PERCEIVED STRESS AND COTININE AMONG AFRICAN AMERICAN SMOKERS: 
VARIATION BY SKIN COLOR

A Dissertation in 
Biobehavioral Health 
by 
Guy-Lucien Sh’Antu Ya Shembo Whembolua

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The dissertation of Guy-Lucien Sh’Antu Ya Shembo Whembolua was reviewed and approved* by the following:

Gary King
Professor of Biobehavioral Health
Dissertation Advisor
Chair of Committee

Edgar P.Yoder
Professor of Agriculture and Extension Education

Sonia A. Cavigelli
Assistant Professor of Biobehavioral Health

Lori A. Francis
Assistant Professor of Biobehavioral Health

Collins O. Airhihenbuwa
Professor
Head of the Biobehavioral Health Department

*Signatures are on file in the Graduate School
ABSTRACT

In the United States, skin color has played an important role in race relations and racial stratification. This has consequences in the daily lives of African Americans, one of which is stress. Numerous studies have looked at the consequences of stress in African American communities. Cigarette smoking has been linked to stress in different pathways and a substantial amount of literature addresses the relationship between stress and cigarette smoking. However, there is no empirical evidence that links skin color and perceived stress to cotinine. This study tested 1) whether African American smokers with higher perceived stress levels had higher cotinine levels; 2) whether self-perception of skin color (subjective measurement) was a better predictor of perceived stress than either facultative or constitutive melanin (objective measure) among African Americans; and 3) whether skin color-based discrimination questions was a better predictor of perceived stress than more objective measures. The results of this study indicated that perceived stress among African American smokers was not significantly associated with cotinine levels. Facultative melanin was found to be significantly and positively associated with cotinine level. This relationship was found to be partially mediated by years of smoking, as smoking duration was significantly associated with higher cotinine levels. Constitutive melanin was not found to be significantly associated with cotinine levels. Self-perception of skin color was found to be a stronger predictor of perceived stress levels than either facultative melanin or constitutive melanin. A composite discrimination measure based on skin color was found to be a stronger predictor of perceived stress among African Americans than the use of an objective measurement or reflectometer.
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“An ant cannot cross a river without a liana” (Tetela Proverb)

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Chapter 1

Introduction

Smoking behavior among African Americans

Smoking has been identified as the single most preventable cause of illness and death in American society and it is thought to be responsible for deaths due to cancer, heart disease, stroke, complications of pregnancy, and respiratory illness (Centers for Disease Control and Prevention [CDC], 2002a, 2002b, 2005b; U.S. Department of Health and Human Services [USDHHS], 2004).

Socio-structural factors such as education, tobacco industry promotion, and health practices play a major role in differences seen in smoking (de Beyer et al., 2001; Gardiner et al., 2004; Gilman et al., 2003; Orr et al., 2005). Additionally, the variable “race” or “ethnicity” is often used in social sciences regarding differences in smoking although notable criticisms have raised questions about its significance (King, 1997; Griesler et al., 1998; Lee et al., 2001). According to the Center for Disease Control and Prevention in 2006, smoking prevalence rates are similar among African American adults (23.0%) and white adults (21.9%) in the United States. African American men (27.6%) smoked at a higher rate than white men (24.3%); African American women (19.2%) and white women (19.7%), however, smoked at a similar rate. While these rates are associated with sociodemographic variables and smoking behavior patterns, it should be
noted that considerable heterogeneity by region exists among African Americans (King et al., 1999; King et al., 2006). For example, African American women residing in the South were more likely to never be smokers than were those living in the Northeast, Midwest, and West.

The African American population has experienced specific consequences due to smoking. For example, African American women have endured higher rates of smoking-related cancers and cardiovascular diseases when compared to Caucasian women (CDC, 2002b).

Cessation rates in African American women are, on the other hand, lower than their White counterparts (Harris et al., 2004; Lawrence, Graber, Mills, Meissner, & Warnecke, 2003). This is despite the fact that African American women in general make more quit attempts than their White American counterparts and express more confidence in their ability to make a successful quit (CDC, 1994; Hahn et al., 1990; Royce et al., 1993; Tessaro et al., 1997; Vander-Martín, Cummings, & Coates, 1990; Velicer et al., 1995). Additionally, African Americans have higher rates of tobacco-related deaths from coronary heart disease, stroke and lung cancer. African Americans are the only ethnic group in which the prevalence of smoking increases by adulthood according to the Center for Disease Control and Prevention (1991). Dornelas (2005) also reveals that 96% of African American adolescents live with another smoker.

African American smoking characteristics differ significantly from those of their Caucasian counterparts. A striking example of this difference is the type of cigarette smoked. Seventy percent of African American smokers consume mentholated cigarettes compared to 30% of Caucasian Americans (U.S. Department of Health and Human
Services [USDHHS], 1998; Ahijevych & Wewers, 1994; Hahn, Folsom, Sprafka, & Norsted, 1990; Ramirez & Gallion, 1993; Royce, Hymowitz, Corbett, Hartwell, & Orlandi, 1993). The adoption of these specific products is largely thought to be due to the intense marketing campaign targeting African American inner city consumers during the late 1970s-1990s (Yerger, 2007). This advertising surge was supported by the use of psychographic profiles and ethnic marketing as well as the distribution of free cigarettes samples (Burrell Advertising, 1978; Fraser, 1987; Philip Morris, 1985). This targeting led to what Gardiner (2004) called the “African Americanization” of menthol cigarettes by the tobacco industry.

Several of the biomarkers used for average smoke exposure have also shown distinct results in African American populations when compared to Caucasian counterparts. In several studies, even when taking into account the number of cigarettes smoked per day, carbon monoxide and cotinine (a primary proximate metabolite of nicotine) levels are higher among African American women when compared to Caucasian women (Ahijevych & Gillespie, 1997; Ahijevych, Gillespie, Demirci, & Jagadeesh, 1996; Ahijevych & Parsley, 1999; Ahijevych, Tyndale, Dhatt, Weed, & Browning, 2002; Wagenknecht et al., 1990). The implications of these different studies are that nicotine dependence should be assessed beyond self-reported cigarettes (Ahijevych & Gillespie, 1997) and increased cotinine level may be due to an increase in exposure (Ahijevych & Parsley, 1999). Understanding these differences may provide clues to the reasons for the higher rates of some smoking-related cancers in African Americans (Wagenknecht et al., 1990).
Skin Color in African American communities

The study of skin color can be approached from different perspectives. The biology of skin color (skin pigmentation) and the sociological perspective will be discussed in this section.

From a biological perspective, there is a wide variation in human pigmentation and most of it is due to the presence of the pigment melanin. The visual appearance of individual pigmentation reflects the biological adaptation to aspects of the environment (Jablonski et al., 2000). Melanin can be found in two different forms: eumelanin and phaeomelanin (Parsad et al., 2003). Melanin is the pigment that gives color to the skin, eyes, and hair (Hedin & Larsson, 1978). Melanocytes synthesize melanin in the basal layer of the epidermis (Baker & Joseph, 1960; Brues et al., 1975). It is then transferred into organelles called the melanosomes (Harper et al., 2007). Differences in skin color are related to the concentration and size of the melanosomes (Yerger et al., 2006). The number of melanocytes is not specific to any racial or ethnic group (Staricco et al., 1957; Soames et al., 1974). Skin pigmentation is divided into two types: constitutive skin color that is the result of individual genetic content and is not affected directly by sun exposure (Kollias et al., 1991); and facultative skin color or “tan” that is influenced by exposure to ultraviolet radiation, hormonal changes and diseases (Fizpatrick et al., 1974).

Pigment variation has led some societies to attach labels and fabricate myths about people with different skin and/or hair colors (Diop, 1981). This is the result of racial stratification, a subtype of social stratification. Social stratification can be defined as the unequal distribution of scarce goods and services in society. In a society
experiencing extensive class stratification, individuals who are members of the ruling class will seek to maintain their power advantages by reinforcing divisions in society (Geschwender, 1980).

In the United States, the importance of skin tone has played an important role in race relations and racial stratification. One of the most defining features in the presence of Africans in the Americas is the existence of the institution of slavery. This institution was an exploitative system that subjugated millions of Africans in the Americas in order to maximize profits on plantations. This coercive system was maintained by racial “differential treatment of Blacks in terms of an attitude of rejection based on group differences, with a special emphasis on skin color.” Jordan (1974) stated that Englishmen viewed Africans as different and a lower order of humans; he viewed slavery and prejudice as interacting constantly and reinforcing each other. In his model, Africans in the Americas were debased not only for profits but because they were different. Bettelheim and Janovitz (1964) suggested that prejudice based on skin color was associated with social mobility. They argued that prejudice occurred to limit social mobility of members of the Black community. White racism and prejudice helped thus perpetuate the social hierarchy with Whites in position of dominance. Hunter et al. (2007) stated that the notion that dark skin represents inferiority supports the maintenance of white aesthetic, ideological and material supremacy.

This ideology was forcibly instilled in the African American community. During the slavery period, individuals of mixed race were more likely to be trained to do household jobs than were their darker peers (Mullins et al., 1984). E. Franklin Frazier (1957) argued that “mulattoes” (individuals with at least one Caucasian parent) led a
privileged existence when compared with their dark-skinned counterparts. Mulattoes even formed a class on their own in the South (Williamson, 1980). During the pre-civil war era, lighter skinned blacks in the South were taller than darker skinned counterparts, which could suggest differential access to health resources (Bodenhorn, 2002). Mullins et al. (1984) have also shown that mulattoes were overrepresented in the elites of the African American community. Despite the enforcement of the “one-drop” rule, a color gradient internally stratifies the African American community (Davis, 1991). Several researchers have suggested a continuing relationship between variations in skin tone and life opportunities of African Americans (Glenn, 1963; Ransford, 1970). Keith and Herring (1991) showed that despite the re-evaluations of “blackness” during the civil rights movements, light complexion continued to be a significant predictor of such outcomes as educational attainment, occupation and income in African Americans. These advantages for light-skinned individuals were again demonstrated by the work of Rondilla et al. in 2007. Dark skin color was also found to be associated with higher levels of perceived discrimination (Allen et al., 2000).

Colonial history and racial discrimination enforced by individuals from European descent in different part of the world (India, South Africa, Americas) has given birth to what Margareth Hunter (2007) called a persistent problem for people of color today: colorism. She defines it as “the process that privileges light-skinned people of color over dark skin in areas such as income, education, housing, and the marriage market.” Colorism becomes then a second system of discrimination after racism for individuals with darker skin tone. Two recent events have unfortunately reminded the world of the existence of this phenomenon. Last September, Shahrukh Khan, a famous Bollywood
actor, was criticized by Asian activists for participating in an advertisement promoting a skin lightening cream called “Fair and Handsome” (British Broadcasting Corporation website, 2007). The following month, in United States, an African American promoter, DJ Ulysses, planned an event with free admission to all fair-complexioned women (Appendix A). These separate events in two different continents demonstrate how skin tone is still used as a tool for division within members of the same community. Colorism has been shown to be both from intraracial and interracial origin (Jones, 2000). The hegemony of White ideals of beauty has created an aversion within some parts of the African American community for “African” phenotype such as dark skin (Freeman et al., 1966; Udry et al., 1971).

In work dating from two decades ago, African American children have been shown to have lighter skin tone preference (Porter, 1991). This was also demonstrated for adolescents and college students (Robinson et al., 1995; Bond et al., 1992). The perception of Whites about African American skin color is seldom investigated, but Terkildsen (1993) reported that White respondents evaluate fictitious Black political candidates more negatively if they have darker skin.

Issues of color are also present in other parts of the American continent. Light skin and European features were privileged in colonial Mexico (Almaguer, 1994). Color stratification has also been demonstrated in the Mexican communities regarding income as well as psychological stress (Arce et al., 1987; Codina et al., 1994). The island nation of Haiti has yet to come to the terms with this issue. Political unrest in this Caribbean country is also partly associated with the conflict between the ethnic French-speaking minority of “Creoles” and the dark-skin Creole-speaking majority (Claude Ribbe, Grioo
The preference for lighter skin individuals is also present in different Asian cultures such as the Japanese (“bihaku”) and Chinese. It is estimated that a third of all the expenses made on the beauty products market ($ five billion yearly) in China are made for whitening lotions (Sylvie Kaufman, Le Monde, 2007).

Although there is evidence of a relationship, the interaction between these three factors- stress levels, skin color and cotinine among African American smokers- has not been sufficiently explored. This study highlights the need to identify key theories that could help the understanding of the processes involved in the individuals studied.

**Study Aims**

The purpose of this study is to examine the relationship between perceived stress and cotinine levels as well as the relationship between self perception of skin color and perceived stress in an African American population in Harrisburg, Pennsylvania. Using behavioral surveys and specific biological markers, the study will:

- Compare the saliva level of cotinine (a biomarker for nicotine) to the results obtained on the 10-item Cohen Perceived Stress Scale.
- Compare the objective score of melanin content obtained through a device (reflectometer) as well as the self-obtained scores of skin color as predictors of perceived stress.
Research Questions

The purpose of the research study is to examine if the correlation between cotinine levels, perceived stress, and skin color among African American smokers are significant. Specifically, the following hypotheses will be explored:

**Hypothesis 1**: African American smokers with higher perceived stress levels will have higher cotinine levels (See Figure 1-1).

![Diagram](image.png)

**Figure 1-1**: Hypothesis 1
**Hypothesis 2:** Self perception of skin color measurements or subjective measures will be a better predictor of perceived stress than more objective measures among African American smokers (See Figure 1-2).

![Diagram](image)

**Figure 1-2:** Hypothesis 2
**Hypothesis 3:** Skin color-based discrimination questions will be a better predictor of perceived stress than objective measures of skin color obtained with a reflectometer among African American smokers (See Figure 1-3).

![Diagram showing the relationship between skin color-based discrimination, perceived stress, and objective skin color measure.](image)

Figure 1-3: Hypothesis 3
Significance

Public recognition of the health risks of smoking has been increasing in the last few years. According to the Surgeon General Report (2004), “Tobacco smoking remains the number one cause of preventable disease and death in the United States, accounting for 440,000 deaths each year.” Cigarette smoking is the cause of most cases of lung cancer reported in the U.S., and lung cancer is the leading cause of all deaths resulting from cancer (American Cancer Society, 2005). Several additional adverse outcomes have also been linked to smoking. Compared to nonsmokers, men who smoke are about twenty-three times more likely to develop lung cancer, resulting in nearly ninety percent of lung cancer deaths in men. Similarly, women who smoke are about thirteen times more likely to develop lung cancer, resulting in almost eighty percent of lung cancer deaths in women (Surgeon Report, 2001). Smoking also accounts for cancers of the oral cavity, pharynx, larynx, esophagus and bladder (Garavello et al., 2006). In addition, coronary heart disease and stroke, the two primary types of cardiovascular disease caused in part by smoking, have been identified as the first and the third leading causes of death in the United States. These two cardiovascular ailments touch 61 million Americans and result in other health complications such as high blood pressure, stroke, and congestive heart failure. The consequent death toll is 2,600 Americans daily (Surgeon Report, 2001). More than ninety percent of deaths resulting from Chronic Obstructive Pulmonary Disease (COPD) have been attributed to smoking (Novotny et al., 1998). Furthermore, women who smoke are at an increased risk for infertility, since smoking makes it more
difficult for them to become pregnant by increasing risks for ectopic pregnancy and spontaneous abortion (Surgeon Report, 2001).

In addition to the foregoing, non-smokers are also exposed to the harmful effects of cigarette smoke. Second-hand smoke, also known as Environmental Tobacco Smoke, is defined as “the mixture of the smoke given off by the burning end of tobacco products (sidestream smoke) and the smoke exhaled by smokers (mainstream smoke)” (National Institute on Drug Abuse, 2006).

African American adults are exposed to daily events that affect their lives, such as lower incomes and racial inequities (Tomaskovic-Devey, 1993), which can lead to adverse health outcomes, such as hypertension, coronary artery disease and depression (Brown et al., 2004).

Stress has been shown to be associated with several mental health outcomes in African Americans. In their 1968 book “Black Rage”, psychiatrists Price Cobbs and William Grier studied the case histories of African American men and women who underwent psychotherapy and argued that individuals living in a racist society are psychologically damaged by the effects of racist oppression. James et al. (1984) created the concept of John Henryism defined as “the predisposition to confront barriers to upward social mobility.” African-Americans with low socioeconomic status, or of low education, with high John Henryism scores have been shown to have an increased blood pressure and prevalence of hypertension (James et al., 1992; James et al. 1994).

Experiencing racist events has been found to be associated with anxiety, depression and somatization in African Americans (Landrine et al., 1996). Personal
experiences of racism have been found to have adverse immediate and cumulative effects on the physical and mental well-being of African Americans (Jackson et al., 1996). This negative effect has been found to be the result of the interaction of race and socioeconomic status (SES) (Kessler et al., 1986). Williams et al. (2000) revealed that reports of discrimination due to race were positively related to psychological distress. Poussaint and Alexander (2000) found an association between stress-related illness and self-destructive behaviors. They argued that the conflict inherent in being Black in America has led many African-Americans to commit suicide.

