A Study of the Dimensionality of the Brief Wisconsin Inventory of Smoking Dependence Motives and Dependence Motive Profiles Among African American Daily Smokers

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Abstract

Introduction: African American (AA) smokers consume fewer cigarettes per day (CPD) and are less likely to report nicotine withdrawal, yet quit smoking at lower rates than White smokers. Racial differences in dependence-related factors suggest that facets of dependence may be differentially salient to the maintenance of smoking across groups. An important step toward understanding dependence among AA smokers is the evaluation of the psychometric properties of measures within this population. The present study describes validation of the Brief Wisconsin Inventory of Dependence Motives (B-WISDM) and derivation of dependence motive profiles in AA daily smokers.

Methods: The study sample comprised AA daily smokers participating in the Kick It at Swope IV randomized controlled trial of varenicline. The factor structure of the B-WISDM was tested using confirmatory factor analysis (CFA). Dependence motive profiles were derived using factor-mixture latent profile analysis (LPA). Profile membership was regressed on clinical characteristics to validate and characterize the obtained profiles.

Results: The 11-factor CFA model resulted in an improper solution, requiring the elimination of the Tolerance factor. A 10-factor model had satisfactory fit. Multiple pairs of factors were highly collinear ($r > .80$). The LPA of the 10-factor model yielded a 3-profile model that provided the best fit and no invalid parameter estimates. The profiles could be distinguished by factors related to compulsive smoking, or primary dependence motives (PDM), Taste, and Affiliative Attachment factor means. Light smoking ($\leq$ 10 cpd) was associated with membership in profiles with lower PDM scores and higher Taste and Affiliative Attachment scores ($OR = 2.4$, $p = .02$).

Conclusions: This study supports the psychometric adequacy of the B-WISDM for AA daily smokers. However, the high degree of collinearity observed among multiple pairs of factors

suggest the B-WISDM may benefit from reducing the number of subscales in the measure. The profile analysis has implications for understanding dependence motivation among AA smokers and the potential treatment needs of particular subgroups.

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Introduction

Cigarette smoking is the leading cause of preventable death in the United States (Danaei et al., 2009). Although prevalence rates of smoking among African American (AA) and Whites are similar, epidemiological and clinical data from the United States demonstrate marked disparity in rates of smoking cessation and relapse among AA smokers (American Lung Association, 2011; Babb, Malarcher, Schauer, Asman, & Jamal, 2017; Caraballo et al., 2014; Kulak, Cornelius, Fong, & Giovino, 2016; Piper et al., 2010). African American smokers are disproportionately burdened by smoking-related morbidity and mortality (CDC, 2015; Haiman et al., 2006). Thus, it is imperative to better understand the factors contributing to racial disparities in cessation outcomes.

Nicotine dependence (ND) is the construct hypothesized to account for chronic smoking that occurs despite adverse health consequences and the desire to quit (Niaura, 2013; Piasecki, Piper, & Baker, 2010b). In addition to having lower rates of quitting, racial differences in smoking behavior among AA smokers, including the smoking of fewer cigarettes per day (CPD), the predominant smoking of mentholated cigarettes and being more likely to make an unsuccessful quit attempt in a given year, suggest that AA smokers may be as nicotine dependent as White smokers, but at lower levels of smoking (Kulak et al., 2016; Rock, Davis, Thorne, Asman, & Caraballo, 2010; Trinidad et al., 2009). Common measures of ND operationalize the construct narrowly and may not fully generalize to the lighter smoking patterns that are more characteristic of smoking among AA compared to Whites (Shiffman, 2009; Trinidad, Perez-Stable, White, Emery, & Messer, 2011). Thus, a fuller characterization of ND among AA smokers is likely an important step in understanding racial disparities in smoking cessation.

Given that the AA smoker population is heterogeneous, understanding how ND varies within the AA smoker population is also necessary for the characterization of ND (Smith, Ramsay, & Mazure, 2014). Sex[1] differences among AA smokers in behavioral, biological, social and psychological factors are suggestive of differences in ND (Mickens, Ameringer, Brightman, & Leventhal, 2010). Females may have more difficulty quitting when unassisted than males and sex differences in response to pharmacotherapy for cessation have also been demonstrated (Smith et al., 2015; Smith, Zhang, Weinberger, Mazure, & McKee, 2017). Females smoke fewer CPD than males, smoke menthol cigarettes at higher rates, metabolize nicotine more quickly and evidence greater negative affect reduction expectancies for smoking (Mickens et al., 2010; Okuyemi, Ahluwalia, Richter, Mayo, & Resnicow, 2001; Pang, Zvolensky, Schmidt, & Leventhal, 2015; Pogun & Yararbas, 2009). Similar to racial differences in ND, sex differences among AA smokers are also inconsistently observed and ambiguous, in part due to the common approaches to ND measurement.

The Wisconsin Inventory of Dependence Motives (WISDM) is a multidimensional measure of ND motives (Piper et al., 2004). The particular motivations assessed (e.g., smoking to enhance cognition) are the motivational correlates of fundamental mechanisms of nicotine dependence (e.g. positive reinforcement). The multidimensional factor structure of the WISDM reflects the different motivational processes underlying persistent smoking that are implicit in different theories of drug dependence (Piper et al., 2004). The WISDM has been used to empirically derive qualitatively and quantitatively distinct motivational profiles that may help to

[1] In this study, the constructs of race and sex are knowingly conflated with ethnicity and gender, respectively. Race and sex refer to biological differences reducible to genotypic homogeneity while ethnicity and gender refer to specific forms of self-definition and shared experience (Obasi et al., 2013). This conflation serves to reflect that ND is likely influenced by both biological factors and factors related to shared experience (e.g., culture, socioeconomic disparity).

explain the motivational bases of ND among smokers across the continuum of smoking intensity (Piasecki et al., 2010b; Piper, Bolt, et al., 2008).

The WISDM is a promising measure for the characterization of ND motives among AA smokers. The Brief WISDM (B-WISDM; Smith et al., 2010) is an abridged version of the WISDM that is widely used and will be used in the present study. To date, there is conflicting evidence regarding the validity of the B-WISDM to assess ND motives among AA smokers (Ma, Li, & Payne, 2012; Smith et al., 2010). The existing validation studies of the WISDM have derived a measurement model describing the relationship between the items and their respective subscales, or factors (i.e., factor structure). Some of these studies have derived a structure describing how individual smokers are related to one another based upon their pattern of scores across the different ND motives subscales (i.e., latent profiles) (Piper, Bolt, et al., 2008; Piper et al., 2004; Smith et al., 2010). The purpose of this study is to assess the validity of these structures in a sample of AA smokers and to use sex and other ND-related variables to characterize and validate profiles of ND motives.

Literature Review

Sociohistorical Context of Racial Disparities in Smoking Outcomes

Smoking and smoking-related morbidity and mortality disproportionately burden socioeconomically deprived communities and AAs (CDC, 2010; Jamal et al., 2016). The association of non-White race and disadvantage in the United States has deep social and historical roots. Racial categorization in the United States has historically served to solidify and perpetuate social, political and economic dominance, resulting in a historical legacy of systematic racial inequality that persists to the present day (American Sociological Association, 2003; Williams, Priest, & Anderson, 2016). In 2016, the rate of poverty among AAs was more than double that of non-Hispanic Whites; the median income for AAs was $39,490 compared to $65,041 for non-Hispanic Whites (Proctor, Semega, & Kollar, 2017). The enduring effect of historical disenfranchisement is persistent; for example, one recent study demonstrated that geographic segregation in the 1880s was associated with less social mobility in the 1980s (Andrews, Casey, Hardy, & Logan, 2017).

Smoking and smoking-related health disparities are maintained by a complex network of intrapersonal, interpersonal, community, social and policy factors (Moolchan et al., 2007; National Cancer Institute, 2017), which has developed out of historical processes of cumulative disadvantage. For example, the net effect of historical disenfranchisement is the development communities of lower socioeconomic status, where AAs are more likely to reside. Higher poverty communities are more likely to have higher densities of tobacco retailers (Yu, Peterson, Sheffer, Reid, & Schnieder, 2010). Higher community density of tobacco retailers is associated with greater rates of uptake of youth smoking (Novak, Reardon, Raudenbush, & Buka, 2006). The experience of living in disenfranchised and marginalized communities promotes

discrimination, which is known to be associated with smoking and nicotine dependence (Borrell et al., 2010; Reitzel et al., 2012).

African Americans experience disparities in health care access and health outcomes (Bailey et al., 2017). With respect to tobacco treatment specifically, low socioeconomic status influences whether an individual uses smoking cessation pharmacotherapy (Shiffman, Brockwell, Pillitteri, & Gitchell, 2008) and is associated with a lower likelihood of receiving smoking cessation advice from a healthcare provider (Houston, Scarinci, Person, & Greene, 2005). African American smokers are less likely than White smokers to use cessation pharmacotherapy, even after accounting for markers of socioeconomic status (Fu et al., 2008). African American smokers are more likely to hold historically-informed negative beliefs about smoking cessation pharmacotherapy; namely, that the development and regulation of medications by the government and pharmaceutical industries is dictated by private interests (Carpenter, Ford, Cartmell, & Alberg, 2011). Thus, sociodemographic differences and historical experience likely influence the extent to which AA smokers engage in smoking cessation treatment, which thereby contributes to disparities in the burden of smoking.

There are numerous other complex pathways by which the historical disenfranchisement has influenced the socioeconomic position of AAs, which thereby contributes to smoking and smoking-related health disparities in the U.S. (National Cancer Institute, 2017). Given the insidious influence of the historical legacy of racism on health generally (Williams et al., 2016), the sociohistorical context of disparities in smoking must serve as the general frame within which to understand racial differences in smoking outcomes.

Race and Sex Differences in Smoking Related Health and Cessation Related Outcomes

Morbidity and mortality. Cigarette smoking is responsible for 467,000 deaths annually in the United States (Danaei et al., 2009). Smoking accounts for a significant proportion of all cancer deaths; in 2014, 29% of all cancer deaths in the United States could be attributed to smoking (Lortet-Tieulent, Goding Sauer, Siegel, & et al., 2016). Smoking causes many chronic diseases including diabetes, stroke, heart disease, chronic obstructive pulmonary disease (COPD) and rheumatoid arthritis (USDHHS, 2014). The health consequences of smoking differ by sex. Females experience unique health consequences as smoking during pregnancy causes premature birth, low birth weight and increases risk of still birth (WHO, 2010). African American smokers suffer from smoking-related morbidity and mortality at higher rates than White smokers (Harris, Zang, Anderson, & Wynder, 1993). The smoking-related health consequences are particularly dire for AA females, who generally exhibit the highest rates of rates of diabetes, breast and cervical cancer and heart disease (Belgrave & Abrams, 2016).

Across the continuum of cigarette consumption from heavy to light smoking, White smokers have 27-55% lower relative risk of incident smoking-related lung cancer compared to AA smokers (Haiman et al., 2006). In the state of Missouri from 2003-2007, the Centers for Disease Control (CDC) (2010) observed that the smoking-attributable mortality rate was 18% higher among AA than White smokers. For both AA and White smokers, males had higher rates of smoking-related mortality than females. African American males had a 28% higher rate of smoking-attributable mortality relative to White males, while AA females had an 11% higher rate of smoking attributable mortality compared to White females (CDC, 2010).

Rates of smoking, quit attempts and cessation. From 2005 to 2015, the prevalence of current smoking in the US population decreased from 20.9 to 15.1% (Jamal et al., 2016). Historically, smoking has been more prevalent among African Americans; however, by the

1990s, these rates began to converge (Kulak et al., 2016). By 2015, 16.6 and 16.7% of White and AA adults reported current smoking, respectively (Jamal et al., 2016). In 2015, AA males had higher rates of current smoking (20.9%) compared to White males (17.2%) and AA females (13.3%). White females had a higher rate of smoking than AA females (16%). Importantly, the prevalence of smoking varies markedly by socioeconomic status. In one study of AA smokers residing in the 39 poorest census tracts in Detroit, the overall prevalence of smoking was 42%, with AA males having higher rates of smoking than AA females (59% vs. 41%, respectively) (Delva et al., 2005). These findings are consistent with observed disparities in smoking prevalence among adults of lower versus higher socioeconomic status in the general population (USDHHS, 2014). Although current rates of smoking in the general population are nearly equivalent among AA and White smokers, differences exist with respect to interest in quitting and annual quit attempts.

Research using population and community-based samples demonstrates that AA smokers are more likely to be interested in quitting and more likely to make a quit attempt in a given year than White smokers (Babb et al., 2017; Keeler et al., 2017; Trinidad et al., 2015). In 2015, 63% of AA smokers reported a past-year quit attempt compared to 53% of White smokers (Babb et al., 2017). Race × sex interactions are evident with respect to quit attempts. Data from the 2000 NHIS demonstrated that although AA smokers were more likely to attempt to quit versus White smokers (48.5 vs. 43.7%), AA males and AA females attempted to quit at nearly equivalent rates (49 vs. 48%) whereas White females attempted to quit at higher rates than White males (46 vs. 43%) (Barbeau, Krieger, & Soobader, 2004). Thus, AA smokers, on average, appear to be highly interested in quitting smoking and regularly attempt to do so.

Despite the increased likelihood of making a quit attempt in a given year, the rate of annual cessation among AA smokers during 2000-2015 was 4.9 % compared to a cessation rate of 7.1% rate for White smokers (Babb et al., 2017). In addition to annual cessation rates, studies adjusting for potential confounders such as age, education, income and ND have observed that AA smokers are 43 - 49% less likely to maintain continuous abstinence than White smokers (Caraballo et al., 2014; Trinidad et al., 2011). From 1990 - 2000, the simultaneous examination of race and sex on successful quit ratio (number of smokers reporting to have quit for at least 12 months/number of ever smokers) revealed lower successful quit ratios for AA males and AA females compared to White male and White female smokers with evidence of a slight comparative disadvantage for AA females compared to AA males (King, Polednak, Bendel, Vilsaint, & Nahata, 2004). Thus, evidence at the population and community level is clearly indicative of disparities in smoking cessation among AA smokers.

Disparities in the use of cessation treatments and other environmental factors likely contribute to disparities in cessation (Cokkinides, Ward, Jemal, & Thun, 2005; Fu et al., 2008). However, disparities in cessation remain evident in efficacy trials, where the provision of cessation resources is constant and some environmental influences may be mitigated. In a placebo controlled randomized controlled trial (RCT) (mean CPD = 20.2) of nicotine replacement therapy (NRT) and bupropion mono/combination therapy, AA smokers were significantly less likely than White smokers to be abstinent at 7 days, 8 weeks and 6 months post-quit day. Notably, these differences were evident in the placebo group and the treatment arms despite AA and White smokers having similar levels of treatment adherence (Piper et al., 2010). In a randomized 2 × 2 factorial experiment of counseling and NRT/pharmacotherapy, AA smokers were significantly less likely to maintain continuous abstinence from weeks 6 to 52 of

follow-up (Gariti et al., 2009). These findings are also consistent with a placebo-controlled RCT of bupropion in an AA sample that found that the rate of abstinence at 26 weeks in the treatment arm (21%) was lower than that observed in comparable trials with samples comprised predominantly White, middle class smokers (25 – 27%) (Ahluwalia, Harris, Catley, Okuyemi, & Mayo, 2002). The observation of cessation-related disparities in efficacy trials suggests that AA smokers have significant difficulty quitting, even within the more controlled context of the RCT. Understanding the factors underlying this disparity is imperative to increasing rates of cessation in the AA smoker population.

Summary. African American smokers are particularly vulnerable to smoking related morbidity and mortality. Data from epidemiological and clinical studies suggest that AA smokers are highly motivated to quit and actively attempt to do so, but struggle to succeed at rates comparable to White smokers. The examination of smoking-related health outcomes and cessation-related outcomes suggest that AA females may suffer particular disadvantage. This is highlighted by the fact that AA females have the lowest rates of current smoking but have higher smoking-related mortality than White females (Mickens et al., 2010). Furthermore, the finding that AA females smoke at lower rates than AA males but attempt to quit at almost identical rates suggest that AA females may have particular difficulty quitting. Disparities evident in clinical trials for smoking cessation and in the population suggest that racial and sex disparities in smoking cessation are not artifacts of sampling biases inherent in clinical and observational research. Racial and sex differences in levels of cigarette consumption, cigarette type smoked, metabolism, withdrawal, craving, cue reactivity and outcome expectancies may underlie heterogeneity in ND and disparities in cessation.

Nicotine dependence (ND) is the construct hypothesized to underlie persistent smoking that occurs despite deleterious consequences and the desire to quit (APA, 2013; NIDA, 2012; Piasecki et al., 2010b). Nicotine dependence is a syndrome, meaning it is a collection of physical, cognitive, affective and motivational symptoms (APA, 2013; Shadel, Shiffman, Niaura, Nichter, & Abrams, 2000). Nicotine dependence is hypothesized to exist on a continuum of severity and the mechanisms underlying it are theorized to change over the course of one's smoking history (George & Koob, 2017; Koob, 2013; Tiffany, Conklin, Shiffman, & Clayton, 2004).

Shadel and colleagues (2000) delineated the features of nicotine dependence, which include the elicitation of pleasurable cognitive, affective, physical and motivational states through use; a decreased response to drug use and the need for larger doses to achieve an equivalent response (i.e., tolerance); the presence of aversive affective, physical and cognitive states when drug is no longer administered or the dose is reduced (i.e., withdrawal); subjective 'need' for drug use and a concomitant urge to use that is often under the control of cues (i.e., craving); compulsive drug administration; being in a state of ambivalence regarding continued use; and, the use of substances to manage negative emotional states. An important implication of the conceptualization of ND as a syndrome is that individuals vary in the expression of ND and that there may be inter-individual differences in the salience of particular ND symptoms. Racial and sex differences are observed with respect to ND related factors including cigarette consumption, nicotine metabolism, withdrawal, reactivity to smoking cues and use of smoking to regulate affective states. By reviewing how these factors vary by race and sex, it may be possible to better understanding how smoking is differentially maintained.

Racial and Sex Differences in Nicotine Dependence Related Factors

Nicotine exposure, metabolism and menthol. Nicotine is the psychoactive substance absorbed from cigarette smoke that is largely responsible for the addictive potential of cigarettes (Niaura, 2013). The pharmacological response of the binding of nicotine to nicotinic acetylcholine receptors is the release of dopamine through the activation of the mesolimbic dopamine reward pathway as well the modulation of other neurotransmitters, including serotonin, acetylcholine, glutamate and γ-Aminobutyric acid (GABA) (Balfour, 2004; Benowitz, 2008a, 2010). The resulting subjective effects including pleasure, tension reduction and increased concentration contribute to the subjective reinforcement value of smoking (Benowitz, 2008a). For a given smoker, blood levels of nicotine fluctuate from cigarette to cigarette, causing smokers to self-titrate nicotine levels to maintain the desired effect and to avoid the aversive symptoms of nicotine deprivation (Benowitz, 2008a). Over time, prolonged exposure to nicotine results in the desensitization of nicotinic receptors and their subsequent upregulation, resulting in tolerance and withdrawal (Benowitz, 2008b; Wang & Sun, 2005).

Persistent and escalating drug consumption is the paradigmatic symptom of drug dependence (MacKillop & Murphy, 2013). Over time, regular and increasingly frequent smoking may become highly overlearned, resulting in smoking characterized by automaticity and compulsivity (Tiffany, 1990). The degree of cigarette consumption is measured using average cigarettes per day (CPD). Cigarettes per day is used as both a proxy measure of nicotine exposure and a measure of ND, insofar that it is an index of smoking *ad libitum* (Fagerstrom & Schneider, 1989; USDHHS, 2010).

Despite evidence of poorer cessation outcomes, research reliably demonstrates that AA smokers smoke fewer CPD and are more likely to be light (≤ 10 CPD) or non-daily smokers than White smokers (Branstetter, Mercincavage, & Muscat, 2015; Cubbin, Soobader, & LeClere,

2010; Perez-Stable, Herrera, Jacob, & Benowitz, 1998). Trinidad and colleagues (2009) found that 37% of AA smokers were light smokers compared to 19% of White smokers. African American females smoke fewer CPD than AA males (Okuyemi et al., 2001; Trinidad et al., 2009). Furthermore, both AA males and females begin smoking regularly at older ages than their White counterparts (Roberts, Colby, Lu, & Ferketich, 2016). The combination of lower CPD and shorter duration of regular smoking suggests that AA smokers experience less cumulative exposure, as measured using pack-years (Holford, Levy & Meza, 2016). However, this does not translate to a comparative advantage for quitting.

