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Examining Weight Gain in Treatment-Seeking African American Smokers: A Biopsychosocial Approach

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UNIVERSITY OF MIAMI

EXAMINING WEIGHT GAIN IN TREATMENT-SEEKING AFRICAN AMERICAN
SMOKERS: A BIOPSYCHOSOCIAL APPROACH

By

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Research has shown that African Americans gain excessive, or more than average, weight after smoking cessation. However, African Americans have been underrepresented in post-cessation weight gain research. Associations between weight gain and sex, age, cortisol levels, depressive symptoms, weight concern, and socioeconomic status have been documented, but few studies have examined 1) the pattern of weight gain and 2) factors associated with weight gain among African American smokers. The current study aimed to examine biopsychosocial predictors of weight gain in a sample of treatment-seekers.

Data were drawn from a randomized controlled trial (RCT) testing the efficacy of a culturally specific smoking cessation intervention among African Americans. The intervention consisted of eight sessions (four weeks) of group cognitive behavioral therapy (CBT) and eight weeks of transdermal nicotine patches (TNP). Participants (N=342) completed assessments at baseline, the end of counseling (EOC), 3-, 6-, and 12-month follow ups. Baseline measures included salivary cortisol, depressive symptoms, and weight concern. Weight and self-reported smoking status were measured at all assessments. Random effects (mixed effects) multilevel modeling including all time-points was used to examine the pattern and predictors of weight gain over twelve months

post CBT. A fully unconditional model was specified to test the pattern of weight gain over time and the appropriateness of using random effects modeling for this data. Next, smoking status was included as a time-varying variable on level 1. The group level effects of biopsychosocial variables were then examined on level 2. Finally, cross-level interactions of the biopsychosocial factors and smoking status on weight were explored.

Results of a univariate model revealed that weight significantly increased among those who remained abstinent over twelve months post CBT. Controlling for intervention condition and baseline obesity, smoking status positively predicted weight gain in the full sample, such that abstinence was associated with increased weight. A multivariate analysis revealed that male sex and weight concern were positively associated with baseline weight. Exploratory analyses indicated that depressive symptoms moderated the association between smoking status and weight.

In this sample of African American smokers, weight gain was comparable to previous post-cessation weight gain research. We also found that psychosocial factors emerged as most important in predicting weight gain. Future research is needed to assess further the psychosocial variables, specifically depressive symptoms, which predict weight gain in African American smokers. Examining predictors of weight gain will inform future smoking cessation interventions and help elucidate factors that contribute to tobacco- and obesity-related health disparities.

Table of Contents

Chapter 1: Introduction

African Americans suffer disproportionately from tobacco-related diseases compared to whites. Post-cessation weight gain, or weight gain attributable to quitting smoking, may be a contributing factor to these disparities, and African Americans have been found to gain excessive weight after smoking cessation (Klesges et al., 1998; Williamson et al., 1991). Sex, age (Williamson et al., 1991), cortisol levels (Bjorntorp, 2001, depressive symptoms (Killen et al., 1996), weight concern (Borrelli & Mermelstein, 1998), and socioeconomic status (SES; Swan & Carmelli, 1995; Williamson et al., 1991) have been found to influence weight gain in primarily white samples of smokers. However, few studies have examined the factors associated with weight gain among African American smokers. The purpose of this study was to examine weight gain among African American smokers enrolled in a smoking cessation intervention.

Cigarette smoking is the single leading cause of preventable death and disease in the U.S., and the prevalence of smoking has remained steady among African American adults (18.1%) in recent years (Agaku, King, & Dube, 2014). Smokers are at a higher risk than nonsmokers of developing chronic diseases, including heart disease, stroke, and various cancers [Centers for Disease Control and Prevention (CDC), 2013]. Tobacco-related illnesses have caused more deaths per year than all other drugs, homicides, suicides, motor vehicle accidents, and HIV/AIDS combined [U.S. Department of Health and Human Services (HHS), 2010]. African Americans, specifically, experience the highest rates of tobacco-related morbidity and mortality of all racial/ethnic groups (Fiore, 2008). For example, African American males are 34% more likely to develop lung cancer

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than white males (American Lung Association, 2010), despite a similar smoking prevalence in both groups (25.5% and 23.6%, respectively).

Obesity is the second leading cause of preventable death in the U.S., leading to health conditions, such as heart disease, stroke, and type 2 diabetes (Khan et al., 2009). Approximately 35% of American adults are obese [body mass index (BMI) > 30; Ogden, Carroll, Kit, & Flegal, 2012], and the prevalence of overweight (BMI 25-30) is approximately 73.9% for males and 63.7% for females (Flegal, Carroll, Kit, & Ogden, 2012). African Americans have a higher rate of obesity (49.5%) and being overweight (76.7%) than whites (34.3% and 66.7%, respectively; Flegal, 2012; HHS, 2012). African American females (82.1%) are the most overweight and obese compared to women of other U.S. racial/ethnic groups (HHS, 2012). Taken together, examining factors related to smoking and weight gain among African American smokers has implications for health disparities.

Post-cessation Weight Gain: Overview and Mechanisms

The benefits of quitting smoking outweigh the effects of weight gain (Clair et al., 2013); however, post-cessation weight gain remains a pertinent issue to address among African American smokers. Approximately 80% of smokers report weight gain after quitting, which usually occurs within the first three months post cessation (Aubin, Farley, Lycett, Lahmek, & Aveyard, 2012). The general population gains an average of one to two pounds (lbs; 0.5 to 1 kg) within one year (Hutfless et al., 2013), yet former smokers gain an average of four to ten lbs (2 to 5 kg) within the first twelve months after quitting (Aubin et al., 2012; Flegal, 2012; Perkins, 1993). The weight status of former smokers approximates never smokers (Reas, Nygard, & Sorensen, 2009). After the initial period

of abstinence, former smokers weigh twice as much as current smokers (Reas et al., 2009; Flegal, 2012). Therefore, incorporating information on weight gain is important in smoking cessation interventions.

Mechanisms of post-cessation weight gain. Mechanisms that drive post-cessation weight gain have been studied. The absence of nicotine is among the key factors involved in post-cessation weight gain. Many individuals use smoking to control weight and appetite because nicotine increases energy expenditure (Audrain-McGovern & Benowitz, 2011). Nicotine promotes the release of the neurotransmitters norepinephrine, dopamine, and serotonin (McGehee & Role, 1995), which are important in increasing metabolic rate (Benowitz, 1996a; Schwid, Hirvonen, & Keesey, 1992). Therefore, the cessation of nicotine considerably slows the metabolic rate, resulting in weight gain (Filozof, Fernandez Pinilla, & Fernandez-Cruz, 2004). Consequently, nicotine replacement therapy [e.g., transdermal nicotine patch (TNP)] has been examined as a method to prevent post-cessation weight gain (Farley, Hajek, Lycett, & Aveyard, 2012). Studies have found that the use of nicotine replacement therapy may delay weight gain (Filozof et al., 2004) or reduce the amount after quitting (Schnoll, Wileyto, & Lerman, 2012).

Furthermore, nicotine has an anorectic effect, as the release of dopamine and serotonin also promotes the suppression of eating (Miyata, Meguid, Fetissov, Torelli, & Kim, 1999; Schwid et al. 1992). As a result, appetite increase, a symptom of nicotine withdrawal, occurs with the cessation of nicotine (Filozof et al., 2004; Hughes & Hatsukami, 1986). This specific symptom of withdrawal has been found to uniquely affect weight gain post-cessation. For example, Stamford et al. (1986) showed that an

increase in mean caloric intake accounted for 69% of weight gain after cessation in females. John, Meyer, Rumpf, Hapke, and Schumann (2006) found that appetite increase predicted BMI in the long-term, independent of other nicotine withdrawal symptoms. Particularly, ex-smokers who reported increased appetite had a higher BMI at a three year follow up than those who did not.

Post-cessation weight gain and health. Post-cessation weight gain contributes to negative health behaviors and related conditions. One of which is smoking relapse. Previous studies have shown that post-cessation weight gain may positively predict of smoking relapse in the short-term (Hall, Ginsberg, & Jones, 1986; Klesges, Meyers, Klesges, & La Vasque, 1989; Klesges & Shumaker, 1992; Mukhopadhyay & Wendel, 2011). For example, weight gain within the first six months of abstinence predicted relapse at one year for continued abstainers (Hall et al., 1986).

Post-cessation weight gain also increases the risk of chronic conditions, such as hypertension and diabetes (Chinn et al., 2005; Davey Smith et al., 2005; Flegal, Troiano, Pamuk, Kuczmarski, & Campbell, 1995; Janzon, Hedblad, Berglund, & Engstrom, 2004; Yeh, Duncan, Schmidt, Wang, & Brancati, 2010). The risk of developing diabetes was partially explained by weight gain after smoking cessation in a sample of non-diabetic adult smokers (N= 10,892). Specifically, abstainers gained significantly more weight and developed diabetes faster compared to never smokers over three years (Yeh et al., 2010). Similarly, post-cessation weight gain is associated with increased glucose intolerance (Davey Smith et al., 2005; Nilsson, Lundgren, Soderstrom, Fagerstrom, & Nilsson-Ehle, 1996; Yeh et al., 2010) and decreased lung functioning (Chinn et al., 2005), both of which contribute to chronic illnesses.

One of the most substantial negative influences of post-cessation weight gain is its contribution to obesity. Ex-smokers are significantly more likely to become overweight than never smokers (Flegal et al., 1995). Reas et al. (2009) illustrated that quitters (<5 years quit) were more likely to be obese compared to ex- (>5 years quit), never, and current smokers. Given their high risk of developing obesity, post-cessation weight gain may be particularly detrimental to African Americans. Further, African Americans also have more difficulty losing weight (DeLany et al., 2014; West, Prewitt, Bursac, & Felix, 2008) and demonstrate a lower resting metabolic rate compared to whites (DeLany et al., 2014; Forman, Miller, Szymanski, & Fernhall, 1998; Weyer, Snitker, Bogardus, & Ravussin, 1999). With the significant health consequences of obesity and increased risk for African Americans to become obese, the effect of weight gain after smoking cessation may be compounded in African American smokers. Therefore, it is imperative to consider post-cessation weight gain in this population during smoking cessation treatment.

Post-cessation Weight Gain and African Americans

African Americans have been underrepresented in post-cessation weight gain research. Research has shown that African Americans tend to gain more weight and gain excessively [i.e., more than the average ten lbs (5kg)] than other racial/ethnic groups after quitting (Klesges et al., 1998; Williamson et al., 1991). Williamson et al. (1991) was among the first to examine the association between smoking cessation and weight gain among African Americans. This longitudinal study examined weight gain among adult males (n=3365) and females (n=5639) participating in the first National Health and Nutrition Examination Survey (NHANES I). Adjusted for age, BMI, physical activity,

and alcohol consumption at baseline, and chronic health conditions at follow up, results demonstrated that abstainers (quit during the study) were more likely to gain excessive weight than never, ex- (abstinent at baseline), and continuing smokers. African American abstainers gained significantly more weight than those categorized as non-African American. Furthermore, research on excessive post-cessation weight gain has shown that after one year of cessation, approximately 47% of those who remained abstinent had gained above average weight (Aubin et al., 2012). Younger age and a greater number of cigarettes smoked per day are associated with excessive weight gain one year post cessation (O'Hara et al., 1998; Swan & Carmelli, 1995; Williamson et al., 1991).

Subsequently, two studies prospectively examined weight gain specifically among African American smokers (Klesges et al., 1998; Vander Weg, Klesges, Eck Clemens, Meyers, & Pascale, 2001). Klesges et al. (1998) examined smoking status and weight change among African Americans and whites (*N=3868*). Adjusting for age, energy intake, alcohol intake, and physical activity, African American ex-smokers (i.e., abstinent at baseline) gained more weight [28 lbs (12.5kg)] compared to white ex-smokers [21 lbs (9.7kg)] over seven years. African Americans (87%) were twice as likely to gain excessive weight, compared to whites (59%). Additionally, Vander Weg et al. (2001) examined changes in weight and energy balance following cessation among African Americans and whites (*N*=95). Abstainers gained weight [average of 2.3 lbs (1kg)] over two weeks, with no significant difference between racial/ethnic groups. Baseline carbon monoxide (CO) reading and resting energy expenditure positively predicted weight gain. To date, research on African Americans and post-cessation weight gain is scant.

Theoretical Approach: Biopsychosocial model

The present study used a biopsychosocial approach to examine predictors of weight gain among African American smokers. This multifaceted model is a framework to propose how biological and psychosocial variables collectively contribute to the expression of health and illness (Engel, 1977). In previous decades, the biomedical model was the dominant disease model and suggested that basic biological factors wholly explain illness (Engel, 1977). In contrast, the biopsychosocial model posits that the interaction of biological, psychological, and social factors influences health (Figure 1). Therefore, the consideration of each of these levels is important for diagnosis, optimal treatment, and overall patient care; focusing on a single factor limits the conceptualization of health conditions. As an example, obesity may be influenced by not only genetic factors (biological), but also depression that leads to overeating (psychological/behavioral) and cultural norms, such as of acceptance of larger body sizes (social). The biopsychosocial model has been used to examine and explain a variety of health behaviors, physical conditions, and mental illnesses (Galizio & Maisto, 1985; Gerrity, 2001), including obesity (Goetz & Caron, 1999; Wildes, Marcus, & Fagiolini, 2006) and depression (Hammack, 2003).