The relationship between race and perceived stress has been proven to be mediated by racial identity (Sellers et al., 2003). Minorities who had a greater ethnic identity reported more personal experiences of discrimination than did less identified minorities (Operario et al., 2001). The findings of these studies, taken together, suggest that stress could be a mediating link between racial discrimination and mental health in African Americans. The results obtained by Sellers et al. (2003) support the existence of a pathway between racial discrimination and psychological distress with perceived stress as a mediating variable. They also point out that individuals for whom race is a more central identity were more likely to report lower levels of psychological distress.

Additionally, the current project adds to the literature on tobacco research especially in the underserved and understudied populations. Public health experts have studied the mechanisms behind the disproportionately high number of smoking related problems in members of low socioeconomic status as well as minority groups in the U.S. (Fagan et al., 2004). This project, because it incorporates elements of epidemiology as
well as stress measurements and basic biology, presents a different perspective of the issue of tobacco use and known health effects in underserved populations. It could help illustrate the understanding of tobacco’s harmful effects and the health related effects. Understanding these disparities could help generate more effective smoking cessation programs. This project will provide a valuable source of information regarding stress, cotinine levels and smoking behaviors.
Chapter 2

Literature Review

This chapter presents a review of the literature on the stress, the link between smoking and stress, an overview of the stress in African American communities and the biological and sociological nature of skin color. In addition, the relationship between cotinine as a biomarker and tobacco will be addressed.

The Stress Process

The stress process is associated with the “wear and tear” that minds and bodies experience when they attempt to cope with the changing environment. Walter Cannon (1932) developed the concept of “homeostasis” by demonstrating the importance of the autonomic nervous system in the production of a fight or flight reaction as the body’s response to aggression. He linked the subsequent adaptive responses to stress with catecholamine excretion and its actions (Chrousos & Gold 1992).

In 1936, stress was defined by Hans Selye as the response of the individual to an environmental stimulus. It includes interaction between individuals and their social environment (Lazarus et al., 1984). In the case of an unfavorable interaction, this could result in an undesired outcome, such as poor health or disease.

Selye (1946) describes the chronological development of the response to stressors as the General Adaptation Syndrome which includes three distinct phases:
• The “Alarm” phase in which the body prepares itself for immediate action (secretion of pro-inflammatory corticoids from the adrenal glands).
• The “Adaptation” phase: if the source of stress persists, the body prepares for long-term protection by secreting hormones to increase blood levels.
• The “Exhaustion” phase: This phase is the final one and is only present during chronic stress situations. It can lead to long term damage due to the exhaustion of the immune system. This phase can result in illnesses (i.e. cardiovascular diseases, ulcers).

Later on, researchers tried to re-conceptualize Selye’s notion of stress. Harold Wolff (1953) suggested a more holistic view of stress by connecting diseases to psychological stress (Black et al., 1996). In 1968, John Mason suggested that hormones may be critical mediators between psychological processes and body tissues in the development of some psychosomatic disorders. Two linked set of factors related to health in different ways have been identified as part of the stress process (Cobb et al., 1976):

• The” stressors” or risk factors which are the events or circumstances that occur and increase the probability that an individual will become ill. Traumas and stressful events such as physical abuses during childhood have been demonstrated to have detrimental impact on mental and physical health status (Adler, 1994; Lesserman et al., 1998). Niedhammer et al. (2008) found that low social support and decision latitude were risk factors for poor self-reported health.
• The “resistance factors”, the social relationships and beliefs and values that when possessed by an individual lower the chances of falling ill. Psychosocial
characteristics such as optimism and health-related hardiness have been studied to provide direction for prevention interventions (Lightsey, 1996; Smith et al., 2004). Hardiness in particular has been found to be associated with positive health behaviors (Pollock et al., 1990).

Dressler (1987) went on to elaborate that stress occurs “when an individual cannot meet the environmental challenges, this could be due to either the challenges are too great or the individual resources are too meager.” Dressler’s theory includes the notion that individuals in general are perpetually looking for a preferred state of balance (homeostasis) between activity level and energy intake. Chrousos et al. (1992) further defined stress as a state of disharmony or as a threat to homeostasis.

Two primary types of stressors have been identified: external and internal factors. External stressors include factors such as social class (Adler et al., 1994; Cohen et al., 2006), life events (Holmes et al., 1967) or lifestyle choices (Dressler et al., 1987). On the other hand, internal factors have also been studied. Psychological beliefs about the efficacy of an intervention have proven to be associated with lower reported stress (Hastings et al., 2001). Recent works like the one of Brandao et al. (1990) claiming that an individual’s thoughts and feelings may influence their metabolism have led to a renewed perspective on stress. In this perspective, stress may cause organic changes generated from the body’s failure to adapt to stressful situations (Kaplan et al., 1997).

Stress provokes multiple organic reactions, presenting several disorders. One of the primary physical consequences of stress has been identified as hypertension (Anderson et al., 1989; Cooper et al., 1996; Taylor et al., 2007). African Americans adults have been reported to have higher prevalence of high blood pressure than
In the case of hypertension, it can serve as a risk factor or stress can cause its clinical onset (Lutgendorf et al., 2000; McCarty et al., 1996).

Evidences have accumulated about the influence of stress on the onset of cardiovascular illness (Franco et al., 2003). One of the first studies to link personality traits and coronary disease was performed by cardiologists Milton Friedman and Ray Rosenbaum in the late 1950s. They concluded that the incidence of coronary disease was lower in Type B subjects (Byrne et al., 1986). Personality traits were later found to be associated with increased sympathetic nervous activity with its resultant increased blood pressure and heart rate (Farmer et al., 1999). Being male gender and subject to stress was a risk factor for the development of coronary events (Makino et al., 1998). Rozanski et al. (1999) demonstrated that acute stress increases blood viscosity, promoting myocardial ischemia, coronary vasoconstriction and arrhythmias while Esch et al. (2002) associated psychosocial stress with the process of atherosclerosis.

Perceived stress has been found to be associated with increased release of neurohormonal factors that predisposes a person to abdominal obesity, insulin resistance, and other features of the metabolic syndrome (Zafari et al., 2005).

Currently, stress is viewed as a system that coordinates adaptive responses of the organisms to stressors. The neurohormonal response to stress is mediated by two components: the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system (SNS) (Zafari et al., 2005). Several centers in the hypothalamus and in the brain stem regulate the stress response. These centers have neurons that secrete corticotrophin releasing hormone (CRH) and arginine-vasopressin; they also have noradrenergic
neurons (located in the locus ceruleus in the brain stem) that secrete norepinephrine (Chrousos et al., 1992). These different hormones have a unique basal and stress-related pattern of activity. The end-product of the HPA axis is cortisol which fluctuates within a narrow range and is maintained by a tightly regulated feedback system. An excessive and prolonged secretion of it would be detrimental to the organism (Chrousos et al., 2000). The activation of the stress system produces a variety of other effects such as depressive symptoms (Lincoln et al., 2007); it can also have a negative effect on the quality of life of terminally ill patients (Grassi et al., 1996).

**Measure of Stress**

Different scales have been used to measures stress. The Perceived Stress Scale (PSS) was designed originally by Cohen et al. (1983) as a 14-item measure that assesses the degree to which a person appraises the situations in his or her life as stressful. It measures the level of predictability and control with which a person views his or her life (Cohen et al., 1988). PSS scores have been shown to be associated with biological markers of stress and risks factors for coronary artery diseases (Malarkey et al., 1995) as well as other health behaviors such as drinking and exercising (Cohen et al., 1988). This scale has been used in several different populations with $\alpha=.82$ in African American populations (Sellers et al., 2003); $\alpha=.67$ in Spanish populations (Remor and Carrobles, 2001). Several studies have used the PSS scale to relate stress to health outcomes:

- Marucha et al. (1998) demonstrated that high stress level could have significant negative consequences on wound healing.
Golden-Kreutz (2005) found that stress was a predictor of quality of life among breast cancer survivors.

Stress has been shown to be associated with being a smoker (Croghan et al., 2006).

Sims et al. (2008) demonstrated the link between stress and high fat food among African Americans.

**Smoking and Stress**

A substantial amount of literature addresses the relationship between stress and cigarette smoking. Nicotine may start the reaction that induces the relationship between stress and smoking. When inhaled, nicotine enters the bloodstream and moves quickly to the brain. Nicotine’s psychoactive properties are due to its affinity to a class of receptors called nicotinic acetylcholine receptors, located in the peripheral and central nervous systems (Watkins, Koob, & Markou, 2000). When nicotine binds to these receptors it causes the increased release of several neurotransmitters. Specifically, acetylcholine, dopamine, serotonin, norepinephrine, and β-endorphin are released (N. L. Benowitz, 2008). These neurotransmitters are involved in several important bodily functions: cognitive functions, such as performance and memory, pleasure and reward (Pomerleau & Pomerleau, 1984); body weight regulation and mood (e.g., anxiety and tension) (Palfai & Jankiewicz, 1997). Further, nicotine also increases the release of norepinephrine and epinephrine from the adrenal glands, which results in increases in heart rate, blood pressure and gastro-intestinal activity. Nicotine is rapidly absorbed through the alveoli in
the lungs when smokers inhale (Winger, Hofman, & Woods, 1992) and is quickly distributed (within seconds) to the brain (Russell, 1987). It also has a brief half-life of approximately two hours (Tricker, 2003). The rewarding properties of nicotine include mild euphoria (Pomerleau & Pomerleau, 1992), increased energy, heightened arousal and appetite suppression (Stolerman & Jarvis, 1995). Cigarette smokers have reported that smoking produces arousal (N. L. Benowitz, 1988), and relaxation during periods of stress (Parrott, 1999). The rapid absorption, distribution and short half-life of nicotine require frequent self-administration if a smoker is to maintain nicotine levels and its associated rewarding properties.

The majority of smokers (>77%) are dependent on nicotine (Douglas, 1997; Stolerman, 1991). Thus, periods of tobacco abstinence produce an aversive withdrawal syndrome. Smokers who attempt to abstain from using tobacco products experience somatic and affective withdrawal symptoms. These symptoms include, but are not limited to, increased food intake, anxiety, restlessness, depressed mood, headache, irritability, sleep disturbances, an inability to concentrate (APA, 2000; Hughes, Gust, Skoog, Keenan, & Fenwick, 1991; Hughes & Hatsukami, 1986).

Among persons who have been smoking long-term, withdrawal symptoms can appear within minutes (Schuh & Stitzer, 1995) after a cigarette. Further, withdrawal symptoms peak in intensity within one to four days after cessation (Hatsukami, Hughes, Pickens, & Svikis, 1984). The aversive withdrawal symptoms that accompany a quit attempt contribute to relatively low cessation rates, especially considering that relapse to cigarette use is an effective method for suppressing these symptoms (Skjei & Markou,
Therefore, the suppression of this aversive withdrawal syndrome by continued nicotine self-administration (i.e., cigarette smoking) is thought to perpetuate smoking behavior (USDHHS, 1988; Watkins, Stinus, Koob, & Markou, 2000). Nicotine dependence contributes enormously to smoking-related death and disease because it is difficult to quit.

Studies have demonstrated that stress in several forms can be associated with smoking behaviors. Lawton (1962) showed that initiation and heavy smoking could be induced by distress. Pomerleau et al. (1984) advanced the explanation that the act of smoking relieves psychological stress. Mangan and Golding (1978) found that stressful events induce smoking. Psychological stressors have been shown to increase tobacco consumption among established smokers (Rose et al., 1983). Gunn (1983) demonstrated that high life stress scores in males were a stronger predictor of failing at quitting smoking in clinical program. In 1991, Romano et al. recognized an association between hassles and smoking behavior which lead them to hypothesize that smoking may be a coping behavior that mitigates the harmful psychological impact of a stressful environment. A survey done by Manfredi et al. (1992) indicated that young Black female smokers living in public housing are heavier smokers and have weaker motivation to quit when compared to Caucasian females. Being in a stressful environment (living in communities plagued by crime in substandard housings) for females has been shown to represent a significant barrier to smoking cessation (Lacey et al., 1993).

Financial stress measured by low socio-economic status (SES) has been shown to be highly correlated with smoking (Kirsch, 1999; Mulatu et al., 2002). Several aspects of
SES seem to be involved in that relationship: individuals with low SES seem to be more prone to begin smoking (Hersch, 2000; CDC, 2002) while purchases of tobacco products has been shown to at least exacerbate low SES (Zagorsky, 2004). Smoking rates have been declining since the mid-1990s. However, the decline has not been evenly divided with most of the decline occurring among upper SES and men. Being a member of lower SES has been proven to significantly influence three different stages of cigarette use. It increased the risk of initiation, increases the risk of progression to regular uses and finally decreases the likelihood of cessation (Gilman et al., 2003).

Initiation rates of smokers also differ depending on the socioeconomic class, with a significantly increased risk of smoking initiation observed among people from lower socioeconomic backgrounds. Low SES in childhood also increases the risk for progression to regular smoking, and was associated with a reduced likelihood of smoking cessation. Progression to regular smoking and smoking persistence is also associated with lower adult SES (USDHHS, 2004). Another dimension usually associated with low SES, low parental education, is also associated with higher rates of lifetime and current smoking (Griesler et al., 1998).

Despite the fact that the relation between SES and smoking patterns is complex, Gilman (2003) and his group demonstrate that the conditions accumulated while living in low SES groups reduce the rates of cessation among those groups. Other studies have taken a look at the relation between smoking and SES in other industrialized nations. The England-based NatWest group (Boseley, 2006) found that 48% of men in the poorest social class died before they reached 70, and half of that difference was due to smoking. It illustrates the point that in 2003, the poorest 10% of households in the British Isles
spent 2.43% of their income on cigarettes a week while the richest 10% spent 0.52%.
Tobacco thus has an additional damage on these families, as scarce family resources are
spent on tobacco products instead of on food, or other essential needs (de Beyer, et al.
2001).

An important idea relating to poverty in high income countries such as the United
States is relative deprivation or relative poverty, which is defined when an individual is
poorer than others in the social hierarchy (Bobak et al. 2001). The socio-economic class
“poverty” is a composite of several items and can therefore be approached using different
perspectives. Winship et al. (1992) believe that occupation and its specific model
Occupational Structure allow to assess poverty although it does not account for
discrimination. American governmental agencies such as the Census Bureau, for
example, have used income levels (Uchitelle, 2001). Winkleby et al. (1992) highlight in
their work the use of education as the strongest measure of SES linked to health, but they
warn against the use of an only SES indicator in any studies. Internationally, studies have
revealed that differences in smoking prevalence between poor and rich groups are greater
in low-income countries than in high-income countries (Bobak et al., 2000) Furthermore,
in high-income countries such as the United States, the prevalence of smoking is closely
and inversely associated with socio-economic status. Smoking prevalence in these
countries follows a continuous upward gradient from high to low socio-economic groups.
In the United States, in particular, educational status also predicts differences in smoking
prevalence more consistently than income, sex, or race (Pamuk et al.1998). In the United
States, smoking prevalence declined between 1974 and 1987 nine times faster in the most
educated group than in the least educated group (USDHHS 1989). Daily cigarette
consumption is 14% higher in smokers with a high-school education or less, than in those with more than 17 years of schooling (Rogers, Nam et al. 1995).

Landrine and Klonoff (2000) found that African American smokers self-reported experiencing significantly more lifetime racism than their non-smoker counterparts. Byrne et al. (2003) found a strong association between stress and smoking onset. In 2004, Baker et al. concluded that anxiety and depression may set the stage for smoking initiation. Bennett et al. (2005) reported that African American college students who experienced racial harassment were twice as likely to use tobacco products daily. Additionally, another study with African Americans by Manning et al. (2005) found that higher stress levels were associated with not being abstinent. The specific stressor, self-reported racial discrimination, proved to be associated with smoking among African Americans (Borrell et al., 2007). Borrell et al. work which used more than a thousand subjects, confirmed the findings several other researches (Resnicow et al., 1999; Guthrie et al., 2002; Kwate et al., 2003). Research performed among the Asian American community has also brought to light similar findings, with experienced racial discrimination identified as risk factor for smoking (Chae et al., 2008). Recently, socio-cultural stress has been recognized for playing a role in smoking behaviors among African American females (Fernander et al., 2008). These findings are consistent with past works confirming the association between stress and smoking, as well as works demonstrating that a greater identification with African American culture is associated with race-related stress. This has the possible consequences of using coping mechanisms which include smoking (Landrine et al., 1996).
Several studies have suggested that cigarette smoking can help reduce stress. Ikard et al. (1969) demonstrated that a large amount of smokers qualified cigarette smoking as something relaxing. This is in contrast to the adverse moods (stress, irritability) smokers have been shown to experience when they have not smoked recently (Hughes et al., 1990). Schachter et al. (1978) suggested that the pleasant feeling experienced during smoking is the simple reversal of the unpleasant effects of abstinence or mood normalization. Parrott et al. (1998) demonstrated that stress levels of smokers are only similar to non-smokers after they have just smoked. A study performed by Jones et al. (1997) reported higher stress levels in cigarette smokers than in nonsmokers during both day and night.

