OK” compared with obese whites (11%) (213).

Since the prevalence of obesity is so high, it is important to continue to look for associations with anthropometric, health, and socioeconomic factors. In this way, interventions can be designed that may help lessen depression in these women.

Relationship between Depression and the Number of Chronic Diseases

In our study, the strongest association was between reported depression and the number of chronic diseases. Depressed women reported more chronic diseases than non-depressed women. Depression and physical chronic diseases were recorded by asking one question with multiple parts, that is, “has a doctor ever told you that you have: heart disease, high blood pressure etc.” Analysis of these responses is difficult, since the temporal association between depression and chronic diseases might not match; thus, we decided to look at the relationship of the sum of the diseases, rather than the individual responses.

A reciprocal relationship between depression and the presence of chronic diseases has been reported previously in various populations. For example, post-MI depression was related with the severity of the MI (161, 162). The co-morbidity of depression with MI was associated with a 2 to 2.5 fold increased risk of impaired cardiovascular outcome and cardiac mortality (165). Thus, preventing both physical and mental diseases is important for the improvement of quality of lives in our study population.

Relationship between Depression and the Health Insurance

A large percentage of our study individuals lacked health insurance, but there was no difference between groups for either having insurance or with the relationship with depression. Among participants with health insurance, the majority of them were covered by Medicaid, Medicare, or both, rather than private insurance. The Medicaid program provides medical benefits to low-income people who may have no medical insurance or inadequate medical insurance (214). The Medicare program provides the specific medical care needs for the elderly, disabled persons, and people with end-stage renal disease (215).

Lack of health insurance is a barrier for using health services (216). Taylor et al. analyzed data from the 2000 Medical Expenditure Panel Survey, and reported that uninsured women were less likely to use any health services (71%) than women with either public (94%) or private (92%) insurance. These health services included ambulatory care services, the use of prescription drugs and preventive health services, and inpatient hospital care (216).

Individuals with public health insurance may perceive that they experience a lower quality of health care than individuals with private health insurance (217). Using data from the 1996 Medicare Current Beneficiaries Survey, Pourat et al. analyzed the experiences of chronically ill elderly with respect to overall quality, access to care, and physicians' technical, interpersonal, and information-giving skills (217). Results showed that Medicare beneficiaries often rated their physician's skills lower than did those with private supplement coverage. Moreover, fewer individuals with chronic diseases were satisfied with overall care or rated their physician's skills highly, compared to those without chronic diseases (217).

In addition, some sociodemographic characteristics are related to low frequency of use of any health services. Black women reported fewer ambulatory health service visits than white women. Women from the South (90%) were less likely to reported use of any health services than women in the Midwest (94%). Women who lived in rural area were less likely to use any health services, have an ambulatory visit, or obtain prescriptions compared to women in near rural area. Poor or near poor women was less likely to use of any health services and ambulatory care compared with women in the highest income category (216).

Our study participants are almost all black, live in rural area, and come from Southeast Louisiana. Most reported that they did not have trouble accessing health care and even reported they underwent routine screening procedures. To improve overall health in this population,

policy makers need to decrease this gap and how to improve Medicare and Medicaid services provided to the low income population.

Relationship between Depression and Unemployment

The majority of our study participants were unemployed, and the odds of depression for unemployed women was four times the odds for employed women. Our results agree with two previous studies using national data sets. Marcotte et al. analyzed data from the 1990-1992 NCS and reported that subjects who were employed were significantly less likely to report a lifetime history of depression compared the unemployed. Among the unemployed, 21.9% reported a lifetime history of depression, compared to 15.2% for the employed (218). Dooley et al. analyzed data from the National Longitudinal Survey of Youth for the years 1992-1994 for subjects who were employed in 1992, and used CES-D scale to assess depressive symptoms. Results showed that the employed reported fewer symptoms of depression compared to the unemployed. In that study, 15.5% of the employed reported an increased number of symptoms of depression in comparison to 33.5% the unemployed in 1992. Individuals who remained employed from 1992 to 1994 reported less depressive symptoms than those who became unemployed (219).