Previous research has applied this model to health concerns among African Americans (e.g., Betancourt & Lopez, 1993; R. Clark, Anderson, Clark, & Williams, 1999; Griffith, 2009). For example, anger (psychological) has been associated with blood pressure in this population (Pointer, Livingtson, Yancey, McClelland, & Bukoski, 2008), and low SES African American females (social) have a greater risk of obesity compared to their higher SES counterparts (Coogan, Wise, Cozier, Palmer, & Rosenberg, 2012).

The biopsychosocial theory also has been proposed as a framework to explore tobacco-related health disparities in racial/ethnic minority smokers (Fernander, Shavers, & Hammons, 2007). Fernander et al. (2007) discussed the limitations of considering only genetic variations as explanatory factors of tobacco-related health outcomes (e.g., lung cancer) among African Americans. The authors argued that biological mechanisms (e.g., nicotine dependence) are important in explaining disparities, yet the influences of behavioral (e.g., diet/nutrition, alcohol consumption) and psychosocial (e.g., stress, environmental cues) variables may affect those biological mechanisms and outcomes. Previous research on post-cessation weight gain has focused heavily on biological mechanisms, such as changes in metabolism (Filozof et al., 2004). Needed are studies to examine how psychosocial factors may also contribute to weight gain among African American smokers. Therefore, a biopsychosocial approach was used in the current study to examine factors that may influence weight gain in an African American sample of smokers.

Factors Associated with Post-cessation Weight Gain

Many factors have been examined as predictors of post-cessation weight gain in smokers. A history of smoking greater than fifteen cigarettes per day (CPD), greater nicotine dependence, lower SES, younger age (<55 years old), decreased physical activity, and baseline BMI are positively associated with weight gain within one year of abstinence (Kmetova et al., 2014; Lycett, Hajek, & Aveyard, 2010; O'Hara et al., 1998; Swan & Carmelli, 1995; Williamson et al., 1991). However, most studies examining these factors were epidemiologic, and few focused on treatment-seeking smokers. O'Hara et al. (1998) examined post-cessation weight gain among adult smokers ($N= 5,887$), aged

35-60, who received either 12-week behavioral smoking intervention plus nicotine gum or assessment only. Both male (33.9%) and female (37.0%) quitters gained excessive weight over five years, although most weight was gained within first year. Predictors of weight gain included younger age and greater baseline BMI. The effect of race/ethnicity was examined, and no significant differences were found. African Americans (n=235) represented only 4% of the sample, and the proportion of African American quitters was even less (2% males, 4% females). Prod'hom et al. (2013) found that older age and male sex predicted weight gain over one year in adults enrolled in a 9-week cognitive-behavioral smoking cessation intervention plus nicotine replacement therapy. Similar to previous research on predictors of post-cessation weight gain, African Americans were underrepresented in this study. More research examining predictors of weight gain among treatment-seeking African American smokers is needed.

Biological. Biological mechanisms have been proposed to explain post-cessation weight gain, such as decreased resting metabolic rate and increased lipoprotein lipase (Filozof, 2004). As physiological processes may help explain disparities in weight gain, research is needed on biological factors in African Americans.

Sex. One established biological factor associated with post-cessation weight gain is sex. Females, on average, gain more weight than males after quitting (Klesges et al., 1989; Williamson et al., 1991), with females gaining eight lbs (3.8kg) and males gaining six lbs (2.8kg; Williamson et al., 1991). Females were also more likely to gain excessive weight compared to males, and female ex-smokers gained more weight compared to female current smokers (Williamson et al., 1991). Racial/ethnic differences in overall weight gain in the total population have been documented, with African American

females gaining more weight than white females (Baltrus, Lynch, Everson-Rose, Raghunathan, & Kaplan, 2005). Yet, there is no previous research on sex differences in weight gain among African American smokers.

    Cortisol. Cortisol is another biological factor that may predict weight gain among smokers. Cortisol, a biomarker for stress, is often examined in the context of health outcomes, such as weight gain and BMI, among nonsmokers (Epel, Lapidus, McEwen, & Brownell, 2001; Kivimaki et al., 2006; Korkeila, Kaprio, Rissanen, Koshenvuo, & Sorensen, 1998; Rosmond, Lapidus, Marin, & Bjorntorp, 1996). Although brief stress responses are functional (Kalat, 2012), chronic levels of cortisol lead to poor health outcomes, such as obesity (Bjorntorp, 2001; Kumari, Chandola, Brunner, & Kivimaki, 2010; Tataranni et al., 1996). Higher cortisol levels predict visceral adiposity and BMI (Bjorntorp, 2001; Tataranni et al., 1996).

    Research on cortisol has shown that the cortisol awakening response (CAR), a reliable, commonly used method to measure cortisol, is positively associated with poor health outcomes (Wust, Federenko, Hellhammer, & Kirschbaum, 2000; Fries, Dettenborn, & Kirschbaum, 2009; Roberts, Wessely, Chalder, Papadopoulos, & Cleare, 2004; J. C. Pruessner, Hellhammer, & Kirschbaum, 1999; M. Pruessner, Hellhammer, Pruessner, & Lupien, 2003; Schulz, Kirschbaum, Prubner, & Hellhammer, 1998). The CAR describes the initial peak in cortisol (50-60% average increase) within the first hour of waking. Smokers have been found to have a higher CAR compared to non-smokers (Badrick, Kirschbaum, & Kumari, 2007; Kunz-Ebrecht et al., 2004; Steptoe & Ussher, 2006). Number of cigarettes smoked is positively associated with CAR (Badrick et al., 2007) as nicotine exposure increases cortisol levels (Steptoe & Ussher, 2006). Therefore,

the absence of nicotine results in a sharp decline in cortisol levels, which is associated with smoking relapse (al'Absi, Hatsukami, Davis, & Wittmers, 2004). However, no studies have examined cortisol and post-cessation weight gain among smokers.

Stress is a proposed mechanism by which African Americans experience health-related disparities (Bennett, Merritt, Sollers, et al., 2004; Moolchan et al., 2007). High cortisol levels have been found to predict high blood pressure and cardiovascular disease reactivity among African Americans (Kapuku, Trieber, & Davis, 2002). Compared to whites, African Americans exhibit higher levels of chronic stress, which increases disease risk (e.g., atherosclerosis; Cohen et al., 2006; Hajat et al., 2010). In addition, African Americans have a different pattern of cortisol secretion compared to whites; the diurnal decline of cortisol throughout the day demonstrates greater blunting, or a flatter curve, among African Americans (Cohen et al., 2006; DeSantis et al., 2007). There is evidence that a flattened cortisol slope is associated with being obese (BMI >31; Kumari et al., 2010). However, there is little research examining the CAR in African Americans, and there is virtually no research among African American smokers. Bennett, Merritt, and Wolin (2004) compared the CAR between African Americans (n=30) and whites (n=26) using salivary cortisol samples, and found a significant ethnicity x education interaction. No differences in CAR between African Americans and whites were found, but results showed that higher educated African Americans had a higher CAR compared to those with lower education. As African Americans have been shown to exhibit unique stress responses, and higher cortisol levels are associated with obesity, it may be important to examine cortisol and weight among African American smokers seeking to quit.

Psychological. Numerous studies have examined psychological factors as predictors of obesity in the general population. Positive associations between psychological factors, specifically depression, and weight gain have been documented (Blaine, 2008; Kress, Peterson, & Hartzell, 2008; Lim, Thomas, Bardwell, & Dimsdale, 2008; Onyike Crum, Lee, Lyketsos, & Eaton, 2003). However, previous research on psychological factors and post-cessation weight gain is scarce. Identifying modifiable risk factors for weight gain, such as depression and concern about gaining weight after cessation, among African American smokers is important for future intervention design.

Studies have shown that the prevalence of depression among African Americans is higher than the national average (Miller et al., 2004; Collins et al., 2010). In addition, treatment-seeking African American smokers exhibit greater depressive symptoms compared to white and Hispanic smokers (Webb Hooper, Baker, & McNutt, 2014; Webb Hooper & Kolar, 2015). Therefore, the observed prevalence of depressive symptoms among African American smokers warrant additional research on depressive symptoms and weight gain in a treatment-seeking population.

Depressive symptoms. Depressive symptoms have been linked to negative health behaviors (Green & Pope, 2000), such as smoking initiation, smoking behavior, and nicotine dependence (Weinberger, Mazure, Morlett, & McKee, 2013). Depressed individuals are more likely to initiate smoking and report greater nicotine dependence than those with no history of depression. Among African Americans, increased levels of depressive symptoms predict lower rates of smoking cessation (Catley et al., 2005). In addition, research on the general population has found that depressed individuals have a higher risk of developing obesity compared to non-depressed (Blaine, 2008). A positive

association between depressive symptoms and BMI has been documented, as well (Lim et al., 2008). However, few studies have examined the association between depressive symptoms and weight gain among smokers. Killen et al. (1996) found that weight gain was positively associated with smoking relapse among smokers who reported more depressive symptoms. Chiriboga et al. (2008) found a positive association between depressive symptoms and weight gain over one year, while controlling for smoking status. More research is needed on depressive symptoms and post-cessation weight gain, given the documented effect of depression on obesity and BMI. To date, no studies have examined this association among African American smokers.

   Weight Concern. Weight concern has been widely studied among smokers (e.g., M. M. Clark et al., 2006; Copeland, Martin, Geiselman, Rash, & Kendzor, 2006; Meyers et al., 1997; Pomerleau, Zucker, & Stewart, 2001) because weight control is a well-established reason for smoking initiation and maintenance (Audrain-McGovern & Benowitz, 2011). Weight concern is a barrier to smoking cessation (M. M. Clark et al., 2006; Meyers et al., 1997) and is associated with weight gain after cessation, particularly among females (Copeland et al., 2006; Borrelli & Mermelstein, 1998). Weight concern is also associated with fewer smoking cessation attempts (Meyers et al., 1997) and short-term relapse (M. M. Clark et al., 2006). In addition, previous research suggested that African American smokers, particularly females, were less weight-concerned compared to whites (Cunningham, Roberts, Barbee, Druen, & Wu, 1995). However, African American female smokers are, indeed, weight-concerned (Pomerleau, Zucker, Namenek Brouwer, et al., 2001; Sanchez-Johnsen, 2005; Webb & Carey, 2009). Webb and Carey (2009) found that African Americans who were older, had greater perceived stress, had

family history of heart disease, and were more connected to the African American culture, were more likely to report greater smoking-related weight control expectancies. In a sample consisting of 40% African Americans receiving cognitive behavioral therapy (CBT) for smoking cessation, Borrelli and Mermelstein (1998) found that weight concern positively predicted weight gain at the end of treatment and at the 3-month follow up. Still needed are studies on weight concern and actual weight gain among African American smokers. Understanding psychological predictors of weight gain in this population will help guide targeted interventions.

Social. There is a dearth of research on social factors associated with post-cessation weight gain. The current study focused on SES as a social factor due to its associations with smoking and weight. Low SES individuals are more likely to initiate and maintain smoking (CDC, 2008), have a higher prevalence of smoking (Delva et al., 2005), and have a harder time quitting compared to their higher SES counterparts (Barbeau, Krieger, & Soobader, 2004). Furthermore, research has shown that SES inversely predicts weight gain in the general population (Ball & Crawford, 2005). Specifically, education level is inversely associated with weight gain among males and females (Ball, Crawford, Ireland, & Hodge, 2003; Burke et al., 1996). For example, individuals with less than a high school degree have greater increases in BMI over time compared to those who completed high school (Kahn & Williamson, 1991). Using family income as a proxy for SES, Kahn & Williamson (1991) also found that low income females were at a higher risk of excessive weight gain than high income females. However, research on SES and obesity among African Americans has demonstrated findings contradictory to the aforementioned results. In the general population, African

American women weigh significantly more than whites as their education level increases (Lewis et al., 2005). Additionally, African American women with at least a high school degree had a higher prevalence of obesity compared to their lower educated counterparts (Wang & Beydoun, 2007). As evidence of a unique association between SES and weight gain exists among African Americans, it is important to examine how these factors may be related to post cessation weight gain among smokers.

The Current Study

Because African Americans suffer from tobacco-related disparities, it is critical to examine contributing factors, such as post-cessation weight gain. The majority of smokers gain weight within three months after cessation, but African Americans tend to gain more weight than whites. Furthermore, African Americans are more likely to gain excessive weight after cessation. Only a few studies have examined post-cessation weight gain specifically among African American smokers, and this group has been underrepresented in research on factors related to weight gain. Previous research has examined associations between weight gain and sex, age, cortisol, depressive symptoms, weight concern, and SES in primarily white samples. Examining predictors of weight gain among African Americans will inform the design of targeted smoking cessation interventions. The current study contributes to the literature, as it is the first to our knowledge to examine weight gain post treatment among African Americans enrolled in a randomized controlled trial testing the efficacy of a smoking cessation intervention.