African American smoke cigarettes more efficiently than White smokers. Across all levels of CPD, AA smokers have higher levels of serum cotinine and nicotine per cigarette than White smokers (Caraballo et al., 1998; Perez-Stable et al., 1998; Rostron, 2013). The absorption of greater levels of nicotine per cigarette among AA smokers suggests that smoking may be reinforced at lower levels of CPD. When stratified by sex and controlling for CPD and other covariates, serum cotinine levels are significantly lower among women than men (Gan, Cohen, Man, & Sin, 2008). These findings suggest that the relationship between CPD and nicotine exposure is complicated by differences in absorption by race and sex. These differences suggest that differences in metabolism may play a role in influencing nicotine exposure and ND.

Nicotine is primarily metabolized by the cytochrome P450 2A6 enzyme (CYP2A6) in the liver and is converted to its metabolite cotinine, which is further metabolized to trans-3'-hydroxycotinine (3HC) (Benowitz, Hukkanen, & Jacob, 2009). The ratio of 3HC/cotinine, or nicotine metabolite ratio (NMR), is used as an index of CYP2A6 activity (Benowitz et al., 2009). African American smokers are more likely than White smokers to be slow metabolizers, thus providing one hypothesis for higher serum cotinine among AAs (Kandel, Hu, Schaffran, Udry, &

Benowitz, 2007). Results from a large clinical trial found that 42% of AA smokers were in the slowest metabolizing quartile versus 15% in the fastest metabolizing quartile. In contrast, 16% of White smokers were in the slowest metabolizing quartile and 30% were in the fastest metabolizing quartile (Schnoll et al., 2014). Females have higher rates of nicotine metabolism than males, thus providing an explanation for lower average serum cotinine levels among females (Ho et al., 2009; Schnoll et al., 2014). Rates of nicotine metabolism may differ by race and sex and may thereby influence smoking behavior and aspects of ND.

Slow metabolizers are more likely to be lighter smokers, experience less abstinence-induced craving and withdrawal and are more likely to quit smoking (Kaufmann et al., 2015; Kubota et al., 2006; Ray, Tyndale, & Lerman, 2009). Faster metabolizers may clear nicotine at higher rates and thus keep lower concentrations of nicotine. Consequently, this may result in more rapid nicotinic receptor inactivation and greater withdrawal and craving symptoms. However, the association between NMR and withdrawal has been observed inconsistently (Kubota, et al. 2006, p. 118). Although NMR has a plausible relationship with ND, it appears to be more reliably associated with markers of smoking intensity (i.e., CPD) than measures of ND, such as the Fagerström Test of Nicotine Dependence (FTND) (Allenby, Boylan, Lerman, & Falcone, 2016; West, Hajek, & McRobbie, 2011). Variability in the rate of nicotine metabolism in the AA smoker population suggests that it may contribute to intra-population heterogeneity in the manifestation of ND. Although differences in metabolic rates of nicotine may partially explain intra-population heterogeneity in cessation, it is presently unclear why quit rates among AA smokers are as low as they are relative to White smokers given the relatively high rates of slow metabolizers in this smoker population.

Menthol smoking. Menthol is a flavor additive that produces a cooling sensation when smoked and anesthetizes the throat to irritation triggered by cigarette smoke (Lawrence, Cadman, & Hoffman, 2011). African American smokers are far more likely to smoke menthol cigarettes than White smokers (Caraballo & Asman, 2011; Cubbin et al., 2010). In the U.S., 82% of AA smokers reported smoking menthol cigarettes compared to 23% of White smokers (Rock et al., 2010). Among both AA and White smokers, females are significantly more likely to smoke menthol cigarettes than males (Rock et al., 2010; Caraballo & Asman, 2011). Menthol cigarette smoking is associated with lower odds of cessation, particularly among AA smokers (Foulds, Hooper, Pletcher, & Okuyemi, 2010; Stahre, Okuyemi, Joseph, & Fu, 2010). A large longitudinal study found that in addition to menthol smoking being associated with lower odds of cessation, AA menthol smokers were 45% less likely to achieve 6-month abstinence than White menthol smokers. Sex moderated this effect, such that AA female menthol smokers were significantly more likely to not quit than White female menthol smokers while AA male and White male menthol smokers had similar odds of cessation failure (Smith, Fiore, & Baker, 2014).

Multiple mechanisms may account for the association between menthol cigarette smoking and difficulty quitting (Wickham, 2015). Menthol cigarette smoking has been found to decrease CYP2A6 activity, suggesting that menthol smokers obtain greater amounts of nicotine per cigarette, although this effect may not survive when accounting for race (Benowitz, Herrera, & Jacob, 2004; Caraballo & Asman, 2011; Fagan et al., 2016; Jones, Apelberg, Tellez-Plaza, Samet, & Navas-Acien, 2013). Although menthol smokers may smoke more efficiently, menthol smoking has not been consistently associated with measures of physical ND, such as the FTND (FDA, 2013). This suggests that facets of menthol smoking other than its more efficient delivery

of nicotine may mediate the association of menthol cigarettes and greater difficulty quitting smoking.

The minty flavor and cooling sensation induced by menthol may function as a sensory cue for smoking in that the sensations associated with menthol are conditional upon smoking (Ahijevych & Garrett, 2010; Wickham, 2015). Habitual menthol cigarette smokers reported less satisfaction from non-menthol cigarettes than menthol cigarettes with comparable levels of nicotine and tar (Rose & Behm, 2004). In a behavioral study using rats, the presentation of a menthol cue elicited nicotine self-administration at greater rates than control cues and reinstated nicotine self-administration after extinction, providing a possible animal model for relapse (Wang, Wang, & Chen, 2014). In sum, the sensations elicited by menthol may pose as potent behavioral cues for smoking and may be associated with relapse.

Withdrawal. Withdrawal is a core symptom of the ND syndrome and refers to the collection of affective (e.g., dysphoria, irritability, anhedonia), physical (e.g., lower heart rate), cognitive (e.g., reduced concentration) and motivational/appetitive (e.g., craving) symptoms that develop in response to nicotine levels dropping below a certain level (Kassel, Veilleux, Heinz, Braun & Weber, 2013, p. 285; Piper, 2015). The affective features of withdrawal are robustly associated with relapse and serve to bias information processing and narrow motivational and behavioral repertoires, resulting in reinstated smoking (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004; Piasecki et al., 2000). Thus, withdrawal may contribute to continued smoking and may trigger relapse through negative reinforcement.

Little research exists on racial differences in withdrawal. Two laboratory-based experiments tested for racial differences in abstinence-induced changes in withdrawal symptoms (Bello et al., 2016; Robinson, Pickworth, Heishman, & Waters, 2014). Both studies utilized an

experimental design in which one group of smokers was instructed to smoke *ad libitum* prior to the session and another group in which smokers were required to be abstinent for 12-16 hours prior to the session. Assessments of withdrawal symptoms were obtained at both sessions and the difference between the scores served as an index of abstinence-induced changes in symptoms. In one study, AA smokers had smaller abstinence induced changes in withdrawal-induced hunger, cigarette craving, negative affect, positive affect and attentional bias toward smoking cues, but effect sizes were small- to-moderate and many did not survive correction (Robinson et al., 2014). In the other study, no significant differences in abstinence-induced withdrawal were observed between AA and White smokers (Bello et al, 2016).

Evidence from observational studies suggest that AA smokers are less likely to experience withdrawal symptoms than White smokers (Breslau, Kilbey, & Andreski, 1992; Riedel, Robinson, Klesges, & McLain-Allen, 2003; Weinberger, Platt, Smith, & Goodwin, 2017). In a representative sample of U.S. smokers, AA smokers indicated experiencing nearly all withdrawal symptoms less frequently than White smokers. In addition, White smokers were more likely to report a withdrawal-related relapse in the past year. In a regression analysis, total number of withdrawal symptoms was associated with continued smoking at study follow-up, however the association was moderated by race such that withdrawal symptoms were more robustly associated with continued smoking among White smokers (*OR* = .89) than among AA smokers (*OR* = .94) (Weinberger et al., 2017). Lower rates of self-reported, abstinence-induced withdrawal symptoms among AA smokers have also been observed in smaller community-based samples of adult (Breslau et al., 1992) and adolescent smokers (Riedel et al., 2003).

Evidence from interventions and epidemiological studies demonstrates that females are more likely to relapse to smoking than males (Smith, Bessette, Weinberger, Sheffer, & McKee,

2016). Experimental and epidemiological research have shown that females evidence greater abstinence-induced increases in self-reported negative affect relative to males, whereas sex differences in changes in physical symptoms, objective measures of cognitive performance, urges and positive affect are not consistently observed (Leventhal et al., 2007; Weinberger, Platt, Shuter, & Goodwin, 2016; Xu et al., 2008). Importantly, abstinence-induced increases in negative affect have been found to mediate the association of sex and a behavioral measure of motivation to delay smoking, suggesting a plausible mechanism underlying the increased likelihood of relapse among females (Pang & Leventhal, 2013).

Racial differences in physiological dimensions of ND, including withdrawal and nicotine metabolism, are apparent; however, their significance to disparities in cessation among AA smokers is unclear. Differences in psychosocial dimensions of ND including weight control expectancies, negative affect and cue reactivity also contribute to both inter-population and intra-population heterogeneity in ND.

Weight control expectancies. Outcome expectancies, subsequently denoted as expectancies, refer to the anticipated effects of using a drug and/or the effects of abstaining from its use (Metrik & Rohsenow, 2013). Expectancy is a construct derived from cognitive and social learning theories of dependence, which posit that cognitive representations that routinize and perpetuate drug use develop from learning that either occurs through one's experience or vicariously (Brandon, Herzog, Irvin, & Gwaltney, 2004; Metrik & Rohsenow, 2013) Though not a symptom of ND *per se*, expectancies may underlie many of the perceived costs and benefits of quitting that may sustain smokers' ambivalence toward abstinence.

Weight control expectancies refer to beliefs that smoking facilitates weight management and appetite suppression and that cessation leads to compensatory weight gain. Weight control

expectancies also overlap with related constructs including weight gain concern and post-cessation weight control self-efficacy (Germeroth & Levine, 2018). Concerns regarding post-cessation weight gain may be particularly salient for AA females, given that they are at the greatest risk for post-quit weight gain and have among the highest rates of obesity (Klesges et al., 1998; Ogden, Lamb, Carroll, & Flegal, 2010; Sanchez-Johnsen, 2005; Williamson et al., 1991). Compared to overweight and normal weight smokers, obese female AA smokers and obese smokers generally report greater concern regarding smoking related weight-gain (Beebe & Bush, 2015; Levine, Bush, Magnusson, Cheng, & Chen, 2013) and less self-efficacy for post-quit weight control (Levine et al., 2013).

Females report greater weight control expectancies, cessation-related weight concern and less post-cessation weight control self-efficacy than males (Aubin, Berlin, Smadja, & West, 2009; Germeroth & Levine, 2018; Hendricks et al., 2014; Sánchez-Johnsen, Carpentier, & King, 2011; Thomas et al., 2008). Weight control expectancies and weight gain concerns are inconsistently related to cessation among AA smokers (Germeroth & Levine, 2018; Thomas et al., 2008). However, studies have associated low levels of post-cessation weight control self-efficacy with faster time to relapse and lower odds of cessation at follow-up among AA smokers (Faseru et al., 2013; Levine, Marcus, Kalarchian, Houck, & Cheng, 2010). Thus, the association between weight control expectancies and cessation outcomes among AA smokers may be conditional upon the specific weight control expectancy construct assessed.

Negative affect. Negative affect, including depression, anxiety, depression, specific stress and general stress, has long been hypothesized to contribute to the maintenance of smoking and relapse through negative reinforcement (Kassel, Stroud, & Paronis, 2003). Stress reduction and relaxation are the most commonly cited reason for smoking among AA smokers

(Ahluwalia, Resnicow, & Clark, 1998). The motivation to smoke as a means to reduce negative affect may be particularly salient among low-income urban AA smokers, who may experience a greater burden of exposure to stressors resulting from living in unsafe neighborhoods, trauma exposure and unequal access to resources (Hatch & Dohrenwend, 2007; Slopen et al., 2012). However, two studies using nationally representative data found that race moderated the relationship between symptoms of anxiety and depression and current smoking, such that the relationship was non-significant for AA smokers but was significant for White smokers (Ellis, Orom, Giovino, & Kiviniemi, 2015; Kiviniemi, Orom, & Giovino, 2011). In contrast, a large study of non-treatment seeking AA smokers found that after controlling for sociodemographic characteristics, specific situational stressors including relationship stress and perceived neighborhood safety were associated with increased odds of self-reported current smoking (Slopen et al., 2012). These conflicting results suggest that among AA smokers, situational stressors may have more validity or weight than inventories of anxiety and depressive symptomology and reflect the affective states most closely associated with smoking.

African American female smokers may be motivated to smoke to relieve negative affect to a greater extent than AA males (Pulvers et al., 2004). Consistent with subjective report, experiments have found that females report greater craving, take in more smoke and report greater liking of smoking following negative mood induction than males (Perkins, Giedgowd, Karelitz, Conklin, & Lerman, 2012; Perkins, Karelitz, Giedgowd, & Conklin, 2013). Smoking may be a mediator of the relationship between cumulative stressors from social and economic discrimination and premature health declines among AA adults. Compared to AA males and White females, AA females may shoulder a disproportionate physiological burden from such stressors (Geronimus, Hicken, Keene, & Bound, 2006). Consistent with this idea, among AA

females of low socioeconomic status, a diverse set of stressors including low perceived neighborhood cohesion, neighborhood disorder, exposure to violence and discrimination are associated with current smoking (Andrews et al., 2014; Mickens et al., 2010). Thus, the combination of a predisposition to reactivity to negative mood states and environmental and social stresses suggests that persistent smoking among AA females may be influenced by the motivation ameliorate stress.

Cue reactivity. Cue reactivity refers to the extent to which stimuli that are paired with drug use elicit subjective craving, autonomic activation and drug seeking behavior (Carter & Tiffany, 1999). Cue reactivity is often tested in the laboratory using paradigms that operationalize smoking cues using a diverse set of stimuli including cigarettes, other smoking paraphernalia, individuals who are smoking and places/situations associated with smoking (Reynolds & Monti, 2013). However, more recently, in vivo cue reactivity has been assessed naturalistically using ecological momentary assessment (EMA).

As discussed above, AA smokers are more likely than White smokers to be light or intermittent smokers (LITS). Unlike heavier smokers, LITS do not evidence marked symptoms of withdrawal, even after periods of prolonged abstinence (Ahluwalia et al., 2006; Shiffman, Paty, Gnys, Kassel, & Elash, 1995). Shiffman and colleagues (2006) hypothesized that the smoking of LITS is governed by situational cues whereas the smoking of heavier smokers is independent of external cues. Heavier smoking is theorized to be under the control of largely unconscious interoceptive cues related to withdrawal, resulting in the maintenance of steady-state nicotine levels (Shiffman & Paty, 2006). Using EMA to assess the extent to which smoking is under the control of situational cues, studies have demonstrated that compared to heavier smokers, both LITS are more likely to smoke when away from home, when drinking alcohol, in

social contexts with other smokers and smoking while drinking caffeinated drinks (Ferguson, Shiffman, Dunbar, & Schuz, 2016; Shiffman et al., 2014; Shiffman & Paty, 2006). This pattern of smoking has been labeled, 'indulgent smoking'; the cues associated with indulgent smoking likely acquire their incentive value in that smoking potentiates the reward of these naturally rewarding situations (Shiffman & Paty, 2006). Importantly, a limited number of studies using laboratory cue reactivity paradigms have found that lighter smokers are more reactive to smoking cues than heavier smokers (Hogarth, Mogg, Bradley, Duka, & Dickinson, 2003; Watson, Carpenter, Saladin, Gray, & Upadhyaya, 2010). Given that LITS comprise a large proportion of AA smokers, it is likely that there is significant variability with respect to the degree to which smoking is under stimulus control among AA smokers.

Although no studies specifically assessing sex differences in cue reactivity among AA smokers have been conducted, females are generally less sensitive than males to the rewarding and reinforcing effects of nicotine (Perkins, 2001; Perkins, Jacobs, Sanders, & Caggiula, 2002). To account for disparities in cessation among females and the evidence that they may be less sensitive to the rewarding effects of nicotine, it has been hypothesized that females may be particularly sensitive to the non-nicotine reinforcers of smoking, namely cues (Perkins, 2009). However, laboratory and in vivo cue exposure studies using EMA testing this hypothesis have obtained inconsistent results with regard to sex differences in cue reactivity. Whereas laboratory studies have observed evidence of greater cue reactivity among female smokers (Doran, 2014; Field & Duka, 2004), studies of in vivo cue exposure using EMA have not (Ferguson, Frandsen, Dunbar, & Shiffman, 2015; Wray et al., 2015). Thus, sex differences in cue reactivity provide an important theoretical basis to explain treatment outcome disparities for female smokers, however

more research is needed to reconcile the discrepant results observed between laboratory and naturalistic cue exposure studies.

Summary. The above review highlights that mechanisms underlying ND may be differentially influential among AA and White smokers. African American smokers smoke cigarettes more efficiently than White smokers resulting from higher rates of genotypic variations that promote slower metabolism of nicotine. In combination with higher rates of menthol smoking, AA smokers require fewer CPD in order to achieve desired nicotine levels. One consequence of this may be that AA smokers may spend less time smoking, resulting in smoking that may be less compulsive and automatic in nature. Given the higher rates of light smoking among AA smokers, it may be that indulgent smoking and stimulus control may be more salient mechanisms of ND among AA than among White smokers. In addition, predominately AA neighborhoods have greater density of tobacco advertising compared to predominately White neighborhoods, providing further suggestion of a robust role for stimulus control among AA smokers (Robinson et al., 2015).

It is apparent from the literature that sex is an important source of heterogeneity within the AA smoker population. While AA smokers are on average slower metabolizers than White smokers, female smokers are faster metabolizers than male smokers and experience greater withdrawal than males. Furthermore, female smokers may be less sensitive to the reward of nicotine and may be more sensitive to non-nicotine cues to smoke including negative mood. Menthol smoking may also have a particularly deleterious effect on the ability of AA females to quit smoking. This may be attributable to the formidable combination of sensitivity to withdrawal coupled with the presence of sensory cues inherent in menthol smoking. Gender roles may also inform mechanisms of among AA females relative to AA males. African American

females may smoke to control weight and appetite and avoid quitting to avoid weight dyscontrol to a greater extent than AA males do. Additionally, AA females may assume the "superwoman role", in which they feel the responsibility to exude emotional and physical strength and act selflessly (Woods-Giscombé, 2010). Role specific stresses in addition to neighborhood factors and higher sensitivity to nicotine withdrawal may mean that AA females smoke to alleviate negative affect to a greater extent than AA males do.

Nicotine dependence is subserved by the same mechanisms among AA and White smokers, however the influence of these mechanisms likely differs both between and within these populations. Assessments of ND thus should reflect the multi-determined and multidimensional nature of the construct; however, this has not historically been undertaken until recently. The Wisconsin Inventory of Smoking Dependence Motives (WISDM) is a theory-informed, multidimensional measure of the motivational processes that underlie dependent smoking. Though the WISDM provides insight into the motivational mechanisms underlying persistent smoking across the continuum of smoking intensity, the extent to which it is valid for AA smokers is unclear.

Measures Used to Assess Nicotine Dependence in African American Smokers

Nicotine dependence is generally assessed using self-report measures; these measures vary considerably in scope, ranging from single items to multidimensional questionnaires (Colby, Tiffany, Shiffman, & Niaura, 2000). The heterogeneity among measures of ND reflects the absence of a gold standard of measurement of the construct. Although many measures of ND exist, the majority of studies assessing ND among AA adult smokers utilize the Fagerström Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker & Fagerström, 1991), whereas a growing number are using the Wisconsin Inventory of Dependence Motives (WISDM;

Piper et al., 2004). Multiple versions of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) have also been used to classify AA smokers as nicotine dependent, predominately in epidemiological studies. The *DSM* has limited utility in the assessment of ND in that it is atheoretical and uninformative regarding the mechanisms of ND; some of the substance dependence criteria may not apply to nicotine dependence; and, the *DSM* criteria have poor predictive validity with respect to smoking outcomes (Hughes, 2006; Hughes, Baker, Breslau, Covey, & Shiffman, 2011; Piper, McCarthy, & Baker, 2006).