Employment may be a protector for the risk of depression. Although, individuals with depression were more likely to lose their jobs compared to those without depression (59), individuals who were employed reported less depressive symptoms than those who were unemployed (220). Danziger et al. recruited 426 individuals who lost cash benefits when Michigan terminated its General Assistance Program (GA) in 1991. All individuals were interviewed one and two years after the GA ended, and symptoms of depression were assessed using CES-D scales. Results showed only 38% individuals were employed at any time during the two years after GA ended, and only 29% had worked in a regular job prior to the second

interview. Individuals who worked in the more recent period after GA termination were less likely to be at risk of depression (depressive symptoms in CES-D scales ≥ 16 indicate a risk of depression) than those who did not (220).

Thus, providing employment for FSP recipients should be beneficial and improve their mental health. The Food Stamp Employment Training Program (FSETP) has been created to help food stamp recipient gain skills, training, or experience which will increase their ability to obtain employment. However, FSETP participants constitute less than 9% of the food stamp population because many food stamp recipients are exempt from work requirements due to age, health, or involved in child care. At this time, there are no nationwide data available to describe whether the FSETP actually helps participants get a job. Outcome data at the state level (a total 15 states were included in this study, including: California, Texas, Florida, and New Mexico; Louisiana was not included in this study) showed there were barriers for FSETP participants to find a job successfully. Since FSETP participants generally have limited education (below high school graduate), limited work history, few job skills, lack of transportation, mental health issues or substance abuse, and are homeless, it is often difficult for employers to hire them. Some state officials noted that program participants often depend on seasonal employment and many of them rarely hold a job more than 3 months. Unfortunately, the USDA and Departments of Labor and Health and Human Services have no plans to evaluate the effect of FSETP (221).

Use of Logistic Regression Analysis

The major aim of our study was to determine what socioeconomic or health variables (e.g., number in household, educational level, and food security) had an effect on depression. Because of the small number of study participants, we elected to class our study as exploratory. Thus we used a conventional criterion for selecting a model in logistic regression analysis. The

criterion used in our study was that a variable has to be significant at 0.05 level before it could enter into a model; and a variable in the model had to be significant at the 0.1 for it to remain in the model.

Our logistic regression analysis results showed few relationships, and showed that other relationships, such as the ones between depression and the number in household or educational level fell just short of significance. These results are tantalizing and suggest that a larger sample size may have shown that number in household and educational levels affect depression. One approach for future investigations would be to change the criterion for statistical significance to a variable has to be significant at the 0.1 level before it can enter into a model, and a variable in the model has to be significant at the 0.15 for it to remain in the model; thus making the study a true exploratory study. However, this criterion might increase the chance of committing a type I error (a type I error occurs when a true null hypothesis is rejected incorrectly; α : denotes the probability of making a type I error) (222), although a type II error (a type II error occurs when an untrue null hypothesis is failed to be rejected; β : denotes the probability of making a type II error) may decrease and the power of a test (“the power of a test is the probability of correctly rejecting the null hypothesis when it is false”; $1 - \beta$: denotes the power of a test) may increase (222). In order to better understanding the relationship between depression and number in household or educational levels, future studies should increase the sample size, which can both decrease type I and type II errors and increase the power of a study (222). The relationships that approached significance might give clues as to what variables might be related, and the variability in this study could be used to calculate sample size (223).

Although a large sample yields more accurate information about a population than a small sample, one cannot ignore some critical issues such as: cost, time, and labor, which will be

needed in such a study. One also needs to recruit an appropriate population for the study, and design the best sampling design (such as random sampling or the non-probability sampling that was used in this study) (224). Whether such an extensive study is feasible should be determined.