Aims and Hypotheses

Aim 1. To examine the pattern of weight gain (e.g., increase, decrease, or remain steady) among treatment-seeking African American smokers and abstainers over twelve months post CBT.

Hypotheses:

a. In the overall sample, participants would demonstrate an increase (i.e., significantly greater than zero pounds) in weight over the 12-month follow-up period.

b. Among abstainers, participants would demonstrate an increase (i.e., significantly greater than zero pounds) in weight over the 12-month follow-up period.

c. Among continued smokers, participants would demonstrate no change in weight over the 12-month follow-up period.

Aim 2. To examine smoking status as a predictor of weight gain over twelve months post CBT among treatment-seeking African American smokers, controlling for intervention condition, appetite increase, TNP use, CPD, medication use, changes in diet and exercise post CBT, and baseline BMI.

Hypothesis: Smoking status would predict weight gain, such that abstainers would gain more weight than continued smokers over the 12-month follow-up period.

Aim 3 (Figure 2)**.** To examine biopsychosocial predictors of weight gain [sex, age, baseline CAR (biological), baseline depressive symptoms, weight concern (psychological), education level, and income (social)] over twelve months post CBT,

controlling for smoking status, intervention condition, appetite increase, TNP use, CPD, medication use, changes in diet and exercise post CBT, and baseline BMI.

Hypotheses:

    a. Females would gain more weight than males.

    b. Age would negatively predict weight gain.

    c. Cortisol awakening response would positively predict weight gain.

    d. Depressive symptoms would positively predict weight gain.

    e. Weight concern would positively predict weight gain.

    f. Education level would positively predict weight gain.

    g. Income level would negatively predict weight gain.

Aim 4. To explore whether biopsychosocial variables predicted the variability in the effect of smoking status on weight gain (i.e., cross-level interactions).

Chapter 2: Method

Participants

Data were drawn from a randomized controlled trial testing the efficacy of a culturally specific smoking cessation intervention among African Americans (Webb Hooper et al., 2013). Participants were recruited via flyers, word-of-mouth, the public transit system, internet ads, doctors' offices, and community organizations. Eligibility criteria included the following: 1) self-reported African American, 2) smoked at least five cigarettes per day, 3) aged 18-65, 4) motivated to quit smoking (> 5 on a scale from 1 to 10), 5) spoke and read fifth to sixth grade level English, and 6) had access to transportation. Participants were ineligible if they 1) were involved in another smoking cessation or drug treatment program, 2) reported serious mental illness, 3) reported contraindications to TNP use, (i.e., pregnant/breastfeeding and acute heart or lung condition), 4) did not have permanent contact information, and 5) reported current illicit drug use. Participants provided informed consent and were compensated up to $300 for participation through the 12-month follow-up. The study was approved by the University of Miami's Institutional Review Board.

Interventions and Procedures

Participants (*N*=342) were randomized into either a standard CBT smoking cessation intervention condition, or a CBT smoking intervention condition that included components targeted to the African American culture. Both conditions included an orientation session and eight sessions of group counseling administered by trained tobacco counselors over a four week period. All participants received psychoeducation and were taught strategies on coping with urges to smoke, general weight control, basic

stress management, and relapse prevention. Participants were also given eight weeks of

TNP to use daily. Transdermal nicotine patch use dosage was 21mg for the first four

weeks, then decreased to 14mg for two weeks, then decreased again to 7mg for two

weeks. Participants were instructed to refrain from smoking while using TNP.

Participants completed follow up assessments either in-person or via telephone at

three, six, and twelve months post CBT. At baseline, the end of counseling (session

eight), and all follow up assessments, questionnaires were administered, weight was

measured, and CO and saliva cortisol samples were collected.

Measures

Outcome variable.

Weight. Weight was measured in pounds using a digital scale, and height was

self-reported. Weight was measured at each intervention session and follow up

assessments at the same time each day (e.g., afternoon, early evening). Participants wore

light clothing and shoes when weighed. Weight was recorded to the nearest whole

number.

Predictor variables.

Demographics. Demographic variables (sex, age in years, education level, and

income level) were self-reported. Education level was reported on a Likert scale [0-

Elementary School, 1-*Junior High School*, 2-*Partial High School*, 3-*High School*, 4-

Business or Technical Training, 5-*Some College (no degree)*, 6-*University Degree,*

Bachelor level or equivalent, 7-*Post-graduate Degree*]. Household yearly income level

was also reported on a Likert scale (0-*Under $10,000*; 1-*$10,000-$20,000*; 2-*$20,001-*

$30,000; 3-*$30,001-$40,000*; 4-*$40,001-$50,000*; 5-*$50,001-$60,000*; 6-*$60,001-*

$70,000; *7-$70,001- $80,000*; *8-$80,001-$90,000*; *9-Over $90,000*). Both education and income level were examined as continuous variables.

Smoking status. Smoking status was self-reported at each session and follow up assessment; participants were classified as either smoking or abstinent. Continuous abstinence was assessed at the end of counseling (abstinence for previous two weeks), and 3-, 6-, and 12-month follow ups (abstinence for previous four weeks; 28-day continuous abstinence), using the timeline follow-back method (TLFB; Brown et al., 1998). This method utilizes a calendar to help individuals recall smoking over a specified time period. In the current study, abstinence was defined at each assessment as those who maintained continuous abstinence.

Cortisol. Salivary cortisol was collected at baseline, the end of counseling, and each follow up assessment. Samples were collected at four time points within a 24-hour period: waking, thirty minutes after waking, 4pm, and 6pm. Participants were instructed to refrain from eating, drinking, smoking, or brushing teeth within sixty minutes of each collection, and to refrain from engaging in vigorous exercise or alcohol consumption within twelve hours of collections. Participants completed samples by saturating a Salimetrics oral swab for at least two minutes, then storing tubes in freezer or insulated bag with frozen ice pack. All samples were collected by the research team and stored in lab freezers until analysis. Cortisol levels (nmol/L) were processed using radioimmunoassays techniques. Baseline cortisol data was analyzed in the current study. The baseline CAR was determined by calculating the percent change value of cortisol levels from the samples taken at the first (waking) and second (thirty minutes after

waking) time points: [(sample 1)/(sample 2) x 100; Pruessner et al., 1997]. For example, if sample 1=14.21 and sample 2=17.63, then the CAR=80.6.

Depressive symptoms. The Centers for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977) measured baseline depressive symptoms. The 20-item scale assessed symptoms/behaviors during the past week. Responses were recorded on a Likert scale ranging from 0 (*less than 1 day—rarely or none of the time*) to 3 (*5 to 7 days—most or all of the time*), and higher scores indicated greater depressive symptoms (range: 0-60). The scale has demonstrated good reliability across studies ($\alpha = .90$; Windle & Windle, 2001) and in the current study ($\alpha=0.84$). The CES-D was administered at baseline, end of counseling, and follow up assessments. Baseline depressive symptoms were analyzed in the current study.

Weight concern. At baseline, weight concern was assessed using a single-item question "If you stopped smoking cigarettes today, how concerned would you be that you might start smoking again because of any weight gain?" Participants responded on an ordinal scale indicating either 0 (*not at all concerned*), 1 (*somewhat concerned*), or 2 (*very concerned*).

Covariates.

Transdermal nicotine patch use. The frequency and accuracy of TNP use was recorded at each CBT session. Accuracy was recorded as 'yes' if the patch was applied to and remained on skin for the entire day. The frequency of days used was totaled and examined as a continuous variable in the analysis.

Appetite increase. Appetite increase was measured by one item from the 13-item Minnesota Nicotine Withdrawal Scale (Hughes & Hatsukami, 1986), which assessed both

somatic and psychological symptoms of nicotine withdrawal. At each session, the intensity of appetite increase experienced since the previous session was rated on a Likert scale (*0= not at all, 4=extremely*). Scores from the single item were summed and averaged across all sessions to compute a total score (α=0.89). Higher scores indicated greater appetite increase.

Cigarettes per day. At baseline, participants self-reported the average number of cigarettes smoked per day (CPD), which was included as a continuous variable in the analyses.

Body mass index. Body mass index was calculated using height and weight measurements $[(lbs)/(in)^2 \text{ x } 703 \text{ (CDC, 2014)}]$. Baseline BMI was used in the analyses.

Changes in diet and exercise. Changes in diet and changes in exercise were assessed at the end of counseling using single-items: "As a result of the quit smoking group, I changed my eating habits to avoid gaining weight" and "As a result of the quit smoking group, I started to get more physical activity." Participants responded on a Likert scale ranging from 0(*Not at all*) to 7(*Yes, definitely*). Higher scores indicated greater changes. Items were included as continuous variables in the model.

Medication use. Participants self-reported at screening all medications that they were prescribed. Medication use was recorded as "Yes" if at least one medication was reported.

Data Analyses

Chi-squared and t-tests were conducted to examine differences in predictors and weight between intervention conditions (Table 1). There were no significant differences in sample characteristics with one exception. Participants in the culturally specific

condition were significantly more likely to report 28-day continuous abstinence compared to the standard condition. Thus, intervention conditions were collapsed, and then included as a covariate in the model. Baseline descriptive statistics were obtained, and data was examined for normality. Non-normal data was log transformed. Variables were also examined for multicollinearity using variance inflation factor (VIF), and no significant intercorrelations were observed. Correlations between covariates and weight at baseline, end of counseling, 3-, 6-, and 12-months post CBT were examined. Variables that were significantly correlated with weight were further examined as control variables in multilevel model analyses. Alpha was set at a p level of 0.05.

Analyses were conducted using PROC MIXED in SAS 9.3 statistical software (SAS Institute, Inc. Cary, NC). Random effects (mixed effects) multilevel modeling, using restricted maximum likelihood (REML) estimation, was used to test predictors of weight gain. There were several advantages to using this technique compared to repeated measures ANOVA to assess longitudinal change. First, this method allowed for the simultaneous examination of intra-individual and inter-individual change within a single statistical test. Second, using this technique, the assumption of sphericity (i.e., equal variance at each time point and equal covariance between all pairs of time points) was relaxed; therefore, it was not assumed that all individuals gain weight at the same rate over time (Holden, Kelley, & Agarwal, 2008; MacCallum, Kim, Malarkey, & Kiecolt-Glaser, 1997). Third, this method used all available information and weighted estimates in cases of missing data. If a participant was missing measurements at a specific time point, the entire case was not removed from analyses (Singer & Willett, 2003).

In the multilevel model, level 1 modeled individual weight gain, and level 2 tested the variability in weight gain (predictor variables). To account for differences in individual weight gain over time, random effects were included in the model. Random effects estimated the variation in weight gain within participants. In the current study, both intercept and slope were included as random effects, which modeled the variation in average baseline weight and rate of weight gain. The intercept described the average baseline weight of the sample, and the slope described the rate of weight gain. Each biopsychosocial and control variable was added to level 2 of the model to predict level 1 slope, or the variation in rate of weight gain. Estimates of the predictor variables were interpreted as the average differences in rate of weight gain per one-unit change in the predictor variables. Variables on level 2 could also predict other variables on level 1 (i.e., time-varying covariates), which were known as cross-level interactions. The estimate of a cross-level interaction described the average difference in the association between the time-varying covariate and weight gain given one unit change in the predictor variable. Associations of both within-person (level 1) and between-person (level 2) characteristics with weight gain were analyzed in the current study. To address each aim, a series of models was analyzed.

Aim 1. First, a fully unconditional level 1 model was specified using five time points (baseline, end of counseling, 3-, 6-, and 12-month follow up assessments) to examine the average baseline weight and the rate of weight gain over 12 months post CBT. The within-person variability in weight gain was represented in level 1, and the effect of time (months that have passed since baseline assessment) was examined at this level. The variances in the intercept and slope were represented in level 2. Results of this

analysis indicated the average baseline weight of the sample and the rate of weight gain

(i.e., increases, decreases, or remain steady). This analysis also tested the significance of

the variance in the intercept and slope. Significant variances indicated whether the

baseline weight and/or rate of weight gain varied among individuals, and whether

multilevel modeling was an appropriate analysis for the data. A fully unconditional

model was specified separately for a) the entire sample, b) abstainers (i.e., participants

who were abstinent at all follow-up periods over twelve months), and c) continued

smokers (i.e., participants who did not meet criteria for 28-day continuous abstinence on

at least one occasion over the 12-month follow up period).

Level 1: $Weight_{it} = \pi_{0i} + \pi_{1i}(Time_{it}) + e_{it}$

Level 2: $\pi_{0i} = \beta_{00} + r_{0i}$

$\pi_{1i} = \beta_{10} + r_{1i}$

Combined: $Weight_{it} = \beta_{00} + \beta_{10}(Time_{it}) + r_{0i} + r_{1i}(Time_{it}) + e_{it}$

Next, to determine which variables to control for in the model, covariates were

included to examine their associations with both baseline weight and rate of weight gain.