The valid assessment of ND among AA smokers is an important for ultimately redressing disparities in smoking cessation outcomes. However, the insensitivity of measures of ND, particularly the FTND, to racial differences in smoking behavior and other factors may undermine the validity of commonly used measures of ND for AA smokers (Moolchan et al., 2007). The present section will review the FTND and WISDM with respect to their content, their dimensionality and their assessment race and sex differences in ND.

Fagerström Test for Nicotine Dependence (FTND). The FTND was derived from its parent measure, the Fagerström Tolerance Questionnaire (FTQ; Fagerström, 1978) and was created in order to improve upon the psychometric limitations of the FTQ (Heatherton et al., 1991). The FTND is a six-item measure that indexes physical dependence or, from a dependence motive perspective, withdrawal-induced motivation to smoke (Fagerstrom, 1978; Heatherton et al., 1991; Piper et al., 2006). The first item, known as Time to First Cigarette (TTFC), ("How soon after your first cigarette do you smoke?") and the fourth item, cigarettes per day (CPD), ("How many cigarettes per day do you smoke?") of the FTND are the most influential items from the measure insofar that they are often used as single-item indicators or as a two-item measure of ND known as the Heaviness of Smoking Index (HSI) (Heatherton, Kozlowski,

Frecker, Rickert, & Robinson, 1989). Despite its narrow focus on physical dependence, the FTND and its constituent measures are nearly ubiquitous in smoking cessation research.

Psychometric performance of the FTND. Although the FTND was developed in order to compensate for the multifactorial structure and low internal consistency of the FTQ, the FTND possesses similar psychometric deficiencies. Internal consistency of the FTND is low by common standards ($\alpha > .80$), ranging from .56 - .64 (Etter, Duc, & Perneger, 1999). Many factor analytic studies have observed that the FTND is multidimensional, with a 2-factor model commonly providing the better fit than a 1-factor model (e.g., Chabrol, Niezborala, Chastan, Montastruc & Mullet, 2003; Johnson, Morgan-Lopez, Breslau, Hatsukami & Bierut, 2008; Korte, Capron, Zvolensky & Schmidt, 2013; Radzius et al., 2003). Despite its well-replicated multifactorial structure, the FTND is typically represented by a sum of the six items, which may not yield a meaningful total score.

Insensitivity to dependence at low levels of smoking. Among light smokers, it has been observed that the FTND and HSI suffer from floor effects and that neither measure demonstrates incremental predictive validity over CPD (Etter et al., 1999). In one study, 90% of smokers with FTND scores of zero reported at least some degree of self-reported loss of autonomy over smoking, suggesting that features of ND are present even in the absence of marked physical dependence (Wellman et al., 2006). Given that AA smoke more efficiently than White smokers (i.e., achieve greater nicotine exposure per cigarette), it is possible that the FTND underestimates dependence in this population of smokers (Ahluwalia et al., 2002).

Despite these limitations, the FTND and its constituent measures have clear predictive validity. A meta-analysis of clinical trial data of adult smokers observed that the FTND and HSI are equivalently predictive of smoking cessation failure (Fagerstrom, Russ, Yu, Yunis, & Foulds,

2012). However, there is evidence to suggest that TTFC may be a particularly robust predictor of smoking cessation outcomes and that the other items of the FTND have little incremental validity beyond it (Baker et al., 2007). Despite having strong predictive validity, the reliance upon the FTND and TTFC provides little opportunity for the multidimensional characterization of ND. Though able to convey information about risk of cessation failure, the FTND and its constituent measure do little to provide insight into the nature of ND.

Wisconsin Inventory of Dependence Motives (WISDM). The WISDM is a multidimensional measure of the motivational processes that contribute to the development of ND. In contrast to measures of ND such as the FTND that asses the 'end-states' of ND, the WISDM is a measure intended to assess "the motivational press to use drugs in a dependent manner" (Piper et al., 2004; Piper et al., 2006, p. 348). The full-length WISDM (WISDM-68; Piper et al., 2004) is comprised of 68 items and 13 subscales. Each subscale and its constituent items operationalizes a different theory of drug-use motivation. For example, items on the Positive Reinforcement subscale assesses the degree to which a smoker is motivated to smoke based upon the hedonic effects of smoking (e.g., item 3: "Smoking makes a good mood better"). The Brief WISDM (B-WISDM; Smith et al., 2010) is comprised of 37 items and 11 subscales. The B-WISDM excludes one of the original subscales of the WISDM (Behavioral Choice-Melioration) due to its poor predictive validity and condensed two of the original subscales (Positive Reinforcement and Negative Reinforcement) into a single subscale (Affective Enhancement) (Smith et al., 2010). Each subscale from the B-WISDM is listed and described in the Appendix.

The WISDM has been used to identify distinct profiles of dependence motivation. Using Latent profile analysis (LPA) and cross-validation in multiple samples, Piper and colleagues

(2008) used mean scores from each of the 13 subscales of the WISDM-68 to empirically derive motive profiles in order to discern "obligatory and non-obligatory" motivational features of ND (Piper et al., 2008, p.750). Latent profile analysis attempts to empirically derive discrete subpopulations of observations based on their patterns of scores across a set of variables (Vermunt & Magidson, 2002). Piper and colleagues (2008) found that across four independent samples, a 5-profile model provided optimal fit and interpretive clarity. Four out of the five profiles were indicative of quantitative difference in the extent of motive endorsement. In other words, when plotted with each motive on the x-axis and the mean score for each motive on the y-axis, the lines indicative of mean scores across the profiles were roughly parallel. The fifth profile (17% of the total sample), labeled as the Automatic-Atypical profile, was qualitatively different from the other four. This profile had elevated scores on the automaticity, craving, tolerance and loss of control subscales with relatively low scores on the remaining six subscales.

Subsequent analyses have found that this group of dependence motives, known collectively as the Primary Dependence Motives (PDM), to be uniquely associated with measures reflecting heavy, compulsive smoking and physical dependence including FTND score, TTFC, CPD, withdrawal and nicotine self-administration (Piasecki, Piper, & Baker, 2010a; Piasecki, Piper, Baker, & Hunt-Carter, 2011; Tarantola, Heath, Sher, & Piasecki, 2017). The other seven subscales (Affiliative Attachment, Behavioral-Choice Melioration, Cognitive Enhancement, Cue exposure, Negative Reinforcement, Positive Reinforcement, Taste/Sensory Processes) have been labeled as Secondary Dependence Motives (SDM) and have been characterized as motivations for smokers who are not physically dependent and reflect instrumental motivations for smoking (Piasecki et al., 2010b). This distinction has been confirmed with respect to daily smokers and intermittent smokers. One study comparing

intermittent smokers (mean CPD = 3.3) and daily smokers (mean CPD = 15) found that after centering each participant's scores relative to their total score across all WISDM subscales, daily smokers had significantly greater mean scores on all PDM subscales compared to intermittent smokers. The majority of SDM subscales were significantly higher among intermittent compared to daily smokers (Shiffman, Dunbar, Scholl, & Tindle, 2012). Thus, the subscales of the PDM and SDM provide insight into the different motivational processes sustaining persistent smoking among lighter and heavier smokers.

Factor analyses of the B-WISDM. The B-WISDM is more widely used than the WISDM-68 and thus has been subjected to more confirmatory studies of its dimensionality. Validation studies of the dimensionality of the B-WISDM have found that the factor structure is generally satisfactory, however potentially to a lesser degree among AA smokers. In the original derivation of the B-WISDM, Smith and colleagues (2010), model fit was found to be satisfactory in both a predominately White clinical sample (RMSEA = .042, 95% CI [.04, .05], TLI = .95) and a small AA clinical sample (RMSEA = .06, 95% CI [.056, .064], TLI = .91) although, the fit was lower in the AA smoker sample. In another study of heavy smokers (mean CPD for White = 28, mean CPD for AA = 26), model fit of the 11-factor B-WISDM was poor for both White smokers and AA smokers. Given that heavy smoking is not characteristic of the average AA smoker in the population, it is unclear how informative this analysis is with respect to how the B-WISDM performs among AA smokers (Ma et al., 2012). Confirmatory factor analyses in predominately White samples and European samples have found that the 11-factor structure of the B-WISDM fit adequately (Adkison, Rees, Bansal-Travers, Hatsukami, & O'Connor, 2016; Pancani et al., 2015; Vajer, Urban, Tombor, Stauder, & Kalabay, 2011).

Latent Profile Analysis of B-WISDM. Only one study has empirically derived dependence motive profiles using the B-WISDM. Pancani and colleagues (2015) used *k*-means cluster analysis, a methodology similar to LPA, in a non-clinical sample of Italian young adults (mean CPD = 10). A five-cluster solution was obtained that was characterized by two qualitatively distinct subgroups. In Group A (clusters 1, 2 and 3), clusters had higher mean scores on the SDM subscales compared to the PDM subscales; the clusters within this group differed quantitatively. In Group B (clusters 4 and 5), mean scores on the PDM and SDM subscales were roughly equivalent; within the group, clusters 4 and 5 differed in degree only. This study found that clusters with the lowest scores on the PDM subscales (clusters 1 and 2) could be differentiated from clusters with intermediate and high scores on the PDM subscales using CPD and FTND.

Racial and sex differences in dependence motives. Little research has been conducted on racial and sex differences in ND motives. In one study of a combined sample of two large clinical trials (overall mean CPD = 22), AA and White smokers had nearly equivalent scores on the FTND while significant scores were evident across multiple WISDM-68 subscales. African American smokers scores were lower on all subscales except one (Tolerance), with statistically significant differences for Loss of Control, Cognitive Enhancement and Cue Exposure subscales (Piper, McCarthy, et al., 2008). In a study of non-treatment seeking heavy smokers, subscales scores on the WISDM were nearly equivalent across all 13 subscales (Ma et al., 2012).

Three studies have assessed sex differences in ND motives. Results among the studies are inconsistent. Consistent with sex differences in ND, females in one study had significantly greater mean scores on Affiliative Attachment, Cue Exposure, Negative Reinforcement and Weight Control dependence motives (Piper, McCarthy, et al., 2008). However, in a study of sex

differences in ND motives in a multiethnic sample, no differences in any of the 11 subscales of the B-WISDM were observed among male and female daily smokers. Mean scores on all of the ND motive subscales except Weight Control were significantly lower among female non-daily smokers compared to male non-daily smokers(Allen, Scheuermann, Nollen, Hatsukami, & Ahluwalia, 2016). Lastly, in a study of light AA smokers that used a preliminary version of the B-WISDM, females had higher mean scores on the Social Goads subscale only (Bronars, 2011).

Summary. The FTND and its constituent measures have utility insofar that they convey information on the risk of cessation failure, however they are hampered by psychometric concerns and limited scope to describe ND. The WISDM and its PDM and SDM subscales describe dependence motivations related to compulsive and elective smoking, respectively (Piasecki et al., 2010b). The existing literature on racial and sex differences in ND motives is presently inconclusive. The finding that female smokers have higher scores on dependence motives related to cue exposure and negative reinforcement is consistent with what is known about sex differences in sensitivity to non-nicotine cues and withdrawal. However, more research is needed. The WISDM may be particularly valuable for the description of ND among AA smokers and AAF, in particular, who are especially light smokers. However, the utility of the WISDM for the assessment of ND among AA is predicated upon its validity for the population, of which there is limited and inconsistent evidence.

Specific Aims

The literature review demonstrates that racial differences in cigarette consumption and menthol cigarette use, withdrawal, outcome expectancies and cue reactivity may contribute to differences in ND among AA and White smokers. Moreover, within the AA smoker population, there are multiple sources of heterogeneity that likely influence the manifestation of ND. Systematic differences in facets of ND may be observed with respect to sex and factors that covary with sex, including light smoking, menthol smoking use and stress. The WISDM measures ND motives, that is the motivational processes underlying ND. Given that heterogeneity exists with respect to the expression ND among AA smokers, it is likely that heterogeneity also exists with respect to ND motives. The application of subgroup analyses (i.e., LPA and cluster analysis) to the WISDM have resulted in the derivation of qualitatively and quantitatively distinct ND motives profiles and the PDM/SDM analytic distinction. To date, subgroup analyses of the WISDM have utilized samples predominately White and European samples, leaving open the question of whether results from these analyses generalize to AA smokers.

This study has three aims. The first aim is to conduct a confirmatory factor analysis (CFA) of the factor structure of the B-WISDM. As described above, conflicting evidence exists regarding the adequacy of the B-WISDM's factor structure for AA smokers. The factor structure of the B-WISDM is a statistical model that describes how the set of hypothesized ND motives (i.e., latent variables, or factors) account for covariation among the items (Brown, 2006). Results from the CFA will provide further information on the viability of the model implied by the B-WISDM for AA smokers and will inform whether the specific factors are appropriate for use in

the LPA. The hypothesis for Aim 1 is that the proposed factor structure of the WISDM will adequately fit this sample of AA smokers.

The second aim of this study is to empirically derive ND motive profiles using LPA. Consistent with previous research, it is hypothesized that a five-profile solution will provide the best model fit and that a qualitative distinction suggestive of the PDM and SDM will be evident in the profile solution. Lastly, the third aim is to use covariates to validate and characterize the obtained latent profiles. Figure 1 represents the statistical model that will be tested. Profiles with higher mean scores on the PDM are hypothesized to be associated with higher scores on the PROMIS Nicotine Dependence-short form, heavier smoking, being male, menthol cigarette use and beginning to smoke regularly at a younger age. Profiles with higher scores on the SDM are hypothesized to be associated with lower PROMIS Nicotine Dependence scores, female sex, stress and light smoking.

Results from this study will make three contributions to the literature. First, this study will provide additional information regarding the dimensionality of the WISDM for AA smokers. Second, this is first to model heterogeneity in ND motives among AA smokers and will validate the PDM/SDM distinction in a non-White/European sample. Lastly, the validation and characterization of these ND motive profiles may provide information useful for the development of tailored interventions for subgroups of AA smokers.

Methods

Participants and Procedures

The proposed study will use the baseline survey data from the Kick-It-at-Swope IV (KIS-IV) study, a randomized, double-blind placebo controlled trial testing the efficacy of varenicline for smoking cessation among AA daily smokers. Participants were recruited in the Kansas City, Missouri and Kansas City, Kansas areas using multiple means including radio and television advertisements, referrals from local hospitals and primary care clinics, community health fairs, local churches and other community resources. Participant eligibility was determined in three steps involving an initial telephone screening, a brief medical evaluation from a study provider and a final eligibility survey at the beginning of the baseline study visit. Study inclusion criteria included self-identification as African American or Black, being 18 years or older, smoking 1 or more CPD, smoking for 25 or more days out of the past 30, having interest in quitting and being willing to complete all study visits and to take study medication. Race was assessed by the open-ended question, "How would you describe your race or ethnicity?" Participants self-identifying as "Black", "African American" or any variants of these terms (e.g., "Black American") were included in the study.

Exclusion criteria included renal impairment, history of allergy to varenicline, history of alcohol or drug treatment in past year, history of major depression requiring treatment in past year, use of other tobacco products in past 30 days, use of smoking cessation pharmacotherapy in month prior to enrollment, pregnant (current or planning to be in next six months) or breast feeding, planning to move from Kansas City area in next six months or living with another study participant. Five-hundred participants were randomized to receive either varenicline (n = 300) or

placebo (n = 200) for 12-weeks, along with individualized health education counseling for both treatment arms. The primary study outcome was biochemically verified abstinence at 26 weeks.

Baseline assessment was conducted in person by a study research assistant following the determination of final eligibility and informed consent. Questions from the baseline assessment were read to the participant and Likert responses were visually displayed in order to accommodate different levels of literacy among participants.

Measures

Sociodemographic variables. Age, education and sex and number of participants with monthly family income < 200% of the federal poverty level will be reported for the sample.

Brief Wisconsin Inventory of Dependence Motives (B-WISDM). The B-WISDM is a theory-informed measure of drug-use motivation (Smith et al., 2010). The B-WISDM has 37-items that comprise 11 subscales, or factors. Each subscale assesses a theoretically distinct ND motive. The items on the B-WISDM are scored on a 7-point Likert scale ranging from 1 (*Not true of me at all*) to 7 (*Extremely true of me*).

PROMIS Nicotine Dependence-short form. The Patient Reported Outcomes Measurement Information System (PROMIS) Nicotine Dependence-short form (PROMIS-ND) is a 4-item measure of ND (Shadel et al., 2014). The PROMIS Possible total scores range from 0 -16. Items are scored on a five-point Likert scale ranging from 0 (*Not at all*) to 4 (*Very much*). The measure is intended to be a brief measure of ND that samples the different facets of ND. The item pool for the initial measure derivation was sampled from existing measures including the FTND and the full-length WISDM (WISDM-68; Piper et al., 2004) (Edelen, Tucker, Shadel, Stucky, & Cai, 2012).

Smoking-related variables. Light smoking is derived from average cigarettes per day (CPD). Smokers reporting smoking less than 11 CPD were classified as a light smoker (Trinidad et al., 2011). Menthol-cigarette smoking status was assessed using self-report ("Do you currently smoke menthol or non-menthol cigarettes?"). Self-reported age at initiation of regular smoking (How old were you when you started smoking regularly?) is an index of persistent smoking, with earlier age of onset associated with greater odds of continued smoking, however this pattern may not hold for AA smokers (Roberts et al., 2016).

Stress. The Perceived Stress Scale (PSS) is a 4-item measure of past-month general distress designed for individuals residing in the general population (Cohen, Kamarck, & Mermelstein, 1983). Possible scores on the PSS range from 0 to 16. Items are scored on a five-point Likert scale from 0 (*Never*) to 4 (*Very often*). The PSS has been associated with smoking cessation outcomes and has been validated for use among community-dwelling African Americans (Sharp, Kimmel, Kee, Saltoun, & Chang, 2007). The total score from this measure will be used.

Analysis Plan

Data analysis will be conducted in R, version 3.3.1 and in Mplus, version 7 (Muthen & Muthen, 1998-2012; R Core Team, 2018). Descriptive statistics for variables of interest will be presented with respect to the total sample and stratified by sex. For each item on the B-WISDM, the proportion of responses endorsing each point on the 7-point Likert scale along with skewness and kurtosis will be reported to characterize response patterns on the individual items.

Aim 1: Assessing the Dimensionality of the B-WISDM. Confirmatory factor analysis (CFA) will be used to assess the adequacy of the 11-factor measurement model of the B-WISDM. In accordance with recommendations on the selection of model estimator when using

ordinal response data with seven response categories, the robust maximum likelihood (MLR) estimator will be used (Rhemtulla, Brosseau-Liard, & Savalei, 2012). Model fit will be assessed using the chi-square test of absolute fit, root mean squared error of approximation (RMSEA, acceptable fit ≤ .08), the standardized root mean square residual (SRMR, fit ≤ .08), the comparative fit index (CFI, acceptable fit ≥ .90) and the Tucker Lewis Index (TLI, fit ≥ .90) (Little, 2013). Should estimation difficulties arise (e.g., non-positive definite matrix) out-of-bounds values be evident in the model output, individual measurement models will be estimated to try to identify problematic items or factors. Should there be poor model fit, modification indices and the standardized residual variance-covariance matrix will be consulted to locate specific sources of model miss-fit (Brown, 2006).

Alternative models will also be tested relative to the 11-factor model and will use chi-square difference tests with the Satorra-Bentler correction for nested model comparison (Satorra & Bentler, 2010). The 11 factors correspond to the subscales described in the Appendix. The alternative models include a 1-factor model, a model with the hypothesized 11 factors plus four error covariances that were observed in the original validation study of the B-WISDM and a model with two higher-order factors corresponding to the PDM and SDM constructs (Smith et al., 2010). A non-significant chi-square difference test indicates that the estimation of additional parameters in the less restrictive model does not contribute appreciable model miss-fit compared to the more restrictive model. McDonald's omega will be calculated to estimate the reliability of each factor of the B-WISDM. Omega is a preferable estimate of internal consistency to Cronbach's alpha because it is does not assume tau-equivalence and is thus not susceptible to bias (Dunn, Baguley, & Brunsden, 2014). Point estimates of omega and their 95% confidence

intervals will be estimated using a function found in the R package, MBESS (Dunn et al., 2014; Kelley & Lai, 2018).