Conclusion

This study adds to the evidence of associations between depression and the number of diseases or unemployment. Results suggest that depression is more prevalent among FSP recipients with increased physical diseases and among those with unemployment. In addition, increased food insecurity, increased obesity, low dietary intake of folate and iron, and low educational level are common in FSP recipients. Our results showed a potential relationship between depression and educational level or number in household, but future research is needed to confirm these associations.

Limitations

The major limitation in our study was our small sample size. The second limitation was that all of the information was self-reported, and may be biased. We cannot determine the effects of an unknown degree of measurement error on our findings. The third limitation is that the study population included only female food stamp recipients from rural area in Southeast Louisiana. The characteristics of this population were female, black, food insecure, and obese. This homogeneous population had few differences between depressed and non-depressed groups and limited statistical analysis. Finally, with the cross-sectional design of this study, we were unable to determine whether risk factors (food security status, low nutrient intakes, BMI, and chronic diseases) preceded or followed depression.

Future Research

Two approaches could be used depending on the study question in future studies. The first one is to increase sample size from a more diverse population, for example, from different locations, ethnic groups (white, black, Hispanic, Asian) and genders. The second one focuses on further study of low income population. Different low income populations may have different characteristics; for example, the prevalence of food insecurity is more common in FSP recipients than eligible or near-eligible non-participants (201). So, it would be interest to recruit female, black, eligible non-food stamp participants to our study, and compare different characteristics between these two low income populations. We could also evaluate whether participating in the FSP is a risk factor for reducing depression. A national data set might be used to compare different characteristics between food stamp participants and general population.

Future studies should use psychological instruments to quantify the symptoms of depression, and collect data about nutrient blood levels and extend dietary recalls to multiple days. That way the relationships among depressive symptoms, nutrient intakes, and nutrient deficiencies can be understood more fully. We have collected data about specific chronic diseases such as heart disease, high blood pressure, diabetes, and cancer, and one could examine the relationship between depression and specific diseases rather than the number of diseases.

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APPENDIX A: INFORMED CONSENT

TITLE OF RESEARCH PROJECT Effects of Weight History, Resource Cycling, and Fast Food on Overall Diet Quality and Health in Low-Income Louisiana Women

The purpose of this study conducted by the Louisiana State University (LSU) Agricultural Center investigators is to evaluate your diet. This will be done by completing two 24-hour diet recalls—one in person and one approximately 30 days later by telephone. This is a standard test used to assess food intake and consists of asking you what you had to eat the day prior to the recall. You will also be asked to keep your food receipts at during this same time period.

This information will be used to understand more fully what foods people are eating and the impact they have on overall diet quality. There are no risks associated with this study. Although you may receive no personal benefit from participating in this study, society as a whole may benefit through improved understanding of what foods people are choosing to eat and where they are purchasing these foods.

Only LSU researchers involved in this study will have access to these recalls. Results of this study, including any publications, will not identify individuals by name. Data will be presented either in summary form or stripped of individual identifiers. You may choose not to participate in this aspect of the study. You may withdraw from this study at any time without prejudice.

The study has been discussed with me and all questions have been answered to my satisfaction. I may direct additional questions regarding this study to Dr. Carol O'Neil, School of Human Ecology, at 225-578-1631. If I have questions about subjects' rights or other concerns, I can contact Dr. David Morrison at 225-578-8236.

With full knowledge of the above information, I voluntarily consent to take part in this study.

Name of participant (please print): _____

Signature of participant: _____ Date: _____

Mailing address: _____

(Street)

(City)

(Zip)

Phone: _____

Witness (please print): _____

Signature of witness: _____ Date: _____

INFORMED CONSENT

TITLE OF RESEARCH PROJECT Effects of Weight History, Resource Cycling, and Fast Food on Overall Diet Quality and Health in Low-Income Louisiana Women

The purpose of this study conducted by the Louisiana State University (LSU) Agricultural Center investigators is to study the relationship between diet, weight, and income. To do this, you will be asked questions about your weight and history of weight, your perceptions of weight and diet, income, nutrition education, and perceptions of diet and health.