Intervention condition (0=Standard and 1=Culturally Specific), TNP use (continuous),

BMI (continuous), appetite increase (continuous), CPD (continuous), medication use

(0=No, 1=Yes), and changes in diet (continuous) and exercise (continuous) were added to

level 2 of the model. Nonsignificant variables were excluded from further analyses.

Level 1: $Weight_{it} = \pi_{0i} + \pi_{1i}(Time_{it}) + e_{it}$

Level 2: $\pi_{0i} = \beta_{00} + \beta_{01}(Int_i) + \beta_{02}(TNP_i) + \beta_{03}(BMI_i) + \beta_{04}(Appet_i) +$

$\beta_{05}(CPD_i) + B_{06}(Meds_i) + \beta_{07}(Diet_i) + \beta_{08}(Exer_i) + r_{0i}$

$\pi_{1i} = \beta_{10} + \beta_{11}(Int_i) + \beta_{12}(TNP_i) + \beta_{13}(BMI_i) + \beta_{14}(Appet_i) +$

$$\beta_{15}(CPD_i) + B_{16}(Meds_i) + \beta_{17}(Diet_i) + \beta_{18}(Exer_i) + r_{1i}$$

Combined: $Weight_{it} = \beta_{00} + \beta_{01}(Int_i) + \beta_{02}(TNP_i) + \beta_{03}(BMI_i) + \beta_{04}(Appet_i) +$

$$\beta_{05}(CPD_i) + B_{06}(Meds_i) + \beta_{07}(Diet_i) + \beta_{08}(Exer_i) + \beta_{10}(Time_{it}) +$$

$$\beta_{11}(Int_i \ x \ Time_{it}) + \beta_{12}(TNP_i \ x \ Time_{it}) + \beta_{13}(BMI_i \ x \ Time_{it}) +$$

$$\beta_{14}(Appet_i \ x \ Time_{it}) + \beta_{15}(CPD_i \ x \ Time_{it}) + B_{16}(Meds_i \ x \ Time_{it}) +$$

$$\beta_{17}(Diet_i \ x \ Time_{it}) + \beta_{18}(Exer_i \ x \ Time_{it}) + r_{0i} + r_{1i}(Time_{it}) + e_{it}$$

Aim 2. To examine smoking status as a predictor of weight gain, controlling for

confounders, smoking status (0=Abstinent, 1=Smoking) was added to level 1 of the

model as a time-varying covariate, which accounted for changes in smoking status over

time. Time-varying covariates may have multiple types of variances, including within-

person, between-person, and/or an interaction of within- and between-person, which all

may have a different effect on the outcome (Hoffman, 2014). For example, the effect of

reporting abstinence more often than usual is distinct from the effect of reporting more

abstinence compared to others. Therefore, it has been recommended that both the within-

person and between-person variances are considered when assessing time-varying

predictors (Hoffman, 2014). The level 1 effect of time-varying smoking status

demonstrated the within-person effect of smoking on weight gain. To control for the

between-person variance, the effect of the percentage of time reporting smoking was

added as a continuous variable to level 2. Between-person smoking status was calculated

by averaging how often each participant reported smoking over all assessments (i.e., 20-

100%; Hoffman, 2014). Results of this analysis indicated the effect of smoking on weight

gain over time, holding constant the effects of covariates.

Level 1: $Weight_{it} = \pi_{0i} + \pi_{1i}(Time_{it}) + \pi_{2i}(Smoke_{it}) + e_{it}$

Level 2: $\pi_{0i} = \beta_{00} + \beta_{01}(Int_i) + \beta_{02}(TNP_i) + \beta_{03}(BMI_i) + \beta_{04}(Appet_i) +$

$\beta_{05}(CPD_i) + B_{06}(Meds_i) + \beta_{07}(Diet_i) + \beta_{08}(Exer_i) + \beta_{09}(BSmoke_i) + r_{0i}$

$\pi_{1i} = \beta_{10} + \beta_{11}(Int_i) + \beta_{12}(TNP_i) + \beta_{13}(BMI_i) + \beta_{14}(Appet_i) +$

$\beta_{15}(CPD_i) + B_{16}(Meds_i) + \beta_{17}(Diet_i) + \beta_{18}(Exer_i) + \beta_{19}(BSmoke_i) + r_{1i}$

$\pi_{2i} = \beta_{20}$

Combined: $Weight_{it} = \beta_{00} + \beta_{01}(Int_i) + \beta_{02}(TNP_i) + \beta_{03}(BMI_i) + \beta_{04}(Appet_i) +$

$\beta_{05}(CPD_i) + B_{06}(Meds_i) + \beta_{07}(Diet_i) + \beta_{08}(Exer_i) + \beta_{09}(BSmoke_i) +$

$\beta_{10}(Time_{it}) + \beta_{11}(Int_i \ x \ Time_{it}) + \beta_{12}(TNP_i \ x \ Time_{it}) +$

$\beta_{13}(BMI_i \ x \ Time_{it}) + \beta_{14}(Appet_i \ x \ Time_{it}) + \beta_{15}(CPD_i \ x \ Time_{it}) +$

$\beta_{16}(Meds_i \ x \ Time_{it}) + \beta_{17}(Diet_i \ x \ Time_{it}) + \beta_{18}(Exer_i \ x \ Time_{it}) +$

$\beta_{19}(BSmoke_i \ x \ Time_{it}) + \beta_{20}(Smok_{it}) + r_{0i} + r_{1i} \ (Time_{it}) + e_{it}$

Aim 3. To examine the associations with weight gain, controlling for smoking

status and covariates, sex (0=Male, 1=Female), baseline cortisol (CAR; continuous), age

(continuous), depressive symptoms (continuous), weight concern [0=Not concerned,

1=Somewhat concerned, 2=Very concerned (dummy coded, with "Not concerned" as

reference group)], education level (continuous), and income (continuous) were added to

level 2 of the model. Univariate analyses were conducted to examine the variables as

predictors of the individual weight gain. Significant variables were then added to a final

multivariate model. Results of this analysis indicated the predictors of weight gain over

the 12-month follow-up, controlling for differences in average baseline weight and

covariates.

Level 1: $Weight_{it} = \pi_{0i} + \pi_{1i}(Time_{it}) + \pi_{2i}(Smoke_{it}) + e_{it}$

Level 2: $\pi_{0i} = \beta_{00} + \beta_{01}(Int_i) + \beta_{02}(TNP_i) + \beta_{03}(BMI_i) + \beta_{04}(Appet_i) +$

$$\beta_{05}(CPD_i) + \beta_{06}(Meds_i) + \beta_{07}(Diet_i) + \beta_{08}(Exer_i) + \beta_{09}(BSmoke_i) +$$

$$\beta_{010}(Age_i) + \beta_{011}(CAR_i) + \beta_{012}(Sex_i) + \beta_{013}(Depress_i) +$$

$$\beta_{014}(Concern1_i) + \beta_{015}(Concern2_i) + \beta_{016}(Edu_i) + \beta_{017}(Income_i) + r_{0i}$$

$$\pi_{1i} = \beta_{10} + \beta_{11}(Int_i) + \beta_{12}(TNP_i) + \beta_{13}(BMI_i) + \beta_{14}(Appet_i) +$$

$$\beta_{15}(CPD_i) + \beta_{16}(Meds_i) + \beta_{17}(Diet_i) + \beta_{18}(Exer_i) + \beta_{19}(BQuit_i) +$$

$$\beta_{110}(Age_i) + \beta_{111}(CAR_i) + \beta_{112}(Sex_i) + \beta_{113}(Depress_i) +$$

$$\beta_{114}(Concern1_i) + \beta_{115}(Concern2_i) + \beta_{116}(Edu_i) + \beta_{117}(Income_i) + r_{1i}$$

$$\pi_{2i} = \beta_{20}$$

Combined: $Weight_{it} = \beta_{00} + \beta_{01}(Int_i) + \beta_{02}(TNP_i) + \beta_{03}(BMI_i) + \beta_{04}(Appet_i) +$

$$\beta_{05}(CPD_i) + B_{06}(Meds_i) + \beta_{07}(Diet_i) + \beta_{08}(Exer_i) + \beta_{09}(BSmoke_i) +$$

$$\beta_{010}(Age_i) + \beta_{011}(CAR_i) + \beta_{012}(Sex_i) + \beta_{013}(Depress_i) +$$

$$\beta_{014}(Concern1_i) + \beta_{015}(Concern2_i) + \beta_{016}(Edu_i) + \beta_{017}(Income_i) +$$

$$\beta_{10}(Time_{it}) + \beta_{11}(Int_i \times Time_{it}) + \beta_{12}(TNP_i \times Time_{it}) +$$

$$\beta_{13}(BMI_i \times Time_{it}) + \beta_{14}(Appet_i \times Time_{it}) + \beta_{15}(CPD_i \times Time_{it}) +$$

$$\beta_{16}(Meds_i \times Time_{it}) + \beta_{17}(Diet_i \times Time_{it}) + \beta_{18}(Exer_i \times Time_{it}) +$$

$$\beta_{19}(BSmok_i \times Time_{it}) + \beta_{110}(Age_i \times Time_{it}) + \beta_{111}(CAR_i \times Time_{it}) +$$

$$\beta_{112}(Sex_i \times Time_{it}) + \beta_{113}(Depress_i \times Time_{it}) + \beta_{114}(Concern1_i \times Time_{it}) +$$

$$\beta_{115}(Concern2_i \times Time_{it}) + \beta_{116}(Edu_i \times Time_{it}) + \beta_{117}(Income_i \times Time_{it}) +$$

$$\beta_{20}(Smoke_{it}) + r_{0i} + r_{1i}(Time_{it}) + e_{it}$$

Aim 4. To explore whether biopsychosocial variables predicted the effect of smoking status on weight gain (i.e., cross-level interactions), the predictor variables were added to level 2 of the model to explain variation in smoking status. Each variable was examined separately. Significant cross-level interactions were then added to the final

multivariate model from Aim 3. Results of this analysis indicated whether each variable predicted the association between smoking status and weight gain over time, controlling for the influences of other predictor and confounder variables.

Level 1: $Weight_{it} = \pi_{0i} + \pi_{1i}(Time_{it}) + \pi_{2i}(Smoke_{it}) + e_{it}$

Level 2: $\pi_{0i} = \beta_{00} + \beta_{01}(Int_i) + \beta_{02}(TNP_i) + \beta_{03}(BMI_i) + \beta_{04}(Appet_i) +$

$\beta_{05}(CPD_i) + \beta_{06}(Meds_i) + \beta_{07}(Diet_i) + \beta_{08}(Exer_i) + \beta_{09}(BQuit_i) +$

$\beta_{010}(Age_i) + \beta_{011}(CAR_i) + \beta_{012}(Sex_i) + \beta_{013}(Depress_i) +$

$\beta_{014}(Concern1_i) + \beta_{015}(Concern2_i) + \beta_{016}(Edu_i) + \beta_{017}(Income_i) + r_{0i}$

$\pi_{1i} = \beta_{10} + \beta_{11}(Int_i) + \beta_{12}(TNP_i) + \beta_{13}(BMI_i) + \beta_{14}(Appet_i) +$

$\beta_{15}(CPD_i) + \beta_{16}(Meds_i) + \beta_{17}(Diet_i) + \beta_{18}(Exer_i) + \beta_{19}(BSmoke_i) +$

$\beta_{110}(Age_i) + \beta_{111}(CAR_i) + \beta_{112}(Sex_i) + \beta_{113}(Depress_i) +$

$\beta_{114}(Concern1_i) + \beta_{115}(Concern2_i) + \beta_{116}(Edu_i) + \beta_{117}(Income_i) + r_{1i}$

$\pi_{2i} = \beta_{20} + \beta_{21}(Int_i) + \beta_{22}(TNP_i) + \beta_{23}(BMI_i) + \beta_{24}(Appet_i) +$

$\beta_{25}(CPD_i) + \beta_{26}(Meds_i) + \beta_{27}(Diet_i) + \beta_{28}(Exer_i) + \beta_{29}(BQuit_i) +$

$\beta_{210}(Age_i) + \beta_{211}(CAR_i) + \beta_{212}(Sex_i) + \beta_{213}(Depress_i) +$

$\beta_{214}(Concern1_i) + \beta_{215}(Concern2_i) + \beta_{216}(Edu_i) + \beta_{217}(Income_i)$

Combined: $Weight_{it} = \beta_{00} + \beta_{01}(Int_i) + \beta_{02}(TNP_i) + \beta_{03}(BMI_i) + \beta_{04}(Appet_i) +$

$\beta_{05}(CPD_i) + B_{06}(Meds_i) + \beta_{07}(Diet_i) + \beta_{08}(Exer_i) + \beta_{09}(BSmoke_i) +$

$\beta_{010}(Age_i) + \beta_{011}(CAR_i) + \beta_{012}(Sex_i) + \beta_{013}(Depress_i) +$

$\beta_{014}(Concern1_i) + \beta_{015}(Concern2_i) + \beta_{016}(Edu_i) + \beta_{017}(Income_i) +$

$\beta_{10}(Time_{it}) + \beta_{11}(Int_i \times Time_{it}) + \beta_{12}(TNP_i \times Time_{it}) + \beta_{13}(BMI_i \times Time_{it}) +$