Research on adolescent populations confirms the “mood normalization” model with occasional smokers expressing greater stress versus lower levels of stress in nonsmokers (Lloyd et al., 1997). Adolescents who passed from experimental to regular smoking experienced an increase in affective distress (Hirschman et al., 1984).

Important works have linked quitting smoking to a decrease in stress. Hughes et al. (1992) found that tobacco cessation led to anxiety in the first few days and that by day 14, individual moods had improved. A significant reduction in self-rated stress and anxiety has been observed in individuals who successfully quit smoking (Carey et al., 1993; West et al., 1997). Falkin et al. (2007) examined the difficulties for adolescent to quit smoking highlighting this event as much more stressful for youth.
The Stress Process among African Americans

African American adults are exposed to daily events that affect their lives, such as lower incomes and racial inequalities (Tomaskovic-Devey, 1993), which can lead to adverse health outcomes, such as hypertension, coronary artery disease and depression (Brown et al., 2004).

One of these identified stressors is racism. In 1989, Armstead et al. examined the relationship between racism and blood pressure in a study by showing excerpts containing racist situations involving African Americans. Their analyses revealed that blood pressure significantly increased during the presentation of racist stimuli. After observing that African-American infants with teen mothers experience a survival advantage relative to infants whose mothers are older, Geronimus (1992) proposed the "weathering hypothesis" that the health of African-American women may begin to deteriorate in early adulthood as a physical consequence of social inequalities.” She (1996) went on to cite racism as a reason as why African Americans may be “weathering”.

Another group of researchers led by Fang et al. (2001) found that exposure to film excerpts that depicted racist situations resulted in elevated diastolic blood pressure. Race-based discrimination at work is associated with increased systolic and diastolic blood pressure. It also has been demonstrated to increase the likelihood of hypertension in African-Americans (Din Dzietham et al., 2004). Work done by Davis and colleagues (2005) studied the intensity of an adverse biopsychosocial stressor and the prevalence of hypertension. They conclude that the magnitude of stress derived from encounters was a
significant predictor of hypertension. The higher levels of psychosocial stressors in the African American community are also thought to contribute to the higher rates of sleep paralysis, an altered state of consciousness experienced when falling asleep or awakening (Paradis et al., 2005).

**Cotinine and Tobacco**

Tobacco research on smoking behavior has used cotinine as an indicator of “smoke constituent exposure”, which is highly specific and sensitive to tobacco use. Its level in blood, saliva, semen and urine correlates with passive and active smoking (Benowitz, 1982). In saliva, Bramer et al. (2003) suggest a breakpoint of 10 ng/ml between smokers and non-smokers while the regular active smoking threshold is above 100 ng/ml. Etter et al. (2000), on the other hand, suggest that the breakpoint smokers/non-smokers was located between 7 and 13 ng/ml.

The use of a biomarker is essential for quantifying human exposure to Environmental Tobacco Smoke (ETS) and for predicting potential health risks for exposed individuals. At present, cotinine-measured in blood, saliva, or urine-appears to be one of the most specific and the most sensitive biomarkers. It outlasts the presence of nicotine in the human body with a half-life of 14-20 hours (Buccafusco et al., 2007). Cotinine levels in nonsmokers are primarily the consequence from tobacco smoke under conditions of sustained exposure to ETS (i.e., over hours or days) (CDC, 2006). The exposure to other components of second-hand smoke can also be revealed by cotinine levels.
Nicotine intake is regulated by smokers in order to maintain body levels of nicotine (Benowitz et al., 1985). Nicotine is metabolized in the liver and around 80% of it is metabolized into cotinine (Figure 3). The metabolism of nicotine to cotinine varies with individuals (Benowitz et al., 1982). Cytochrome P450 2A6 (CYP2A6) is the hepatic enzyme that mediates approximately 90% of this reaction (Nakajima et al., 1996; Messina et al., 1997). Variable expressions of the cytochrome \textit{P4502A6 (CYP2A6)} gene are associated with individual cotinine differences. As part of the inactivation and the removal of nicotine from the body, it plays an important role in the C-oxidation of nicotine to generate cotinine. Approximately, 70 to 80% of the nicotine is metabolized into cotinine, and 90% of it is mediated by CYP2A6 (Malayandi et al., 2005). It therefore contributes to individual differences in the quantitative relationships between cotinine and nicotine intake. Nakajima et al (2000) showed that deficient cotinine formation after cigarette smoking is associated with deletion of CYP2A6 gene. Several studies have highlighted CYP2A6 function regarding cotinine: cotinine is both generated and metabolized by it (Bramer et al., 2003). Drugs that are substrate for CYP2A6 (Coumarin, Dicoumarol) result in diminished levels of cotinine while inhibitors like Methoxsalen and Pilocarpin have the opposite effect. Individuals who were heterozygous were described as having decreased CYP2A6 enzyme activity. Individual with CYP2A6*2 allele had a longer cotinine half-life than their counterparts (Sellers et al., 1998). The gene \textit{CYP2A6} is highly polymorphic and presents many variations in its DNA sequence. Xu et al. (2002) showed that \textit{CYP2A6} polymorphisms can influence the activity of the enzyme which can alter nicotine metabolism. Factors such as diet (Nakajima et al., 2006) and disease (Kirby et al., 1996) can modulate the CYP2A6 activity.
The term “race” has been shown to have limitations and is often viewed as playing a role in the acceptance of “race biology” in which specific genetic differences between races are accepted. Alternatively, the term “racially classified social group” (RCSG) was added to the public health lexicon as it refers to the social conception of race (King, 1997). This new term highlights the need for caution regarding the use of the “race” concept in public health research.

The association between RCSG and cotinine among smokers has been the subject of studies in past years (Caraballo et al., 1998; Perez-Stable et al., 1998; Ahijevych et al., 2002). Berlin et al. (2001) found that saliva cotinine is significantly higher in African Americans than in Caucasians for equal numbers of smoked cigarettes, suggesting that the clearance of nicotine may be slower in African Americans. These differences are thought to be the product of differences in metabolic variations among adult smokers (Caraballo et al., 1998; Sellers et al., 2000; Benowitz et al., 2002; Fujieda et al., 2004)). The recent work of Moolchan et al. (2006) on adolescents demonstrates a trans-3-hydroxycotinine (3HC)/ cotinine (COT) metabolite ratio lower in African American adolescents compared to the Caucasian adolescents. This finding could support the hypothesis that the presence of ethnic based differences in nicotine metabolism among adolescent smokers could be the result of the phenotypic expression of Cytochrome P450 2A6 (CYP2A6), the primary liver enzyme responsible for the conversion of nicotine in cotinine. CYP2A6 alleles vary among ethnicities. The frequency of specific alleles resulting in the reduced enzyme activity is more common among Chinese, Japanese, and Korean populations (Hsia et al., 2003; Spruitz-Metz et al., 2004). Differences in these alleles have direct consequences on health as an outcome.
The role of RCSG in smoking when using cotinine as a biomarker, although a complex one, can be seen from different perspectives: the biological aspect (i.e. presence of different alleles of one genes) or the cultural ones (i.e. higher number of a specific type of cigarettes). Ahijevych et al. in their 2002 work focused on the biological aspect, they compared women from two different ethnic groups in the United States: African Americans and their Caucasian counterparts. They concluded that African American menthol smoking was a significant predictor of cotinine half-life in comparison to Caucasian non-menthol half-life. Bramer et al. (2003) show that cotinine clearance and half-life is ethnically dependent. Significant differences were detected for clearance (total, renal and non-renal). Chinese Americans had 40% slower renal clearance than Caucasian, while their non-renal clearance was 30% faster (Bramer et al., 2003). As stated earlier, several Asian populations have higher concentration of variant alleles resulting in the reduced enzyme activity CYP2A6 (Hsia et al., 2003; Spruitz-Metz et al., 2004). Clearance rates, genetic variation, and cultural habits, which are all included in the factor of RCSG, are important factors that affect cotinine levels. Understanding the cotinine levels in their context is essential to solve health issues such as smoking related diseases and their risks.

Several factors have been identified as playing a role in cotinine individual differences. Gender has been demonstrated to partially explain the difference in cotinine levels (Etter et al. 2000). These gender differences may be due to difference in metabolism, sex-specific bias in self-reported smoking habits, and/or different ways to smoke (rate and depth of inhalation) (Perkins et al., 1996; Benowitz et al., 1984).
The variable “age of the smoker” has led to contradicting results with older smokers having higher cotinine levels than do younger smokers (Swan et al., 1993) and the opposite in Etter et al. (2000). Higher cotinine levels in subjects with lowest body mass index (BMI) have been observed (Jaakola et al., 2003). Jaakola approached the issue of nicotine, finding higher cotinine concentration increase per cigarette in groups that had a lower median level of cotinine. The authors suggested that smokers may regulate their smoking behaviors according to their nicotine concentration to achieve a certain serum nicotine level and consequently salivary cotinine level.
Social History of Skin Color

One of the most defining features in the historical presence of Africans in the Americas is the institution of slavery. This institution exploited and subjugated millions of Africans in the Americas in order to maximize profits on plantations. The existence of this human tragedy was justified by an ideology based primarily on skin color, but
included other discriminatory features such as hair texture, facial features, and morphology.

In the French Caribbean, legislators passed what is known as the “Code Noir” (Black Code) in 1685 (Tobner, 2007). This decree restricted the activity of free Africans and defined the conditions of slavery in the French colonial empire (Tobner, 2007). It later served as a model for the code drafted in Louisiana in 1725 and that spread in the American colonies (UNESCO, 2004). For two centuries, the “Code Noir” continued to be supported in the Americas by a Biblical theology called the Curse of Ham. The interpretation of this Biblical verse (Genesis 9:20-27) has been controversial. It described how Noah cursed the descendants of his son Ham with servitude due to a dishonorable act. The belief that individuals from African descent were descendants of Ham has been considered a primary justification for slavery among Christians (Haynes, 2002). As early as 1885, Haitian anthropologist Antenor Firmin contested the idea of any curse on Africans. In his book *Essay on the Inequality of Human Races*, he exposed the absurdity of the different racist theories claiming to demonstrate the superiority of some humans based solely on pigmentation.

The arrival of Europeans on the African continent, combined with the history of oppression, has led to the persistence of a deeply embedded racial paradigm that defines character, merit, and prestige through the distorted lens of skin color (Jordan 1962; Jordan, 1968; Hughes and Hertel, 1990). This racial paradigm was based on a supremacy ideology which states that individuals of African descent were innately inferior to Whites or those of European origins (Jefferson, 1853; Thompson, 1939; Frazier; 1957; Allport, 1958; Jordan, 1974; Ani, 1994; Hill, 2002). The history of this racialized context serves
as a basis for understanding the contemporary importance of phenotypic traits such as eye color, hair texture, and more importantly, skin color.

The study of the image of Blacks in the European psyche was one of the topics examined by Martinique born psychiatrist Frantz Fanon in his 1952 book “Black Skin, White Masks.” Fanon argued that years of colonization and cultural domination have left Black subjects with few choices but to internalize feeling of inferiority in a world under European domination. The Senegalese anthropologist Cheikh Anta Diop also challenged the White supremacist theories through his groundbreaking archeological works. Diop studied Ancient Egypt and argued that Egypt was part of the cultural and genetic context of Africa and not a separate entity (1954). In response to the White supremacist theory, Dr. Welsing proposed in her 1991 book “The Isis Papers: the Keys to Colors” the controversial psychoanalytic theory of “color confrontation.” This theory, which stipulates that color-deficient individuals have tried to conquer people of color out of a feeling of inadequacy, has been heavily criticized as promoting an overtly racist ideology.

**Skin Color in Americas and Africa**

After its independence was obtained in 1804 through a revolution led by General Toussaint l’Ouverture, the newly independent republic of Haiti saw the emergence of a new ethnic group: mulattoes (Davis, 1991). This group, benefiting from years of the in-between status (between the European slave owner and the African slave), quickly became the dominant elite both economically and politically after the French departure. According to Nicholls (1981), it is only since the 1960’s that this ethnic group lost some
of its political power during struggles with some of the members of the Noiriste (Black Nationalism) movement. Haiti continues to face political instability due to social divisions based on color distinctions (Dupuy, 2007). Other plural societies such as the Caribbean island of Jamaica, despite political independence and modernization, are still facing the persistence of racial, cultural, and color distinctions of the colonial order (Charles, 2003).

An analysis of the multicultural society of Brazil reveals a typology of racial classification based on skin pigmentation. This country’s population is the product of the mixture of, at least initially, three different ethnic groups: Amerindians, Europeans and Africans. In his 1964 experiment, Harris showed nine portraits to a hundred people. He found that more than forty terms for racial classification were used to describe the portraits. Despite having the largest number of individuals from African descent and an official doctrine of multi-culturalism, a European ethnocentric view still dominates Brazilian politics and business. The notable exceptions are the elections of Benita da Silva Sampaio as governor of the state of Rio de Janeiro in 2002 and Joaquim Benedito Barbosa Gomes at the Supreme Court in 2003. Telles, in his award-winning 2006 book Race in Another America: The significance of skin color in Brazil, concluded that inequalities in earnings and life expectancies still existed, and that most of them were at the disadvantage of dark skinned Brazilians of African descent.

On the African continent, colonial regimes often imported different groups that played what economists called the “middleman minorities”, whose sole purpose was to perform duties that the European majority could or did not want to do. These communities (such as Indians in Uganda and in Kenya) were distinct from the respective
indigenous groups not only culturally but also in skin tone (Davis, 1991). These communities faced severe reprisals during African independences. The Apartheid regime of the Republic of South Africa maintained drastic laws to perpetuate the European minority’s dominant position in society. This regime maintained racial categories that include two distinct buffer groups: Asian and Colored. Although the classification used by this regime has been shown to be highly inconsistent (Watson, 1970), the Apartheid system lasted until the election of Nelson Mandela as president in 1994.

**Skin Color as a Stigma**

The traumatizing experience of slavery in the Americas, as well as colonization and social domination, has been accompanied by the elevation of European values among Africans (Russell, 1992; Blay, 2007). The internalization of these Eurocentric values among Africans has led to several attempts to denature or modify two distinct yet important African racial features: hair and skin (Morrow, 1984). These two symbols of African ancestry were marked by White racism as stigma especially for women (Smeralda, 2004).

By the end of the 19th century, two African American women, Madam C.J. Walker and Mrs. Annie Turnbo, had developed several hair products conceived to alter hair texture and appearance, giving it a more acceptable look by European standards (Russell et al., 1992). In another attempt to alleviate the weight of discrimination, certain members of these non-European groups practiced skin bleaching which artificially
lightens the skin (Singham, 1968; Charles, 2003). Hall (1995) described skin bleaching as a universal phenomenon and as the result of cultural domination.

The practice of skin bleaching was particularly popular in the 1920s in the United States with the emergence of products such as Ro-Zol. It was followed by new skin-bleaching products such as Bel Dam ©, Fair and Handsome ©. Additionally, skin bleaching products resulted in $44 million worldwide in the year 1990 alone (Russell et al., 1992). Several of these products are primarily marketed to women to relieve acne scars and are sometimes referred to as “skin toners”. The use of these products has spread to continental Africa as well as in other parts of its Diaspora. In Senegal, skin bleaching termed “Xessal” is considered by women of some circles as a standard of beauty and elegance (Del Giudice and Yves, 2002).

Fanon (1952) referred to the existence of laboratories researching different creams that would allow individuals from African descent to “whiten” in order to avoid the “weight of the body curse”. Robinson (2004) in her research found that a number of Ghanaians are turning to skin-bleaching products to lighten their skin. Her survey of 600 residents of Accra found that 26.6% practice skin bleaching. It is estimated that about 10 to 15 % of the patient’s visits to dermatologists in the island of Jamaica are caused by the use of skin bleaching products (Charles, 2003). The practice of skin bleaching has often been associated with self-hate and low self-esteem. Notwithstanding, Charles did not find an association between self-hate and bleaching. He argued that personal identity and reference group orientation are not correlated suggesting a difference with respect to personal self-esteem and group identity.
Skin Color in the African American Context

In the United States, legislators of European descent applied the “one-drop rule”, a law stating that any person with even a drop of Black blood would have the same status as a “pure” African (Jordan, 1968; Davis, 1991; Russell et al., 1992). This law is also referred to as “any traceable amount rule”. In anthropological terms it is referred to as a “hypo-descent” rule meaning that racially mixed persons are assigned the status of the subordinate group (Harris, 1964).

During the slavery period, individuals of mixed race were more likely to be trained to do household jobs, rather than working in fields and manual labor, than were their darker peers (Mullins et al., 1984). Mulattoes even formed a class on their own in certain parts of the South (Williamson, 1980). During the pre-civil war era, lighter skinned blacks in the South were taller than darker skinned counterparts, which could suggest access to health resources (Bodenhorn, 2002). Mullins et al. (1984) have also shown that mulattoes were overrepresented in the elites of the African American community.