Aim 2: Derivation and Validation of Dependence Motive Profiles. Latent profile analysis (LPA) will be used to empirically derive dependence motive profiles. Latent profile analysis belongs to a family of statistical models known as mixture models. Mixture models are person-centered analyses, meaning that they describe unobserved subpopulations that comprise a heterogeneous parent population. This is in contrast to variable-centered approaches (e.g., factor analysis, regression), which describe the relationship among variables (Wang & Wang, 2012). Like CFA, LPA is latent variable model. However, unlike CFA, the scale of the latent variable is unordered categorical rather than continuous (Lubke & Muthen, 2005). Latent profile analysis often uses observed variables as model indicators. Because the indicators for the proposed LPA are subscales of the WISDM, the present study will use latent variable latent profile analysis (LPA; Lubke & Muthen, 2005). This model is a factor mixture model with the within-profile covariance matrix constrained to zero. Rather than use the arithmetic means of the subscales as indicators for the LPA, this model will use the factors corresponding to the B-WISDM subscales (Piper et al., 2008; Pancani et al., 2015). The advantage of this approach is that it accounts for measurement error, which is not the case when subscale means are utilized. Two sets of LPAs will be run. The first will constrain all item residual variances to equality across profiles (i.e., assumption of strict invariance) while the second will remove the assumption of equivalent item residual variances, allowing all item residual variances to be uniquely estimated in each profile. The use of these two models allows for the examination of the extent to which non-normality (e.g., bimodal distributions) in the response distributions for individual items influences the derivation of the latent profiles.

Latent profile analyses are modeled iteratively; models with 1 to *k* profiles are estimated and compared with respect to model fit and interpretability. The previous LPA and cluster analysis of the WISDM derived models with five profiles. Thus, the present analysis will hypothesize that a five profile model will provide optimal fit. In accordance with the recommendation of Ram and Grimm (2009), models with 1 to 6 profiles will be estimated in order to provide statistical evidence that the five-profile model is optimal. Fit statistics for the present analysis will include the AIC, BIC and the sample-size adjusted BIC (aBIC). Smaller values on these fit indices indicate better fit. For the AIC, BIC and aBIC, smaller values represent better model fit. Entropy (range = 0 -1) will be used to assess the extent of profile separation, with larger values representing greater profile separation. Lastly, Vuong-Lo-Mendell-Rubin ratio test (VLMR-RT) and bootstrap likelihood ratio test (BLRT) will be used to compare to relative fit of a model with *k* and *k*-1 profiles. A significant test ($p < .05$) on either the VLMR-RT or the BLRT means that the model with *k* profiles should be chosen over the *k*-1 profile model (Ram & Grimm, 2009). Analysis steps outlined by Asparouhov and Muthén (2012) were followed to minimize the runtime required to estimate the LPA models.

Following the derivation of the final model, the latent profiles will be characterized in two steps. First, descriptive statistics for age of beginning to smoke regularly, sex, total PROMIS ND score, menthol cigarette use, light smoking (≤ 10 CPD) and the total score on the Perceived Stress Scale (PSS) stratified by profile will be presented. The statistics will be estimated using the "(e)" AUXILIARY variable option in Mplus (Muthen & Muthen, 1998-2012). The descriptive statistics are estimated such that uncertainty in the classification of individual observation to particular profiles is accounted for. In order to further validate and characterize the observed latent profile, the categorical latent variable will be regressed on the set of clinical

characteristics using multivariate multinomial logistic regression account for covariation among the variables. This step will be conducted using the corrected 3-step method (R3STEP) (Asparouhov & Muthén, 2013).

Results

Sample Characteristics

The individual and clinical characteristics of the sample are presented in Table 1. On average, the sample smoked approximately 13 CPD. More than half of the sample (52%) reported being light smokers (≤ 10 CPD). Nearly 86% of the sample reported smoking menthol cigarettes. Females smoked significantly fewer cigarettes per day, were more likely to be light smokers and started to smoke at a later age than male smokers.

Brief WISDM Item Response Characteristics

Figure 2 and Table 2 visually and numerically characterize the response distributions of the B-WISDM items. Notably, the majority of the 37-items were characterized by large proportions of responses at either, or both, extremes of the 7-point Likert scale. High frequencies of responding at the maximum value of the Likert-scale was observed most among PDM, particularly the Tolerance (TOL) and Loss of Control (LOC) subscales. For example, on item 31 of the TOL subscale, 52% of the sample reported that smoking within 30 minutes of waking was extremely true of them. On item 21 of the LOC subscale, 49% of the sample reported that being "really hooked on cigarettes" was extremely true of them. In contrast, multiple items of subscales comprising the SDM exhibited high frequencies of responding on the least extreme end of the Likert scale, particularly with respect to the Weight Control (WGHT) and Affiliative Attachment (AFF) subscales. On item 19 of WGHT, 64% of the sample reported that smoking to control weight was not a reason for smoking at all. Large numbers of responses at both ends of the Likert scale were observed for items on the Cue Exposure/Associative Processes (CUE) and Social and Environmental Goads (GOAD) subscales.

Confirmatory Factor Analysis of Brief WISDM

Confirmatory factor analysis (CFA) was conducted to assess the degree fit of the measurement model implied by the B-WISDM to the sample variance-covariance matrix. The fit statistics for each model are reported in Table 3. The schematic diagram for each model in Table 3 is depicted in Figure 3. As expected, the 1-factor model (Model 1) fit the observed data poorly (RMSEA [95% CI] = 0.11 [0.10, 0.11], CFI = 0.64, TLI = 0.62). The absolute and relative fit of the 11-factor measurement (Model 2) model was markedly improved compared to the 1-factor model (RMSEA [95% CI] = 0.06 [0.05, 0.06], CFI =0.91, TLI = 0.90) and resulted in a statistically significant chi-square difference test ($\Delta\chi^2_{(55)} = 2672.1$, $p < .001$). The modification indices for Model 2 suggested that the estimation of the 4 pairs of error covariances identified by Smith and colleagues (2010) would result in improved model fit.

The 11-factor model with the four pairs of error-covariances (Model 3) was estimated and resulted in improved fit compared to Model 2. However, examination of the standardized parameter estimates revealed that the correlation between the Tolerance (TOL) and Loss of Control (LOC) latent variables was greater than one ($r = 1.01$, $p < .001$). The significance of this out-of-bounds parameter is that the solution for Model 3 could not be regarded as valid (Brown, 2006). Extreme multicollinearity among latent variables is addressed by either constraining the out-of-bounds latent variable correlation to be less than one, removing it from the model or by combining it with another latent variable (Brown, 2006). The imposition of model constraints was judged to be an unsatisfactory solution because doing so obscures the poor discriminant validity among the latent variables implied by multicollinearity (Grewal, Cote, & Baumgartner, 2004).

To inform whether TOL would be eliminated or combined with LOC, TOL was estimated separately as an independent measurement model to assess the viability of the factor.

The fit for TOL was poor (RMSEA [95% CI] = 0.53 [0.48, 0.58], CFI = 0.49, TLI = -0.54) suggesting that the measurement model implied by the TOL factor fit the observed data poorly. Thus, to ensure that the measurement model would be appropriately specified and yield valid parameter estimates while maintaining the meaning of the other latent variables, it was decided that TOL would be eliminated from the measurement model and subsequent analyses.

The measurement model excluding TOL (Model 4) was estimated and resulted in satisfactory fit. Model 5 was identical to Model 4 with the addition of the error covariance from Smith (2010) that remained following the removal of TOL. Model 5 fit the observed data well (RMSEA [95% CI] = 0.04 [0.04, 0.05], CFI = 0.95, TLI = 0.94). All of the standardized factor loadings for Model 5 were greater than 0.60 and statistically significant. The inclusion of the error covariance in Model 5 resulted in significantly improved fit in comparison to Model 4 ($\Delta\chi^2_{(1)} = 72$, $p < .001$).

The measurement model with two higher-order latent variables corresponding to PDM and SDM was estimated (Model 6). Although the fit for Model 6 was satisfactory, Model 5 fit significantly better ($\Delta\chi^2_{(33)} = 136.6$, $p < .001$) suggesting that measurement model in which higher-order factors explained the covariance among the latent variables did not fit as well as the measurement model in which the covariation among the latent variables was estimated. In addition, higher-order PDM and SDM latent variables were highly correlated ($r = 0.82$, $p < .001$).

Brief WISDM Subscale Characteristics

The latent variable correlation matrix is presented in Table 4 and is also represented as a heatmap to facilitate visual inspection in Figure 4. With respect to the bivariate correlations, the PDM latent variables (range: $r = 0.78 – 0.93$) were generally more highly correlated than those

of the SDM (range: $r = 0.19 – 0.92$). Among the PDM, the correlation of LOC and CRAV was close to one ($r = 0.93$). The Automaticity (AUTO) and CRAV latent variables were also highly correlated ($r = 0.85$). Among the SDM, robust inter-correlations among the Affective Enhancement (AE), Cognitive Enhancement (COG) and Affiliative Attachment (AFF) subscales (range: $r = 0.79 – 0.92$) were observed.

A number of robust bivariate associations were observed between PDM and SDM latent variables. The correlation between CRAV and CUE ($r = 0.85$) was equivalent to the correlation of LOC and AUTO ($r = 0.85$), both of which are PDM latent variables. In addition, the correlation between CRAV and Affective Enhancement ($r = 0.80$) was slightly greater than the correlation among the two PDM, AUTO and CRAV ($r = 0.78$).

The large correlations existing between PDM and SDM latent variables also explain the finding of a large correlation between the SDM and PDM higher order latent variables (Model 6). The presence of large correlations between PDM and SDM latent variables also explain why Model 6 may not have fit as well as Model 5. In Model 6, only the factors hypothesized to belong to the PDM loaded onto the PDM higher-order latent variable and only the SDM subscales hypothesized to belong to the SDM loaded onto the SDM latent variable. No cross-loadings were estimated. Consistent with this idea, relatively large modification index values were observed for the factor loading of LOC on SDM ($\Delta\chi^2_{(1)} = 40.7$) and CUE on PDM ($\Delta\chi^2_{(1)} = 44.9$). In other words, these modification index values suggest that model fit for Model 6 would have improved with LOC was allowed to load on SDM and if CUE was allowed to load on PDM.

The subscale means and reliability estimates are presented in Table 4. The subscales of the 10-factor B-WISDM demonstrated adequate reliability, based on the estimates of McDonald's ω.

Latent Profile Analysis

Determination of final model. Factor-mixture latent profile analysis (LPA) of the 10-factor measurement model was conducted to empirically derive dependence motive profiles. The fit statistics for the LPA models are presented in Table 5. A visualization of the changes in AIC, BIC and aBIC by model are presented in Figure 5 to facilitate understanding of how model fit changed as function of model complexity. Smaller AIC, BIC and aBIC values are indicative of better model fit.

Relative to the 1-profile model, model fit improved markedly with the estimation of 2-profile and 3-profile models. The gains in model fit resulting from the estimation of the 4-profile, 5-profile and 6-profile models were comparatively modest to that observed for the previous three models. The marginal improvement in model fit gained from the estimation of the four to six profile models is reflected in the results from VLMR tests (Table 5). Specifically, the VLMR test comparing the gain in fit when estimating the 4-profile model versus the 3-profile indicated that the improvement in model fit relative to the increase in model complexity was non-significant ($p = 0.12$). Similarly, the VLMR test comparing the increase in model fit of the 6-profile model versus the 5-profile model showed that increasing model complexity did not appreciably improve fit ($p = 0.37$). A similar result was evident in the VLMR test comparing the 4-profile and 5-profile models was evident, however the results of this test were not considered trustworthy because the k-1 model loglikelihood value could not be replicated. With respect to the BLRT (Table 5), none of the p-values were deemed trustworthy because the k-1 model loglikelihood

could not be replicated for any of the models. Thus, the BLRT could not be used as a criterion to inform model selection in this analysis.

Differences in the precision of parameter estimates across models also factored into deciding upon a final profile model. With increased model complexity resulting from the estimation of each additional profile, estimates of the latent means became increasingly imprecise and unstable. This pattern became most extreme in the 5-profile and 6-profile models. For example, the latent mean for the AFF subscale in the 5-profile model was numerically large ($\alpha_{AFF} = 16.04$, $p = 0.13$), yet not significantly different from zero, suggesting a high degree of uncertainty in the estimation of this parameter. In the 6-profile model, the instability for the same latent variable mean had become more extreme ($\alpha_{AFF} = 40.81$, $p = 0.13$). Because of the instability of some of the estimated latent means and the lack of evidence of incremental fit in the 5-profile and 6-profile models, these models were ruled out as final models.

The remaining possible models for selection were the 3-profile and 4-profile models. The 3-profile and 4-profile were generally similar with respect to the pattern of latent means within each models' respective profiles. Compared to the 3-profile model, however, the 4-profile model had profiles that were highly parallel with profile means that did not appear appreciably different from one another. The presence of relatively indistinct profiles, coupled with the fact that the improvement in fit observed when estimating the 4-profile versus the 3-profile model was not statistically significant using the VLMR test lent evidence of the 3-profile model as the optimal model for this sample.[2]

[2] The estimation of a set of LPA models with the assumption strict invariance relaxed was planned to ascertain the effect of free item residual variances on the model profile solutions. However, this set of models could not be estimated with this constraint relaxed due to estimation difficulties and thus this part of the analysis plan could not be completed.

Characterization of final model. The profile plot for the 3-profile model is presented in Figure 6. The estimated latent mean is depicted on the y-axis and the label for each B-WISDM subscale is on the x-axis. Each line corresponds to one of the three profiles. The three profiles were uniformly differentiated by the subscales comprising the PDM, namely Automaticity (AUTO), Loss of Control (LOC) and Craving (CRAV). The model implied posterior probabilities of membership in each profile was 22.5% (113/500) for the High PDM profile, 43.5% (217/500) for the Medium PDM profile and 34% (170/500) for the Low PDM profile.

In addition to the PDM subscales, the three profiles could be differentiated by the pattern of latent means of the seven subscales comprising the SDM. Compared to the High PDM profile, the Low PDM profile had lower latent means on the Cognitive Enhancement (COG), Cue Exposure/Associative Processes (CUE), Social Goads (GOAD), Weight Control (WGHT) and Affective Enhancement (AE) factors, but higher mean scores on the Affiliative Attachment (AFF) and Taste (TAST) factors. The Medium PDM profile was largely intermediate between the Low PDM and High PDM profiles.

Descriptive statistics for the set of clinical characteristics used to characterize the latent profiles is presented in Table 6[3]. Study participants classified in the Low and Medium PDM groups started smoking regularly at significantly older ages than those in the High PDM group. Participants in the Low PDM group were significantly more likely to be light smokers than participants in the Medium PDM group (73% vs. 48%, $p < .001$) and the High PDM group (73% vs. 29%, $p < .001$). With respect to the PROMIS-ND and PSS-4, scores increased in a stepwise fashion from the Low PDM to the High PDM groups.

[3] The prevalence of menthol smoking in each group was estimated. However, parameter estimates were outside of the range of possible values due to large standard errors and thus are not reported. This is likely attributable to the low rates of non-menthol smoking in the present sample.

Multivariate multinomial logistic regression was used to test the degree of association of the variables in Table 6 with class membership (Table 7). The results of the multinomial logistic regression, with membership in the Low PDM profile relative to the High PDM profile as the outcome variable, demonstrated that light smokers were more likely to be in the Low PDM profile relative to the High PDM profile ($OR = 2.44$, $p = 0.02$). Participants with greater levels of nicotine dependence, as measured using the PROMIS-ND, and stress were significantly more likely to belong to the High PDM profile than the low PDM profile. With respect to membership in the Medium PDM profile relative to the High PDM profile, higher scores on the PROMIS-ND were associated with significantly greater odds of being in the High PDM profile ($OR = 0.75$, $p < 0.01$).

Discussion

The present study aimed to validate the psychometric adequacy of the B-WISDM in a sample of AA daily smokers and to empirically derive and characterize dependence motive profiles. Similar to other confirmatory analyses, this study largely confirmed the factor structure of the B-WISDM. However, limited differentiation was observed among many of the B-WISDM factors, suggesting little discriminant validity among particular pairs of subscales. With respect to the factor mixture latent profile analysis (LPA) of the B-WISDM, a novel 3-profile model proved to be the best fitting relative to model complexity and was characterized using a set of clinical characteristics.

Factor Structure and Psychometric Performance of the B-WISDM

This analysis of the dimensionality of the B-WISDM found that a 10-factor measurement was the best fitting model that resulted in no invalid parameter estimates. The original 11-factor model with estimated error covariances was the optimal measurement model in two previous studies (Smith et al., 2010; Vajer et al.,2011), but resulted in an improper solution due to the correlation between the Loss of Control (LOC) and Tolerance (TOL) factors in the present study. Similar to the results presented in this analysis, Adkison et al. (2016) found that the 11-factor model plus error covariances did not estimate normally, potentially due to a large latent correlation between TOL and LOC ($r = 0.92$). Notably, although the 11-factor model with estimated error covariances ostensibly estimated normally in the study by Vajer and colleagues (2011), the reported latent correlation between LOC and TOL was close to one ($r = 0.91$).

Beyond the lack of differentiation among TOL and LOC, this study observed correlations approaching one for factors among the SDM, namely Cognitive Enhancement (COG) and Affective Enhancement (AE), suggesting that these factors may represent a general state-

enhancement dependence motive. From a neuropsychopharmacological perspective, the high degree of overlap between AE and COG is not unexpected given that the acutely positively reinforcing effects of nicotine, including mild euphoria and increased arousal, are largely mediated by a common midbrain dopamine reward pathway of the ventral tegmental area (Watkins, Koob, & Markou, 2000).

Correlations greater than 0.80 were also observed with respect to factors spanning the SDM and PDM distinction, including Craving (CRAV) and Cue Exposure/Associative Properties (CUE) and CRAV and Affective Enhancement (AFF). The robust association between CRAV and CUE is also not unexpected given the well-documented association between the presence of smoking related stimuli and the elicitation of subjective and objective states indicative of craving (Carter & Tiffany, 1999). The large degree of association between the PDM and SDM higher-order latent variables that was observed in this analysis and a previous analysis (Pancani et al., 2015) is expected given the robust correlations between specific lower-order PDM and SDM factors.

A noteworthy finding in this study was the evidence of the misspecification of the TOL subscale. Four pairs of error covariances (item 3 with item 31, item 2 with item 16, item 35 with item 36, item 28 with item 36) were estimated in the original derivation of the B-WISDM because they improved model fit of the 11-factor measurement model (Smith et al., 2010). Three out of the four error covariances estimated by Smith (2010) involved items from the TOL factor (items 3, 28, 31, 36) and two out of the four concerned TOL items exclusively.

Error covariances are typically estimated to account for systematic measurement error that occurs due to a common item characteristic (e.g., similar wording and/or meaning) (Byrne, 2016). Item 3 (“I usually want to smoke right after I wake up”) and Item 31 (“I smoke within the

first 30 min of awakening in the morning") measure Time to First Cigarette whereas item 28 ("Other smokers would consider me a heavy smoker") and item 36 ("I consider myself a heavy smoker") assess identification as a heavy smoker. These items comprising the TOL factor represent two qualitatively distinct subsets of items, that when estimated as a single factor without accounting for the shared meaning among its constituent items, did not fit the observed data. The fact that the out-of-range latent variable correlation was present in Model 3 (with error covariances), but not Model 2 (no error covariances) may imply that once the common error variance among the items of the TOL factor was accounted for in Model 3, there was no variance in remaining in TOL that was distinct from LOC. Thus, the operationalization of the TOL factor should be revisited.

Implications. The importance of the lack differentiation among select B-WISDM subscales is that it implies that the number of subscales may be able to be reduced, thus making the measure faster to administer and more tractable to use in structural equation modeling. The AE and COG subscales and the CUE and LOC subscales may be candidate pairs of subscales for combination. Importantly, given the conceptual linkages among these pairs of latent variables, it is likely that the number of factors could be reduced in a theoretically driven manner.