$\beta_{14}(Appet_i \times Time_{it}) + \beta_{15}(CPD_i \times Time_{it}) + B_{16}(Meds_i \times Time_{it}) +$

$$\beta_{17}(Diet_i \text{ x } Time_{it}) + \beta_{18}(Exer_i \text{ x } Time_{it}) + \beta_{19}(BSmok_i \text{ x } Time_{it}) +$$

$$\beta_{110}(Age_i \text{ x } Time_{it}) + \beta_{111}(CAR_i \text{ x } Time_{it}) + \beta_{112}(Sex_i \text{ x } Time_{it}) +$$

$$\beta_{113}(Depress_i \text{ x } Time_{it}) + \beta_{114}(Concern1_i \text{ x } Time_{it}) +$$

$$\beta_{115}(Concern2_i \text{ x } Time_{it}) + \beta_{116}(Edu_i \text{ x } Time_{it}) + \beta_{117}(Income_i \text{ x } Time_{it}) +$$

$$\beta_{20}(Smoke_{it}) + \beta_{21}(Int_i \text{ x } Smoke_{it}) + \beta_{22}(TNP_i \text{ x } Smoke_{it}) +$$

$$\beta_{23}(BMI_i \text{ x } Smoke_{it}) + \beta_{24}(Appet_i \text{ x } Smoke_{it}) + \beta_{25}(CPD_i \text{ x } Smoke_{it}) +$$

$$\beta_{26}(Meds_i \text{ x } Smoke_{it}) + \beta_{27}(Diet_i \text{ x } Smoke_{it}) + \beta_{28}(Exer_i \text{ x } Smoke_{it}) +$$

$$\beta_{29}(BQuit_i \text{ x } Smoke_{it}) + \beta_{210}(Age_i \text{ x } Smoke_{it}) + \beta_{211}(CAR_i \text{ x } Smoke_{it}) +$$

$$\beta_{212}(Sex_i \text{ x } Smoke_{it}) + \beta_{213}(Depress_i \text{ x } Smoke_{it}) + \beta_{214}(Concern1_i \text{ x } Smoke_{it}) +$$

$$\beta_{215}(Concern2_i \text{ x } Smoke_{it}) + \beta_{216}(Edu_i \text{ x } Smoke_{it}) + \beta_{217}(Income_i \text{ x } Smoke_{it}) +$$

$$r_{0i} + r_{1i}(Time_{it}) + e_{it}$$

Chapter 3: Results

Overall participant demographics are found in Table 1. The sample consisted of mostly males (57%) with an average age of 49.31 (SD=9.09). Participants were mostly low income (61% less than $10,000 household income) years with a high school education or less (69%). Most were concerned about gaining weight after quitting smoking (59%). The average weight of the sample at baseline was 192.79 (SD=46.10), and the mean BMI was in the upper end of the overweight category (29.69, SD=7.28). The average weight gain over twelve months post CBT was .60 lbs (SD=16.16; Mdn=1.5). About 27% of the sample reported 28-day continuous abstinence at twelve months post CBT, and 23% reported abstinence at every follow-up. Participants reported elevated depressive symptoms (21.19, SD=10.83) and exhibited an average CAR of 20.17 (SD=51.10).

Primary Analyses

Aim 1. A fully unconditional level 1 model was specified to examine the rate of weight gain over twelve months post CBT in the overall sample (Table 2). Contrary to the hypothesis, the fixed effect of time (i.e., the average rate of weight gain per one month increase in time) was not significant (p=.622). The fixed effect of time also was not significant among sample of continued smokers (p=.157), indicating no significant weight gain over twelve months. Among abstainers, the average baseline weight was 205.36 (p<.0001). Time was positively associated with weight (p<.0001), indicating that abstainers significantly gained weight (approximately 6.828 lbs) over the 12-month follow-up.

Potential confounding variables were included in model to examine their associations with baseline weight and the pattern of weight gain. Participants in the culturally-specific condition weighed less than those in the standard condition (β=-9.1169, SE=4.9988, p=0.069). Baseline BMI was positively related to baseline weight (β=193.31, SE=6.85, p<.0001). Due to the high correlation between continuous BMI and weight, BMI was coded into a dichotomous variable indicating obesity status (BMI >=30; 0=Not obese, 1=Obese) and included in further analyses. No significant associations between baseline weight and other variables were found (TNP use, appetite increase, CPD, medication use, and changes in diet and exercise). No variables were significantly associated with the rate of weight gain.

The variations in the individual intercept (covariance estimate=2129.52, SE=164.70, z=12.93, p<0.0001) and slope (covariance estimate=1.3267, SE=0.1544, z=8.59, p<0.0001) were significant, indicating that each individual had a unique baseline weight and rate of weight gain. Therefore, multilevel modeling was appropriate to use for analysis for this data. Smoking status and biopsychosocial variables were then tested to further predict individual weight gain over the 12-month follow up, controlling for baseline differences and covariates.

Aim 2. Controlling for the effects of intervention condition and baseline obesity, smoking status significantly predicted weight gain over the twelve month follow-up (Table 3). The within-person effect of smoking status was significantly associated with weight, indicating that a participant weighed more over time if he/she reported abstinence rather than smoking (p=.012). The between-person effect was also associated with weight gain, such that the rate of weight gain decreased by 0.78 lbs each month as the average

number of times the participant reported smoking increased ($p<.001$). These results indicated that average time smoking was predictive of the rate of weight gain over time (i.e., how rapidly an individual gained weight).

Aim 3. To examine biopsychosocial predictors of weight gain, sex, CAR, age, depressive symptoms, weight concern, education level, and income were added separately to level 2 of the model (Table 4). The effects of intervention condition, obesity, and smoking status were controlled for in each model.

Sex. Sex negatively predicted baseline weight, indicating that the mean weight for females was 21.74 lbs less than for males ($p<.0001$). However, there was no significant difference in rate of weight gain between sexes ($p=.577$), which did not support the hypothesis. Sex was included in the final multivariate model to control for the observed baseline differences in weight.

Age. Age was not significantly associated with baseline weight ($p=.910$) or the rate of weight gain over time ($p=.734$), which did not support the hypothesis.

Cortisol awakening response. Baseline CAR was not significantly associated with baseline weight ($p=.627$) or the rate weight gain over time ($p=.597$), which did not support the hypothesis.

Depressive symptoms. Depressive symptoms were not significantly associated with baseline weight ($p=.270$) or the rate weight gain over time ($p=.888$), which did not support the hypothesis.

Weight concern. Those who were very concerned about gaining weight due to smoking cessation weighed significantly more at baseline compared to those who were not concerned ($p=.017$). Weight concern was not significantly associated with the rate of

weight gain over time (p=.350), which did not support the hypothesis. Weight concern was included in the final multivariate model to control for the observed baseline differences in weight.

Education level. A positive trend was found between education level and baseline weight (p=.059), such that baseline weight increased as education level increased. Education level was not significantly associated with the rate weight gain (p=.423), which did not support the hypothesis. Education was included in the final multivariate model as a control.

Income level. Income level was not significantly associated with baseline weight (p=.802) or the rate weight gain (p=.689), which did not support the hypothesis.

Final model. To test predictors of weight gain over twelve months post CBT, smoking status, sex, weight concern, and education level were then included in a multivariate model (Table 5). Intervention condition (p=.044), sex (p<.0001), and weight concern (p<.01) were significantly associated with weight at baseline. An inverse relationship was found between smoking status and individual weight gain (p=.054), such that the rate of weight gain decreased over the 12-month follow-up as the average number of times a participant reported smoking increased.

Exploratory Analyses

Aim 4. To explore the effects of cross-level interactions between biopsychosocial variables and smoking status on weight gain, sex, age, CAR, depressive symptoms, weight concern, education level, and income were added to level 2 of the model to predict smoking status (Table 6). Depressive symptoms significantly predicted the effect of time-varying smoking status (p=.006). As baseline depressive symptoms increased,

participants significantly weighed more if they reported abstinence compared to if they reported smoking. Therefore, as participants reported greater baseline depressive symptoms, the difference in weight gain between abstainers and smokers was larger. No other interactions were significant.

     Final model. The significant cross-level interaction of depressive symptoms and smoking status was added to the final model in Aim 3 (Table 7). Controlling for all covariates, participants who remained abstinent over the follow-up gained an average of 0.5 lbs each month (p=.009). Females weighed significantly less than males (p<.0001), and those who were very concerned about weight gain weighed significantly more than those who were not concerned (p=.008). The average number of times a participant reported smoking was negatively related to rate of weight gain over the 12-month follow up (p=.005). The interaction of depressive symptoms and within-person effect of smoking status remained significant (p=.006), such that as depressive symptom severity increased, participants weighed even more if they reported abstinence versus smoking (Figure 4). However, the simple effect of smoking status was not significant. Furthermore, the regions of significance were calculated to determine the values of depressive symptoms after which the simple main effect of smoking status becomes significant, or when the simple effect of smoking status is not contingent upon depressive symptom severity (Hoffman, 2014). Results showed that the upper threshold was 42.987, indicating that the simple effect of smoking status on weight would become significant when mean depressive symptoms scores were greater than 42.987. Therefore, the greatest weight gain would occur if a participant reported severe depressive symptoms as well as abstinence.

Chapter 4: Discussion

The aim of the current study was to examine predictors of weight gain over twelve months post CBT among treatment-seeking African Americans enrolled in randomized controlled smoking cessation trial. The overall sample of African American smokers did not gain weight significantly over the 12-month follow-up period. However, weight significantly increased among participants who reported continuous abstinence at each follow-up. Smoking status predicted an individual's weight gain, and those who reported abstinence at any follow-up weighed more than those who reported smoking. The rate of weight gain was reduced as the average number of times a participant reported smoking increased. Furthermore, the difference in weight between abstainers and smokers was moderated by depressive symptoms, such that participants who were abstinent at any time point weighed more if they reported greater baseline depressive symptoms.

Weight Gain in African American Smokers

Overall, the average weight gain in this sample was minimal. However, when examined among abstainers only, weight gain was significant. Post-cessation weight gain within a 12-month follow-up period averaged about seven lbs. The results are consistent with previous research indicating that smokers gained an average of four to ten lbs after abstaining (Aubin et al., 2012; Flegal, 2012; Perkins, 1993). This phenomenon of post-cessation weight gain has been well-documented. Yet, our findings are inconsistent with literature indicating that African Americans gained more than average weight after cessation (Klesges et al., 1998; Williamson et al., 1991). These differing findings may be a function of the variation in study designs and analyses. Previous studies in this

36

population lacked established smoking cessation intervention components (Williamson et al., 1991; Klesges et al., 1998;, Vander Weg et al., 2001). The current study was the first, to our knowledge, to examine weight in African Americans enrolled in a CBT efficacy trial. Therefore, the absence of excessive weight gain in this sample may be due to aspects of the intervention content. For example, one session of CBT focused on post-cessation weight gain and taught strategies to reduce weight gain (e.g., increasing physical activity, decreasing fatty food intake). Teaching weight control strategies in the context of tobacco cessation treatment may attenuate weight gain (Danielsson, Rossner, & Westin, 1999; Spring et al., 2004; Spring et al., 2009; Farley et al., 2012). Thus, exposure to content on post-cessation weight gain and weight management among treatment-seeking African Americans may have a positive impact on weight control. Considering the dearth of current research on African Americans and post-cessation weight gain, our findings help fill a gap in smoking cessation literature.

Smoking and Weight Gain

We found that smoking status influenced individual weight gain over time, which significantly varied among participants. Similar to previous studies (Reas et al., 2009; Flegal, 2012; O'Hara et al., 1998), abstainers weighed more than smokers. However, the current study examined multiple effects of smoking status (i.e., within-person and between-person) on weight. We found that reporting smoking at any time point (EOC, 3-, 6-, or 12-month follow up), as well as the average number of times a participant reported smoking, were associated with less weight gain. We also found a marked difference in weight each month between abstainers and smokers when the effects of biopsychosocial variables were controlled. Most research has analyzed weight gain using

predefined smoking categories (e.g., abstainers, continued smokers), and the variability

of individual smoking status during the follow-up period was not considered. Smokers

tend to make multiple quit attempts before sustaining long-term abstinence (Caraballo et

al., 2014), so the prevalence of lapsing or relapsing during post-treatment follow-up may

be high.   For example, approximately 50% of smokers in evidence-based CBT

treatments relapsed during one-year post treatment (Kolar & Webb Hooper, 2015).

Allowing smoking status to vary over time captured its inconsistent nature within

individuals. Distinguishing the effects of smoking status on weight is important to inform

the design of smoking interventions that also address weight management.