Legislation was passed in certain states to preserve the racial hierarchy (Davis, 1991). The colony of Virginia passed its first law in 1662 relegating its mulatto population to slave status (Davis, 1991). Despite the existence of privileged status for mulattoes in South Carolina and in Louisiana, anti-“miscegenation” rules spread throughout the states during the post Civil War era (Davis, 1991). The enforcement of this legislation was meant to limit the social mobility of individuals of African descent. In this regard, Jordan (1968) stated that the “color of Africans was useful as it was in itself
enough to put them in a distinct category.” Despite the abolition of slavery in the United States in 1865, inequalities in access to wealth persisted and were reinforced by the adoption of Black Codes in eight Southern States. These codes were quickly declared unconstitutional but left a legacy of racial discrimination (Davis, 1991).

Racial discrimination remained a problem in the USA and reinforced the color hierarchy developed during slavery, with additional privileges accruing to individuals with visible traits from European descent (Hunter, 2007).

The notion of the inferiority of dark skin was forcibly instilled in the African American community while segregation was enforced in American cities. In 1954, in the case of Brown vs. the Board of Education, the United States Supreme Court declared that public school segregation did not provide equal educational opportunities to Black children (Blaustein et al., 1957). During this trial, civil rights attorneys presented results from the 1947 Clark study. This doll experiment tested the hypothesis of self-hate. In this study, Black and White children were given the choice of a Black or White doll. The results indicating a preference of White dolls by Black children were perceived at that time as an indication of self-hate. These results were used to demonstrate the harm of internalized racism caused by stigmatization and segregation.

In 1957, E. Franklin Frazier (1957) argued in Black Bourgeoisie that the Black middle class was culturally rootless and beset by feelings of inferiority and self-hatred. He went on to argue that many members of this group were descendent of “mulattoes” (individuals with at least one Caucasian parent) and led a privileged existence when compared with their dark-skinned counterparts during slavery. Thus, a color gradient continued to internally stratify the African American community (Davis, 1991).
During the political upheavals of the 1960s, the Civil Rights movement strengthened the unity of the African American community. The Black Power movement encouraged pride in being black and having darker skin (Davis, 1991). This movement was accompanied by a cultural revival “Black is Beautiful” that aimed to restore African aesthetics. The movement encouraged individuals to wear Afro hairstyles and reject the use of products that alters skin color or hair texture (Van Derburg, 1993).

Studies of discrimination have documented how members of minority groups in the United States (namely African Americans and Hispanics) are still granted opportunities and rewards based solely on how their appearance approximates Eurocentric standards (Telles and Murguia, 1990; Arce et al., 1987; Murguia et al. 1996). In their 1991 work, sociologists Keith and Herring found that persistent discrimination against dark African-Americans resulted in continuing disadvantages in educational attainment, occupation and income.

**Conclusions**

Historically, populations in American society have been stratified by skin color via racism and systematic discrimination against individuals (King, 1997); the darker the skin, the greater likelihood of increased economic and social prejudice (Harburg et al., 1973; Keil et al., 1977). Consequently, an analysis of skin color and tobacco consumption as it relates to perceived stress entails sociological perspectives.

On average, African Americans with darker skin have been shown to be employed in lower incomes and less prestigious occupations when compared to African
Americans with lighter skin (Hughes et al., 1990; Keith et al., 1991; Hill et al., 2000). Hunter (2007) argued that in the case of African American women, possessing light skin is a form of social capital that can be converted into economic or educational capital.

Edwards (1973) noted that African Americans with darker complexion had a greater awareness of racial ethnic identification as well as greater awareness of racial discrimination and hostility. Discrimination has been linked to significant increases in blood pressure (Armstead et al., 1989; Fang et al., 2001). Studies examining the skin color gradient in blood pressure among African Americans have produced different findings. In 1978, Harburg et al. found that skin color combines with socially induced stress to induce higher blood pressures in lower class African Americans. Klag et al. (1991) demonstrated an association between skin color and blood pressure in low economic strata. They argued that it could be due to the psychosocial stress associated with darker skin color. However, Schwan et al. (1995) concluded that dark skin did not predict higher intraocular pressure in a sample of African Americans. Recently, Sweet et al. (2007) demonstrated that the protective gradient of income on systolic blood pressure seen among African Americans with lighter skin is not observed to the same degree among those with darker skin.

Studies indicate that although African Americans smoke fewer cigarettes than some other RCSGs, they have higher intake of nicotine per cigarette smoked (Benowitz, 1999; Caraballo et al., 1998, Moolchan et al., 2006; Wagenenknecht et al., 1990) (Benowitz, 1999; Caraballo et al., 1998; Moolchan, Franken, & Jaszyna-Gasior, 2006; Perez-Stable, Herrera, Jacob, & Benowitz, 1998; Wagenknetcht et al., 1990) and report greater difficulty quitting (King, Polednak, Bendel, Vilsaint, & Nahata, 2004). Using
smoking as a means of coping with stress and poor intrapersonal relationships has been more observed more among African American females (Mermelstein & The Tobacco Control Network Writing Group, 1999).

The presence of skin color-based stratification among African Americans (Franklin, 1994; Frazier, 1957; Jordan, 1968) suggests that smokers would be more likely to be of darker skin. Thus, skin color may represent an additional dimension by which health varies in the United States and, as such, should be understood in relation to perceived stress and tobacco consumption.
Chapter 3

Methodology

Study Sample

A criterion based non-probability design (Carter-Edwards et al., 2002) was used to select a pilot sample (N=149) of African Americans smokers from three selected sites in Harrisburg, PA (total population estimate: 47,164; U.S. Census Bureau, 2006). The city of Harrisburg was chosen due to the presence of a large number of Americans of African descent (54 %) (U.S. Census Bureau, 2006) and its proximity to State College, PA. Three different sites (The Harambee United Church of Christ, the Bethesda Men’s Shelter and the Children’s Check-Up Center) were selected for their convenience because of location and recruitment sites for African American smokers.

Study Sites

The Harambee Church of Christ (HC) is located in the northern section of Harrisburg (Census Tract 207), an area with 54.8% of African Americans, 71.8 % of the population are 25 years old or over (U.S. Census, 2000). This church is heavily involved in the surrounding African American communities and several social services besides spiritual guidance. These activities include tutoring programs for the youth, financial advice, and health programs (Harambee Church website, 2007).
The Bethesda Men’s Shelter (MS) is also located in the northern section of Harrisburg, on Reily Street. It is the only men’s shelter in Harrisburg and provides shelter and social services including free health care to its residents. The residents who seek refuge in this shelter come from the wider Harrisburg region and surrounding communities (Bethesda Men’s Shelter website, 2007).

The Children’s Check-Up Center is located in Hall Manor, Harrisburg. It provides healthcare to uninsured and underinsured individuals within the South Harrisburg area. The Hall Manor community (Census Tract 214) has 54.8% of African Americans, 14.3% of individuals with a bachelor degree or higher. The center is located in the middle of over 2,500 public housing residencies and facilitates the care of more than 3,500 Medicaid-eligible children who live within this area (The Children’s Check Up Center website, 2007).

The three recruitment different sites for this study were contacted six months prior to the data collection by the principal investigator (PI), Dr. Gary King. Several meetings were organized to present the study goals as well as its methodology. Letters were received from three sites indicating their desire to participate as locations and recruitment sites for the study.

Institutional Review Board

Upon approval by the Pennsylvania State University Office of Research Compliance (Institutional Review Board approval #24899), the data collection phase of the study of 149 African Americans smokers started June 23, 2007. A copy of the
informed consent form and the IRB approval document are provided in Appendices B and C.

Sample Size

The sample size was selected due to time and logistical constraints as well as project resources. Each participant was paid $20.00 for participating fully in the data collection process. Payment to participants helped to increase our sample size but also limited the number of individuals. It was apparent that more participants could have been recruited but we had depleted available funds. Payment to individuals for participating in the study also resulted in another problem: some individuals tried to participate in the study more than once. These individuals were recognized by members of the research team. However, one participant was allowed in order to avoid a public disturbance. His data was later removed from the dataset.

Study Recruitment

This research project targets an African American population of smokers. Several studies have highlighted the lack of willingness of African Americans to participate in medical research (Shavers et al., 2001). This attitude is often misunderstood but it is the direct consequence of having to experience scientific works such as the Tuskegee Syphilis Study. Other factors also influenced the recruitment in this specific community including: refusal to be “guinea pigs”; the high importance attributed to the race of the physician; and the perception that benefits of studies do not reach the African American
community (Corbie-Smith et al., 1999). Several strategies have been designed to improve African American recruitment in medical research. Some of the techniques have been:

- The use of reactive recruitment strategies such as the use of a hotline (Harris et al., 2003).
- A better understanding of the importance of interpersonal trust in the clinical relationship (Corbie-Smith et al., 1999; Shavers et al., 2002).
- Showing respect to the participants (Adderley-Kelly et al., 2005).
- Intending to “give back” to the community (Adderley-Kelly et al., 2005).

Respondents were recruited using different methods. During contacts with the three different sites, all agreed to advertise their participation in our study and help our recruitment effort (Figure 1 and Table 1).

- At the Harambee Church, an announcement was made during the Sunday service before our first visit and on the members’ listserv at the Church by Mrs. Robin Perry-Smith. The research team also left postings on the church activities board with time and date of the next study after each visit.
- At the Children’s Check-Up Center, flyers were placed by the personnel in the clinic (Mrs. Gretchen Ballard) in the hallway board the day before a study visit.
- At the Bethesda Men’s shelter, Mr. Bill Christian, an officer in charge at the shelter, made announcements to publicize the study and passed on a sign-up sheet for all willing participants before each of our visits.
At the first two sites (HC & CC), a large standing street sign and several posters were also placed in the surroundings of the study site about an hour before the start of the study. In addition, the research coordinator (Guy-Lucien Whembolua) with the help of the other research assistants (Emile Rochon, Maia Early, and Ajabeyang Amin) was active in street recruitment. It should also be noted that we received the help and guidelines from the PI on several days. The street recruitment included passing out flyers and a five minute exposé on the study where all questions regarding the project could be answered (See Table 3-1). The flyers (See Appendix D) included the time and location of the study as well the information that participants would receive a compensation of twenty dollars. The entire data collection process for each participant took around 45 minutes.

Visits were scheduled to the different sites according to site schedules, funds and research assistant availability. Each visit was planned to include an hour at the site for setup and recruitment and a half-hour for data quality control check as well as packing all items. Sixteen sessions were necessary to recruit the 149 participants. Double sessions (2) with visit on two separate sites on the same day were also organized (See Table 1). The PI and the research coordinator held debriefing sessions after each day. During these communications, the research coordinator communicated the number of participants as well as any problems regarding the data collection on that day.
Figure 3-1: Site sources of participants
Table 3-1: Schedule of Site Visits

<table>
<thead>
<tr>
<th>Date</th>
<th>Data Collection</th>
<th>Location</th>
<th># of Subjects</th>
<th>On-site Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>6/23/2007</td>
<td></td>
<td>Harambee Church</td>
<td>6</td>
<td>1 PM- 6:30 PM</td>
</tr>
<tr>
<td>6/30/2007</td>
<td></td>
<td>Men's Shelter</td>
<td>16</td>
<td>12 PM- 6:30 PM</td>
</tr>
<tr>
<td>7/2/2007</td>
<td></td>
<td>Check-Up Center</td>
<td>8</td>
<td>1 PM- 5 PM</td>
</tr>
<tr>
<td>7/5/2007</td>
<td></td>
<td>Check-Up Center</td>
<td>7</td>
<td>8 AM- 12:30 PM</td>
</tr>
<tr>
<td>7/5/2007</td>
<td></td>
<td>Men's Shelter</td>
<td>5</td>
<td>1 PM- 4 PM</td>
</tr>
<tr>
<td>7/12/2007</td>
<td></td>
<td>Check-Up Center</td>
<td>6</td>
<td>8 AM- 12:30 PM</td>
</tr>
<tr>
<td>7/12/2007</td>
<td></td>
<td>Men's Shelter</td>
<td>3</td>
<td>1 PM- 3 PM</td>
</tr>
<tr>
<td>7/19/2007</td>
<td></td>
<td>Check-Up Center</td>
<td>17</td>
<td>8 AM- 4 PM</td>
</tr>
<tr>
<td>7/27/2007</td>
<td></td>
<td>Check-Up Center</td>
<td>6</td>
<td>1 PM- 4:30 PM</td>
</tr>
<tr>
<td>8/2/2007</td>
<td></td>
<td>Harambee Church</td>
<td>4</td>
<td>8 AM- 11:30 AM</td>
</tr>
<tr>
<td>8/4/2007</td>
<td></td>
<td>Harambee Church</td>
<td>8</td>
<td>8 AM- 1 PM</td>
</tr>
<tr>
<td>8/7/2007</td>
<td></td>
<td>Harambee Church</td>
<td>14</td>
<td>1 PM- 5:30 PM</td>
</tr>
<tr>
<td>8/11/2007</td>
<td></td>
<td>Harambee Church</td>
<td>14</td>
<td>9 AM- 3:30 PM</td>
</tr>
<tr>
<td>8/18/2007</td>
<td></td>
<td>Harambee Church</td>
<td>10</td>
<td>9 AM- 3:30 PM</td>
</tr>
<tr>
<td>8/21/2007</td>
<td></td>
<td>Harambee Church</td>
<td>7</td>
<td>1 PM- 5:30 PM</td>
</tr>
<tr>
<td>8/23/2007</td>
<td></td>
<td>Harambee Church</td>
<td>18</td>
<td>1 PM- 5:30 PM</td>
</tr>
</tbody>
</table>

Total= 16 sessions  
Total= 149 subjects

¹ Double sessions
Protocol Procedure

After the subjects were recruited, a specific protocol was followed. This protocol was organized during the month of May. During that time, the PI, a research assistant (Mr. Emile Rochon) and the research coordinator met several times to discuss the most efficient ways for data collection. The PI directed that distinct phases should be followed in which specific tasks should be performed. The different phases were tested and timed in the lab and adjustments were made until the phases were deemed satisfactory. After the completion of each phase, participants were given a colored sticker on their hand. This technique gave a visual clue on the exact standing of the participant in our protocol. Each phase was located at a different station. Subjects moved from station to station in order to complete the phases. Variables from participants were entered on study logs (See Appendix E). All researchers wore safety gloves and followed universal protection practices during the entire data collection. Five distinct phases of data collection were established as follows:
Phase 1
- Preliminary Questions
  - age, 18+, smoker, Black or African American
  - Informed Consent
  - Carbon Monoxide Reading

Phase 2
- Demonstration
  - Saliva Sample Collection

Phase 3
- Reflectometer Reading

Phase 4
- Questionnaire
  - 30-40 min

Phase 5
- Closing:
  - Subject Signature
  - Payment ($20.00)
  - Sample Storage
  - Quality Control Checks

Figure 3-2: Protocol Diagram
Phase 1

Each participant was asked a series of screening questions to ensure their initial eligibility. All participants had to be current cigarette smokers, at least 18 years of age, and African American or Black. The specific eligibility questions were (See Appendix F):

- “Do you consider yourself to be African American or Black?”
- “Are you a smoker?”
- “Are you 18 years old or older?”

Exclusionary criteria had to be used due to the use of different instruments in the study. The use of a Carbon monoxide meter required the following exclusion criteria. Individuals who were either pregnant, breastfeeding or diagnosed with chronic obstructive lung disease or other pulmonary diseases were rejected (Chatkin et al., 2007). The use of saliva required the following exclusion criteria. Individuals who were presently using nicotine replacement therapies or medication affecting salivary functions were rejected (Binnie et al., 2004; Blackford et al., 2006; Rubinstein et al., 2007). The use of a reflectometer required the following exclusion criteria: individuals were presently using skin lighteners or who were pregnant (Park et al., 2005).

Those who met these study requirements were given copies of the informed consent form while the others were rejected (see Appendix G).

Each individual was required to read and sign two copies of the informed consent form prior to providing any data (see Appendix B). Participants were asked if they had any questions. Each of the questions was answered by the PI or the research coordinator. One copy of the questionnaire was given to the participant and the other is being
maintained by the project PI, Dr. King, in accordance with the Institutional Review Board guidelines.

In order to deter non-smokers from presenting themselves as smokers, participants were asked to expel their breath into a carbon monoxide reader. Subjects who met the smoking criterion of 10 parts per million (ppm) were allowed to continue with the study (Tonnesen et al., 1993; Jorenby et al.; 1995; Middleton et al., 2000). Those who did not were notified that they wereineligible. The carbon monoxide reader took several minutes to reset to zero which meant that some of the participants did not have to go through the carbon monoxide reader test. Individuals (25) who stated that they smoked a large number of cigarettes were chosen to skip the carbon monoxide reader test. A sub-analysis of the cotinine assays will inform the researchers on their validity as participants. This adjustment was necessary to keep the waiting line to a minimum.

**Phase 2**

After the completion of phase 1, subjects were given a glass of water to drink in order to clear any particles or residue. Subjects were then given a questionnaire on socio-demographic characteristics, sociological questions on skin color, stress and tobacco use to complete which took approximately 20-25 minutes (see Appendix H).