There is evidence that the distinction between SDM and PDM is both analytically and theoretically useful (Piasecki et al., 2010b). However, this study along with other confirmatory factor analyses of the B-WISDM provide evidence that both individual SDM and PDM subscales and the SDM and PDM higher-order latent variables are relatively indistinct. Studies (e.g., Tarantola, Heath, Sher, & Piasecki, 2017) have used PDM and SDM subscales rather than the individual factor subscales to examine the discriminant validity of these general categories of dependence motives. The present results offer reasons to be cautious in this practice. The Loss of

Control factor was as highly correlated with the CUE factor as it was the CRAV factor. The LOC factor was more highly correlated with AE than AUTO. In addition, the Higher-Order CFA model had relatively large modification indices, suggesting that model fit would have improved if LOC was allowed to load on the SDM factor and if CUE was allowed to load on the PDM factor. Although there is a well-developed theoretical basis to classify the subscales as they are in the WISDM (Piper et al., 2004), the empirical associations observed among the latent variables in this analysis do not support the use of PDM and SDM as subscales as they are currently specified.

Dependence Motive Profiles among African American Smokers

The LPA conducted in the present analysis exhibited similarities and differences compared to the two other profile analyses of the B-WISDM. Consistent with the present analysis, the two previous analyses of dependence motive profiles demonstrated that the profiles could be differentiated by uniform differences across the PDM (Pancani et al., 2015; Piper et al., 2004). As in the present analysis, the SDM did not differentiate the profiles in a uniform fashion in the other analyses. For example, in Piper (2008), the distribution of mean scores across the SDM for each profile were roughly parallel, with the exception of Cue Exposure/Associative Properties and Social/Environmental Goads. Commonalities were also observed with respect to the characterization of the latent profiles. Like the study by Pancani and colleagues (2015), and consistent with the relationship hypothesized in this analysis, profiles with higher PDM scores were associated with measures of nicotine dependence and a greater degree of cigarette consumption.

Despite these general similarities, a number of key differences were observed in the present analyses that were different from the others. Contrary to expectation, this best fitting

model had fewer profiles than that of the other analyses. The present analysis derived three profiles whereas the other two studies derived five. Although larger sample sizes are generally known to be conducive to the observation of greater numbers of groups in mixture models (Cudeck & Henly, 2003), the sample size in Pancani (2015) and three out of five of the cross-validation samples in Piper (2004) were smaller than in the present study. Thus, sample size was likely not the determinative difference. It may be that the shape of multivariate parent distribution from which the profiles were derived in this study supported fewer groups relative to the distributions in the other two studies.

The LPA by Piper and colleagues (2004) observed the "Automatic-Atypical" profile, which was characterized by elevated mean scores across the four PDM and relatively low scores across all of the SDM. From this analysis and the body of literature proceeding it, evidence has suggested that the four PDM alone may be "sufficient for dependence manifestation" (Piper et al., 2004, p. 758). Of note, the four samples used for cross-validation in this study were comprised by a majority of White participants (68.5 – 89.4%) and relatively low rates (5.4 - 24.9%) and absolute numbers (*n*s = 25 – 130) of AA participants. Thus, although this study has provided greater knowledge regarding dependence motive phenotypes, how generalizable these findings are to AA smokers has been unclear.

A profile consistent with the Automatic-Atypical profile was not observed in this analysis. Like the Automatic-Atypical Profile, the High PDM profile had relatively high PDM scores compared to the other profiles. However, the High PDM profile in this analysis also had elevations on the CUE, COG, GOAD and AE subscales. The Low and Medium PDM profiles in the present analysis do not have clear analogues to the other profiles presented in Piper (2004) or

Pancani (2014). This does not imply that the Automatic-Atypical profile does not exist among AA smokers, rather it was not evident in this sample.

Beyond uniform differences across the PDM, this analysis showed that subgroups of smokers could be differentiated based upon select SDM, in particular TAST, COG, AFF and AE.

Taste and Sensory Dependence Motives. Consistent with previous research (Shiffman et al., 2012), mean scores for the TAST subscale were highest in the groups with lower levels of cigarette consumption, namely the Low PDM and Medium PDM profiles. These two profiles, and particularly the low PDM profile, were the lightest smoking profiles. Of note, menthol smokers smoke fewer CPD on average than non-menthol smokers and have more difficulty quitting potentially due to menthol's potentiation of the reinforcing effects of nicotine (Biswas et al., 2016; Jones et al., 2013; Smith, Fiore, et al., 2014). Despite the robust association of menthol smoking with difficulty quitting, the association of menthol smoking with measures of 'physical dependence' (e.g., TTFC, FTND) has been observed in some studies (Fagan et al., 2010), but not others (Hyland, Garten, Giovino, & Cummings, 2002; Jones et al., 2013; Muscat et al., 2009). In addition, AA smokers smoke menthol cigarettes at much higher rates than White smokers, but are less likely to experience symptoms of withdrawal (Weinberger et al., 2017). In addition, although nearly 86% of the sample reported smoking menthol-cigarettes, only approximately 22% of participants were in the high PDM group.

The independent reinforcing effects of the sensory qualities of menthol have been known to tobacco companies for decades according to internal industry documents (Yerger, 2011) and have been demonstrated in experimental research not affiliated with industry (Rose & Behm, 2004). The TAST subscale of the WISDM has been found to discriminate taster phenotypes for bitter compounds among smokers, suggesting it is a valid measure of dependence motivation

governed by the sensory qualities of smoking (Cannon et al., 2005). In the study by Cannon and colleagues (2005), mean scores on the TAST subscale were significantly lower among smokers with 'non-taster' versus 'taster' haplotypes. The Low and Medium PDM profiles are indicative of subgroups within the present sample that are relatively driven by the sensory qualities of smoking. Understanding the systematic sources of heterogeneity in sensitivity to the sensory reward of menthol would help explain the present findings and would be clinically valuable. One such factor, female sex, did not discriminate membership in the Low PDM or Medium PDM profiles from the High PDM profile. This was contrary to what was hypothesized for this analysis and to what would be expected given findings of sex differences in sensitivity to the sensory properties of smoking (Perkins, 2009; Perkins et al., 2002). However, none of the previous studies of sex differences in dependence motives to date have observed greater taste dependence motivation among females compared to males (Allen et al., 2016; Bronars, 2011; Piper et al., 2006).

Clinical implications. Given the independently reinforcing sensory features of menthol cigarettes, treatment strategies that specifically address this dimension of the reward of smoking may be particularly important among subgroups of smokers who are particularly sensitive to the non-nicotine smoking stimuli. Laboratory evidence suggests that switching habitual menthol smokers to non-menthol cigarettes reduces the subjective reward of smoking (Rose & Behm, 2004). In one study, the majority of habitual menthol smokers who switched to non-menthol cigarettes reported them as having an unpleasant aftertaste, unpleasant smell, causing throat irritation and being overall less enjoyable than menthol cigarettes (Watson et al., 2017). Thus, switching from menthol to non-menthol cigarettes over a prolonged period of time may be a

means to reduce to the reward of smoking or aid in reduction. To this author's knowledge, no clinical intervention has tested this hypothesis.

Rose and Behm (2004) showed that switching from menthol to non-menthol cigarettes that occurs simultaneously with the administration of NRT or medication that blocks the reward of nicotine results in no reduction in the subjective reward of smoking. The authors hypothesized that because the menthol cue is no longer present, the learned conditional association between the sensory effect of menthol and nicotine administration cannot be extinguished. Thus, it may be important for menthol smokers who use cessation medication to utilize pharmacotherapy that also features aspects of the sensory stimuli inherent in menthol, for example mint flavored nicotine gum or lozenges.

Enhancement and Affiliative Attachment Dependence Motives. The profiles comprising the final LPA model in the present analysis could be differentiated by means scores on the COG, AE and AFF subscales. The High PDM subscales had higher COG and AE scores relative to the Low and Medium PDM profiles. The Low and Medium PDM profiles had higher scores on the AFF subscale compared to the High PDM profile. This configuration of dependence motive scores can be understood through a developmental perspective regarding substance dependence. As dependence progresses, the motivational mechanisms underlying persistent substance use shift gradually from positive reinforcement due to the hedonic qualities of substance to negative reinforcement to ameliorate the aversive affective, cognitive and physiological states caused by withdrawal (Koob, 2013). Nicotine withdrawal is comprised of multiple symptoms, including anxiety, dysphoria and difficulty concentrating (APA, 2013). Thus, it is possible that the elevated COG and AE scores in the High PDM group reflect smoking to alleviate the cognitive and affective symptoms of withdrawal. Although symptoms of

withdrawal may be less common among AA smokers generally, the fact that the High PDM group was associated heavier smoking, higher scores on the PROMIS-ND and earlier age of smoking initiation may suggest that this subgroup of participants may have been more likely to experience withdrawal than smokers in the Medium and Low PDM profiles.

The configuration of scores among the latent profiles also suggests that dependence motives described by the AFF subscale may be influential of persistent smoking in the absence of significant compulsivity or heavy smoking. The Affiliative Attachment (AFF) dependence motives are grounded in the observation that some smokers experience their cigarettes as an "attractive social stimuli" and that quitting smoking entails an experience of personal loss (Piper et al., 2004, p. 151). Previous research has demonstrated that AFF mediates the relationship between social anxiety and nicotine dependence and has also been shown to be related to depressive symptoms above and beyond AE (Buckner & Vinci, 2013; Vinci, Copeland, & Carrigan, 2012). Thus, it may be that AFF describes affectively driven motivational processes to smoke in response to loneliness and perceived isolation whereas AE represents the motivation to smoke driven by the need to regulate the autonomic stress response and cognitive disturbance promoted by physiological withdrawal (Koob, 2013).

Many of the participants in this study resided in high poverty and high crime areas of Kansas City, Kansas and Kansas City, Missouri. Approximately one third of the sample had incomes greater than 200% of the federal poverty limit. Previous studies of AA smokers have demonstrated that greater perceived discrimination and neighborhood threat are associated with both SDM and PDM WISDM scale scores (Kendzor et al., 2014; Reitzel et al., 2012). These ecological stressors may contribute to the maintenance of smoking to the extent that individuals smoke to cope with the negative affect resulting from these ecological stressors, such as

perceived discrimination and neighborhood dangerous. The distinction between the prominence of AFF in the Low and Medium PDM profiles and COG and AE in the High PDM profiles suggest that ecological stressors may motivate sustained smoking differently for heavier and lighter AA smokers. Among lighter smokers, smoking in response ecological stressors may be motivated by the desire reduced perceived loneliness or alienation. For heavier smokers, the aversive affective state stemming from ecological stressors may induce craving and smoking through conditioned withdrawal (Oleson, Cachope, Fitoussi, & Cheer, 2014).

Clinical implications. Though both AE and AFF are affect-related dependence motives, they likely influence the maintenance of smoking in different ways and thus require different treatment approaches in order to facilitate cessation. Insofar that AE is a motivation that develops in response to nicotine withdrawal, the treatment of withdrawal may subdue this motivation to smoke. Nicotine replacement therapy reduces the symptoms of acute nicotine withdrawal (Shiffman, Ferguson, Gwaltney, Balabanis, & Shadel, 2006). In addition, mindfulness and lowering of affective volatility may be important components of future cognitive-behavioral interventions that could address the affective dimensions of acute withdrawal (Adams et al., 2014).

Specific interventions concerned with the barriers to quitting posed by Affiliative Attachment are not known. Subgroups of smokers who are strongly motivated to remain smoking due to affiliative attachment may hold negative outcome expectancies regarding quitting. Challenging expectancies is an evidenced-based practice for smoking relapse prevention (Shiffman, Kassel, Gwaltney, & McChargue, 2005). Theories of health behavior, particularly the Theory of Planned Behavior, also posit that attitudes are important to the formation of intentions for behavior change (Ajzen, 1991). Thus, empathetically challenging

cognitions associated with affiliative attitudes may be one clinical strategy to address dependence motivations consistent with the AFF subscale. In addition, other cognitive-behavioral strategies such as graded exposure may be helpful for subgroups of smokers concerned with their ability to tolerate the perceived loss of giving up smoking.

Limitations. This study had multiple limitations related to the sample, construct measurement and analytical methodology used. The sample included in the present analysis was treatment seeking. It is well established that treatment seeking smokers differ from smokers in the general population with respect to demographic and clinical characteristics (Gilbert, Sutton, & Sutherland, 2005; Hughes, Giovino, Klevens, & Fiore, 1997). With respect to AA smokers in particular, the present study excluded smokers who smoked non-cigarette tobacco products (cigars, cigarillos, Black & Milds, blunts). Rates of polytobacco use among AA smoker are particularly high (Corral, Landrine, Simms, & Bess, 2013). Polytobacco use presents a potentially important source of heterogeneity in nicotine dependence that is not captured by the sample of smokers in this study. An important extension of the present study would be the replication of the present analysis using a sample of non-treatment seeking smokers.

With respect to the B-WISDM itself, the measure was constructed to measure dependence motives along a continuum (Piper et al., 2004) and was not constructed with the intention of assessing groups[4] (Templin & Jiao, 2012). The potential of consequence of the mismatch between the latent scale of the measure and the goal of recovering groups is that one is effectively breaking a continuous scale into finite components, which has the potential of introducing error into classification (MacCallum, Zhang, Preacher, & Rucker, 2002).

4 Dr. Jonathan Templin is credited with suggesting this point.

There are several limitations related to the factor mixture LPA that deserve comment. First, it was not possible to estimate LPA models with the assumption of strict invariance relaxed due to estimation difficulties. It is possible that a better fitting model would have been derived with the relaxation of strict invariance. This limitation is indicative of a more general limitation of the current study. The LPA is a constrained permutation of the family of factor mixture models, which can achieve a very high degree of model flexibility (Clark et al., 2013; Lubke & Muthen, 2005). Given the previous use of LPA in studies of the B-WISDM, the computational complexity of the estimation of factor mixture models and the size of the B-WISDM measurement model relative to the study sample size, the LPA was deemed an appropriate statistical model for the present study. However, the relaxation of any number of parameter constraints can lead to different model results (Masyn, 2013). Therefore, it is possible that a less constrained factor mixture model could yield different results.

Lastly, a word of caution is necessary regarding the interpretation of mixture models generally. The derived groups observed in mixture models can be interpreted as qualitatively distinct subgroups within a population *or* as the simplified individual components, or "landmarks," of an unknown multivariate parent distribution (Bauer & Shanahan, 2007, p.270). The decision of how to interpret the model cannot be informed by the estimated model, but rests with the researcher (Bauer & Curran, 2003). Thus, theories of population heterogeneity are necessary to better appreciate the plausibility of the interpretation of mixture models as representative of discrete subpopulations (Bauer & Curran, 2003).

Conclusions

The goal of this study was evaluate the adequacy of the B-WISDM to assess dependence motivation among AA smokers and to apply the measure to better understand how dependence

motivation varies within the AA smoker population. The present study found that the B-WISDM demonstrated general psychometric adequacy for the assessment of dependence motives for AA smokers. The analysis also observed correlations approaching one for several pairs of latent variables and found that the TOL factor did not fit the observed data, likely due to the misspecification of the factor. The highly collinear latent variables in the B-WISDM occurred both among the PDM and SDM dependence motives and between individual PDM and SDM latent variables. This finding suggests that the use of the PDM and SDM as higher-order subscales should be done with caution and that the PDM and SDM distinction may be of value as a heuristic only.

The factor mixture LPA of the B-WISDM yielded a model that differed from previous profile analyses of the B-WISDM and provides additional insight into how dependence motivation may vary within the AA smoker population. The 3-profile model obtained in this analysis was not comprised of a factor suggestive of the sufficiency of the PDM alone to manifest nicotine dependence. Smokers in the lowest PDM group were the lightest smokers and evidenced marked elevations in dependence motives related to the sensory reward of smoking and social reward due to perceived affiliation with cigarettes and the act of smoking itself. In contrast, the High PDM group was characterized greater scores across all PDM subscales and dependence motives indicative of smoking motivated by the drive undo the aversive cognitive and affective sates created by physiological dependence. Thus, this study provides greater understanding of the complex of interplay of sensory and affective motivational processes that may maintain smoking among AA light smokers.

Bibliography

Adams, C. E., Chen, M., Guo, L., Lam, C. Y., Stewart, D. W., Correa-Fernández, V., Cano, M. A., Heppner, W. L., Vidrine, J. I., Li, Y., Ahluwalia, J. S., Cinciripini, P. M., & Wetter, D. W. (2014). Mindfulness Predicts Lower Affective Volatility among African Americans During Smoking Cessation. *Psychol Addict Behav, 28*(2), 580-585 10.1037/a0036512.

Adkison, S. E., Rees, V. W., Bansal-Travers, M., Hatsukami, D. K., & O'Connor, R. J. (2016). Psychometric Characteristics of the Brief Wisconsin Inventory of Smoking Dependence Motives Among a Nonclinical Sample of Smokers. *Nicotine Tob Res, 18*(4), 470-476 10.1093/ntr/ntv113.

Ahijevych, K., & Garrett, B. E. (2010). The Role of Menthol in Cigarettes as a Reinforcer of Smoking Behavior. *Nicotine Tob Res, 12*(Suppl 2), S110-116 10.1093/ntr/ntq203.

Ahluwalia, J. S., Harris, K., Catley, D., Okuyemi, K. S., & Mayo, M. S. (2002). Sustained-release bupropion for smoking cessation in african americans: A randomized controlled trial. *Jama, 288*(4), 468-474 10.1001/jama.288.4.468. http://dx.doi.org/10.1001/jama.288.4.468.

Ahluwalia, J. S., Okuyemi, K., Nollen, N., Choi, W. S., Kaur, H., Pulvers, K., & Mayo, M. S. (2006). The effects of nicotine gum and counseling among African American light smokers: a 2 x 2 factorial design. *Addiction, 101*(6), 883-891 10.1111/j.1360-0443.2006.01461.x.

Ahluwalia, J. S., Resnicow, K., & Clark, W. S. (1998). Knowledge about smoking, reasons for smoking, and reasons for wishing to quit in inner-city African Americans. *Ethn Dis, 8*(3), 385-393

Ajzen, I. (1991). The Theory of Planned Behavior. *Organizational Behavior & Human Decision Processes, 50*, 179-211

Allen, A. M., Scheuermann, T. S., Nollen, N., Hatsukami, D., & Ahluwalia, J. S. (2016). Gender Differences in Smoking Behavior and Dependence Motives Among Daily and Nondaily Smokers. *Nicotine & Tobacco Research, 18*(6), 1408-1413 10.1093/ntr/ntv138. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4906260/.

Allenby, C. E., Boylan, K. A., Lerman, C., & Falcone, M. (2016). Precision Medicine for Tobacco Dependence: Development and Validation of the Nicotine Metabolite Ratio. *J Neuroimmune Pharmacol, 11*(3), 471-483 10.1007/s11481-016-9656-y.

American Lung Association. (2011). *Trends in Tobacco Use*. http://www.lung.org/assets/documents/research/tobacco-trend-report.pdf. Accessed on: September 9, 2017.

American Sociological Association. (2003). *The Importance of Collecting Data and Doing Social Scientific Research on Race*. http://www.asanet.org/sites/default/files/savvy/images/press/docs/pdf/asa_race_statement.pdf. Accessed on: September 13, 2018.

Andrews, J. O., Mueller, M., Newman, S. D., Magwood, G., Ahluwalia, J. S., White, K., & Tingen, M. S. (2014). The Association of Individual and Neighborhood Social Cohesion, Stressors, and Crime on Smoking Status Among African-American Women in Southeastern US Subsidized Housing Neighborhoods. *J Urban Health, 91*(6), 1158-1174 10.1007/s11524-014-9911-6.

Andrews, R., Casey, M., Hardy, B. L., & Logan, T. D. (2017). Location matters: Historical racial segregation and intergenerational mobility. *Economics Letters, 158*, 67-72

https://doi.org/10.1016/j.econlet.2017.06.018.

http://www.sciencedirect.com/science/article/pii/S0165176517302458.

APA. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing.

Asparouhov, T., & Muthén, B. (2012). *Using Mplus TECH11 and TECH14 to test the number of latent classes*. https://www.statmodel.com/examples/webnotes/webnote14.pdf. Accessed on: July 1, 2018.

Asparouhov, T., & Muthén, B. (2013). *Auxiliary variables in mixture modeling: A 3-step approach using Mplus*. http://statmodel.com/examples/webnotes/AuxMixture_submitted_corrected_webnote . Accessed on: July 1, 2018.