Biopsychosocial Predictors of Weight Gain

Biopsychosocial factors were examined as predictors of weight gain in this

sample. We found no association between biological factors and weight gain over the 12-

month follow up. Similar to previous studies (Klesges et al., 1989; Williamson et al.,

1991; Vander Weg et al., 2001; Borrelli & Mermelstein, 1998; Collins, Nair, Hovell, &

Audrain-McGovern, 2009; Ludman et al., 2002), sex and weight concern were associated

with baseline weight, indicating greater weight among females and participants who were

very concerned about weight gain. However, there was no effect of these variables on the

rate of weight gain over time; females and males gained weight similarly despite the

differences at baseline. Additionally, concern about weight gain at baseline was not

indicative of the amount of weight gained over the follow-up period. In contrast, studies

have shown that, controlling for smoking status and other covariates, weight over time

increased more for females (Klesges et al., 1989; Williamson et al., 1991; Vander Weg et

al., 2001) and for those who were concerned (Borrelli & Mermelstein, 1998; Collins, Nair, Hovell, & Audrain-McGovern, 2009; Ludman et al., 2002).

Age and CAR also were not predictive of weight gain in the current study. Previous research has demonstrated an effect of age on weight gain in adults (Lycett, Hajek, & Aveyard, 2010; O'Hara et al., 1998; Swan & Carmelli, 1995; Williamson et al., 1991). Yet, the average follow-up period for these studies was about five years. The follow-up period in our study was only twelve months. In addition, when measuring cortisol, a very complex variable, several factors should be considered. Namely, the circadian rhythm, which controls secretion of cortisol, determines the CAR (Pruessner et al., 1997; Fries et al., 2009). Future research should assess circadian rhythm when measuring cortisol levels. In addition, cortisol samples were collected over one day rather than across multiple days, which may have affected the stability of intra-individual levels.

The lack of biological effects may indicate that psychosocial factors were the primary contributors to weight gain in this sample. In contrast to most previous research among smokers, the current study accounted for the effects of psychosocial variables in predicting weight gain. Specifically, we found a positive trend between education level and baseline weight gain. This finding corroborates previous research, indicating that higher educated African American women were more obese compared to their lower educated counterparts (Wang & Beydoun, 2007). In our exploratory analysis, we also found significant effects of psychological factors on weight gain over the follow-up period. Baseline depressive symptoms were not directly associated with weight gain. However, the difference in weight between abstainers and smokers was greater as the severity of baseline depressive symptoms increased. Therefore, it may be important to

address psychosocial symptoms that may affect abstinence and weight during smoking cessation interventions.

Psychosocial Factors and African American Smokers

Previous research has considered the unique influences of psychosocial factors among treatment-seeking African American smokers. For example, lower levels of acculturation to the majority U.S. culture (i.e., stronger identification with traditional African American culture) was related to a reduced likelihood of quitting smoking (Webb Hooper, Baker, Rodriguez de Ybarra, McNutt, & Ahluwalia, 2012). In addition, distress has been explored among African American treatment-seeking smokers (Hooper, Baker, & McNutt, 2013; Webb Hooper & Kolar, 2015). Results of these studies have shown that African Americans demonstrate moderate to severe depressive symptoms, similar to the observed rates in the current study, and the symptoms have been found to be greater compared to other racial/ethnic groups. Previous research also has shown that change in depressive symptom scores differ among African American smokers compared to other groups. Webb Hooper & Kolar (2015) found that depressive symptoms of African Americans decreased significantly over the course of a four-week smoking cessation intervention compared to that of white and Hispanic smokers. Further, reduced depressive symptoms were associated with a higher likelihood of abstinence. Thus, assessing and treating psychosocial factors, specifically depressive symptoms, may have an impact on the reduction of chronic disease risk factors (e.g., smoking) in African Americans.

Little to no research has explored the interactive effect of depressive symptoms and smoking status on weight gain, another risk factor of chronic disease. In the current

study, African American abstainers weighed more if they reported greater depressive symptom severity at baseline. Thus, the magnitude of weight gained over the study was influenced by baseline depressive symptom scores. The overlap of behaviors related to depression and smoking cessation may explain this finding. One of which is eating. For example, depression has been found to positively predict emotional eating (Ouwens, van Strien, & van Leeuwe, 2009; Goldschmidt et al., 2104; van Strien, Konttinen, Homberg, Engels, & Winkens, 2016; Michopoulous et al., 2015). This relationship has been documented in African Americans; in a primarily African American, low SES sample (N=1110), depressed individuals engaged in severe emotional eating compared to their non-depressed counterparts (Michopoulous et al. 2015). Emotional eating has been found to predict BMI and weight gain (Goldbacher, La Grotte, Komaroff, Vander Veur, & Foster, 2016; Hayman Jr., McIntyre, & Abbey, 2015; Koenders & van Strien, 2011). Specifically, this maladaptive coping strategy has been proposed as a mechanism by which depression predicts obesity (van Strien, Konttinen, Homberg, Engels, & Winkens, 2016).

Eating is also a replacement for smoking after cessation (Filozof et al., 2004). Many individuals use smoking as a maladaptive strategy to cope with life stressors. Upon cessation, smokers may continue engaging in maladaptive strategies, such as overeating. Research has shown that maladaptive coping is positively correlated with negative affect among African American smokers (Hooper, Baker, & McNutt). Therefore, maladaptive eating may occur especially in abstinent smokers experiencing negative affect. Among depressed smokers who already engage in maladaptive eating behaviors, quitting smoking may sustain and worsen these habits.

Findings of the current study suggest that depressive symptoms may further increase weight after quitting smoking. It appeared that the difference in weight between abstainers and smokers was dependent upon depressive symptom severity. The psychological factor moderated the association between smoking cessation and weight by strengthening the deleterious impact of quitting on weight. As post-cessation weight gain may contribute to obesity, targeting psychosocial factors during smoking and weight interventions appears to be critical. The prevalence of depressive symptoms is high among African American smokers, a group that suffers disproportionately from tobacco- and obesity-related diseases. Unaddressed psychological symptoms may increase the likelihood of developing obesity during smoking cessation treatment in this population; reducing one chronic disease risk factor may, in turn, increase prevalence of another. Therefore, understanding the associations among chronic disease risk factors within African Americans is particularly important.

Strengths and Limitations

There are several strengths to the current study. First, the current study included a large sample of African American smokers, who have been primarily underrepresented in post-cessation weight gain research. Second, the prospective design allows for examination of predictive versus cross-sectional associations, and rigorous, longitudinal data modeling was used to delineate effects of the predictors on the rate of weight gain. Lastly, objective measures of weight were used (i.e., participants weighed in lab on same scale at each assessment).

The results of this study should be interpreted in light of its limitations. First, this was a secondary analysis, so variables pertinent to the current research question, such as

details on diet/eating habits and physical activity, were not available for analysis. Second, the limitations of using self-report measurements, such as the possibility of social desirability and response bias, are widely known. Lastly, we are unable to generalize findings to non-treatment-seeking African American smokers.

Conclusions

This study is the first, to our knowledge, to examine predictors of weight gain among treatment-seeking African American smokers. With the racial/ethnic disparities that exist in smoking outcomes and obesity-related disease, it is important to understand the distinctive factors that are associated with weight gain in this population. The weight gain of African Americans in this sample was comparable to the average four to ten pounds documented in literature. Smokers in this study may have benefited from an evidenced-based smoking cessation intervention that provided nicotine replacement therapy and addressed weight management strategies. Using a biopsychosocial approach, we also found that psychosocial, rather than biological, factors influenced weight gain. Our exploratory findings demonstrated that higher rates of depressive symptoms led to an increase in the difference in weight between abstainers and smokers. The results of our study may have implications for the design of targeted smoking cessation and weight interventions in African Americans.

Future Directions

Future research is needed to assess further the factors that predict weight gain in African American smokers. First, studies including data on diet and exercise history, as well as changes in energy balance and other biopsychosocial factors (i.e., depressive symptoms, cortisol, etc.) over time, are warranted. Data on eating behavior may be

particularly important. Next, to examine whether exposure to weight strategies helps reduce weight gain in African Americans, an RCT testing the efficacy of an added weight management component to an evidence-based smoking cessation treatment (treatment group: smoking cessation CBT plus weight management; control: smoking cessation CBT only) is needed. Finally, longitudinal studies that include multiracial/multiethnic samples are important for direct comparisons of weight gain among racial/ethnic groups. These comparison studies also would help identify factors unique to each group that may contribute to disparities.

As previous research has highlighted the prevalence of greater depressive symptoms in treatment-seeking African American versus white smokers, targeted interventions addressing depressive symptoms should be explored further. Assessing depression at baseline will allow for treatment of the symptoms within the context smoking cessation treatment. To delineate the impact of psychological factors on weight gain in this population, the efficacy of smoking plus depression treatment should be tested in an RCT, comparing an established smoking cessation CBT plus depression condition to an established smoking cessation CBT alone. Findings from the RCT would highlight the role that untreated depressive symptoms may have in post-cessation weight gain. Overall, more longitudinal research conducted with large African American samples is important to understand and address factors that are related to weight gain among treatment-seeking smokers.

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Figures

Figure 1. Biopsychosocial Theory
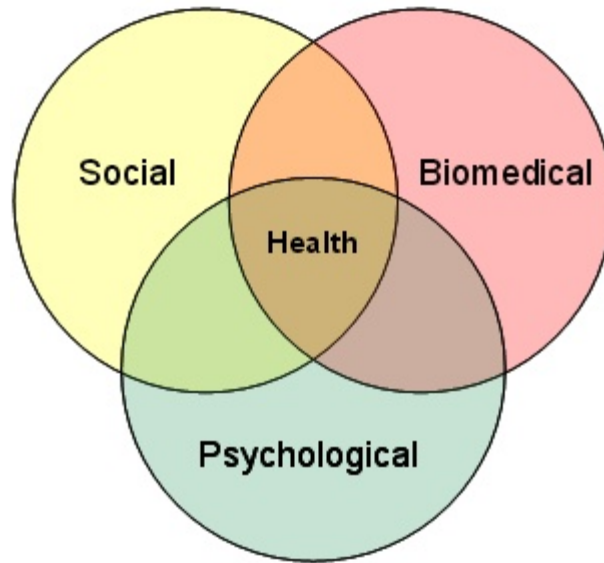
Figure 2. Biopsychosocial Theory of Weight Gain among Treatment-Seeking African American Smokers
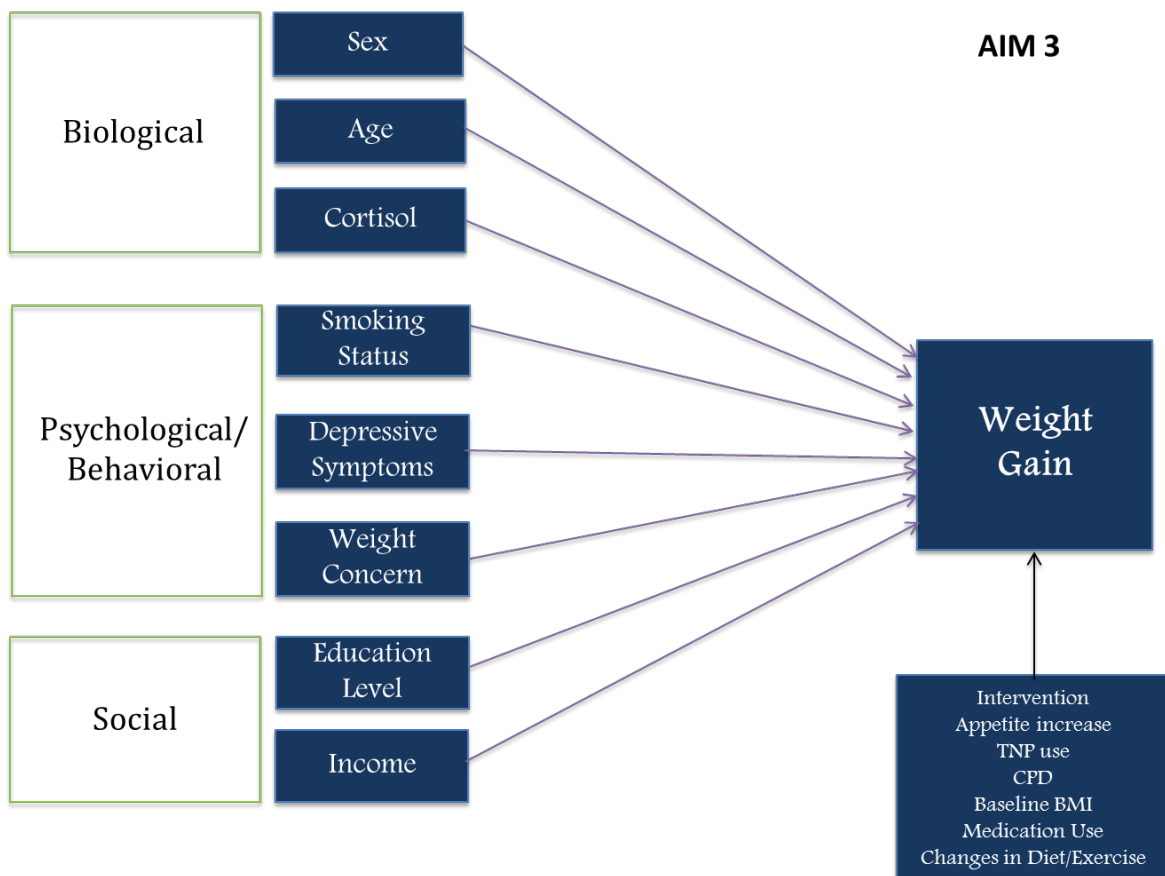
Figure 3. Interactive Effect of Biopsychosocial Factors and Smoking Status on Weight Gain Over Twelve Months Post CBT (Exploratory Aim 4)
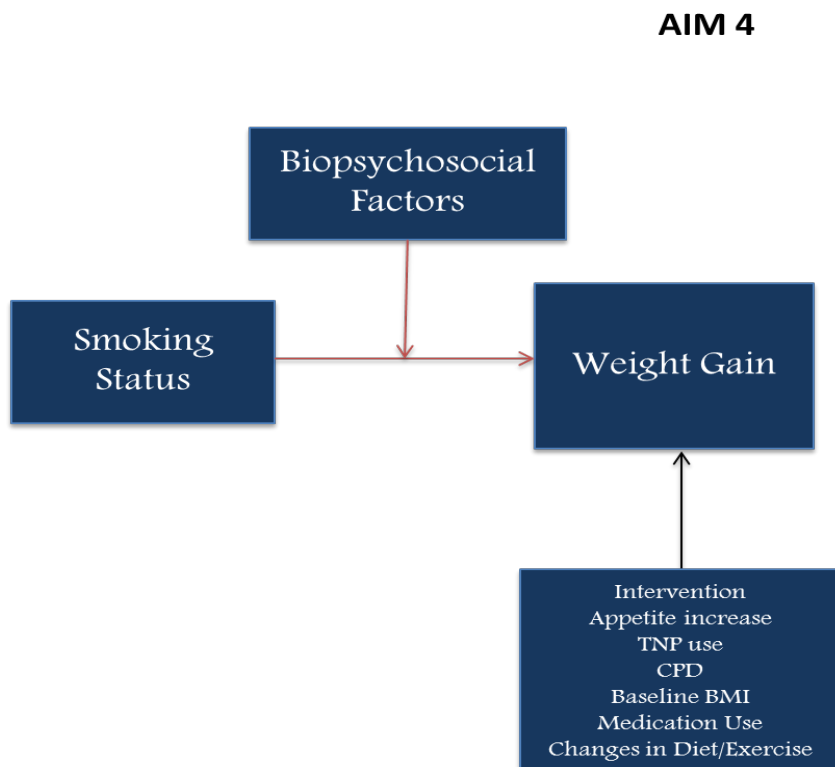
Figure 4: Crosslevel Interaction of Depressive Symptoms and Smoking Status on Weight at 12-month Follow up