**Phase 3**

After completing all items of the questionnaire, participants were instructed on how to give saliva, they were then asked to expectorate 5cc into a conical saliva collection tube through a straw. Each participant was offered the opportunity to use
gloves, but the vast majority did not use them. This part of the study was performed behind a curtain to give privacy and raise the level of comfort of the participants. The expectoration of saliva took longer than expected for several of the participants (up to 20 minutes). Members of the research team encouraged the participants to be patient and to take all the time necessary to expectorate the required amount of saliva. The saliva sample was first placed in a small plastic bag and then placed in a cooler filled with refrigerant gel packs. It was then transported to Pennsylvania State University by road and placed in -20°C freezer. Each sample was assayed anonymously by a certified laboratory for cotinine concentration (See Appendix I).

**Phase 4**

Lastly, a battery operated reflectometer was used to measure skin color. Two separate measurements were recorded for each physical location (i.e., the forehead and the upper underarm) and an average M value was used in all the analyses for both measurements. The use of the reflectometer resulted in many questions by participants about the procedure and the overall study (See Appendices J and K).

The reflectometer used in this project is the Derma Spectrometer (Cortex Technology). It is an instrument that emits light at two defined wavelengths: 568 nm (green) and 655 nm (red) with a photo detector that measures the light reflected by the skin. It measures the absorbed and the reflected light at wavelengths for melanin. A melanin index is computed from the intensity of the absorbed and the reflected light at 568 nm and 655 nm. The calibration required the use of a black and white plate (Clarys et al., 2000). This handheld device has also been used in other research work regarding skin
color using the technique of measuring the unexposed skin of the upper arm and the exposed skin of the forehead (Shriver et al., 2000). The reflectometer use provides an objective quantification of skin color (Takiwaki et al., 2002). Despite its accuracy, Kittles and his colleagues (2004) emphasized the need to be cautious when using the pigmentation as a proxy of ancestry.

**Phase 5**

Participants were asked to sign a receipt and were then given $20 as compensation. Members of the research team then performed the first data quality control check which included:

- Comparison of the number of saliva samples to the number participants
- Comparison of the number of questionnaires collected to the number of participants.
- Comparison of the number of exclusionary criteria forms collected to the number of participants.
- Comparison of study logs by each research assistant to make sure that they were identical (ID numbers and variables).

The analysis of socio-demographic, behavioral and biomarker data were conducted using the Statistical Package for the Social Sciences.

This work constitutes a pilot study for further work done by Drs. Gary King, Valerie Yerger, Eric Moolchan, Robert Bendel and Rick Kittles.
**Hypotheses**

**Hypothesis 1:** African American smokers with higher perceived stress levels will have higher cotinine levels (See Figure 3-3).

![Diagram](image)

**Figure 3-3:** Hypothesis 1
Hypothesis 2: Self perception of skin color measurements or subjective measures will be a better predictor of perceived stress than more objective measures among African Americans smokers (See Figure 3-4).

Figure 3-4: Hypothesis 2
**Hypothesis 3:** Skin color-based discrimination questions will be a better predictor of perceived stress than objective measures skin color obtained with a reflectometer among African Americans smokers (See Figure 3-5).
1. Measures

To establish and describe the independent variables: frequency, distribution and simple descriptive statistics will be presented for each of the following characteristics:

- **Age of the participants**
- **Gender**
- **Socio-Economic Status (SES) measures:**
  - “A4. What was the highest grade or year of regular school or college that you completed?”
  - “A6. What is your current employment status?”
  - “A7. And just for statistical purposes, we need to know if your total family income per year is”
  - “A8. Do you currently rent or own your place of residence?”

- **Melanin Content**

  The melanin content will be presented using two different techniques:
  - Three measurements of average melanin content in the forehead as well as in the inner upper arm as measured by the reflectometer, procedure used by Kittles (1995) and Parra et al. (2004)
  - Self-scored skin color measurements.

- **Cotinine levels (means, sample range and standard deviations)**

  Tobacco research on smoking behavior has used cotinine as an indicator of smoke constituent exposure. It is highly specific and sensitive to tobacco use. Its level in blood, saliva, semen and urine correlates with passive and active smoking (Benowitz, 1982). In saliva, Bramer et al. (2003) suggest a breakpoint of 10 ng/ml between smokers and non-smokers while regular active smoking is above 100 ng/ml.
• **Perceived Stress:**

Participants’ levels of stress were measured using a version of the Perceived Stress Scale (PSS) (Cohen et al., 1983). This version consists of 14 items that ask how often in the last month the participants experienced symptoms of stress. Participants used a 5-point response scale (1 = never to 5 = very often). Higher score on the scale denote experiencing more stress. The total perceived stress will be presented as well as the Cronbach’s α. This scale has been used in several different populations with α > .80 in African American populations (Sellers et al., 2003; Manning et al., 2005); α = .67 in Spanish populations (Remor and Carrobles, 2001). Different version of the scale (14-items, 12-items, 10-items) will be tested for internal consistency. Preliminary analyses revealed that the PSS 10 provided the most reliable or valid measure of perceived stress in this sample.

• **Discrimination Questions:**

The respective questions for the four summated Likert response variables (D1, D2, D3, and D9) are:

- **D1.** During your lifetime, how often do you think that you have been treated unfairly or badly because you are African American or Black?
- **D2.** During your lifetime, while shopping, how often were you ignored as if you were not a serious customer?
- **D3.** During your lifetime, how many times have you been stared at as if you did not belong in a place or situation?
- **D9.** I often feel stressful because how I think people may react to my skin color.

The response categories for the three questions (D1, D2, D3) were “1: often”, “2: sometimes”, “3: seldom”, “4: never”. The response categories for the question (D9) were “1: strongly agree, 2: agree”, “3: disagree”, “4: strongly disagree”. The lower the cumulative score, the higher an individual may feel stressed.

The Cronbach’s alpha was found to be .775 which is considered acceptable (Kline, 1998). It should be noted that when the item deletion test was performed, deleting one item only increased the Cronbach’s Alpha marginally from .775 to .825. Thus, all four items were included in the stress scale.
2. **Normality**

The assumption of normality was tested for all the variables studied. This was done by drawing a visual representation of the distribution of scores (the normal curve). The measures of central tendency were calculated (mean, median, and mode). All variables were examined for extreme outliers (individuals falling more than three standard deviations from the mean) and skewed distributions (skewness statistic > |1|). Distributions were recoded if necessary. Specifics variables were examined to construct optimal groups for recategorization according to mean frequencies distributions.

Variables were entered based on their conceptual significance in the study. Missing data were reported and excluded from the data set. Categorical variables were recorded according to frequency distributions and the optimal way to analyze the data.

3. **Analysis**

   a. **Exploratory Analysis: Bivariate correlation**

   To control for the effects of potential confounds, a series of preliminary analyses were conducted to identify variables, such as participants characteristics (age, gender, SES measures) that might influence salivary cotinine and PSS scores. Independent sample t-tests or bivariate correlations were used to examine first the relationship between participants’ cotinine and potential confounds and secondly the PSS scores and potential confounding variables.
b. **Cross-tabulations and Analysis of Variance (ANOVA)**

**Hypothesis 1:** African American smokers with higher perceived stress levels will have higher cotinine levels.

Analysis of variance (ANOVA) was used to examine the relationship between PSS scores and salivary cotinine levels. Potential confounds including age, gender, SES measures, racial discrimination and color related issues were assessed.

**Hypothesis 2:** Self perception of skin color (subjective measure) will be a better predictor of perceived stress than either facultative or constitutive melanin (objective measure) among African American smokers.

Two independent variables self-scored skin color and reflectometer measures were examined in regard to perceived stress scale (the dependent variable). The two predictors (self-scored skin color and reflectometer measures) are interval ratio variables. The single dependent variable is interval ratio. After testing for the normal distribution of errors of prediction and the assumption of homeodasticity (similar variance in the dependent variable across the values of the predictor variables) regression models were developed (Cohen, 1987).
Hypothesis 3: Skin color-based discrimination questions will be a better predictor of perceived stress than objective measures of skin color obtained with a reflectometer among African American smokers.

Skin color-based discrimination questions and reflectometer measures were examined in regard to perceived stress scale (the dependent variable). One of the predictors (a reflectometer measure) is an interval ratio variable while the other (skin color-based discrimination questions) has been dummy-coded into an interval ratio variable. The single dependent variable is interval ratio. After testing for the normal distribution of errors of prediction and the assumption of homeodasticity (similar variance in the dependent variable across the values of the predictor variables) regression models were developed (Cohen, 1987).

c. Multivariate Analysis

The relationship between the dependent variable and the independent variables was modeled using a linear regression method. It was done to examine the primary relationship in the two hypotheses. First, the one between PSS scores and cotinine levels and secondly the one between skin color measures (subjective and objective) and PSS scores. This regression model was developed after testing for the normal distribution of errors of prediction, linearity between the interval ratio variables and the assumption of homeodasticity (Cohen, 1987). In addition to these assumptions for regression, the researcher examined the normality distribution for interval ratio variables. The skewness value for cotinine was 1.04; for small sample such as this one, Field (2000, p 41)
suggested that the guidelines should be increased to +/-2.5. Being very careful with the normality assumption, the researcher transformed the cotinine variable using a natural log transformation (Cohen, 1987).
Chapter 4

Results

Descriptive Statistics

The sample (N=147) was 61.6 % male and 38.4 % female. The mean age was 42.3 years ± (11.4) with 61.3 % of the respondents below 45 years old. Most participants (47.3%) were high school graduates (Table 4-1). Average reflectometer mean readings of constitutive and facultative melanin were 56.3 ±10.2 (range 34.6-80.7) nm, and 66.5, ±13.6 (range 31.7-103.9) nm respectively. Almost two-thirds of the respondents (65.5%) began smoking before 18 years of age ( x = 17 yrs, ±5.4). On average, participants had smoked for 25.2 ± 12.2 years, and over 90% consumed menthol cigarettes (data not shown). The mean number of cigarettes smoked per day (CPD) was 19 ± 10.6 (range 3-75), and the average cotinine level was 435 ng/mL ± (234) (range= 1273.38). The PSS10 mean score was 18.8 ± 6.0 (range 4-34).

Bivariate Correlations

Pearson correlations (Table 4-1) revealed that facultative melanin was positively and significantly related to CPD (r =.18, p<.05) and cotinine (r =.23, p<.05). Constitutive melanin scores were not significantly related to the dependent variables CPD (r =.07, p>.05) and cotinine (r =.13, p>.05). Age was positively and significantly related to cotinine (r =.25, p<.01) and women had significantly lower concentrations than men.
Cotinine level ($r = -.19$, $p<.05$) and CPD ($r = -.20$, $p<.05$) were significantly associated with age when started smoking. Cotinine level ($r = .33$, $p<.01$) and CPD ($r = .17$, $p<.05$) were also associated with smoking duration. Subjective measure of skin color were significantly associated with facultative melanin ($r = -.29$) and constitutive melanin ($r = -.30$).
Table 4-1: Frequencies Overall Means, and Pearson Correlations

<table>
<thead>
<tr>
<th>Variable</th>
<th>Sample Frequency</th>
<th>Cigarette Per Day</th>
<th>Cotinine Level (ng/mL)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td><strong>Constitutive Melanin Reading</strong></td>
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</tr>
<tr>
<td>34.6-50.6</td>
<td>33.3</td>
<td>18.1</td>
<td>393.3</td>
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<td>50.7-60.9</td>
<td>33.3</td>
<td>18.6</td>
<td>445.6</td>
</tr>
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<td>33.3</td>
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</tr>
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<td></td>
<td>$\bar{x}$ = 56.3, SD = 10.2</td>
<td>$r$ = 0.07</td>
<td>$r$ = 0.13</td>
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<tr>
<td><strong>Facultative Melanin Reading</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>31.7-58.6</td>
<td>33.3</td>
<td>17.2</td>
<td>351.8**</td>
</tr>
<tr>
<td>58.7-71.1</td>
<td>33.3</td>
<td>18.7</td>
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<td>33.3</td>
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<td></td>
<td>$\bar{x}$ = 66.5, SD = 13.6</td>
<td>$r$ = 0.18*</td>
<td>$r$ = 0.23**</td>
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<tr>
<td><strong>Age</strong></td>
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<td></td>
</tr>
<tr>
<td>18-30</td>
<td>12.7</td>
<td>18.3</td>
<td>297.9**</td>
</tr>
<tr>
<td>31-45</td>
<td>48.6</td>
<td>19.0</td>
<td>421.0</td>
</tr>
<tr>
<td>&gt;45</td>
<td>38.7</td>
<td>19.1</td>
<td>496.3</td>
</tr>
<tr>
<td></td>
<td>$\bar{x}$ = 42.3, SD = 11.4</td>
<td>$r$ = 0.10</td>
<td>$r$ = 0.25**</td>
</tr>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>61.6</td>
<td>18.7</td>
<td>484.0**</td>
</tr>
<tr>
<td>Female</td>
<td>38.4</td>
<td>19.6</td>
<td>357.4</td>
</tr>
<tr>
<td></td>
<td>$r_{ptbis}$ = -0.06</td>
<td>$r_{ptbis}$ = -0.25**</td>
<td>$r_{ptbis}$ = -0.04</td>
</tr>
<tr>
<td><strong>Education</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; High School</td>
<td>26.0</td>
<td>22.7*</td>
<td>442.8</td>
</tr>
<tr>
<td>High School Grad</td>
<td>47.3</td>
<td>17.1</td>
<td>423.9</td>
</tr>
<tr>
<td>Some College or Graduate</td>
<td>26.7</td>
<td>18.7</td>
<td>448.5</td>
</tr>
<tr>
<td></td>
<td>$\bar{x}$ = 17.0, SD = 5.4</td>
<td>$r$ = -0.20*</td>
<td>$r$ = -.19*</td>
</tr>
<tr>
<td><strong>Age of Smoking Initiation</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before 18 yrs.</td>
<td>65.5</td>
<td>20.6*</td>
<td>453.2</td>
</tr>
<tr>
<td>18 yrs. or Older</td>
<td>34.5</td>
<td>15.9</td>
<td>390.0</td>
</tr>
<tr>
<td></td>
<td>$\bar{x}$ = 17.0, SD = 5.4</td>
<td>$r$ = -0.20*</td>
<td>$r$ = -.19*</td>
</tr>
<tr>
<td><strong>Smoking Duration (yrs.)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-10</td>
<td>14.5</td>
<td>16.0**</td>
<td>292.7***</td>
</tr>
<tr>
<td>11-20</td>
<td>21.7</td>
<td>16.2</td>
<td>390.8</td>
</tr>
<tr>
<td>21-30</td>
<td>25.4</td>
<td>22.1</td>
<td>399.3</td>
</tr>
<tr>
<td>&gt;30</td>
<td>38.4</td>
<td>19.4</td>
<td>522.2</td>
</tr>
<tr>
<td></td>
<td>$\bar{x}$ = 25.2, SD = 12.2</td>
<td>$r$ = 0.17*</td>
<td>$r$ = 0.33***</td>
</tr>
<tr>
<td><strong>Perceived Stress Scale (10)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low (&lt; 15)</td>
<td>28.6</td>
<td>23.2</td>
<td>432.3</td>
</tr>
<tr>
<td>Medium (16-19)</td>
<td>26.6</td>
<td>18.0</td>
<td>447.6</td>
</tr>
<tr>
<td>Medium to High (20-22)</td>
<td>23.7</td>
<td>19.8</td>
<td>426.6</td>
</tr>
<tr>
<td>High (&gt; 23)</td>
<td>21.6</td>
<td>18.5</td>
<td>427.6</td>
</tr>
<tr>
<td></td>
<td>$\bar{x}$ =18.8, SD =6.0</td>
<td>$r$ = - 0.04</td>
<td>$r$ = - 0.05</td>
</tr>
</tbody>
</table>

$p <= .05$                        
$** p <= .01$                      
$*** p <= .001$                     
$r_{ptbis}$ = point biserial correlation
Hypothesis 1:

*American smokers with higher perceived stress levels will have higher cotinine levels.*

Facultative and constitutive melanin measurements were found to be strongly correlated \((r = .775, p \leq .01)\), therefore two separate analysis were performed to preclude collinearity.

**a. Facultative Melanin--Fully Saturated Regression Model**

Table 4-2 presents the backward regression results where cotinine level was regressed on five variables--facultative melanin, stress, gender, age, and smoking duration. In backward statistical regression analysis all the independent variables are entered and then removed one by one until only statistically significant variables remain in the most parsimonious model (Tabachnick and Fidell, 2007, pp. 138 - 144).

The fully saturated regression model was statistically significant \((F= 5.055, df=5/125, p < .001)\). Collectively, the five variables explained 16.8 % of the variance in cotinine levels.