Aubin, H. J., Berlin, I., Smadja, E., & West, R. (2009). Factors associated with higher body mass index, weight concern, and weight gain in a multinational cohort study of smokers intending to quit. *Int J Environ Res Public Health, 6*(3), 943-957 10.3390/ijerph6030943.

Babb, S., Malarcher, A., Schauer, G., Asman, K., & Jamal, A. (2017). Quitting Smoking Among Adults - United States, 2000-2015. *MMWR Morb Mortal Wkly Rep, 65*(52), 1457-1464 10.15585/mmwr.mm6552a1.

Bailey, Z. D., Krieger, N., Agenor, M., Graves, J., Linos, N., & Bassett, M. T. (2017). Structural racism and health inequities in the USA: evidence and interventions. *Lancet, 389*(10077), 1453-1463 10.1016/s0140-6736(17)30569-x.

Baker, T. B., Piper, M. E., McCarthy, D. E., Bolt, D. M., Smith, S. S., Kim, S. Y., Colby, S., Conti, D., Giovino, G. A., Hatsukami, D., Hyland, A., Krishnan-Sarin, S., Niaura, R., Perkins, K. A., & Toll, B. A. (2007). Time to first cigarette in the morning as an index of

ability to quit smoking: Implications for nicotine dependence. *Nicotine Tob Res, 9*(Suppl 4), S555-570 10.1080/14622200701673480.

Baker, T. B., Piper, M. E., McCarthy, D. E., Majeskie, M. R., & Fiore, M. C. (2004). Addiction motivation reformulated: an affective processing model of negative reinforcement. *Psychol Rev, 111*(1), 33-51 10.1037/0033-295x.111.1.33.

Balfour, D. J. (2004). The neurobiology of tobacco dependence: a preclinical perspective on the role of the dopamine projections to the nucleus accumbens [corrected]. *Nicotine Tob Res, 6*(6), 899-912

Barbeau, E. M., Krieger, N., & Soobader, M. J. (2004). Working Class Matters: Socioeconomic Disadvantage, Race/Ethnicity, Gender, and Smoking in NHIS 2000. *Am J Public Health, 94*(2), 269-278

Bauer, D. J., & Curran, P. J. (2003). Distributional assumptions of growth mixture models: implications for overextraction of latent trajectory classes. *Psychol Methods, 8*(3), 338-363 10.1037/1082-989x.8.3.338.

Bauer, D. J., & Shanahan, M. J. (2007). Modeling Complex Interactions: Person-Centered and Variable-Centered Approaches. In T. D. Little, J. A. Bovaird, & N. A. Card (Eds.), *Modeling Contextual Effects in Longitudinal Studies* (pp. 255-283). Mahwah, N.J: Erlbaum Associates.

Beebe, L. A., & Bush, T. (2015). Post-cessation weight concerns among women calling a state tobacco quitline. *Am J Prev Med, 48*(1 Suppl 1), S61-64 10.1016/j.amepre.2014.09.004.

Belgrave, F. Z., & Abrams, J. A. (2016). Reducing disparities and achieving equity in African American women's health. *Am Psychol, 71*(8), 723-733 10.1037/amp0000081.

Bello, M. S., Pang, R. D., Cropsey, K. L., Zvolensky, M. J., Reitzel, L. R., Huh, J., & Leventhal, A. M. (2016). Tobacco Withdrawal Amongst African American, Hispanic, and White Smokers. *Nicotine Tob Res, 18*(6), 1479-1487 10.1093/ntr/ntv231.

Benowitz, N., Hukkanen, J., & Jacob, P. (2009). Nicotine Chemistry, Metabolism, Kinetics and Biomarks. In J. E. Henningfield, E. D. London, & S. Pogun (Eds.), *Nicotine Psychopharmacology* (pp. 29-60). Berlin: Spring-Verlag.

Benowitz, N. L. (2008a). Clinical pharmacology of nicotine: implications for understanding, preventing, and treating tobacco addiction. *Clin Pharmacol Ther, 83*(4), 531-541 10.1038/clpt.2008.3.

Benowitz, N. L. (2008b). Neurobiology of nicotine addiction: implications for smoking cessation treatment. *Am J Med, 121*(4 Suppl 1), S3-10 10.1016/j.amjmed.2008.01.015.

Benowitz, N. L. (2010). Nicotine Addiction. *N Engl J Med, 362*(24), 2295-2303 10.1056/NEJMra0809890.

Benowitz, N. L., Herrera, B., & Jacob, P., 3rd. (2004). Mentholated cigarette smoking inhibits nicotine metabolism. *J Pharmacol Exp Ther, 310*(3), 1208-1215 10.1124/jpet.104.066902.

Biswas, L., Harrison, E., Gong, Y., Avusula, R., Lee, J., Zhang, M., Rousselle, T., Lage, J., & Liu, X. (2016). Enhancing effect of menthol on nicotine self-administration in rats. *Psychopharmacology (Berl), 233*(18), 3417-3427 10.1007/s00213-016-4391-x.

Borrell, L. N., Diez Roux, A. V., Jacobs, D. R., Shea, S., Jackson, S. A., Shrager, S., & Blumenthal, R. S. (2010). Perceived racial/ethnic discrimination, smoking and alcohol consumption in the Multi-Ethnic Study of Atherosclerosis (MESA). *Prev Med, 51*(3-4),

307-312 10.1016/j.ypmed.2010.05.017. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2939242/.

Brandon, T. H., Herzog, T. A., Irvin, J. E., & Gwaltney, C. J. (2004). Cognitive and social learning models of drug dependence: implications for the assessment of tobacco dependence in adolescents. *Addiction, 99 Suppl 1*, 51-77 10.1111/j.1360-0443.2004.00737.x.

Branstetter, S. A., Mercincavage, M., & Muscat, J. E. (2015). Predictors of the Nicotine Dependence Behavior Time to the First Cigarette in a Multiracial Cohort. *Nicotine Tob Res, 17*(7), 819-824 10.1093/ntr/ntu236.

Breslau, N., Kilbey, M. M., & Andreski, P. (1992). Nicotine withdrawal symptoms and psychiatric disorders: findings from an epidemiologic study of young adults. *Am J Psychiatry, 149*(4), 464-469 10.1176/ajp.149.4.464.

Bronars, C. (2011). *Psychometric Properties of the 30 Item Wisconsin Inventory of Dependence Motives (WISDM-30) among African American Light Smokers.* (PhD), Unveristy of Kansas, Lawrence, KS.

Brown, T. A. (2006). *Confirmatory Factor Analysis for Applied Research*. New York: Guilford.

Buckner, J. D., & Vinci, C. (2013). Smoking and Social Anxiety: The Roles of Gender and Smoking Motives. *Addict Behav, 38*(8), 2388-2391 10.1016/j.addbeh.2013.03.007.

Byrne, B. (2016). *Structural Equation Modeling With AMOS: Basic Concepts, Applications, and Programming* (3rd ed.). New York: Routledge.

Cannon, D. S., Baker, T. B., Piper, M. E., Scholand, M. B., Lawrence, D. L., Drayna, D. T., McMahon, W. M., Villegas, G. M., Caton, T. C., Coon, H., & Leppert, M. F. (2005).

Associations between phenylthiocarbamide gene polymorphisms and cigarette smoking. *Nicotine Tob Res, 7*(6), 853-858 10.1080/14622200500330209.

Caraballo, R. S., & Asman, K. (2011). Epidemiology of menthol cigarette use in the United States. *Tob Induc Dis, 9*(Suppl 1), S1 10.1186/1617-9625-9-s1-s1.

Caraballo, R. S., Giovino, G. A., Pechacek, T. F., Mowery, P. D., Richter, P. A., Strauss, W. J., Sharp, D. J., Eriksen, M. P., Pirkle, J. L., & Maurer, K. R. (1998). Racial and ethnic differences in serum cotinine levels of cigarette smokers: Third National Health and Nutrition Examination Survey, 1988-1991. *Jama, 280*(2), 135-139

Caraballo, R. S., Kruger, J., Asman, K., Pederson, L., Widome, R., Kiefe, C. I., Hitsman, B., & Jacobs, D. R., Jr. (2014). Relapse among cigarette smokers: the CARDIA longitudinal study - 1985-2011. *Addict Behav, 39*(1), 101-106 10.1016/j.addbeh.2013.08.030.

Carpenter, M. J., Ford, M. E., Cartmell, K., & Alberg, A. J. (2011). Misperceptions of nicotine replacement therapy within racially and ethnically diverse smokers. *J Natl Med Assoc, 103*(9-10), 885-894

Carter, B. L., & Tiffany, S. T. (1999). Meta-analysis of cue-reactivity in addiction research. *Addiction, 94*(3), 327-340

CDC. (2010). Racial disparities in smoking-attributable mortality and years of potential life lost -- Missouri, 2003-2007. *MMWR Morb Mortal Wkly Rep, 59*(46), 1518-1522

CDC. (2015). *Health, United States, 2014: With Special on Adults Aged 55-64* https://www.cdc.gov/nchs/data/hus/hus14.pdf. Accessed on: October 10, 2017.

Chabrol, H., Niezborala, M., Chastan, E., Montastruc, J. L., & Mullet, E. (2003). A study of the psychometric properties of the Fagestrom Test for Nicotine Dependence. *Addict Behav, 28*(8), 1441-1445

Clark, S. L., Muthen, B., Kaprio, J., D'Onofrio, B. M., Viken, R., & Rose, R. J. (2013). Models and Strategies for Factor Mixture Analysis: An Example Concerning the Structure Underlying Psychological Disorders. *Struct Equ Modeling, 20*(4) 10.1080/10705511.2013.824786.

Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *J Health Soc Behav, 24*(4), 385-396

Cokkinides, V. E., Ward, E., Jemal, A., & Thun, M. J. (2005). Under-use of smoking-cessation treatments: results from the National Health Interview Survey, 2000. *Am J Prev Med, 28*(1), 119-122 10.1016/j.amepre.2004.09.007.

Colby, S. M., Tiffany, S. T., Shiffman, S., & Niaura, R. S. (2000). Measuring nicotine dependence among youth: a review of available approaches and instruments. *Drug Alcohol Depend, 59 Suppl 1*, S23-39

Corral, I., Landrine, H., Simms, D. A., & Bess, J. J. (2013). Polytobacco use and multiple-product smoking among a random community sample of African-American adults. *BMJ Open, 3*(12), e003606 10.1136/bmjopen-2013-003606.

Cubbin, C., Soobader, M. J., & LeClere, F. B. (2010). The intersection of gender and race/ethnicity in smoking behaviors among menthol and non-menthol smokers in the United States. *Addiction, 105 Suppl 1*, 32-38 10.1111/j.1360-0443.2010.03191.x.

Cudeck, R., & Henly, S. J. (2003). A Realistic Perspective on Pattern Representation in Growth Data: Comment on Bauer and Curran (2003). *Psychol Methods, 8*(3), 378-383 10.1037/1082-989X.8.3.378.

Danaei, G., Ding, E. L., Mozaffarian, D., Taylor, B., Rehm, J., Murray, C. J., & Ezzati, M. (2009). The preventable causes of death in the United States: comparative risk

assessment of dietary, lifestyle, and metabolic risk factors. *PLoS Med, 6*(4), e1000058 10.1371/journal.pmed.1000058.

Delva, J., Tellez, M., Finlayson, T. L., Gretebeck, K. A., Siefert, K., Williams, D. R., & Ismail, A. I. (2005). Cigarette Smoking Among Low-Income African Americans: A Serious Public Health Problem. *Am J Prev Med, 29*(3), 218-220 10.1016/j.amepre.2005.05.004.

DiFranza, J. R., Savageau, J. A., Fletcher, K., Ockene, J. K., Rigotti, N. A., McNeill, A. D., Coleman, M., & Wood, C. (2002). Measuring the loss of autonomy over nicotine use in adolescents: the DANDY (Development and Assessment of Nicotine Dependence in Youths) study. *Arch Pediatr Adolesc Med, 156*(4), 397-403

Doran, N. (2014). Sex differences in smoking cue reactivity: craving, negative affect, and preference for immediate smoking. *Am J Addict, 23*(3), 211-217 10.1111/j.1521-0391.2014.12094.x.

Dunn, T. J., Baguley, T., & Brunsden, V. (2014). From alpha to omega: a practical solution to the pervasive problem of internal consistency estimation. *Br J Psychol, 105*(3), 399-412 10.1111/bjop.12046.

Edelen, M. O., Tucker, J. S., Shadel, W. G., Stucky, B. D., & Cai, L. (2012). Toward a more systematic assessment of smoking: development of a smoking module for PROMIS(R). *Addict Behav, 37*(11), 1278-1284 10.1016/j.addbeh.2012.06.016.

Ellis, E. M., Orom, H., Giovino, G. A., & Kiviniemi, M. T. (2015). Relations between negative affect and health behaviors by race/ethnicity: Differential effects for symptoms of depression and anxiety. *Health Psychol, 34*(9), 966-969 10.1037/hea0000197.

Etter, J. F., Duc, T. V., & Perneger, T. V. (1999). Validity of the Fagerstrom test for nicotine dependence and of the Heaviness of Smoking Index among relatively light smokers. *Addiction, 94*(2), 269-281

Fagan, P., Moolchan, E. T., Hart, A., Jr., Rose, A., Lawrence, D., Shavers, V. L., & Gibson, J. T. (2010). Nicotine dependence and quitting behaviors among menthol and non-menthol smokers with similar consumptive patterns. *Addiction, 105 Suppl 1*, 55-74 10.1111/j.1360-0443.2010.03190.x.

Fagan, P., Pokhrel, P., Herzog, T. A., Pagano, I. S., Franke, A. A., Clanton, M. S., Alexander, L. A., Trinidad, D. R., Sakuma, K. L. K., Johnson, C. A., & Moolchan, E. T. (2016). Nicotine Metabolism in Young Adult Daily Menthol and Nonmenthol Smokers. *Nicotine Tob Res, 18*(4), 437-446 10.1093/ntr/ntv109.

Fagerstrom, K., Russ, C., Yu, C. R., Yunis, C., & Foulds, J. (2012). The Fagerstrom Test for Nicotine Dependence as a predictor of smoking abstinence: a pooled analysis of varenicline clinical trial data. *Nicotine Tob Res, 14*(12), 1467-1473 10.1093/ntr/nts018.

Fagerstrom, K. O. (1978). Measuring degree of physical dependence to tobacco smoking with reference to individualization of treatment. *Addict Behav, 3*(3-4), 235-241

Fagerstrom, K. O., & Schneider, N. G. (1989). Measuring nicotine dependence: a review of the Fagerstrom Tolerance Questionnaire. *J Behav Med, 12*(2), 159-182

Faseru, B., Nollen, N. L., Mayo, M. S., Krebill, R., Choi, W. S., Benowitz, N. L., Tyndale, R. F., Okuyemi, K. S., Ahluwalia, J. S., & Sanderson Cox, L. (2013). Predictors of cessation in African American light smokers enrolled in a bupropion clinical trial. *Addict Behav, 38*(3), 1796-1803 10.1016/j.addbeh.2012.11.010.

FDA. (2013). *Preliminary Scientific Evaluation Of The Possible Public Health Effects Of Menthol Versus Nonmenthol Cigarettes*. https://www.fda.gov/downloads/UCM361598.pdf. Accessed on: September 9, 2017.

Ferguson, S. G., Frandsen, M., Dunbar, M. S., & Shiffman, S. (2015). Gender and stimulus control of smoking behavior. *Nicotine Tob Res, 17*(4), 431-437 10.1093/ntr/ntu195.

Ferguson, S. G., Shiffman, S., Dunbar, M., & Schuz, N. (2016). Higher stimulus control is associated with less cigarette intake in daily smokers. *Psychol Addict Behav, 30*(2), 229-237 10.1037/adb0000149.

Field, M., & Duka, T. (2004). Cue reactivity in smokers: the effects of perceived cigarette availability and gender. *Pharmacol Biochem Behav, 78*(3), 647-652 10.1016/j.pbb.2004.03.026.

Foulds, J., Hooper, M. W., Pletcher, M. J., & Okuyemi, K. S. (2010). Do smokers of menthol cigarettes find it harder to quit smoking? *Nicotine Tob Res, 12 Suppl 2*, S102-109 10.1093/ntr/ntq166.

Fu, S. S., Kodl, M. M., Joseph, A. M., Hatsukami, D. K., Johnson, E. O., Breslau, N., Wu, B., & Bierut, L. (2008). Racial/Ethnic Disparities in the Use of Nicotine Replacement Therapy and Quit Ratios in Lifetime Smokers Aged 25-44. *Cancer Epidemiol Biomarkers Prev., 17*(7), 1640-1647

Gan, W. Q., Cohen, S. B., Man, S. F., & Sin, D. D. (2008). Sex-related differences in serum cotinine concentrations in daily cigarette smokers. *Nicotine Tob Res, 10*(8), 1293-1300 10.1080/14622200802239132.

Gariti, P., Lynch, K., Alterman, A., Kampman, K., Xie, H., & Varillo, K. (2009). Comparing smoking treatment programs for lighter smokers with and without a history of heavier smoking. *J Subst Abuse Treat, 37*(3), 247-255 10.1016/j.jsat.2009.01.006.

George, O., & Koob, G. F. (2017). Individual differences in the neuropsychopathology of addiction. *Dialogues Clin Neurosci, 19*(3), 217-229

Germeroth, L. J., & Levine, M. D. (2018). Postcessation weight gain concern as a barrier to smoking cessation: Assessment considerations and future directions. *Addict Behav, 76*, 250-257 10.1016/j.addbeh.2017.08.022.

Geronimus, A. T., Hicken, M., Keene, D., & Bound, J. (2006). "Weathering" and Age Patterns of Allostatic Load Scores Among Blacks and Whites in the United States. *Am J Public Health, 96*(5), 826-833 10.2105/ajph.2004.060749.

Gilbert, H., Sutton, S., & Sutherland, G. (2005). Who calls QUIT? The characteristics of smokers seeking advice via a telephone helpline compared with smokers attending a clinic and those in the general population. *Public Health, 119*(10), 933-939 10.1016/j.puhe.2005.03.005.

Grewal, R., Cote, J. A., & Baumgartner, H. (2004). Multicollinearity and Measurement Error in Structural Equation Models: Implications for Theory Testing. *Marketing Science, 23*(4), 519-529 http://www.jstor.org/stable/30036686.

Haiman, C. A., Stram, D. O., Wilkens, L. R., Pike, M. C., Kolonel, L. N., Henderson, B. E., & Le Marchand, L. (2006). Ethnic and racial differences in the smoking-related risk of lung cancer. *N Engl J Med, 354*(4), 333-342 10.1056/NEJMoa033250.

Harris, R. E., Zang, E. A., Anderson, J. I., & Wynder, E. L. (1993). Race and sex differences in lung cancer risk associated with cigarette smoking. *Int J Epidemiol, 22*(4), 592-599

Hatch, S. L., & Dohrenwend, B. P. (2007). Distribution of traumatic and other stressful life events by race/ethnicity, gender, SES and age: a review of the research. *Am J Community Psychol, 40*(3-4), 313-332 10.1007/s10464-007-9134-z.

Heatherton, T. F., Kozlowski, L. T., Frecker, R. C., & Fagerstrom, K. O. (1991). The Fagerstrom Test for Nicotine Dependence: a revision of the Fagerstrom Tolerance Questionnaire. *Br J Addict, 86*(9), 1119-1127

Heatherton, T. F., Kozlowski, L. T., Frecker, R. C., Rickert, W., & Robinson, J. (1989). Measuring the heaviness of smoking: using self-reported time to the first cigarette of the day and number of cigarettes smoked per day. *Br J Addict, 84*(7), 791-799

Hendricks, P. S., Westmaas, J. L., Park, V. M. T., Thorne, C. B., Wood, S. B., Baker, M. R., Lawler, R. M., Hooper, M. W., Delucchi, K. L., & Hall, S. M. (2014). Smoking Abstinence-related Expectancies among American Indians. *Psychol Addict Behav, 28*(1), 193-205 10.1037/a0031938.