Depressive Symptoms and Smoking Status Interaction Effect at 12-month Follow up
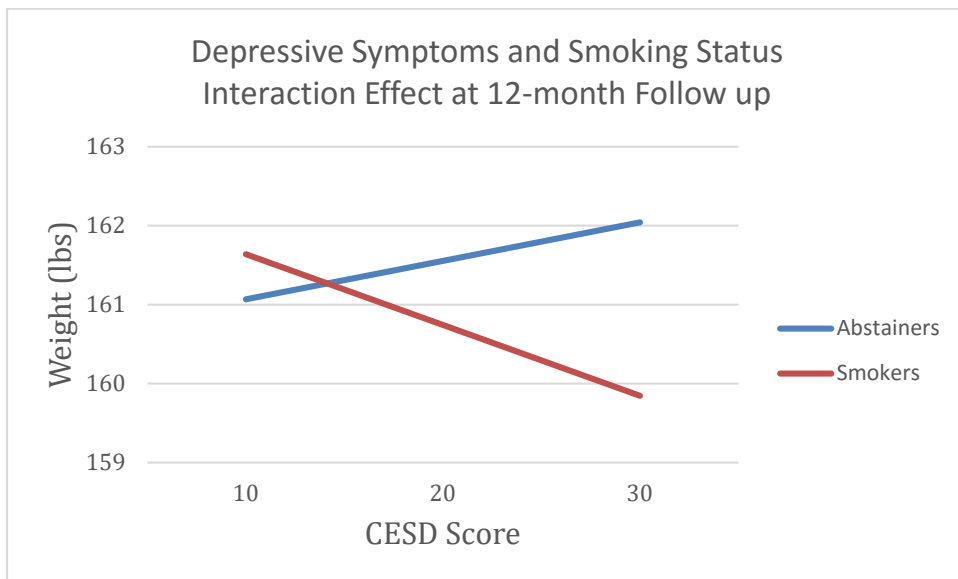
Tables

Table 1: Sample Characteristics

Characteristic	Total (N=342)	Standard (n=173)	Culturally Specific (n=167)	Possible Range	Observed Range	p*
Demographics						
Age, years, M (SD)	49.50 (9.07)	49.52 (8.73)	49.48 (9.44)	18—	(20-67)	ns
Sex (%)						
Male	57	52	61	—		
Female	43	48	39	—		
Household income (%)						ns
Less than $10,000	61	60	62	—		
$10, 000 - $30,000	29	31	27	—		
$30,000 - $50,000	4	5	7	—		
Greater than $50,000	6	4	4	—		
Education level (%)						ns
Less than high school	19	17	20	—		
High school	50	51	48	—		
Greater than high school	31	32	32	—		
Smoking Status (%)						
3 months post CBT	31	26	37	—		p<.05
6 months post CBT	29	28	31	—		ns
12 months post CBT	27	25	28	—		ns
Cigarettes per day, M (SD)	18.08 (10.81)	17.87 (10.03)	18.29 (11.54)	0—	(1.0-60.0)	ns
Transdermal nicotine patch use, total days, M (SD)	21.67 (6.44)	21.19 (6.44)	22.20 (6.41)	(0-25)	(0-25)	
Average appetite increase over 4 week intervention, M (SD)	1.79 (1.09)	1.73 (1.06)	1.85 (1.13)	(0-4)	(0-4)	
Psychological						
Weight concern (%)						ns
No concern	41	40	42	—		
Some concern	36	39	33	—		
Much concern	23	21	25	—		
Depressive symptoms, M (SD)	21.19 (10.83)	20.43 (11.56)	22.12 (10.85)	(0-60)	(0-50)	ns
Biological						

Weight in pounds, M (SD)						
Baseline	192.79 (46.10)	197.08 (46.21)	188.30 (45.70)	—	(93.0-354.0)	ns
12 months	195.74 (46.77)	199.04 (45.06)	192.46 (48.37)	—	(97.6-378.8)	ns
Gain over 12 months	.60 (16.16)	-0.54 (16.39)	1.74 (15.90)	0—	(-64.0-42.8)	ns
Baseline BMI, M (SD)	29.69 (7.28)	30.55 (7.34)	28.86 (7.13)	—	(16.6-58.9)	
Obesity (%)						*p*<.05
Obese (BMI>=30)	42	45	39	—		
Not obese (BMI<30)	58	55	61	—		
Cortisol awakening response, M (SD)	20.17 (51.10)	23.60 (49.66)	17.76 (51.99)	—	(-67.8-203.5)	ns
*X^2 or t-test comparing each variable by intervention condition						
CBT=cognitive-behavioral therapy						

Table 2: Results of Full Unconditional Model (Aim 1)

Model Results					
Fixed Effect	**Estimate***	**Standard Error**	**DF**	**t Value**	**Pr > |t|**
Overall Sample					
Intercept	193.49	2.5077	341	77.16	<.0001
Time (months)	0.03955	0.08016	952	0.49	0.6218
Abstainers					
Intercept	205.36	4.9540	78	41.45	<.0001
Time (months)	0.5694	0.1398	235	4.07	<.0001
Continued Smokers					
Intercept	189.92	2.8697	262	66.18	<.0001
Time (months)	-0.1308	0.09240	716	-1.42	0.1574
*Average baseline weight in lbs. (intercept) or average rate of weight gain per one month increase (time)					

Table 3: Association of Smoking Status and Weight Over Twelve Months Post CBT (Aim 2)

Model Results					
Fixed Effect	**Estimate***	**Standard Error**	**DF**	**t Value**	**Pr > |t|**
Intercept	170.19	3.0528	339	55.75	<.0001
Time (Months)	0.5123	0.1768	945	2.90	0.0039
Smoking status (within)	-1.2914	0.5154	945	-2.51	0.0124
Smoking status (between) x Time	-0.7832	0.2534	945	-3.09	0.0021
Intervention condition	-5.1212	3.6272	339	-1.41	0.1589
Obesity	63.9642	3.6764	339	17.40	<.0001
*Average differences in baseline weight in lbs. (intercept) or rate of weight gain (time) per one-unit change in the predictor variable (if continuous) or between groups (if categorical)					

Table 4. Independent Associations of Predictors and Weight Over Twelve Months Post CBT (Aim 3)

Model Results					
Variable Fixed Effect	**Estimate***	**Standard Error**	**DF**	**t Value**	**Pr > |t|**
SEX					
Intercept	178.25	3.2143	336	55.45	<.0001
Time (months)	0.5472	0.1881	941	2.91	0.0037
Smoking status (within)	-1.3122	0.5165	941	-2.54	0.0112
Smoking status (between) x Time	-0.7719	0.2549	941	-3.03	0.0025
Intervention condition	-6.9850	3.4705	336	-2.01	0.0449
Obesity	69.5882	3.6176	336	19.24	<.0001
Sex (female)	-21.7427	3.6107	336	-6.02	<.0001
Sex (female) x Time	-0.08904	0.1595	941	-0.56	0.5769
AGE					
Intercept	169.05	10.4026	336	16.25	<.0001
Time (months)	0.6661	0.4738	941	1.41	0.1601
Smoking status (within)	-1.2993	0.5168	941	-2.51	0.0121
Smoking status (between) x Time	-0.7940	0.2555	941	-3.11	0.0019
Intervention condition	-5.4216	3.6452	336	-1.49	0.1379
Obesity	64.1713	3.7080	336	17.31	<.0001
Age	0.02242	0.1978	336	0.11	0.9098
Age x Time	-0.00292	0.008576	941	-0.34	0.7339
CAR					
Intercept	172.31	4.1316	217	41.71	<.0001
Time (months)	0.5932	0.2226	662	2.66	0.0079
Smoking status (within)	-1.3011	0.6129	662	-2.12	0.0341
Smoking status (between) x Time	-0.9887	0.3105	662	-3.18	0.0015
Intervention condition	-6.6407	4.5035	217	-1.47	0.1418
Obesity	60.9909	4.5094	217	13.53	<.0001
CAR	-0.02120	0.04359	217	-0.49	0.6272
CAR x Time	0.001019	0.001925	662	0.53	0.5967
DEPRESSIVE SYMPTOMS					
Intercept	175.33	4.7061	283	37.26	<.0001
Time (months)	0.4741	0.2465	798	1.92	0.0548
Smoking status (within)	-0.8984	0.5517	798	-1.63	0.1038
Smoking status (between) x Time	-0.7723	0.2784	798	-2.77	0.0057
Intervention condition	-6.2629	3.8595	283	-1.62	0.1058
Obesity	62.0083	3.9199	283	15.82	<.0001
Depressive symptoms	-0.1907	0.1724	283	-1.11	0.2697
Depressive symptoms x Time	0.001117	0.007896	798	0.14	0.8875

WEIGHT CONCERN					
Intercept	167.03	3.5586	337	46.94	<.0001
Time (months)	0.4061	0.1924	943	2.11	0.0350
Smoking status (within)	-1.2950	0.5154	943	-2.51	0.0121
Smoking status (between) x Time	-0.8418	0.2556	943	-3.29	0.0010
Intervention condition	-5.3357	3.6143	337	-1.48	0.1408
Obesity	61.8532	3.7542	337	16.48	<.0001
Some concern	4.3566	4.1865	337	1.04	0.2988
Some concern x Time	0.2726	0.1847	943	1.48	0.1404
Much concern	11.5076	4.8040	337	2.40	0.0171
Much concern x Time	0.1874	0.2005	943	0.93	0.3503
EDUCATION LEVEL					
Intercept	161.06	5.5932	335	28.80	<.0001
Time (months)	0.6904	0.2814	938	2.45	0.0143
Smoking status (within)	-1.3085	0.5181	938	-2.53	0.0117
Smoking status (between) x Time	-0.7980	0.2554	938	-3.12	0.0018
Intervention condition	-4.8482	3.6170	335	-1.34	0.1810
Obesity	64.7003	3.6640	335	17.66	<.0001
Education level	2.5830	1.3624	335	1.90	0.0588
Education level x Time	-0.04921	0.06144	938	-0.80	0.4234
HOUSEHOLD INCOME LEVEL					
Intercept	169.78	3.2931	333	51.56	<.0001
Time (months)	0.4775	0.1874	933	2.55	0.0110
Smoking status (within)	-1.3250	0.5178	933	-2.56	0.0107
Smoking status (between) x Time	-0.7517	0.2575	933	-2.92	0.0036
Intervention condition	-5.1942	3.6720	333	-1.41	0.1581
Obesity	64.3979	3.7398	333	17.22	<.0001
Income	0.2996	1.1944	333	0.25	0.8021
Income x Time	0.02100	0.05243	933	0.40	0.6889
*Average differences in baseline weight in lbs. (intercept) or rate of weight gain (time) per one-unit change in the predictor variable (if continuous) or between groups (if categorical)					