In the fully saturated model, years of smoking (called smoking duration) was significantly associated \((\beta = .586, p = .001)\) with cotinine levels after statistically controlling for the other variables. A second variable, facultative melanin, was also significant \((\beta = .232, p = .011)\). In the fully saturated model neither stress, gender, nor age
were statistically significant at the .05 alpha level established a priori by the researcher as documented in Table 4-2. Model diagnostics appear in Appendices L and M.

b. Facultative Melanin - Most Parsimonious Regression Model

Based on the work of Hair, Anderson, Tatham and Black (1998, p. 24) the investigator developed a more parsimonious regression model. In developing a more parsimonious model, backward regression analysis was utilized for variable entry. The final model summarized in Table 4-2 represents the most parsimonious regression model. In the final model smoking duration ($\beta = .358, p = <.001$) and facultative melanin ($\beta = .230, p = .006$) were statistically significant in explaining the variance in cotinine. For both variables the pattern was similar. Greater years of smoking was associated with higher cotinine levels, and higher levels of facultative melanin was associated with higher cotinine levels. The final model was statistically significant ($F = 11.636, df = 2/128$) and explained 15.4% of the variance in cotinine level.

As explained earlier, PSS10 ($\beta = .022, p = .786$), gender ($\beta = .010, p = .918$), and age ($\beta = -.254, p = .149$) were not found to be significantly associated with cotinine levels. Facultative melanin and smoking duration were found to be significantly associated with cotinine levels.
Table 4-2. Summary of Fully Saturated and Most Parsimonious Regression Model Using Cotinine Levels as Dependent Variable

<table>
<thead>
<tr>
<th>Variables</th>
<th>Fully Saturated Model</th>
<th>Parsimonious Model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>β*</td>
<td>p-value</td>
</tr>
<tr>
<td>PSS10</td>
<td>0.000</td>
<td>0.999</td>
</tr>
<tr>
<td>Gender</td>
<td>-0.027</td>
<td>0.781</td>
</tr>
<tr>
<td>Age</td>
<td>-0.268</td>
<td>0.152</td>
</tr>
<tr>
<td>Smoking Duration</td>
<td>0.586</td>
<td>0.001</td>
</tr>
<tr>
<td>Facultative Melanin</td>
<td>0.232</td>
<td>0.011</td>
</tr>
</tbody>
</table>

* = standardized βeta

Mediation Assessment

The researcher hypothesized that the direct effect between facultative melanin and cotinine level was partially mediated by years of smoking. Using the guidelines for examining mediation formulated by Baron and Kenny (1986) and Tabachnick and Fidel (2007), a simple mediation analysis was conducted. The Sobel test for mediation was used and the results indicated statistical mediation existed (Sobel test statistic= 2.38, 1
tail $p = .009$, 2 tail $p = .017$). The issue then was to determine if there was complete (perfect) mediation or partial mediation (Tabachnick and Fidel (2007, pp. 159 - 160). The Baron and Kenny (1986) approach was used which involved using two regression equations. The results of those procedures appear in Figure 4-1.

![Figure 4-1. Results of Mediation Analysis](image)

The relationship between facultative melanin and cotinine level was found to be partially mediated by smoking duration ($R^2 = 15.4\%$).

c. **Constitutive Melanin - Fully Saturated Regression Model**

Table 4-3 presents the backward regression results where cotinine level was regressed on five variables--constitutive melanin, stress, gender, age, and smoking duration. This fully saturated regression model was statistically significant ($F = 4.244$, $df = 5/125$, $p < .001$). Collectively the five variables explained 16.8% of the variance in cotinine levels.

In the fully saturated model, years of smoking (called smoking duration) was significantly related ($\beta = .561$ $p = .002$) to cotinine levels when statistically controlling
for the other variables. A second variable, constitutive melanin, approached statistical significance or was marginally significant ($\beta = .153, p = .079$). In the fully saturated model none of the other variables were statistically significant at the .05 alpha level established a priori by the researcher. Model diagnostics appear in Appendices N and O.

d. Constitutive Melanin - Most Parsimonious Regression Model

The final model summarized in Table 4-3 represents the most parsimonious regression model. In the final model smoking duration ($\beta = .367, p < .001$) was statistically significant in explaining the variance in cotinine while facultative melanin ($\beta = .163, p = .06$) remained only marginally significant. Greater years of smoking were associated with higher cotinine levels and higher levels of constitutive melanin were associated with higher cotinine levels. The final model was statistically significant ($F = 11.636, df = 2/128$) and explained 12.7% of the variance in cotinine level.

PSS10 ($\beta = .022, p = .786$), gender ($\beta = .010 p = .918$), and age ($\beta = -.254, p = .149$) were not found not to be associated with cotinine levels.
### Table 4-3. Summary of Fully Saturated and Most Parsimonious Regression Model using Cotinine Levels as Dependent Variable

<table>
<thead>
<tr>
<th>Variables</th>
<th>Fully Saturated Model</th>
<th>Parsimonious Model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>β*  p-value</td>
<td>β*  p-value</td>
</tr>
<tr>
<td>n= 131</td>
<td></td>
<td>n= 131</td>
</tr>
<tr>
<td>PSS10</td>
<td>0.008 0.922</td>
<td>- -</td>
</tr>
<tr>
<td>Gender</td>
<td>-0.108 0.239</td>
<td>- -</td>
</tr>
<tr>
<td>Age</td>
<td>-0.261 0.168</td>
<td>- -</td>
</tr>
<tr>
<td>Constitutive melanin</td>
<td>0.153 0.079**</td>
<td>0.163 0.062**</td>
</tr>
<tr>
<td>Smoking Duration</td>
<td>0.561 0.002</td>
<td>0.367 &lt;.001</td>
</tr>
</tbody>
</table>

* = standardized βeta  
** = marginal significance  

**Mediation Assessment**

The researcher hypothesized that the direct effect between constitutive melanin and cotinine level was partially mediated by years of smoking. Using the guidelines for examining mediation formulated by Baron and Kinney (1986) and Tabachnick and Fidel (2007), a simple mediation analysis was conducted. The Sobel test for mediation was used and the results indicated statistical mediation did not exist (Sobel test statistic= 1.54, 1 tail p = .062, 2 tail p =.123).
Hypothesis 2:

Self-perception of skin color (subjective measurement) will be a better predictor of perceived stress than either facultative or constitutive melanin (objective measure) among African Americans smokers.

a. Facultative Melanin - Fully Saturated Regression Model

Table 4-4 presents the backward regression results where PSS10 was regressed on two variables--Self-perception of skin color and facultative melanin. This fully saturated regression model was not statistically significant (F=2.283, df=2/132, p=.106).

In the fully saturated model only self-perception of skin color was statistically significant ($\beta=-.192$ p = .035) with PSS10 when statistically controlling for the other variable. In the fully saturated model facultative melanin was not statistically significant at the .05 alpha level (Appendices P and Q).

b. Facultative Melanin - Most Parsimonious Regression Model

The final model summarized in Table 4-4 represents the most parsimonious regression model. In the final model self-perception of skin color ($\beta=-.169$, p = .050) was statistically significant in explaining the variance in PSS10 while facultative melanin ($\beta=-.073$, p=.421) remained non-significant. The lower score on the skin color self-perception scale (darker skin) were associated with higher levels of stress. The final
model was statistically significant (F= 3.923, df = 1/133) and explained 2.9% of the variance in Perceived Stress level.

Table 4-4 presents the regression results for the model that includes constitutive melanin, smoking duration, and perceived stress as predictors of cotinine levels.

As explained earlier, facultative melanin was not found not be associated with PSS10 levels while self-perception of skin color was found to be significantly associated with Perceived Stress levels.

Table 4-4. Summary of Fully Saturated and Most Parsimonious Regression Model using Perceived Stress Levels as Dependent Variable

<table>
<thead>
<tr>
<th>Variables</th>
<th>Fully Saturated Model</th>
<th></th>
<th>Parsimonious Model</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>β*</td>
<td>p-value</td>
<td>β*</td>
<td>p-value</td>
</tr>
<tr>
<td>Facultative Melanin</td>
<td>-0.073</td>
<td>0.421</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Self-perception of Skin Tone</td>
<td>-0.192</td>
<td>0.035</td>
<td>-0.169</td>
<td>0.50</td>
</tr>
</tbody>
</table>

*= standardized Beta

n= 135
n= 135
c. Constitutive Melanin - Fully Saturated Regression Model

Table 4-5 presents the backward regression results where PSS10 was regressed on two variables-- Self-perception of skin color and constitutive melanin. This fully saturated regression model was not statistically significant (F= 2.438, df= 2/132, p = .091).

In the fully saturated model only self-perception of skin color was statistically significant (β = -.197 p = .030) with PSS10 when statistically controlling for the other variable. In the fully saturated model constitutive melanin was not statistically significant at the .05 alpha level established a priori by the researcher (Appendices R and S).

d. Constitutive Melanin - Most Parsimonious Regression Model

The final model summarized in Table 4-5 represents the most parsimonious regression model. In the final model, self-perception of skin color (β =-.169, p = .050) was statistically significant in explaining the variance in PSS10 while constitutive melanin (β = -.088, p=.330) remained non-significant. The lower score on the skin color self-perception scale (darker skin) were associated with higher levels of stress. The final model was statistically significant (F= 3.923, df = 1/133) and explained 2.9% of the variance in Perceived Stress level.

Table 4-5 presents the regression results for the model that includes constitutive melanin, smoking duration, and perceived stress as predictors of cotinine levels.
As explained earlier, constitutive melanin was not found not be associated with PSS10 levels, while self-perception of skin color was found to be significantly associated with Perceived Stress levels.

**Table 4-5. Summary of Fully Saturated and Most Parsimonious Regression Model using Perceived Stress Levels as Dependent Variable**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Fully Saturated Model</th>
<th>Parsimonious Model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>β*</td>
<td>p-value</td>
</tr>
<tr>
<td>Constitutive Melanin</td>
<td>-0.088</td>
<td>0.330</td>
</tr>
<tr>
<td>Self-perception of</td>
<td>-0.197</td>
<td>0.030</td>
</tr>
<tr>
<td>Skin Tone</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* = standardized Beta
Hypothesis 3:

*Skin color-based discrimination questions will be a better predictor of perceived stress than more objective measures among African Americans smokers.*

**Table 4-6. Pearson Correlations of Subjective Measures of Discrimination with Perceived Stress Scale (PSS 10)**

<table>
<thead>
<tr>
<th></th>
<th>Treated Unfairly</th>
<th>Ignored</th>
<th>Being Stared at</th>
<th>Feel stressful</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pearson (r)</td>
<td>-0.28</td>
<td>-0.18</td>
<td>-0.28</td>
<td>-0.26</td>
</tr>
<tr>
<td>p-value (2-tail)</td>
<td><strong>0.00</strong></td>
<td>0.03</td>
<td><strong>0.00</strong></td>
<td><strong>0.00</strong></td>
</tr>
<tr>
<td>N</td>
<td>137</td>
<td>138</td>
<td>138</td>
<td>139</td>
</tr>
</tbody>
</table>

The correlations of all four question items were all found negative and statistically significant as it relates to Perceived Stress (PSS 10). This confirms that these variables may be added and used as it relates to PSS10. A new variable was then computed as the addition of all questions (NEWD). This new variable was entered in a regression with PSS10 as the dependent variable NEWD and Facultative (average Forehead) as independent variables.
a. Facultative Melanin- Fully Saturated Regression Model

Table 4-7 presents the backward regression results where PSS10 was regressed on two variables-- variable NEWD, facultative melanin. This fully saturated regression model was statistically significant (F= 7.915, df= 2/134, p = .0.001). Collectively the two variables explained 10.6 % of the variance in cotinine levels.

In the fully saturated model the new variable NEWD was statistically significant (β = -.325, p <= .001) with PSS10 when statistically controlling for the other variables. In the fully saturated model facultative melanin was not statistically significant at the .05 alpha level established a priori by the researcher (Appendices T and U).

b. Facultative Melanin- Most Parsimonious Regression Model

The final model summarized in Table 4-7 represents the most parsimonious regression model. In the final model the variable NEWD (β =-.321, p <= .001) was statistically significant in explaining the variance in PSS10 while facultative melanin (β = -.049, p=.554) remained non-significant. Lower scores on the combined four questions were associated with higher levels of stress. The final model was statistically significant (F= 15.552, df= 1/135) and explained 10.3% of the variance in Perceived Stress level.

Table 4-7 presents the regression results for the model that includes facultative melanin and perceived stress as predictors of PSS10 levels. Facultative melanin was not associated with PSS10 levels. The variable NEWD was found to be significantly associated with Perceived Stress levels.
Table 4-7. Summary of Fully Saturated and Most Parsimonious Regression Model using Perceived Stress Levels as Dependent Variable

<table>
<thead>
<tr>
<th>Variables</th>
<th>Fully Saturated Model</th>
<th></th>
<th>Parsimonious Model</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>β*</td>
<td>p-value</td>
<td>β*</td>
<td>p-value</td>
</tr>
<tr>
<td>Facultative Melanin</td>
<td>-0.049</td>
<td>0.554</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>NewD</td>
<td>-0.325</td>
<td>&lt;0.001</td>
<td>-0.321</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*= standardized Beta

c. Constitutive Melanin - Fully Saturated Regression Model

Table 4-8 presents the backward regression results where PSS10 was regressed on two variables-- variable NEWD, constitutive melanin. This fully saturated regression model was statistically significant (F= 7.765, df= 2/134, p = 0.001). Collectively the two variables explained 10.4 % of the variance in cotinine levels.

In the fully saturated model only NEWD was statistically significant (β = -.322, p <= .001) with PSS10 when statistically controlling for the other variable. In the fully saturated model constitutive melanin was not statistically significant at the .05 alpha level established a priori by the researcher (Appendices T and U).
d. Constitutive Melanin - Most Parsimonious Regression Model

The final model summarized in Table 4-8 represents the most parsimonious regression model. In the final model the variable NEWD ($\beta = -.321$, $p \leq .001$) was statistically significant in explaining the variance in PSS10 while constitutive melanin ($\beta = -.024$, $p = .773$) remained non significant. Lower scores on the combined four questions were associated with higher levels of stress. The final model was statistically significant ($F = 15.552$, $df = 1/135$) and explained 10.3% of the variance in Perceived Stress level.

Table 4-8 presents the regression results for the model that includes constitutive melanin and perceived stress as predictors of PSS10 levels. As explained earlier, constitutive melanin was not found to be associated with PSS10 levels. The variable NEWD was found to be significantly associated with Perceived Stress levels.
Table 4-8. Summary of Fully Saturated and Most Parsimonious Regression Model using Perceived Stress Levels as Dependent Variable

<table>
<thead>
<tr>
<th>Variables</th>
<th>Fully Saturated Model</th>
<th>Parsimonious Model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>β*</td>
<td>p-value</td>
</tr>
<tr>
<td>Constitutive Melanin</td>
<td>-0.024</td>
<td>0.773</td>
</tr>
<tr>
<td>NewD</td>
<td>-0.322</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*= standardized Beta
Chapter 5

Discussion, Summary, Limitations and Recommendations

The purpose of this research study was to examine the association between cotinine levels, perceived stress, and skin color among African American smokers. Using state-of-the-art approaches of behavioral surveys and biological testing, data were obtained from African Americans located in Harrisburg, Pennsylvania. This study included socio-demographic characteristics, attitudes toward RCSG and health, smoking behavior, questions about racial discrimination, and perceptions about skin color and stress.

This chapter presents the summary, discussion and implications of the findings and offers recommendations for future research.

Summary of Findings

The major findings of this study are:

- Perceived stress levels among African American smokers were not significantly associated with cotinine levels.
- Facultative melanin was found to be significantly and positively associated with cotinine level. This relationship was found to be partially mediated by years of smoking.
• Smoking duration was significantly associated with higher cotinine levels.
• Constitutive melanin was not found to be significantly associated with cotinine levels.
• Self-perception of skin color as measured with a scale was found to be significantly associated with perceived stress levels in comparison to either facultative melanin or constitutive melanin.
• The combination of four questions related to discrimination based on skin color was found to be a more significant predictor of perceived stress among African Americans than the use of an objective measurement or reflectometer.

Discussion

The present study’s intent was to examine the use of cotinine as a correlate to stress. The first question sought to identify a significant relationship between perceived stress levels in individuals and their respective cotinine level as a measure of tobacco use. As previously noted, African Americans have been shown to have higher cotinine than their Caucasian counterparts (Ahijevych & Gillespie, 1997; Ahijevych, Gillespie, Demirci, & Jagadeesh, 1996; Ahijevych & Parsley, 1999; Ahijevych, Tyndale, Dhatt, Weed, & Browning, 2002; Wagenknecht et al., 1990). Stress has also been shown to be linked to tobacco use (Lawton et al., 1962; Pomerleau et al., 1984).

Several studies have found an association between duration of smoking and smoking outcomes such as higher CPDs and cotinine (Al-Delaimy et al., 2002; Setty,
Curhan, & Choi, 2007). The results obtained in this work corroborated this positive correlation.

CPD was not a significant variable in the cotinine regression models, with either facultative or constitutive melanin, despite a CPD (\( \bar{x} = 19 \)) which was fairly high for African American smokers, even for African American smokers with low SES (USDHHS, 1998). The results obtained may be explained by the phenomenon that Heatherton (1989) described as ceiling effects for higher numbers of CPD on cotinine. When controlling for CPD, African American smokers have significantly higher serum levels of cotinine than do non-Hispanic White and Mexican American smokers (Caraballo et al., 1998; P. I. Clark, Gautam, & Gerson, 1996; Perez-Stable et al., 1998; Wagenknetcht et al., 1990). Cotinine level has been found to be directly proportional to the quantity of absorbed nicotine (Hill, Haley, & Wynder, 1983). However, CPD has not been shown to be consistently related to cotinine in some studies with African Americans (Kandel, Hu, Schaffran, Udry, & Benowitz, 2007; Mustonen, Spencer, Hoskinson, Sachs, & Garvey, 2005).