Ho, M. K., Mwenifumbo, J. C., Al Koudsi, N., Okuyemi, K. S., Ahluwalia, J. S., Benowitz, N. L., & Tyndale, R. F. (2009). Association of nicotine metabolite ratio and CYP2A6 genotype with smoking cessation treatment in African-American light smokers. *Clin Pharmacol Ther, 85*(6), 635-643 10.1038/clpt.2009.19.

Hogarth, L. C., Mogg, K., Bradley, B. P., Duka, T., & Dickinson, A. (2003). Attentional orienting towards smoking-related stimuli. *Behav Pharmacol, 14*(2), 153-160 10.1097/01.fbp.0000063527.83818.9e.

Holford, T. R., Levy, D. T., & Meza, R. (2016). Comparison of Smoking History Patterns Among African American and White Cohorts in the United States Born 1890 to 1990. *Nicotine Tob Res, 18*(Suppl 1), S16-29 10.1093/ntr/ntv274.

Houston, T. K., Scarinci, I. C., Person, S. D., & Greene, P. G. (2005). Patient Smoking Cessation Advice by Health Care Providers: The Role of Ethnicity, Socioeconomic Status, and Health. *Am J Public Health, 95*(6), 1056-1061 10.2105/ajph.2004.039909.

Hughes, J. R. (2006). Should criteria for drug dependence differ across drugs? *Addiction, 101 Suppl 1*, 134-141 10.1111/j.1360-0443.2006.01588.x.

Hughes, J. R., Baker, T., Breslau, N., Covey, L., & Shiffman, S. (2011). Applicability of DSM criteria to nicotine dependence. *Addiction, 106*(5), 894-895; discussion 895-897 10.1111/j.1360-0443.2010.03281.x.

Hughes, J. R., Giovino, G. A., Klevens, R. M., & Fiore, M. C. (1997). Assessing the generalizability of smoking studies. *Addiction, 92*(4), 469-472

Hyland, A., Garten, S., Giovino, G., & Cummings, K. (2002). Mentholated cigarettes and smoking cessation: findings from COMMIT. *Tob Control, 11*(2), 135-139 10.1136/tc.11.2.135.

Jamal, A., King, B. A., Neff, L. J., Whitmill, J., Babb, S. D., & Graffunder, C. M. (2016). Current Cigarette Smoking Among Adults - United States, 2005-2015. *MMWR Morb Mortal Wkly Rep, 65*(44), 1205-1211 10.15585/mmwr.mm6544a2.

Johnson, E. O., Morgan-Lopez, A. A., Breslau, N., Hatsukami, D. K., & Bierut, L. J. (2008). Test of measurement invariance of the FTND across demographic groups: assessment, effect size, and prediction of cessation. *Drug Alcohol Depend, 93*(3), 260-270 10.1016/j.drugalcdep.2007.10.001.

Jones, M. R., Apelberg, B. J., Tellez-Plaza, M., Samet, J. M., & Navas-Acien, A. (2013). Menthol Cigarettes, Race/Ethnicity and Biomarkers of Tobacco Use in US Adults: The

1999- 2010 National Health and Nutrition Examination Survey (NHANES). *Cancer Epidemiol Biomarkers Prev, 22*(2), 224-232 10.1158/1055-9965.EPI-12-0912.

Kandel, D. B., Hu, M. C., Schaffran, C., Udry, J. R., & Benowitz, N. L. (2007). Urine nicotine metabolites and smoking behavior in a multiracial/multiethnic national sample of young adults. *Am J Epidemiol, 165*(8), 901-910 10.1093/aje/kwm010.

Kassel, J. D., Stroud, L. R., & Paronis, C. A. (2003). Smoking, stress, and negative affect: correlation, causation, and context across stages of smoking. *Psychol Bull, 129*(2), 270-304

Kassel, J. D., Veilleux, J. C., Heinz, A. J., Braun, C., & Weber, S. (2013). The Role of Aftereffects and Withdrawal in Addiction. In J. MacKillop & H. d. Wit (Eds.), *The Wiley-Blackwell Handbook of Addiction Psychopharmacology* (First ed., pp. 281-313). New York: John Wiley & Sons.

Kaufmann, A., Hitsman, B., Goelz, P. M., Veluz-Wilkins, A., Blazekovic, S., Powers, L., Leone, F. T., Gariti, P., Tyndale, R. F., & Schnoll, R. A. (2015). Rate of nicotine metabolism and smoking cessation outcomes in a community-based sample of treatment-seeking smokers. *Addict Behav, 51*, 93-99 10.1016/j.addbeh.2015.07.019.

Keeler, C., Max, W., Yerger, V., Yao, T., Ong, M. K., & Sung, H. Y. (2017). The Association of Menthol Cigarette Use With Quit Attempts, Successful Cessation, and Intention to Quit Across Racial/Ethnic Groups in the United States. *Nicotine Tob Res, 19*(12), 1450-1464 10.1093/ntr/ntw215.

Kelley, K., & Lai, K. (2018). MBESS: MBESS. R Package version 4.4.3. Retrieved from https://cran.r-project.org/web/packages/MBESS/MBESS.pdf

Kendzor, D. E., Businelle, M. S., Reitzel, L. R., Rios, D. M., Scheuermann, T. S., Pulvers, K., & Ahluwalia, J. S. (2014). Everyday Discrimination Is Associated With Nicotine Dependence Among African American, Latino, and White Smokers. *Nicotine Tob Res, 16*(6), 633-640 10.1093/ntr/ntt198.

King, G., Polednak, A., Bendel, R. B., Vilsaint, M. C., & Nahata, S. B. (2004). Disparities in Smoking Cessation Between African Americans and Whites: 1990–2000. *Am J Public Health, 94*(11), 1965-1971

Kiviniemi, M. T., Orom, H., & Giovino, G. A. (2011). Psychological distress and smoking behavior: the nature of the relation differs by race/ethnicity. *Nicotine Tob Res, 13*(2), 113-119 10.1093/ntr/ntq218.

Klesges, R. C., Ward, K. D., Ray, J. W., Cutter, G., Jacobs, D. R., Jr., & Wagenknecht, L. E. (1998). The prospective relationships between smoking and weight in a young, biracial cohort: the Coronary Artery Risk Development in Young Adults Study. *J Consult Clin Psychol, 66*(6), 987-993

Koob, G. F. (2013). Theoretical frameworks and mechanistic aspects of alcohol addiction: alcohol addiction as a reward deficit disorder. *Curr Top Behav Neurosci, 13*, 3-30 10.1007/7854_2011_129.

Korte, K. J., Capron, D. W., Zvolensky, M., & Schmidt, N. B. (2013). The Fagerström Test for Nicotine Dependence: Do revisions in the item scoring enhance the psychometric properties? *Addict Behav, 38*(3), 1757-1763 10.1016/j.addbeh.2012.10.013.

Kubota, T., Nakajima-Taniguchi, C., Fukuda, T., Funamoto, M., Maeda, M., Tange, E., Ueki, R., Kawashima, K., Hara, H., Fujio, Y., & Azuma, J. (2006). CYP2A6 polymorphisms are

associated with nicotine dependence and influence withdrawal symptoms in smoking cessation. *Pharmacogenomics J, 6*(2), 115-119 10.1038/sj.tpj.6500348.

Kulak, J. A., Cornelius, M. E., Fong, G. T., & Giovino, G. A. (2016). Differences in Quit Attempts and Cigarette Smoking Abstinence Between Whites and African Americans in the United States: Literature Review and Results From the International Tobacco Control US Survey. *Nicotine Tob Res, 18*(Suppl 1), S79-87 10.1093/ntr/ntv228.

Lawrence, D., Cadman, B., & Hoffman, A. C. (2011). Sensory properties of menthol and smoking topography. *Tob Induc Dis, 9*(Suppl 1), S3 10.1186/1617-9625-9-s1-s3.

Leventhal, A. M., Boyd, S., Moolchan, E. T., Waters, A. J., Lerman, C., & Pickworth, W. B. (2007). Gender Differences in Acute Tobacco Withdrawal: Effects on Subjective, Cognitive, and Physiological Measures. *Exp Clin Psychopharmacol, 15*(1), 21-36 10.1037/1064-1297.15.1.21.

Levine, M. D., Bush, T., Magnusson, B., Cheng, Y., & Chen, X. (2013). Smoking-related weight concerns and obesity: differences among normal weight, overweight, and obese smokers using a telephone tobacco quitline. *Nicotine Tob Res, 15*(6), 1136-1140 10.1093/ntr/nts226.

Levine, M. D., Marcus, M. D., Kalarchian, M. A., Houck, P. R., & Cheng, Y. (2010). Weight Concerns, Mood, and Postpartum Smoking Relapse. *Am J Prev Med, 39*(4), 345-351 10.1016/j.amepre.2010.05.023.

Little, T. D. (2013). *Longitudinal Structural Equation Modeling*. New York: Guilford.

Lortet-Tieulent, J., Goding Sauer, A., Siegel, R. L., & et al. (2016). State-level cancer mortality attributable to cigarette smoking in the united states. *JAMA Internal Medicine, 176*(12),

1792-1798 10.1001/jamainternmed.2016.6530. http://dx.doi.org/10.1001/jamainternmed.2016.6530.

Lubke, G. H., & Muthen, B. (2005). Investigating population heterogeneity with factor mixture models. *Psychol Methods, 10*(1), 21-39 10.1037/1082-989x.10.1.21.

Ma, J. Z., Li, M. D., & Payne, T. J. (2012). Evaluation of the brief wisconsin inventory of smoking dependence motives in african-american and European-american heavy smokers. *Front Psychiatry, 3*, 36 10.3389/fpsyt.2012.00036.

MacCallum, R. C., Zhang, S., Preacher, K. J., & Rucker, D. D. (2002). On the practice of dichotomization of quantitative variables. *Psychol Methods, 7*(1), 19-40

MacKillop, J., & Murphy, C. (2013). Drug-Self Administration Paradigms: Methods for Quantifying Motivation in Experimental Research. In J. MacKillop & H. d. Wit (Eds.), *The Wiley-Blackwell Handbook of Addiction Psychopharmacology* (Vol. First, pp. 315-344). New York: John Wiley & Sons.

Masyn, K. (2013). Latent Class Analysis and Finite Mixture Modeling. In T. D. Little (Ed.), *The Oxford Handbook of Quantitative Methods* (pp. 551-611). New York, NY: Oxford University Press.

Metrik, J., & Rohsenow, D. J. (2013). Understanding the Role of Substance Expectancies in Addiction. In J. MacKillop & H. d. Wit (Eds.), *The Wiley-Blackwell Handbook of Addiction Psychopharmacology* (Vol. First, pp. 459-487). New York: John Wiley & Sons.

Mickens, L., Ameringer, K., Brightman, M., & Leventhal, A. M. (2010). Epidemiology, determinants, and consequences of cigarette smoking in African American women: an integrative review. *Addict Behav, 35*(5), 383-391 10.1016/j.addbeh.2009.12.014.

Moolchan, E. T., Fagan, P., Fernander, A. F., Velicer, W. F., Hayward, M. D., King, G., & Clayton, R. R. (2007). Addressing tobacco-related health disparities. *Addiction, 102 Suppl 2*, 30-42 10.1111/j.1360-0443.2007.01953.x.

Muscat, J. E., Chen, G., Knipe, A., Stellman, S. D., Lazarus, P., & Richie, J. P. (2009). Effects of menthol on tobacco smoke exposure, nicotine dependence, and NNAL glucuronidation. *Cancer Epidemiol Biomarkers Prev, 18*(1), 35-41 10.1158/1055-9965.epi-08-0744.

Muthen, L. K., & Muthen, B. (1998-2012). *Mplus User's Guide* (Seventh ed.). Los Angeles, CA: Muthen & Muthen.

National Cancer Institute. (2017). *A Socioecological Approach to Addressing Tobacco-Related Health Disparities (NCI Tobacco Control Monograph Series #22)*. https://cancercontrol.cancer.gov/brp/tcrb/monographs/22/index.html. Accessed on: September 12, 2018.

Niaura, R. (2013). Nicotine. In B. S. McCrady & E. E. Epstein (Eds.), *Addictions* (2nd ed.). New York: Oxford University Press.

NIDA. (2012). *Research Report Series: Tobacco/Nicotine*. https://d14rmgtrwzf5a.cloudfront.net/sites/default/files/tobaccorrs_1_2016.pdf. Accessed on: September 20, 2017.

Novak, S. P., Reardon, S. F., Raudenbush, S. W., & Buka, S. L. (2006). Retail Tobacco Outlet Density and Youth Cigarette Smoking: A Propensity-Modeling Approach. *Am J Public Health, 96*(4), 670-676 10.2105/ajph.2004.061622.

Obasi, E. M., Richard, J. L., Pittman, D. M., Ingram, J., Beasley, M. R., & Ratliff, K. L. (2013). The Rolse of Race and Sex in Addiction Research. In J. MacKillop & H. d. Wit (Eds.),

The Wiley-Blackwell Handbook of Addiction Psychopharmacology. New York: John Wiley & Sons.

Ogden, C. L., Lamb, M. M., Carroll, M. D., & Flegal, K. M. (2010). Obesity and socioeconomic status in adults: United States, 2005-2008. *NCHS Data Brief*(50), 1-8

Okuyemi, K. S., Ahluwalia, J. S., Richter, K. P., Mayo, M. S., & Resnicow, K. (2001). Differences among African American light, moderate, and heavy smokers. *Nicotine Tob Res, 3*(1), 45-50 10.1080/14622200020032097.

Oleson, E. B., Cachope, R., Fitoussi, A., & Cheer, J. F. (2014). Tales from the dark side: do neuromodulators of drug withdrawal require changes in endocannabinoid tone? *Prog Neuropsychopharmacol Biol Psychiatry, 52*, 17-23 10.1016/j.pnpbp.2013.07.019.

Pancani, L., D'Addario, M., Cappelletti, E. R., Greco, A., Monzani, D., & Steca, P. (2015). Smoking Behavior: A Cross-Sectional Study to Assess the Dimensionality of the Brief Wisconsin Inventory of Smoking Dependence Motives and Identify Different Typologies Among Young Daily Smokers. *Nicotine Tob Res, 17*(1), 98-105 10.1093/ntr/ntu143.

Pang, R. D., & Leventhal, A. M. (2013). Sex Differences in Negative Affect and Lapse Behavior During Acute Tobacco Abstinence: A Laboratory Study. *Exp Clin Psychopharmacol, 21*(4), 269-276 10.1037/a0033429.

Pang, R. D., Zvolensky, M. J., Schmidt, N. B., & Leventhal, A. M. (2015). Gender Differences in Negative Reinforcement Smoking Expectancies. *Nicotine Tob Res, 17*(6), 750-754 10.1093/ntr/ntu226.

Perez-Stable, E. J., Herrera, B., Jacob, P., 3rd, & Benowitz, N. L. (1998). Nicotine metabolism and intake in black and white smokers. *Jama, 280*(2), 152-156

Perkins, K. A. (2001). Smoking cessation in women. Special considerations. *CNS Drugs, 15*(5), 391-411

Perkins, K. A. (2009). Sex differences in nicotine reinforcement and reward: influences on the persistence of tobacco smoking. *Nebr Symp Motiv, 55*, 143-169

Perkins, K. A., Giedgowd, G. E., Karelitz, J. L., Conklin, C. A., & Lerman, C. (2012). Smoking in response to negative mood in men versus women as a function of distress tolerance. *Nicotine Tob Res, 14*(12), 1418-1425 10.1093/ntr/nts075.

Perkins, K. A., Jacobs, L., Sanders, M., & Caggiula, A. R. (2002). Sex differences in the subjective and reinforcing effects of cigarette nicotine dose. *Psychopharmacology (Berl), 163*(2), 194-201 10.1007/s00213-002-1168-1.

Perkins, K. A., Karelitz, J. L., Giedgowd, G. E., & Conklin, C. A. (2013). Negative mood effects on craving to smoke in women versus men. *Addict Behav, 38*(2), 1527-1531 10.1016/j.addbeh.2012.06.002.

Piasecki, T. M., Niaura, R., Shadel, W. G., Abrams, D., Goldstein, M., Fiore, M. C., & Baker, T. B. (2000). Smoking withdrawal dynamics in unaided quitters. *J Abnorm Psychol, 109*(1), 74-86

Piasecki, T. M., Piper, M. E., & Baker, T. B. (2010a). Refining the tobacco dependence phenotype using the Wisconsin Inventory of Smoking Dependence Motives: II. Evidence from a laboratory self-administration assay. *J Abnorm Psychol, 119*(3), 513-523 10.1037/a0020235.

Piasecki, T. M., Piper, M. E., & Baker, T. B. (2010b). Tobacco Dependence: Insights from Investigations of Self-Reported Smoking Motives. *Curr Dir Psychol Sci, 19*(6), 395-401 10.1177/0963721410389460.

Piasecki, T. M., Piper, M. E., Baker, T. B., & Hunt-Carter, E. E. (2011). WISDM primary and secondary dependence motives: associations with self-monitored motives for smoking in two college samples. *Drug Alcohol Depend, 114*(2-3), 207-216 10.1016/j.drugalcdep.2010.10.005.

Piper, M. E. (2015). Withdrawal: Expanding a Key Addiction Construct. *Nicotine Tob Res, 17*(12), 1405-1415 10.1093/ntr/ntv048.

Piper, M. E., Bolt, D. M., Kim, S. Y., Japuntich, S. J., Smith, S. S., Niederdeppe, J., Cannon, D. S., & Baker, T. B. (2008). Refining the tobacco dependence phenotype using the Wisconsin Inventory of Smoking Dependence Motives. *J Abnorm Psychol, 117*(4), 747-761 10.1037/a0013298.

Piper, M. E., Cook, J. W., Schlam, T. R., Jorenby, D. E., Smith, S. S., Bolt, D. M., & Loh, W. Y. (2010). Gender, race, and education differences in abstinence rates among participants in two randomized smoking cessation trials. *Nicotine Tob Res, 12*(6), 647-657 10.1093/ntr/ntq067.

Piper, M. E., McCarthy, D. E., & Baker, T. B. (2006). Assessing tobacco dependence: a guide to measure evaluation and selection. *Nicotine Tob Res, 8*(3), 339-351 10.1080/14622200600672765.

Piper, M. E., McCarthy, D. E., Bolt, D. M., Smith, S. S., Lerman, C., Benowitz, N., Fiore, M. C., & Baker, T. B. (2008). Assessing dimensions of nicotine dependence: An evaluation of the Nicotine Dependence Syndrome Scale (NDSS) and the Wisconsin Inventory of Smoking Dependence Motives (WISDM). *Nicotine Tob Res, 10*(6), 1009-1020 10.1080/14622200802097563.

Piper, M. E., Piasecki, T. M., Federman, E. B., Bolt, D. M., Smith, S. S., Fiore, M. C., & Baker, T. B. (2004). A multiple motives approach to tobacco dependence: the Wisconsin Inventory of Smoking Dependence Motives (WISDM-68). *J Consult Clin Psychol, 72*(2), 139-154 10.1037/0022-006x.72.2.139.

Pogun, S., & Yararbas, G. (2009). Sex Differences in Nicotine Addiction. In J. E. Henningfield, S. Pogun, & E. D. London (Eds.), *Nicotine Psychopharmacology*. Berlin: Springer-Verlag.

Proctor, B., Semega, J., & Kollar, M. (2017). *Income and Poverty in the United States: 2016*. https://www.census.gov/content/dam/Census/library/publications/2017/demo/P60-259.pdf. Accessed on: September 12, 2017.

Pulvers, K. M., Catley, D., Okuyemi, K., Scheibmeir, M., McCarter, K., Jeffries, S. K., & Ahluwalia, J. S. (2004). Gender, smoking expectancies, and readiness to quit among urban African American smokers. *Addict Behav, 29*(6), 1259-1263 10.1016/j.addbeh.2004.03.028.

R Core Team. (2018). R: A language and environment for statistical computing. Vienna, Austria: R Foundation for Statistical Computing. Retrieved from http://www.r-project.org/

Radzius, A., Gallo, J. J., Epstein, D. H., Gorelick, D. A., Cadet, J. L., Uhl, G. E., & Moolchan, E. T. (2003). A factor analysis of the Fagerstrom Test for Nicotine Dependence (FTND). *Nicotine Tob Res, 5*(2), 255-240

Ram, N., & Grimm, K. J. (2009). Growth Mixture Modeling: A Method for Identifying Differences in Longitudinal Change Among Unobserved Groups. *Int J Behav Dev, 33*(6), 565-576 10.1177/0165025409343765.