Table 5: Multivariate Model of Associations of Predictors and Weight Gain Over Twelve Months Post CBT (Aim 3)

Fixed Effect	Estimate*	Standard Error	DF	t Value	Pr > |t|
Model Results					
Intercept	166.06	5.6737	332	29.27	<.0001
Time (months)	0.1852	0.1160	939	1.60	0.1108
Smoking status (within)	-0.7975	0.6394	939	-1.25	0.2126
Smoking status (between) x Time	-0.2462	0.1281	939	-1.92	0.0548
Sex (female)	-23.1185	3.5779	332	-6.46	<.0001
Some concern	6.8940	3.9570	332	1.74	0.0824
Much concern	14.9868	4.5645	332	3.28	0.0011
Education level	2.1804	1.2818	332	1.70	0.0899
Intervention condition	-6.9161	3.4212	332	-2.02	0.0440
Obesity	67.6955	3.6206	332	18.70	<.0001
*Average differences in baseline weight in lbs. (intercept) or rate of weight gain (time) per one-unit change in the predictor variable (if continuous) or between groups (if categorical)					

Table 6: Biopsychosocial x Smoking Status Interactive Effects on Weight Over Twelve Months Post CBT (Exploratory Aim 4)

Model Results					
Variable Fixed Effect	**Estimate***	**Standard Error**	**DF**	**t Value**	**Pr > |t|**
SEX					
Intercept	177.97	3.2306	336	55.09	<.0001
Time (months)	0.5160	0.1775	941	2.91	0.0037
Smoking status (within)	-0.8498	0.7079	941	-1.20	0.2303
Smoking status (between) x Time	-0.7836	0.2539	941	-3.09	0.0021
Intervention condition	-6.9448	3.4693	336	-2.00	0.0461
Obesity	69.5512	3.6164	336	19.23	<.0001
Sex (female)	-21.1736	3.6740	336	-5.76	<.0001
Sex (female) x Smoking status (within)**	-0.9836	1.0263	941	-0.96	0.3382
AGE					
Intercept	171.98	10.5086	336	16.37	<.0001
Time (months)	0.5218	0.1777	941	2.94	0.0034
Smoking status (within)	-5.3468	2.4781	941	-2.16	0.0312
Smoking status (between) x Time	-0.7949	0.2543	941	-3.13	0.0018
Intervention condition	-5.4220	3.6435	336	-1.49	0.1377
Obesity	64.1610	3.7063	336	17.31	<.0001
Age	-0.03735	0.2002	336	-0.19	0.8521
Age x Smoking status (within)**	0.08305	0.04977	941	1.67	0.0955
CAR					
Intercept	172.60	4.1373	217	41.72	<.0001
Time (months)	0.5966	0.2219	662	2.69	0.0074
Smoking status (within)	-1.7146	0.6693	662	-2.56	0.0106
Smoking status (between) x Time	-0.9637	0.3076	662	-3.13	0.0018
Intervention condition	-6.7204	4.5053	217	-1.49	0.1372
Obesity	61.0520	4.5111	217	13.53	<.0001
CAR	-0.03330	0.04442	217	-0.75	0.4543
CAR x Smoking status (within)**	0.01819	0.01206	662	1.51	0.1320
DEPRESSIVE SYMPTOMS					
Intercept	173.16	4.7548	283	36.42	<.0001
Time (months)	0.5017	0.1917	798	2.62	0.0090
Smoking status (within)	2.0355	1.1903	798	1.71	0.0876

Smoking status (between) x Time	-0.7764	0.2749	798	-2.82	0.0048
Intervention condition	-6.2208	3.8530	283	-1.61	0.1075
Obesity	62.0007	3.9133	283	15.84	<.0001
Depressive symptoms	-0.08657	0.1757	283	-0.49	0.6226
Depressive x Smoking status (within)**	-0.1408	0.05067	798	-2.78	0.0056
WEIGHT CONCERN					
Intercept	166.57	3.5792	337	46.54	<.0001
Time (months)	0.5195	0.1765	943	2.94	0.0033
Smoking status (within)	-0.9679	0.8025	943	-1.21	0.2281
Smoking status (between) x Time	-0.7940	0.2528	943	-3.14	0.0017
Intervention condition	-5.3194	3.6151	337	-1.47	0.1421
Obesity	61.8560	3.7552	337	16.47	<.0001
Some concern	4.8897	4.2587	337	1.15	0.2517
Some concern x Smoking status (within)	-0.1445	1.1864	943	-0.12	0.9031
Much concern	12.5752	4.8739	337	2.58	0.0103
Much concern x Smoking status (within)**	-1.1043	1.2996	943	-0.85	0.3957
EDUCATION LEVEL					
Intercept	160.29	5.6759	335	28.24	<.0001
Time (months)	0.5179	0.1779	938	2.91	0.0037
Smoking status (within)	0.05123	1.4715	938	0.03	0.9722
Smoking status (between) x Time	-0.7852	0.2544	938	-3.09	0.0021
Intervention condition	-4.8668	3.6180	335	-1.35	0.1795
Obesity	64.6897	3.6651	335	17.65	<.0001
Education level	2.8024	1.3896	335	2.02	0.0445
Education x Smoking status (within)**	-0.3889	0.3918	938	-0.99	0.3212
HOUSEHOLD INCOME LEVEL					
Intercept	169.79	3.2982	333	51.48	<.0001
Time (months)	0.4994	0.1792	933	2.79	0.0054
Smoking status (within)	-1.3764	0.5913	933	-2.33	0.0201
Smoking status (between) x Time	-0.7594	0.2565	933	-2.96	0.0031
Intervention condition	-5.1815	3.6716	333	-1.41	0.1591
Obesity	64.3894	3.7394	333	17.22	<.0001
Income	0.2918	1.2096	333	0.24	0.8095
Income x Smoking status (within)**	0.05283	0.2999	933	0.18	0.8602

*Average differences in baseline weight in lbs. (intercept) or rate of weight gain (time) per one-unit change in the predictor variable (if continuous) or between groups (if categorical)

**Average difference in the association between the time-varying smoking status and weight gain given one-unit change in the predictor variable (if continuous) or between groups (if categorical).

Table 7: Multivariate Model Results of Predictors of Weight Gain Over Twelve Months Post CBT (Exploratory Aim 4)

Model Results					
Fixed Effect	**Estimate**	**Standard Error**	**DF**	**t Value**	**Pr > |t|**
Intercept	163.90	6.7879	278	24.15	<.0001
Time (months)	0.5044	0.1922	795	2.62	0.0088
Smoking status (within)	1.9558	1.1914	795	1.64	0.1011
Smoking status (between) x Time	-0.7811	0.2754	795	-2.84	0.0047
Intervention condition	-7.0870	3.6700	278	-1.93	0.0545
Obesity	65.7009	3.9495	278	16.64	<.0001
Sex (female)	-20.2408	4.0429	278	-5.01	<.0001
Some concern	8.7742	4.2756	278	2.05	0.0411
Much concern	13.1543	4.9378	278	2.66	0.0082
Education level	2.3284	1.3403	278	1.74	0.0834
Depressive symptoms	0.04874	0.1760	278	0.28	0.7821
Depressive x Smoking status (within)**	-0.1384	0.05065	795	-2.73	0.0064
*Average differences in baseline weight lbs. (intercept) or rate of weight gain (time) per one-unit change in the predictor variable (if continuous) or between groups (if categorical)					
**Average difference in the association between the time-varying smoking status and weight gain given one-unit change in the predictor variable (if continuous) or between groups (if categorical).					

Appendices

Demographic Questionnaire

The following questions are about you, and your life situation. They are to help us better understand the people we serve. You are under no obligation to answer any question that you find objectionable, however, we would appreciate your answering as many as possible. All answers will be kept confidential.

***Screening #*_____   *Participant #:*_____**

***Date:*_____**

1.   What is your age?_____

2.   What level of education did you complete?
     _____ Elementary School   _____ Business or Technical Training
     _____ Junior High School   _____ Some College (no degree)
        _____ Partial High School   _____   University   Degree,
Bachelor level or equivalent
     _____ High School   _____ Post-graduate Degree

3.   What is your marital status?
     _____ Single   _____ Separated   _____ Widowed
     _____ Married   _____ Divorced

4.   With which ethnic/racial group do you most identify yourself? (please check one)
     _____ Oriental/Asian American/Pacific Islander
     _____ Black/African American
     _____ Native American
     _____ White/Caucasian
     _____ Other

5.   Are you Hispanic?   YES   NO   (Circle one)

6.   What is your approximate personal yearly income?
     _____ Under $10,000   _____ $50,001 - $60,000
     _____ $10,000 - $20,000   _____ $60,001 - $70,000
     _____ $20,001 - $30,000   _____ $70,001 - $80,000
     _____ $30,001 - $40,000   _____ $80,001 - $90,000
     _____ $40,001 - $50,000   _____ Over $90,000

7.   Total Household income?
     _____ Under $10,000   _____ $50,001 - $60,000
     _____ $10,000 - $20,000   _____ $60,001 - $70,000
     _____ $20,001 - $30,000   _____ $70,001 - $80,000
     _____ $30,001 - $40,000   _____ $80,001 - $90,000
     _____ $40,001 - $50,000   _____ Over $90,000

8.   How do you primarily pay for health care?
     _____ Medicare

_____ Medicaid
_____ Insurance
_____ Self-pay
_____ Don't know/not sure

9. Who is your usual health care provider?
_____ Private Doctor
_____ Community Health Clinic
_____ Hospital-Based Health Clinic
_____ Emergency Room
_____ Other

10. Are you currently pregnant or breastfeeding (Women only)?   YES or NO

11. How many times have you EVER tried to quit smoking? _____

12. What is the longest period of time you have been able to quit smoking? (Circle your answer)

_____ Never   _____ 1 week to 1 month
_____ Several hours only   _____ 1 month to 6 months
_____ Less than one day   _____ More than 6 months
_____ 1 day to 1 week

13. Not counting yourself, how many other people who live in your household currently smoke? _____

14. Not counting yourself, how many of your friends smoke?
_____ None of them
_____ Few of them
_____ Some of them
_____ Most of them
_____ All of them

15. During the past week, how many days per week did you drink any alcoholic beverages, on the average?

0   1   2   3   4   5   6   7   (Circle one)

16. On the days when you drank, about how many drinks did you drink on average? _____

17. If you stopped smoking cigarettes today, how concerned would you be that you might start smoking   again because of any weight gain? (Circle your answer)
_____ Not at all concerned
_____ Somewhat concerned
_____ Very concerned

CES-D

Below is a list of some of the ways you may have felt or behaved. Please indicate how often you have felt this way during the past week by circling the appropriate number. **DURING THE PAST WEEK:**

	Rarely or None of the time (Less than 1 day)	Some or A little of the time (1-2days)	Occasionally or A moderate amount of time (3-4 days)	Most or All of the time (5-7 days)
1. I was bothered by things that usually don't bother me.	0	1	2	3
2. I did not feel like eating; my appetite was poor.	0	1	2	3
3. I felt that I could not shake off the blues even with help from my family or friends.	0	1	2	3
4. I felt that I was just as good as other people.	0	1	2	3
5. I had trouble keeping my mind on what I was doing.	0	1	2	3
6. I felt depressed.	0	1	2	3
7. I felt that everything I did was an effort.	0	1	2	3
8. I felt hopeful about the future.	0	1	2	3
9. I thought my life had been a failure.	0	1	2	3
10. I felt fearful.	0	1	2	3
11. My sleep was restless.	0	1	2	3
12. I was happy.	0	1	2	3
13. I talked less than usual.	0	1	2	3
14. I felt lonely.	0	1	2	3
15. People were unfriendly.	0	1	2	3
16. I enjoyed life.	0	1	2	3
17. I had crying spells.	0	1	2	3
18. I felt sad.	0	1	2	3
19. I felt that people disliked me.	0	1	2	3
20. I could not get "going."	0	1	2	3

Minnesota Nicotine Withdrawal Scale

Participant #:_____   Date:_____

Behavior Rating Scale
Self-Report

 Please rate your feelings for the period of the last _____
•**Please circle the MOST APPROPRIATE number next to each symptom**

0 = none   1 = slight   2 = mild   3 =moderate   4 = severe

	None		Mild		Severe
1. Angry, irritable, frustrated	0	1	2	3	4
2. Anxious, nervous	0	1	2	3	4
3. Depressed mood, sad	0	1	2	3	4
4. Desire or craving to smoke	0	1	2	3	4
5. Difficulty concentrating	0	1	2	3	4
6. Increased appetite, hungry, weight gain	0	1	2	3	4
7. Insomnia, sleep problems, awakening at night	0	1	2	3	4
8. Restless	0	1	2	3	4
9. Impatient	0	1	2	3	4
10. Constipation	0	1	2	3	4
11. Dizziness	0	1	2	3	4
12. Coughing	0	1	2	3	4
13. Dreaming or nightmares	0	1	2	3	4