This information will be used to understand more fully the relationships among income, diet, and weight. For participating in this study, you will receive a stipend. Further, society as a whole may benefit through improved understanding of weight, diet, and health in a population of low-income women.

Only LSU researchers involved in this study will have access to these recalls. Results of this study, including any publications, will not identify individuals by name. Data will be presented either in summary form or stripped of individual identifiers. You may choose not to participate in this aspect of the study. You may withdraw from this study at any time without prejudice.

The study has been discussed with me and all questions have been answered to my satisfaction. I may direct additional questions regarding this study to Dr. Carol O'Neil, School of Human Ecology, at 225-578-1631. If I have questions about subjects' rights or other concerns, I can contact Dr. David Morrison at 225-578-8236.

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Signature of participant: _____ Date: _____

Mailing address: _____

(Street)

(City)

(Zip)

Phone: _____

Witness (please print): _____

Signature of witness: _____ Date: _____

INFORMED CONSENT

TITLE OF RESEARCH PROJECT Effects of Weight History, Resource Cycling, and Fast Food on Overall Diet Quality and Health in Low-Income Louisiana Women

The purpose of this study conducted by the Louisiana State University (LSU) Agricultural Center investigators is to evaluate your weight. This will be done by weighing you with a standard scale and measuring your height with an instrument called a stadiometer; a stadiometer is a standard method to measure height. These values will be used to calculate body mass index (BMI), a value designed to categorize people by weight status. Using a tape measure, your waist and hip measurements will also be taken. Together these measurements will allow researchers to assess your weight status and what's called central abdominal obesity.

This information will be used to understand more fully risk factors potentially associated with development of chronic disease, such as type 2 diabetes mellitus. These are standard medical risks and there are no risks associated with this study. You will personally benefit from participating in this study by learning your weight, BMI, waist circumference, and waist-to-hip ratio. Society as a whole may benefit through improved understanding of weight status in a population of low-income women.

Only LSU researchers involved in this study will have access to these recalls. Results of this study, including any publications, will not identify individuals by name. Data will be presented either in summary form or stripped of individual identifiers. You may choose not to participate in this aspect of the study. You may withdraw from this study at any time without prejudice.

The study has been discussed with me and all questions have been answered to my satisfaction. I may direct additional questions regarding this study to Dr. Carol O'Neil, School of Human Ecology, at 225-578-1631. If I have questions about subjects' rights or other concerns, I can contact Dr. David Morrison at 225-578-8236.

With full knowledge of the above information, I voluntarily consent to take part in this study.

Name of participant (please print): _____

Signature of participant: _____ Date: _____

Mailing address: _____

(Street)

(City)

(Zip)

Phone: _____

Witness (please print): _____

Signature of witness: _____ Date: _____

APPENDIX B: HOUSEHOLD SURVEY

Household Survey – SRDC 2003—04

Name: _____ Interview Date: _____

Social Security #: _____

Address: _____
Street City State Zip

Home phone: _____ Work phone: _____

Relative/ Other phone: _____

1. **Education level:** (Check all that apply.)

- a. _____ High school diploma b. _____ GED c. _____ Some college
d. _____ College degree (record highest degree) e. _____ Trade or technical college
f. _____ FIND Work / STEP g. _____ Project Independence h. _____ Nutrition classes (i.e. EFNEP, FNP) (list): _____
i. _____ Other training programs: (list) _____

2. Are you currently attending any school or training programs? _____ Yes _____ No
2a. What? _____ 2b. How many hours? _____

3. Are you currently working? _____ Yes _____ No
3a. Where/ What type of job? _____
3b. How many hours per week? _____
3c. What is your hourly wage? Or weekly salary? _____
3d. What benefits are available at your job? _____