The relationship between facultative melanin and cotinine levels was shown to be strongly explained by the number of years of smoking which accounted for 15.4% of the variance. It should be noted that nicotine has been shown to accumulate in animal tissues containing melanin (Brittebo et al., 1980; Brittebo et al., 1981; Domellof et al., 1987; Iwata et al., 1981, Tjalve et al., 1983; Larsson et al., 1979). Increased melanin production and smoking have been suggested to play a role in the pathogenesis of certain pulmonary diseases such as cryptococcosis (Casadevall, Rosas, & Nosanchuk, 2000; Khan, 2006). The role of melanin in tissue uptake of nicotine could have potential implications for
individuals with high levels of melanin (Yerger, 2006). The relationship between facultative melanin and nicotine may not be explained completely through biological processes-- darker skin African Americans may smoke longer because of stress and discrimination.

The positive association between facultative melanin and nicotine dependence suggests several interpretations. It could suggest that because melanin has been found to bind with nicotine, then an individual with darker pigmentation may sequester more nicotine in the body than a lighter pigmented one. This storage of nicotine in melanin-containing tissues has yet to be explained in regard to nicotine clearance as well as in regard to the biomarker cotinine. Interestingly, Szuts et al. (1978) have found that cotinine does not appear to be sequestered in melanin-containing tissues.

At the present, no biological mechanisms have been proven to link nicotine consumption and melanin. Data collected in this study could be used to hypothesize that melanin-bound nicotine provides a reservoir that allows for slow egress of relatively small concentrations of unmetabolized nicotine to migrate from skin and other melanin-containing tissues through the bloodstream to brain nicotine receptors at concentrations that might not desensitize those nicotine receptors (King et al, unpublished). This would have the potential to lead to significant change in the understanding of nicotine and melanin. Darker skin individuals, according to this paradigm, could be predisposed to greater exposure to nicotine and tobacco-specific toxins, and susceptibility to tobacco-related carcinogens, especially if these toxicants are being slowly released from the reservoir of melanin-containing tissues (King et al, unpublished).
The use of the subjective scale of skin color was found to be significantly associated with perceived stress. This was not true in the case of facultative melanin or constitutive melanin. The combination of four questions related to discrimination based on skin color was found to be a more significant predictor of perceived stress than the use of an objective measurement collected with a reflectometer.

The results obtained supported the hypothesis that perceptions regarding skin color also affect health behaviors, and related outcomes (King, 1997; Williams, 1999). Pigmentation or skin color has been used in several studies regarding adverse health outcomes. An association of skin color with blood pressure was demonstrated in low socioeconomic strata and it also has been suggested to be a marker for hypertension and racial discrimination (Klag et al., 1991; Klonoff et al., 2000). The obtained results also highlight the importance of the psychological perspective of discrimination as opposed to a strictly biological perspective about RCSG. African Americans who experience racism and discrimination based on skin color may internalize certain beliefs creating psychological stress and cognitive dissonance, which could also affect health behaviors such as smoking (R. Clark, Anderson, Clark, & Williams, 1999; Klonoff & Landrine, 2000; Wyatt et al., 2003). Skin color based-discrimination and the stress variables were significant predictors in this study, these indicators have been found to be significant in other studies of tobacco use (Borrell et al., 2007; Fernander et al., 2005; Harrell, Hall, & Taliaferro, 2003; Landrine & Klonoff, 2000; Manning et al., 2005) and therefore further investigation is needed. It is also important to note that each skin-color based question was also found to be negatively associated with perceived stress level.
Study Limitations

This study had several limitations that must be addressed:

Sample:

The sample used in this study was small and limited only to African American smokers residing in Harrisburg. It includes a large portion of individuals residing in a men’s shelter (21.8%) which could have increased the proportion of highly stressed individuals in our sample. The study recruitment was performed in the impoverished areas of Harrisburg and our sample was representative of it, with a large proportion of unemployed participants (30.9%). Therefore, the findings may not be generalizable to all African American smokers. In addition, some of the findings were based on participants’ self-report of their smoking behaviors. A variable such as the facultative melanin reading is also influenced by the choice of the sample as it could vary if taken during different times of the year or in other regions of the country (Jablonski et al., 2000, King et al. unpublished). The presence of skin color stratification in the African American community (Frazier, 1957; Jordan et al., 1968; Goldsmith et al., 2006) may have precluded more variation in melanin readings (King et al. unpublished).

Preliminary questions:

The question regarding a diagnosis of a Chronic Obstructive Pulmonary Disease (COPD) was often misunderstood.
Questionnaires:

Several questions had to be explained to some of the participants. The questions “D10: How would you rate your skin tone?” had to be explained several times. Several participants perceived the question as provocative. The research assistant had to help clarify the meaning of these questions. Several participants seem to have literacy issues. The question regarding the brand of cigarette smoked was asked as an open-ended question which resulted in very general responses (e.g. “menthol”). The presence of two other questions “C12: What is the most usual brand of cigarettes you smoke?” and “C13: Are the cigarettes you smoke mentholated or plain?” were not enough to accurately identify each smoker’s cigarette of use.

The Study logs:

Categories should be added to the questionnaire in order to identify individuals that seem under the influence of any substances. This would allow our research to identify any possible interaction that the use of illegal substances could have on the cotinine results.

Length of study:

Not all the participants could be accommodated during the interval planned for the study. Due to time constraints, several participants did not blow in the CO meter due to the large amount of time needed to “recover” from previous use. This fact forced the investigators to rely on participants responses (N=25) to check their smoking status.
**Logistics and Ethical issues:**

During several of our visits to the Harambee Church, several staff members helped our team in the recruitment. Their presence helped deter the presence of problematic individuals. It leads me to believe that the presence of community leaders during the study is highly desirable and improve the efficiency of the study.

The study was more difficult to manage at the Check-up Center due to the lack of space and the presence of a few individuals who seem intoxicated.

As explained earlier, the number of CO meters was the biggest challenge. In the future, additional CO meters (2 or 3) could help accelerate the process.

**Recommendations**

- Considering the sample size and design, appropriate caution must be exercised in interpreting the results of this study. Future studies should include a more heterogeneous group of African American smokers in term of locations and income.

- The current study included only a small number of non-menthol smokers (n=13), which was insufficient to compare to menthol smokers. Despite mentholated cigarettes being the choice of the majority of African American smokers (Gardiner, 2004). Future studies including more non-menthol smokers are needed to assess its role in the relationship between cotinine and stress outcomes.
• The current work did not provide an analysis of trans-3-hydroxycotinine concentrations (King et al. unpublished). Further research could provide additional information on nicotine metabolism.

• Future studies on smoking in African American communities should include facultative melanin and constitutive melanin at different times of the year to identify any variations. This could be an important finding and exhortation for future research on smoking and melanin among African American adults.

• Future research on skin color and stress could include other American ethnic groups such as Latino or other individuals of color.

• Future studies on stress among African Americans should also include several questions regarding social support as it has been demonstrated to play a mitigating role regarding stress (Din-Dzietham, 2004).

Summary

The results obtained in this work may have particular import for African American smokers, who tend to have higher levels of melanin and are more prone to tobacco-related health consequences of dependence, morbidity and mortality (USDHHS, 1998; USDHHS, 2007). Furthermore, it provides a stronger basis for assessing any hypothesized biological association between nicotine and melanin by differentiating between the sociological significance and the biological nature of skin color with respect to tobacco use, dependence, and exposure.

The results obtained regarding skin based–discrimination questions and the self-perception of skin color could also help provide a better understanding of African
American health. These indicators have been found to be significant in other studies of
tobacco use (Borrell et al., 2007; Fernander et al., 2005; Harrell, Hall, & Taliaferro, 2003;
Landrine & Klonoff, 2000; Manning et al., 2005) and therefore further investigation is
needed.

The gap in the quality of health and health care across racial and socioeconomic
groups has been the subject of much scientific discourse (King, 1997; Turrell, 2000;
Alexander et al., 2003; McGruder et al., 2004; Bravata et al, 2005; Dunlop et al., 2007;
Song et al., 2007) and has helped expand the field of health disparities.

Disparities in health in general have been linked to different factors. One of
these factors is the presence of institutional racism in the medical field with the
occurrence of “racial bias” and inequities in medical care with skin pigmentation used as
a “proxy” of race and the main source of discrimination (Cooper, 1984; Cooper et al.,
1986; King, 1997, Parra et al., 2004; Smedley et al., 2003). The presence of a health
gradient following individual SES has been well documented regarding several diseases,
such as asthma and cancer, etc. (Chu et al., 2007; Shankardass et al., 2007) and SES
gradient is also observed in smoking itself as the increasing prevalence of smoking with
the decreasing SES (Royal College of Physicians, 2000).

Additionally, the health status of racial and ethnic minorities is still
disproportionately affected by tobacco use. In particular, African Americans have higher
rates of tobacco-related deaths from coronary heart disease, stroke and lung cancer.
References

Bethesda Men's Shelter. from http://www.bethesda-mission.org/


Harambee Church from http://www.harambeeucc.org/


Din-Dzietham, R., Nemhhard, W. N., Collins, R., & Davis, S. K. (2004). Perceived stress following race-based discrimination at work is associated with hypertension in


Harper, D. C., Theos, A. C., Herman, K. E., Tenza, D., Raposo, G., & Marks, M. S. (2007). Premelanosome amyloid-like fibrils are composed of only golgi-processed forms of pmel17 that have been proteolytically processed in endosomes. *J Biol Chem*.


Morris, P. (1982). *Recommended Product Sampling & Image Building Program for Benson & Hedges Cigarettes Among Black and Hispanic Consumers.* Richmond, VA.


the Coronary Artery Risk Development in (Young) Adults study. *Am J Public Health, 80*(9), 1053-1056.


APPENDIX A

Party Flyer

![Party Flyer Image]

Dj Lish & P.Y.T Ent
Present

Light Skin
Libra Birthday Bash

Hosted by
Randi a.k.a
Ms. P.Y.T / Ltc Ent

Friday Oct 12th, 2007
@ Club APT
1500 Woodward
Downtown, Detroit

Light Skinned Women &
ALL LIBRA's FREE ENTRY ALL NIGHT!
APPENDIX B

Consent Form

Informed Consent Form
The Pennsylvania State University

Project Title: Harrisburg Pilot Study of African American Smoking Behavior:
Investigating the Role of Melanin.

Investigator: Gary King, Ph.D.
Department of Biobehavioral Health
315 E. Henderson Bldg.
Pennsylvania State University
State College, PA 16802

Dr. King and colleagues are involved in a research project to study the relationship between tobacco use and melanin, which determines the differences in skin color. We invite you to take part in this research study. Taking part in this study is entirely voluntary and you may refuse to participate or withdraw from the study without any penalty or loss of benefits to which you are entitled. You are urged to ask any questions you have about this study with the staff members who will explain it to you. All participants must be African American, a current smoker, and 18 years old or older.

Purpose: Some African Americans have a good deal of difficulty quitting smoking and many African Americans develop smoking related diseases in greater proportion than other ethnic
groups. The objective of this project is to investigate the association of tobacco use among African Americans and melanin which determines differences in skin color. Melanin, which is related to skin color, may be related to why African Americans have higher rates of smoking related diseases (such as lung cancer) than some other groups.

The following tests or procedures are needed for the project.

1. **Carbon Monoxide Test**: To determine if you meet our requirements for being a cigarette smoker, we are going to ask you to breath into a machine to measure the amount of carbon monoxide in your body. Smokers have higher levels of carbon monoxide than non-smokers.

2. **Questionnaire**: As a part of your participation in this project you will be asked to complete a questionnaire that will inquire about your social background such as age, gender, education, and questions about attitudes toward smoking, smoking habits, and stress. Following these questions are a series of questions on basic physical traits such as skin color. Dr. King or someone in his staff will be happy to answer any questions that you may have about this the questionnaire. You may refuse to answer any question.

3. **Skin Color Measures**: You will then have your skin measured with a colorimeter, an instrument that shines a bright light on a surface and measures what is reflected. Skin color is measured in two places, on the inside of the upper arms and on the forehead.

4. **Saliva Samples**: In addition, we will need to collect a sample of saliva so that we can measure the amount of nicotine in your body.

**Duration**: Your entire participation should require about 45 minutes and you can only participate once.
**Risks/Discomforts:** For some people, giving information about skin color may be difficult. Also, some people may be slightly embarrassed to show parts of their upper arms, which is necessary to obtain accurate skin color measurements.

**Sample collection/storage:** The principal investigator will arrange to have your saliva samples stored and analyzed by a certified laboratory. Your name will not be part of your saliva sample and we are only testing the sample to measure nicotine content.

**Right to Ask Questions:** If you have any questions about this research, please contact **Dr. Gary King at (814) 863-8184.** You can also call this number if you feel that you have been harmed by this study. If you have questions about your rights or complaints after completing the survey, contact the Penn State University’s Office for Research Protections at (814) 865-1775.

**Benefits:** There is no direct benefit to you from participating in this study. The benefits of this study will be is to increase our understanding of the relationship between melanin and smoking among African Americans.

**Compensation:** You will be paid by researchers $20 for your participation.

**Confidentiality:** Your participation in this research is confidential. This means that no one will know your answers. The principal investigator and research assistants will have access to the records. The Office of Human Research Protections in the U.S. Department of Health and Human Services, the U.S. the Office for Research Protections at Penn State and the Biomedical Institutional Review Board may review records related to this project.
**Consent Document:** It is suggested that you retain a copy of this document for your later reference and personal records.

**PLEASE COMPLETE ITEM BELOW:**

I have read the explanation about this study and have been given the opportunity to talk about it and ask questions. I hereby consent to take part in this study.

____________________________________________________
Participant’s Signature      Date

____________________________________________________
Investigator’s Signature     Date
APPENDIX C

Institutional Review Board Approval Letter

Date: June 21, 2007

From: Mary B. Becker, IRB Administrator

To: Gary King

Subject: Results of Review of Proposal - Full (IRB #24899)

Approval Expiration Date: February 14, 2008

“Harrisburg Pilot Study of African American Smoking Behavior:
Investigating the Role of Melanin”

The Biomedical Institutional Review Board (IRB) has reviewed and approved your proposal for use of human participants in your research. By accepting this decision, you agree to obtain prior approval from the IRB for any changes to your study. Unanticipated participant events that are encountered during the conduct of this research must be reported in a timely fashion.

Attached is/are the dated, IRB-approved informed consent(s) to be used when recruiting participants for this research. Participants must receive a copy of the approved informed consent form to keep for their records.
If signed consent is obtained, the principal investigator is expected to maintain the original signed consent forms along with the IRB research records for this research at least three (3) years after termination of IRB approval. For projects that involve protected health information (PHI) and are regulated by HIPAA, records are to be maintained for six (6) years. The principal investigator must determine and adhere to additional requirements established by the FDA and any outside sponsors.

If this study will extend beyond the above noted approval expiration date, the principal investigator must submit a completed Continuing Progress Report to the Office for Research Protections (ORP) to request renewed approval for this research.

On behalf of the IRB and the University, thank you for your efforts to conduct your research in compliance with the federal regulations that have been established for the protection of human participants.

MBB/mbb

Attachment

cc: Guy-Lucien S. Whembolua

Please Note: The ORP encourages you to subscribe to the ORP listserv for protocol and research-related information. Send a blank email to: L-ORP-Research-L-subscribe-request@lists.psu.edu
Individuals Wanted for Research on Cigarette Smoking

Must be African American
At least 18 years of age
Current Smoker Only

Smoking status will be verified
12 participants only

Principal Investigator: Professor Gary King
Department of Biobehavioral Health
Penn State University
814-865-8410

Individuals will be paid $20 for their full participation.