4. **Marital status:**

- a. _____ Married b. _____ Single, living with parents/ relatives _____
c. _____ Single, living alone d. _____ Single, living with man
e. _____ Divorced, living alone f. _____ Divorced, living with man g. _____ Widowed

5. **How many children do you have?** _____

- a. Ages of children: _____

6. **Persons living in the household:** (how many)

- a. _____ own children b. _____ other children c. _____ Mother
d. _____ Father e. _____ siblings f. _____ Other relative(s)
g. _____ Female friend h. _____ Male friend i. _____ Other

Medical Insurance and Care:

7. Do you have medical insurance? _____ Yes _____ No
a. Government provided: Medicaid? _____ Yes _____ No Medicare? _____ Yes _____ No
b. Is medical insurance available through your employer? _____ Yes _____ No
c. Does your employer pay all, a portion of, or none of your medical insurance?
d. How much do you pay for medical coverage? _____

8. Do your children have medical insurance? ____ Yes ____ No
 a. Government provided: LaChip ____ Yes ____ No
 b. Is medical insurance available through your employer for your children? ____ Yes ____ No
 c. Does your employer pay all, a portion of, or none of your children's health insurance?
9. Are you able to get the medical care that you need? ____ Yes ____ No
 a. If not, why not? _____
10. Are your children able to get the medical care they need? ____ Yes ____ No
 a. If not, why not? _____
11. Compared with other people your age, how would you rate your overall physical health at the present (circle one)
- | | | | | |
|------|------|------|-----------|------------|
| poor | fair | good | excellent | don't know |
| (1) | (2) | (3) | (4) | (5) |
12. Compared with other people your age, how would you rate your overall physical health over the past five years (circle one)
- | | | | | |
|------|------|------|-----------|------------|
| poor | fair | good | excellent | don't know |
| (1) | (2) | (3) | (4) | (5) |
13. Compared with other people your age, how would you rate your overall mental health at the present (circle one)
- | | | | | |
|------|------|------|-----------|------------|
| poor | fair | good | excellent | don't know |
| (1) | (2) | (3) | (4) | (5) |
14. Compared with other people your age, how would you rate your overall mental health over the past five years (circle one)
- | | | | | |
|------|------|------|-----------|------------|
| poor | fair | good | excellent | don't know |
| (1) | (2) | (3) | (4) | (5) |
15. When was the last time you visited a physician? _____
 a. Did you go to the physician's office or to the emergency room? _____
16. When was the last time you visited a dentist? _____
 a. Was it a routine visit or did you go in on an emergency basis? _____
17. Have you ever had a PAP smear? ____ a. Do you have them regularly? ____ b. When was your last PAP smear? ____ c. Results? ____ d. How did you pay for the PAP smear? _____

18. Have you ever had a mammogram? _____ a. Do you have them regularly? _____
b. When was your last mammogram? _____ c. Results? _____ d. How did you pay for
the mammogram? _____

19. Has a doctor ever told you that you have:
- a) Heart disease _____
 - b) High cholesterol _____
 - c) High blood pressure _____
 - d) Diabetes _____
 - e) Fluid Retention _____
 - f) A problem weighing too much _____
 - g) Anemia _____
 - h) Cancer _____
 - i) Arthritis _____
 - j) Osteoporosis _____
 - k) Depression _____

20. Are you taking any kind of medicines?

List: _____

21. **Sources of Income** : (record amount and frequency)

- a. Wages and salaries (self) _____
- b. Wages and salaries (other household members) _____
- c. Tips, commission, overtime _____
- d. Odd jobs (doing nails, hair, babysitting, transportation, etc.) _____
- e. Social Security _____
- f. SSI _____
- g. Child support _____
- h. Unemployment Compensation _____
- i. Workmen's Compensation _____
- j. Veteran's benefits _____
- k. Regular gifts from family or friends to assist with bills or expenses _____
- l. Other income sources _____

22. **Government Benefits as Sources of Income**

- a. TANF _____
- b. EITC (Earned Income Tax Credit) _____
- c. Child care assistance _____
- d. Housing assistance _____
- e. Energy/Fuel Assistance _____
- f. Transportation Assistance _____
- g. Educational grants or loans _____
- h. Other _____

23. **Expenses**

- a. Rent or house payment _____
- b. Electric/ Gas _____

- c. Sewer/ Water/ Trash collection _____
- d. Cable _____
- e. Telephone _____
- f. Cell phone/ pager _____
- g. Credit card payments _____
- h. Loan payments _____
- i. Rent-to-own payments _____
- j. Life or burial insurance _____

24. Does anyone help you pay your monthly expenses? ____ Yes ____ No

- a. Who helps? _____
- b. How often? _____
- c. How much? _____
- d. What do they help pay for? _____

25. To what extent is your income sufficient to live on?

26. If you do not have enough money to pay your bills, what are some things that you will do without?

_____ What do you do to stretch your money? _____

27. Transportation:

- a. Do you have a valid driver's license? ____ Yes ____ No
- b. Do you own a car? ____ Yes ____ No
- c. If not, do you have reliable transportation? ____ Yes ____ No

28. Feelings about Employment: (If applicable)

- a. Are you satisfied with your current job? ____ Yes ____ No

b. What do you like most about your job? _____

c. Is there a job that you would rather be doing? What? _____

d. Is there something that makes it difficult for you to keep your job? If so, what? _____

APPENDIX C: FOOD SECURITY QUESTIONS

SRDC 2003—04 USDA Food Security Module (modified)

[Administer these items in a fairly standard manner. Upon completion of these items, go on to the height, weight, and waist circumference measures, then the 24-hour food recall]

The next questions are about the food eaten in your household in the last 30 days and whether you were able to afford the food you need.

1. "The food that I bought just didn't last, and I didn't have money to get more." Was that often, sometimes, or never true for you in the last 30 days?
2. "We couldn't afford to eat balanced meals." Was that often, sometimes, or never true for you in the last 30 days?

(1) Often true (2) Sometimes true (3) Never true

Probe: What does "balanced meal" mean to you?

3. In the last 30 days, did you ever cut the size of your meals or skip meals because there wasn't enough money for food?

(1) Yes _____ (2) No _____

4. In the last 30 days, did you ever eat less than you felt you should because there wasn't enough money to buy food?

(1) Yes _____ (2) No _____

5. In the last 30 days, were you ever hungry but didn't eat because you couldn't afford enough food?

(1) Yes _____ (2) No _____

6. In the last 30 days, have you not eaten in order to have enough food for your children?

(1) Yes _____ (2) No _____

7. Which of these statements best described the food eaten in your household in the last 30 days? (Check only one)

- (1) We always have enough to eat and the kinds of food we want
- (2) We have enough food to eat but NOT always the KINDS of food we want
- (3) SOMETIMES we don't have ENOUGH to eat
- (4) OFTEN we don't have ENOUGH to eat

8. Who does the majority of the grocery shopping in your household? (circle one)
- a) Self
 - b) Spouse/significant other
 - c) Parent(s)
 - d) Child(ren)
 - e) Friends/roommate
 - f) Other (describe): _____
9. Who does the majority of cooking for your household? (circle one)
- a) Self
 - b) Spouse/significant other
 - c) Parent(s)
 - d) Child(ren)
 - e) Friends/roommate
 - f) Other (describe): _____
10. Where do you do the majority of your food shopping?
11. Where else do you shop for food?
12. What amount of food stamps do you receive each month? _____
13. How much money do you spend for food above the amount of food stamps that you receive each month? _____
14. If you need to, how do you stretch your food stamps to reach the end of the month?