Thursday August 23, 2007
Place: Harambee Church
Time: 2 pm – 5 pm
## APPENDIX E

### Study Log

<table>
<thead>
<tr>
<th>Subject</th>
<th>Vial Number</th>
<th>CO (ppm)</th>
<th>Date</th>
<th>Time</th>
<th>Last time smoked</th>
<th>Reflectometer Site</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F 1</td>
<td></td>
<td></td>
<td></td>
<td>F 2</td>
<td>Forehead (F)</td>
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<td>F 3</td>
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<td>Underarm (U)</td>
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</tbody>
</table>

APPENDIX F

Exclusion Criteria

Harrisburg Pilot Study on Melanin and Cigarette Smoking

1. Do you consider yourself to be African American or black?
   a) Yes
   b) No

   *If no, please do not continue.*

2. Are you a smoker?
   a) Yes
   b) No

   *If no, please do not continue.*

3. Are you 18 years old or older?
a) Yes  
b) No  

**If YES, continue; if NO, please do not continue any further.**

*On average, how many cigarettes do you smoke?*
Exclusionary Criteria

1. Are you presently using any form of Nicotine Replacement Therapy (NRT) such as the nicotine patch, nicotine gum, spray to help you quit smoking? YES NO

2. Are you taking any medication which affects your ability to produce saliva? YES NO

3. Are you pregnant? YES NO

4. Are you currently breast-feeding? YES NO

5. Have you ever been diagnosed with chronic obstructive lung disease or any other pulmonary disease? YES NO

6. Are you presently using any skin lighteners?
APPENDIX G

List of Rejected Individuals

07/02/07       3 rejected
07/05/07       3 rejected
07/12/07       5 rejected
07/19/07       2 rejected previously participated
07/27/07       1 rejected
08/07/07       2 rejected
08/11/07       2 rejected
08/21/07       2 rejected
08/23/07       4 rejected

TOTAL OR INDIVIDUALS REJECTED: 24
The following questions deal with your thoughts about yourself, cigarette smoking, skin color, racism, and stress. All your answers will be confidential and will be used only for research purposes. Your cooperation is voluntary, and you can refuse to answer any specific question.

Please circle your answers.
DEMOGRAPHICS (1) - Part A

A1. Were you born in the U.S.?
   a) Yes
   b) No

A1b. If no, in what country were you born?

A1c. How long have you been a resident of Harrisburg?

A2. Would you say that in general your health is:
   a) Excellent
   b) Good
   c) Fair
   d) Poor

A3. Are you a male or a female?
   a) male
   b) female

A4. What was the highest grade or year of regular school or college that you completed?
   a) Less than 9 years
   b) Between 9-11 years
   c) Obtained 4 year high school diploma/GED
   d) 1-4 years of college
   e) Obtained college BS. or BA degree
   f) Obtained Master or Ph.D. degree
A5. Please, tell us what your present age is.

_________

A6. What is your current employment status?

a) full-time
b) part-time
c) going to school
d) keeping house
e) unemployed
f) disabled
g) retired

A7. And just for statistical purposes, we need to know if your total family income per year is:

1. $30,000 and below  2. above $30,000
A8. Do you currently rent or own your place of residence?

1. Rent  2. Own  3. Bethesda men’s shelter

A9. What is your current marital status?

a) single, never married  
b) single, living with partner  
c) married  
d) divorced or separated  
e) widow/widower

COMMUNITY- Part B

B1. Do you think that cigarette smoking among African Americans is:

1. increasing  2. has remained the same  3. decreasing
B2. Thinking of your closest friends, about how many would you say are smokers?

a) none
b) a few
c) about half
d) most of them
e) almost all of them

B3. Do you personally think smoking is a physical addiction?

1. Yes  2. No

B4. Do you believe that Blacks who smoke cigarettes are more likely to get lung cancer or other smoking related diseases than Whites who smoke?

1. Yes  2. No

B5. Do you think it is more difficult, biologically, for Blacks to quit smoking as compared to Whites?

1. Yes  2. No
B6. How much of the smoking problem among African Americans do you think is related to the stressful effects of racism in American society?

1. None of it  
2. A little bit  
3. Quite a bit  
4. Very Much
SMOKING- Part C

C1. Have you smoked at least 100 cigarettes in your entire life? Or about five packs?

1. Yes    2. No

C2. How often do you smoke?

a) Every day  
b) Some days  
c) Not at all
C3. On those days that you smoke, on average, about how many cigarettes do you smoke? (please, write the exact number)

C4. What is the highest number of cigarette that you have smoked on an average day?

C5. How soon after you wake up do you smoke your first cigarette?

   a) After 60 minutes
   b) 31-60 minutes
   c) 6-30 minutes
   d) Within 5 minutes

C6. Which cigarette would you hate most to give up?

   a) The first in the morning
   b) Any other
C7. Do you smoke more frequently during the first hours after awakening than during
the
rest of the day?

1. Yes 2. No

C8. Do you smoke even if you are so ill that you are in bed most of the day?

1. Yes 2. No

C9. Do you find it difficult to refrain from smoking in places where it is forbidden (like
churches or theaters)?

1. Yes 2. No

C10. When you think about it, were you smoking this time last year?

1. Yes 2. No

C11. About how old were you when you first started smoking daily?
C12. Is your most usual brand of cigarette…

a) ULTRA-LIGHT  
b) LIGHT  
c) REGULAR  
d) DON’T KNOW

C13. Is your brand Menthol or plain?

a) Menthol  
b) Plain  
c) Don’t know

C14. What is the full name of the cigarette brand that you smoke most often?

C15. Thinking about menthol versus non-menthol cigarettes and the effect they could have on your health, do you think smoking menthol cigarettes is better for your health, worse for your health, or has no greater effect on your health than non-menthol cigarettes?

a) Better for my health  
b) Worse for my health  
c) No greater effect on my health than non-menthol cigarettes
C16. Have you ever made a serious attempt to stop smoking cigarettes entirely?

1. Yes    2. No

C17. Have you ever used any of the following items to quit smoking?

Nicotine patch   Yes   No
Nicotine gum      Yes   No
Prescriptions for Zyban/Wellbutrin/Buproprion Yes   No
Nasal spray      Yes   No
Nicotine inhaler Yes   No
Any other methods _____________________

C18. During the past 12 months, have you quit smoking for one day or longer, because you were trying to stop smoking?
1. Yes  2. No

If yes, about how many days did you quit?

C19. What is the possibility that you will be smoking one year from now? Do you think that you will:

1. Definitely be smoking  2. Probably be smoking  3. Probably not be smoking
4. Definitely not be smoking one year from now

C20. Are you getting pressure from people you know to stop smoking?

   a) No, not all  
   b) Yes, a little
   c) Yes, somewhat
   d) Yes, very much

C21. What do you think might stop you from smoking?

__________________________

C22. Do you allow smoking in your home?

C23. Do you allow smoking in your car?

1. Yes  2. Sometimes  3. Never  4. I don’t have a car

D. PERCEIVED RACISM

For the next three questions, think about your experiences over your lifetime. Tell me about how many times these events have occurred.

D1. During your lifetime, how often do you think that you have been treated unfairly or badly because you are African American or black? Would you say,


D2. During your lifetime, while shopping at a store or when attempting to make a purchase, how often were you ignored as if you were not a serious customer or were followed by store personnel? Would you say,

D3. During your lifetime, how many times have you been stared at as if you did not belong in a place or situation? Would you say,


For the following statements about skin color, please tell me how much you agree or disagree. Do you strongly agree, agree, disagree or strongly disagree.

D4. Whites are influenced more by the color of my skin than blacks.


D5. If my skin color was lighter, I feel that I would have more opportunities in life.


D6. I am very proud of the color of my skin.


D7. I am more comfortable around people who are about the same skin color as I.

D8. Both Blacks and Whites sometimes have problems relating to dark skin African Americans.


D9. I often feel stressful because how I think people may react to my skin color.


D10. On a skin color scale of 1 to 10, where very dark skin color would represent the number 1 (say, Wesley Snipes, Bernie Mac), 5 would be in the middle range (say, Denzel Washington, Janet Jackson), and 10 would be very light skin color (say, Prince, Lena Horne), what number would best represent your skin color?

1 2 3 4 5 6 7 8 9 10
E. PERCEIVED STRESS

The questions in this scale ask you about your feelings and thoughts during the last month. In each case, you will be asked to indicate how often you felt or thought a certain way. Although some of the questions are similar, there are differences between them and you should treat each one as a separate question.

E1. In the last month, how often have you been upset because of something that happened unexpectedly?

<table>
<thead>
<tr>
<th>Never</th>
<th>Almost never</th>
<th>Sometimes</th>
<th>Fairly often</th>
<th>Very often</th>
</tr>
</thead>
</table>

E2. In the last month, how often have you felt that you were unable to control the important things in your life?

<table>
<thead>
<tr>
<th>Never</th>
<th>Almost never</th>
<th>Sometimes</th>
<th>Fairly often</th>
<th>Very often</th>
</tr>
</thead>
</table>
**E3. In the last month, how often have you felt nervous and stressed?**

<table>
<thead>
<tr>
<th>Never</th>
<th>Almost never</th>
<th>Sometimes</th>
<th>Fairly often</th>
<th>Very often</th>
</tr>
</thead>
</table>

**E4. In the last month, how often have you felt successful dealing with irritating life hassles?**

<table>
<thead>
<tr>
<th>Never</th>
<th>Almost never</th>
<th>Sometimes</th>
<th>Fairly often</th>
<th>Very often</th>
</tr>
</thead>
</table>

**E5. In the last month, how often have you felt that you were effectively coping with important changes that were occurring in your life?**

<table>
<thead>
<tr>
<th>Never</th>
<th>Almost never</th>
<th>Sometimes</th>
<th>Fairly often</th>
<th>Very often</th>
</tr>
</thead>
</table>

**E6. In the last month, how often have you felt confident about your ability to handle your personal problems?**

<table>
<thead>
<tr>
<th>Never</th>
<th>Almost never</th>
<th>Sometimes</th>
<th>Fairly often</th>
<th>Very often</th>
</tr>
</thead>
</table>
E7. *In the last month, how often have you felt that things were going your way?*

<table>
<thead>
<tr>
<th></th>
<th>Never</th>
<th>Almost never</th>
<th>Sometimes</th>
<th>Fairly often</th>
<th>Very often</th>
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</thead>
</table>

E8. *In the last month, how often have you found that you could not cope with all the things you had to do?*

<table>
<thead>
<tr>
<th></th>
<th>Never</th>
<th>Almost never</th>
<th>Sometimes</th>
<th>Fairly often</th>
<th>Very often</th>
</tr>
</thead>
</table>

E9. *In the last month, how often have you been able to control irritations in your life?*

<table>
<thead>
<tr>
<th></th>
<th>Never</th>
<th>Almost never</th>
<th>Sometimes</th>
<th>Fairly often</th>
<th>Very often</th>
</tr>
</thead>
</table>

E10. *In the last month, how often have you felt that you were on top of things?*

<table>
<thead>
<tr>
<th></th>
<th>Never</th>
<th>Almost never</th>
<th>Sometimes</th>
<th>Fairly often</th>
<th>Very often</th>
</tr>
</thead>
</table>
E11. *In the last month, how often have you been angered because of things that happened that were outside of your control?*

<table>
<thead>
<tr>
<th>Never</th>
<th>Almost never</th>
<th>Sometimes</th>
<th>Fairly often</th>
<th>Very often</th>
</tr>
</thead>
</table>

E12. *In the last month, how often have you found yourself thinking about things that you have to accomplish?*

<table>
<thead>
<tr>
<th>Never</th>
<th>Almost never</th>
<th>Sometimes</th>
<th>Fairly often</th>
<th>Very often</th>
</tr>
</thead>
</table>

E13. *In the last month, how often have you been able to control the way you spend your time?*

<table>
<thead>
<tr>
<th>Never</th>
<th>Almost never</th>
<th>Sometimes</th>
<th>Fairly often</th>
<th>Very often</th>
</tr>
</thead>
</table>

E14. *In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?*

<table>
<thead>
<tr>
<th>Never</th>
<th>Almost never</th>
<th>Sometimes</th>
<th>Fairly often</th>
<th>Very often</th>
</tr>
</thead>
</table>

*Thank you very much for your cooperation. Please contact the research assistant, if you are finished.*
APPENDIX I

Cotinine Assay

Salivary cotinine.

Salivary levels of cotinine were determined by enzyme immunoassay using commercially available kits (Salimetrics LLC, State College, PA). These assays were conducted at the Penn State University General Clinical Research Center. All samples were tested in duplicate in a single assay batch. Duplicate test values that varied by more than 6% error were subject to repeat testing. The average of the duplicate tests is reported. Intra-assay variation (CV) computed for the mean of 30 replicate tests was less than 5.8%. Inter-assay variation computed for the mean of average duplicates for 12 separate runs was less than 8.2%. The assay sensitivity, based on the minimum cotinine concentration required to produce a two standard deviation from assay A0 is 0.05ng/ml.

Acknowledgement for using GCRC

The Penn State General Clinical Research Center Cytokine Core Laboratory at the Noll Physiological Research Laboratory (NIH Grant M01-RR-10732) provided support for the salivary hormone assays.
APPENDIX J

Questions and Comments regarding the Reflectometer

- “What is this for?”
- “I know what that is, it’s a reflectometer!”
- “Can I see these numbers, please?”
- “Am I going to feel anything?”
- “Is it dangerous?”
- “Is it going to cause me headaches?”
- “What are the meanings of these numbers?”
- “What do these numbers tell you?”
- “Do these numbers change if I stay in the sun all day?”
- “Am I too dark?”
- “Does dark skin protects me?”
- “What does that have to do with smoking?”
APPENDIX K

Questions and comments regarding the Study

- “How long is it going to take?”
- “Can I participate several times?”
- “Do I have to put my names on the form?”
- “Can I get the results of the study?”
- “Is it going to tell me if I am going to have lung cancer?”
- “What do these numbers means?” (regarding CO meter values)
- “Why do you only focus on African Americans?”
- “Do you do this study anywhere else?”
- “Are you going to tell me how to stop smoking?”
- “Are you a doctor?”
- “Are you African American?”
- “Where are you from?”
- “Who is supervising this study?”
- “Is it part of a larger study?”
APPENDIX L

Facultative Melanin- Regression plot I

Normal P-P Plot of Regression Standardized Residual

Dependent Variable: LNCOTIN

APPENDIX M

Facultative Melanin- Scatterplot I

Scatterplot

Dependent Variable: LNCOTIN
APPENDIX N
Constitutive Melanin - Regression plot I

Normal P-P Plot of Regression Standardized Residual

Dependent Variable: LNCOTIN

APPENDIX O
Constitutive Melanin - Scatterplot I

Scatterplot

Dependent Variable: LNCOTIN
APPENDIX P

Facultative Melanin- Regression plot II

Normal P-P Plot of Regression Standardized Residual

Dependent Variable: PSS 10: P6+ P7+P9+P10+ E1+E2+E3+E8+ E11+ E14

APPENDIX Q

Facultative Melanin- Scatterplot II

Scatterplot

Dependent Variable: PSS 10: P6+ P7+P9+P10+ E1+E2+E3+E8+ E11+ E14
APPENDIX R

Constitutive Melanin- Regression plot II

Normal P-P Plot of Regression Standardized Residual

Dependent Variable: PSS 10: P6+ P7+P9+P10+ E1+E2+E3+E8+ E11+ E14

APPENDIX S

Constitutive Melanin- Scatterplot II

Scatterplot

Dependent Variable: PSS 10: P6+ P7+P9+P10+ E1+E2+E3+E8+ E11+ E14
APPENDIX T
Facultative Melanin- Regression plot III

Normal P-P Plot of Regression Standardized Residual

APPENDIX U
Facultative Melanin- Scatterplot III
APPENDIX V

Constitutive Melanin- Regression plot III

Normal P-P Plot of Regression Standardized Residual

Dependent Variable: PSS 10: P6+ P7+P9+P10+ E1+E2+E3+E8+ E11+ E14

APPENDIX W

Constitutive Melanin- Scatterplot III

Scatterplot

Dependent Variable: PSS 10: P6+ P7+P9+P10+ E1+E2+E3+E8+ E11+ E14
VITA

Guy-Lucien S. Whembolua
200 Highland Avenue, Apt 101
State College, Pennsylvania 16801
Phone (814) 861-8779
Email: whembolua@psu.edu

EDUCATION

2008   Ph.D., Department of Biobehavioral Health, Pennsylvania State University
2000   B.S., Biology, Vertebrate Physiology, Pennsylvania State University
        Minor: African Studies

Dissertation Title Perceived Stress and Cotinine among African American Smokers: Variation by
Skin Color.

PROFESSIONAL EXPERIENCE

2001 to present Served as research assistant, instructor, guest lecturer and research coordinator.
Cortisol Salivary Immunoassay Training at the Salimetrics Laboratory.

PUBLICATIONS

Integration of salivary biomarkers into developmental and behaviorally-oriented research: problems and

and its effects on the measurement of cortisol, dehydroepiandrosterone, and testosterone in saliva.Horm
Behav. 2006 Apr;49(4):478-83.

Evidence of an Association. (unpublished)

Gilreath T, Whembolua, GL, King G. “Chapter 25: The Pathway to Substance Abuse: The Use
Tobacco by African-Americans” in “Handbook on African American Psychology” Guilford Press by
Robert Hampton and Ray Crowell (unpublished)

PRESENTATIONS

Poster Presentation, Ethnic differences in environmental tobacco smoke exposure in infancy: relation
between salivary cotinine and behavior at the Annual Meeting of the Society for Research in Child
Development, Boston, 2007

Poster Presentation, Ethnic Differences in Cotinine Levels Among Mother-Infants Dyads
at the 2006 Annual Meeting of the Society for Research on Nicotine and Tobacco, Orlando, 2006

AWARDS/HONORS

President of the African Students’ Association at Penn State University, 2003-2004

The Ardeth and Norman Frisbey International Award for exemplary contributions furthering international
understanding, Pennsylvania State University, April 2000