15. On the average, how much does your household spend per week on food?
- | | | | | | |
|--------|---------|-----------|-----------|-----------|-----------|
| \$0-25 | \$26-75 | \$ 76-125 | \$126-200 | \$201-300 | \$301-500 |
| (1) | (2) | (3) | (4) | (5) | (6) |
16. How many persons does this feed per week? (fill in a number in each of the spaces below; fill in zero if applicable)
- a. _____ number of adults
 - b. _____ number of teenagers
 - c. _____ number of children
 - d. _____ number of infants

17. Do you receive WIC? ____ Yes ____ No
18. How would you rate your eating habits? (circle one)
- | | | | |
|------|------|------|-----------|
| Poor | Fair | Good | Excellent |
| (1) | (2) | (3) | (4) |
19. How would you rate the nutritional quality of your diet? (circle one)
- | | | | |
|------|------|------|-----------|
| Poor | Fair | Good | Excellent |
| (1) | (2) | (3) | (4) |
20. About how many calories do you think you eat a day? (circle one)
- | | | | | |
|-----------------|-----------------|---------------------|------------------|------------------|
| Much
Too Low | Somewhat
Low | Just About
Right | Somewhat
High | Much
Too High |
| (1) | (2) | (3) | (4) | (5) |
21. How would you rate your knowledge of nutrition? (circle one)
- | | | | |
|------|------|------|-----------|
| Poor | Fair | Good | Excellent |
| (1) | (2) | (3) | (4) |
22. On average, how often do you eat in fast-food restaurants? (circle one)
- | | | | | |
|--------------------|----------------------------|---------------------------|---------------|---------------|
| Rarely
Or Never | Several Times
Per Month | Several Times
Per Week | Once a
Day | Most
Meals |
| (1) | (2) | (3) | (4) | (5) |
23. Which fast-food restaurants do you eat in most often?
24. What do you typically order in these fast-food restaurants?
25. On average, how often do you eat in other types of restaurants?
- | | | | | |
|--------------------|----------------------------|---------------------------|---------------|---------------|
| Rarely
Or Never | Several Times
Per Month | Several Times
Per Week | Once a
Day | Most
Meals |
| (1) | (2) | (3) | (4) | (5) |
26. Which restaurants do you eat in most often?

27. What do you typically order in these restaurants?
29. Use the silhouettes above to answer the following questions about yourself (for each item, fill in the number of the corresponding silhouette).

- | | | |
|----|--|-------|
| a. | Which figure is closest to your size? | _____ |
| b. | Which figure is closest to the figure you desire? | _____ |
| c. | Which figure represents you as a child? | _____ |
| d. | Which figure represents you as a teenager? | _____ |
| e. | Which figure is closest to your highest adult body weight? | _____ |
| f. | Which figure is closest to your lowest adult body weight? | _____ |

30. Do you think you were overweight as a child or teenager? (If yes, proceed with the Perception of Teasing Scale - POTS.)

APPENDIX D: HEIGHT AND WEIGHT RECORDING CHART

Name: _____

Date: _____

Machine settings:

Height (stated): _____

Age (stated): _____

Weight & BMI: _____

Weight & BMI: _____

Weight & BMI: _____

Waist Circumference: _____

Waist Circumference: _____

Waist Circumference: _____

Comments:

VITA

Yifang Bai was born on February 1, 1970, in Beijing, People's Republic of China. After graduating from the 66th high school of Beijing in July of 1988, she attended Haidian University. After she graduated from Haidian University in 1991, she worked in Zhongguancun Hospital as a resident. In 1994, she attended Peking Union Medical College and graduated with a Bachelor of Clinical Medicine in 1998. Meanwhile, she continued to work in Zhongguancun Hospital. In 1999, she worked as an attending physician in the department of internal medicine in that hospital. She attended graduate school, Peking Union Medical in 2000. In 2001, she and her husband and daughter moved to Baton Rouge. In the fall of 2004, she began a graduate program at Louisiana State University. She plans to graduate in the fall of 2006 with a Master of Science degree in Nutrition.