Ray, R., Tyndale, R. F., & Lerman, C. (2009). Nicotine dependence pharmacogenetics: role of genetic variation in nicotine-metabolizing enzymes. *J Neurogenet, 23*(3), 252-261 10.1080/01677060802572887.

Reitzel, L. R., Vidrine, J. I., Businelle, M. S., Kendzor, D. E., Cao, Y., Mazas, C. A., Li, Y., Ahluwalia, J. S., Cinciripini, P. M., Cofta-Woerpel, L., & Wetter, D. W. (2012). Neighborhood Perceptions Are Associated With Tobacco Dependence Among African American Smokers. *Nicotine Tob Res, 14*(7), 786-793 10.1093/ntr/ntr285.

Reynolds, E. K., & Monti, P. M. (2013). The Cue Reactivity Paradigm in Addiction Research. In J. MacKillop & H. d. Wit (Eds.), *The Wiley Blackwell Handook of Addiction Psychopharmacology*. New York: John Wiley & Sons.

Rhemtulla, M., Brosseau-Liard, P. E., & Savalei, V. (2012). When can categorical variables be treated as continuous? A comparison of robust continuous and categorical SEM estimation methods under suboptimal conditions. *Psychol Methods, 17*(3), 354-373 10.1037/a0029315.

Riedel, B. W., Robinson, L. A., Klesges, R. C., & McLain-Allen, B. (2003). Ethnic differences in smoking withdrawal effects among adolescents. *Addict Behav, 28*(1), 129-140

Roberts, M. E., Colby, S. M., Lu, B., & Ferketich, A. K. (2016). Understanding Tobacco Use Onset Among African Americans. *Nicotine Tob Res, 18 Suppl 1*, S49-56 10.1093/ntr/ntv250.

Robinson, C. D., Pickworth, W. B., Heishman, S. J., & Waters, A. J. (2014). The acute tobacco withdrawal syndrome among black smokers. *Psychol Addict Behav, 28*(1), 173-181 10.1037/a0031950.

Robinson, C. D., Pickworth, W. B., Heishman, S. J., Wetter, D. W., Cinciripini, P. M., Li, Y., Rowell, B., & Waters, A. J. (2015). Black Cigarette Smokers Report More Attention to Smoking Cues Than White Smokers: Implications for Smoking Cessation. *Nicotine Tob Res, 17*(8), 1022-1028 10.1093/ntr/ntu263.

Rock, V. J., Davis, S. P., Thorne, S. L., Asman, K. J., & Caraballo, R. S. (2010). Menthol cigarette use among racial and ethnic groups in the United States, 2004-2008. *Nicotine Tob Res, 12 Suppl 2*, S117-124 10.1093/ntr/ntq204.

Rose, J. E., & Behm, F. M. (2004). Extinguishing the rewarding value of smoke cues: pharmacological and behavioral treatments. *Nicotine Tob Res, 6*(3), 523-532 10.1080/14622200410001696501.

Rostron, B. (2013). NNAL exposure by race and menthol cigarette use among U.S. smokers. *Nicotine Tob Res, 15*(5), 950-956 10.1093/ntr/nts223.

Sanchez-Johnsen, L. A. (2005). Smoking cessation, obesity and weight concerns in black women: a call to action for culturally competent interventions. *J Natl Med Assoc, 97*(12), 1630-1638

Sánchez-Johnsen, L. A. P., Carpentier, M. R., & King, A. C. (2011). Race and Sex Associations to Weight Concerns among Urban African American and Caucasian Smokers. *Addict Behav, 36*(1-2), 14-17 10.1016/j.addbeh.2010.08.001.

Satorra, A., & Bentler, P. M. (2010). Ensuring Positiveness of the Scaled Difference Chi-square Test Statistic. *Psychometrika, 75*(2), 243-248 10.1007/s11336-009-9135-y.

Schnoll, R. A., George, T. P., Hawk, L., Cinciripini, P., Wileyto, P., & Tyndale, R. F. (2014). The relationship between the nicotine metabolite ratio and three self-report measures of

nicotine dependence across sex and race. *Psychopharmacology (Berl), 231*(12), 2515-2523 10.1007/s00213-013-3421-1.

Shadel, W. G., Edelen, M. O., Tucker, J. S., Stucky, B. D., Hansen, M., & Cai, L. (2014). Development of the PROMIS nicotine dependence item banks. *Nicotine Tob Res, 16 Suppl 3*, S190-201 10.1093/ntr/ntu032.

Shadel, W. G., Shiffman, S., Niaura, R., Nichter, M., & Abrams, D. B. (2000). Current models of nicotine dependence: what is known and what is needed to advance understanding of tobacco etiology among youth. *Drug Alcohol Depend, 59 Suppl 1*, S9-22

Sharp, L. K., Kimmel, L. G., Kee, R., Saltoun, C., & Chang, C. H. (2007). Assessing the Perceived Stress Scale for African American adults with asthma and low literacy. *J Asthma, 44*(4), 311-316 10.1080/02770900701344165.

Shiffman, S. (2009). Light and intermittent smokers: background and perspective. *Nicotine Tob Res, 11*(2), 122-125 10.1093/ntr/ntn020.

Shiffman, S., Brockwell, S. E., Pillitteri, J. L., & Gitchell, J. G. (2008). Individual differences in adoption of treatment for smoking cessation: demographic and smoking history characteristics. *Drug Alcohol Depend, 93*(1-2), 121-131 10.1016/j.drugalcdep.2007.09.005.

Shiffman, S., Dunbar, M. S., Li, X., Scholl, S. M., Tindle, H. A., Anderson, S. J., & Ferguson, S. G. (2014). Smoking patterns and stimulus control in intermittent and daily smokers. *PLoS One, 9*(3), e89911 10.1371/journal.pone.0089911.

Shiffman, S., Dunbar, M. S., Scholl, S. M., & Tindle, H. A. (2012). Smoking motives of daily and non-daily smokers: A profile analysis. *Drug Alcohol Depend, 126*(3), 362-368 10.1016/j.drugalcdep.2012.05.037.

Shiffman, S., Ferguson, S. G., Gwaltney, C. J., Balabanis, M. H., & Shadel, W. G. (2006). Reduction of abstinence-induced withdrawal and craving using high-dose nicotine replacement therapy. *Psychopharmacology (Berl), 184*(3-4), 637-644 10.1007/s00213-005-0184-3.

Shiffman, S., Kassel, J. D., Gwaltney, C. J., & McChargue, D. (2005). Relapse Prevention for Smoking. In G. A. Marlatt & D. M. Donovan (Eds.), *Relapse Prevention: Maintenance Strategies in the Treatment of Addictive Behaviors* (2nd ed., pp. 92-129). New York: Guilford.

Shiffman, S., & Paty, J. (2006). Smoking patterns and dependence: contrasting chippers and heavy smokers. *J Abnorm Psychol, 115*(3), 509-523 10.1037/0021-843x.115.3.509.

Shiffman, S., Paty, J. A., Gnys, M., Kassel, J. D., & Elash, C. (1995). Nicotine withdrawal in chippers and regular smokers: subjective and cognitive effects. *Health Psychol, 14*(4), 301-309

Slopen, N., Dutra, L. M., Williams, D. R., Mujahid, M. S., Lewis, T. T., Bennett, G. G., Ryff, C. D., & Albert, M. A. (2012). Psychosocial Stressors and Cigarette Smoking Among African American Adults in Midlife. *Nicotine & Tobacco Research, 14*(10), 1161-1169 10.1093/ntr/nts011.

Smith, M. V., Ramsay, C., & Mazure, C. M. (2014). Understanding disparities in subpopulations of women who smoke. *Curr Addict Rep, 1*(1), 69-74 10.1007/s40429-013-0002-7.

Smith, P. H., Bessette, A. J., Weinberger, A. H., Sheffer, C. E., & McKee, S. A. (2016). Sex/gender differences in smoking cessation: A review. *Prev Med, 92*, 135-140 10.1016/j.ypmed.2016.07.013.

Smith, P. H., Kasza, K. A., Hyland, A., Fong, G. T., Borland, R., Brady, K., Carpenter, M. J., Hartwell, K., Cummings, K. M., & McKee, S. A. (2015). Gender differences in medication use and cigarette smoking cessation: results from the International Tobacco Control Four Country Survey. *Nicotine Tob Res, 17*(4), 463-472 10.1093/ntr/ntu212.

Smith, P. H., Zhang, J., Weinberger, A. H., Mazure, C. M., & McKee, S. A. (2017). Gender differences in the real-world effectiveness of smoking cessation medications: Findings from the 2010-2011 Tobacco Use Supplement to the Current Population Survey. *Drug Alcohol Depend, 178*, 485-491 10.1016/j.drugalcdep.2017.05.046.

Smith, S. S., Fiore, M. C., & Baker, T. B. (2014). Smoking cessation in smokers who smoke menthol and non-menthol cigarettes. *Addiction, 109*(12), 2107-2117 10.1111/add.12661.

Smith, S. S., Piper, M. E., Bolt, D. M., Fiore, M. C., Wetter, D. W., Cinciripini, P. M., & Baker, T. B. (2010). Development of the Brief Wisconsin Inventory of Smoking Dependence Motives. *Nicotine Tob Res, 12*(5), 489-499 10.1093/ntr/ntq032.

Stahre, M., Okuyemi, K. S., Joseph, A. M., & Fu, S. S. (2010). Racial/ethnic differences in menthol cigarette smoking, population quit ratios and utilization of evidence-based tobacco cessation treatments. *Addiction, 105*(s1), 75-83

Tarantola, M. E., Heath, A. C., Sher, K. J., & Piasecki, T. M. (2017). WISDM Primary and Secondary Dependence Motives: Associations With Smoking Rate, Craving, and Cigarette Effects in the Natural Environment. *Nicotine Tob Res, 19*(9), 1073-1079 10.1093/ntr/ntx027.

Templin, J., & Jiao, H. (2012). Applying model-based approaches to identify performance category classifications. In G. Cizek (Ed.), *Setting performance standards: Theory and application* (2nd ed., pp. 379-398). New York: Routledge.

Thomas, J., Pulvers, K., Befort, C., Berg, C., Okuyemi, K. S., Mayo, M., Nazir, N., & Ahluwalia, J. S. (2008). Smoking-related Weight Control Expectancies among African American Light Smokers Enrolled in a Smoking Cessation Trial. *Addict Behav, 33*(10), 1329-1336 10.1016/j.addbeh.2008.06.011.

Tiffany, S. T. (1990). A cognitive model of drug urges and drug-use behavior: role of automatic and nonautomatic processes. *Psychol Rev, 97*(2), 147-168

Tiffany, S. T., Conklin, C. A., Shiffman, S., & Clayton, R. R. (2004). What can dependence theories tell us about assessing the emergence of tobacco dependence? *Addiction, 99 Suppl 1*, 78-86 10.1111/j.1360-0443.2004.00734.x.

Trinidad, D. R., Perez-Stable, E. J., Emery, S. L., White, M. M., Grana, R. A., & Messer, K. S. (2009). Intermittent and light daily smoking across racial/ethnic groups in the United States. *Nicotine Tob Res, 11*(2), 203-210 10.1093/ntr/ntn018.

Trinidad, D. R., Perez-Stable, E. J., White, M. M., Emery, S. L., & Messer, K. (2011). A nationwide analysis of US racial/ethnic disparities in smoking behaviors, smoking cessation, and cessation-related factors. *Am J Public Health, 101*(4), 699-706 10.2105/ajph.2010.191668.

Trinidad, D. R., Xie, B., Fagan, P., Pulvers, K., Romero, D. R., Blanco, L., & Sakuma, K.-L. K. (2015). Disparities in the Population Distribution of African American and Non-Hispanic White Smokers Along the Quitting Continuum. *Health education & behavior : the official publication of the Society for Public Health Education, 42*(6), 742-751 10.1177/1090198115577376.

USDHHS. (2010). Nicotine Addiction: Past and Present *How Tobacco Smoke Causes Disease: The Biology and Behavioral Basis for Smoking-Attributable Disease*. Atlanta, GA: Center for Disease Control and Prevention.

USDHHS. (2014). Reports of the Surgeon General *The Health Consequences of Smoking-50 Years of Progress: A Report of the Surgeon General*. Atlanta (GA): Centers for Disease Control and Prevention (US).

Vajer, P., Urban, R., Tombor, I., Stauder, A., & Kalabay, L. (2011). Psychometric properties and construct validity of the brief Wisconsin inventory of smoking dependence motives in an Internet-based sample of treatment-seeking Hungarian smokers. *Nicotine Tob Res, 13*(4), 273-281 10.1093/ntr/ntq254.

Vermunt, J. K., & Magidson, J. (2002). Latent class cluster analysis. In J. Hagenaars & A. McCutcheon (Eds.), *Applied Latent Class Analysis*. Cambridge: Camrbidge University Press.

Vinci, C., Copeland, A. L., & Carrigan, M. H. (2012). Exposure to negative affect cues and urge to smoke. *Exp Clin Psychopharmacol, 20*(1), 47-55 10.1037/a0025267.

Wang, H., & Sun, X. (2005). Desensitized nicotinic receptors in brain. *Brain Res Brain Res Rev, 48*(3), 420-437 10.1016/j.brainresrev.2004.09.003.

Wang, J., & Wang, X. (2012). *Structural Equation Modeling: Applications Using Mplus*. New York: John Wiley & Sons.

Wang, T., Wang, B., & Chen, H. (2014). Menthol facilitates the intravenous self-administration of nicotine in rats. *Front Behav Neurosci, 8* 10.3389/fnbeh.2014.00437.

Watkins, S. S., Koob, G. F., & Markou, A. (2000). Neural mechanisms underlying nicotine addiction: acute positive reinforcement and withdrawal. *Nicotine Tob Res, 2*(1), 19-37

Watson, C., Richter, P., de Castro, B. R., Sosnoff, C., Potts, J., Clark, P., McCraw, J., Yan, X., Chambers, D., & Watson, C. (2017). Smoking Behavior and Exposure: Results of a Menthol Cigarette Crossover Study. *Am J Health Behav, 41*(3), 309-319 10.5993/ajhb.41.3.10.

Watson, N. L., Carpenter, M. J., Saladin, M. E., Gray, K. M., & Upadhyaya, H. P. (2010). Evidence for greater cue reactivity among low-dependent vs. high-dependent smokers. *Addict Behav, 35*(7), 673-677 10.1016/j.addbeh.2010.02.010.

Weinberger, A. H., Platt, J., Shuter, J., & Goodwin, R. D. (2016). Gender differences in self-reported withdrawal symptoms and reducing or quitting smoking three years later: A prospective, longitudinal examination of U.S. adults. *Drug Alcohol Depend, 165*, 253-259 10.1016/j.drugalcdep.2016.06.013.

Weinberger, A. H., Platt, J. M., Smith, P. H., & Goodwin, R. D. (2017). Racial/Ethnic Differences in Self-reported Withdrawal Symptoms and Quitting Smoking Three Years Later: A Prospective, Longitudinal Examination of US Adults. *Nicotine Tob Res, 19*(3), 373-378 10.1093/ntr/ntw221.

Wellman, R. J., Savageau, J. A., Godiwala, S., Savageau, N., Friedman, K., Hazelton, J., & Difranza, J. R. (2006). A comparison of the Hooked on Nicotine Checklist and the Fagerstrom Test for Nicotine Dependence in adult smokers. *Nicotine Tob Res, 8*(4), 575-580 10.1080/14622200600789965.

West, O., Hajek, P., & McRobbie, H. (2011). Systematic review of the relationship between the 3-hydroxycotinine/cotinine ratio and cigarette dependence. *Psychopharmacology (Berl), 218*(2), 313-322 10.1007/s00213-011-2341-1.

WHO. (2010). The Impact of Tobacco Use on Women's Health. In J. M. Samet & S. Y. Yoon (Eds.), *Gender, Women, and the Tobacco Epidemic*: World Health Organization.

Wickham, R. (2015). How Menthol Alters Tobacco-Smoking Behavior: A Biological Perspective. *Yale J Biol Med, 88*(3), 279-287

Williams, D. R., Priest, N., & Anderson, N. (2016). Understanding Associations between Race, Socioeconomic Status and Health: Patterns and Prospects. *Health Psychol, 35*(4), 407-411 10.1037/hea0000242.

Williamson, D. F., Madans, J., Anda, R. F., Kleinman, J. C., Giovino, G. A., & Byers, T. (1991). Smoking cessation and severity of weight gain in a national cohort. *N Engl J Med, 324*(11), 739-745 10.1056/nejm199103143241106.

Woods-Giscombé, C. L. (2010). Superwoman Schema: African American Women's Views on Stress, Strength, and Health. *Qual Health Res, 20*(5), 668-683 10.1177/1049732310361892.

Wray, J. M., Gray, K. M., McClure, E. A., Carpenter, M. J., Tiffany, S. T., & Saladin, M. E. (2015). Gender differences in responses to cues presented in the natural environment of cigarette smokers. *Nicotine Tob Res, 17*(4), 438-442 10.1093/ntr/ntu248.

Xu, J., Azizian, A., Monterosso, J., Domier, C. P., Brody, A. L., London, E. D., & Fong, T. W. (2008). Gender effects on mood and cigarette craving during early abstinence and resumption of smoking. *Nicotine Tob Res, 10*(11), 1653-1661 10.1080/14622200802412929.

Yerger, V. B. (2011). Menthol's potential effects on nicotine dependence: a tobacco industry perspective. *Tob Control, 20*(Suppl_2), ii29-36 10.1136/tc.2010.041970.

Yu, D., Peterson, N. A., Sheffer, M. A., Reid, R. J., & Schnieder, J. E. (2010). Tobacco outlet density and demographics: analysing the relationships with a spatial regression approach. *Public Health, 124*(7), 412-416 10.1016/j.puhe.2010.03.024.

Table 1. Sample Descriptive Statistics

	Female		Male		Total Sample	
Age, mean (SD)	51.49	(11.36)	52.43	(11.7)	51.94	(11.52)
Education > HS, n (%)	229	(87.4)	202	(84.87)	431	(86.2)
Income > 200% FPL, n (%)	85	(33.33)	76	(33.93)	161	(33.61)
CPD, mean (SD)	11.65	(6.21)	13.65	(6.92)***	12.6	(6.63)
Smokes ≤ 10 CPD, n (%)	150	(57.3)	110	(46.2)*	260	(52)
Age started smoking, mean (SD)	19.58	(6.51)	17.81	(5.82)**	18.74	(6.25)
Menthol smoker, n (%)	229	(87.4)	200	(84.03)	429	(85.8)
PROMIS ND total, mean (SD)	8.21	(4.13)	7.64	(4.02)	7.94	(4.08)
PSS-4 total, mean (SD)	4.06	(3.18)	4.2	(3.11)	4.13	(3.14)

Note. FPL = federal poverty limit; CPD = average cigarettes per day; PROMIS ND = PROMIS nicotine dependence; PSS – 4 = Perceived stress scale.
$^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$

Table 2. Response Distributions for Brief WISDM Items

Item	Mean	SD	Skew	Kurtosis	Count1	Prop.1	Count2	Prop.2	Count3	Prop.3	Count4	Prop.4	Count5	Prop.5	Count6	Prop.6	Count7	Prop.7
Often_i1	4.5	2.3	-0.2	1.6	72	0.1	52	0.1	73	0.2	51	0.1	50	0.1	32	0.1	170	0.3
Control_i2	3.4	2.2	0.4	1.8	153	0.3	58	0.1	68	0.1	62	0.1	54	0.1	36	0.1	69	0.1
Wake_i3	4.8	2.3	-0.5	1.7	63	0.1	50	0.1	46	0.1	50	0.1	43	0.1	37	0.1	211	0.4
Urge_i4	4.7	2.0	-0.3	2.0	43	0.1	39	0.1	62	0.1	82	0.2	84	0.2	52	0.1	138	0.3
Flavor_i5	4.2	2.1	-0.1	1.7	69	0.1	62	0.1	69	0.1	63	0.1	73	0.2	45	0.1	119	0.2
Focused_i6	3.4	2.1	0.4	1.9	135	0.3	73	0.2	62	0.1	72	0.1	64	0.1	27	0.1	67	0.1
Eating_i7	2.7	2.1	1.0	2.6	236	0.5	68	0.1	52	0.1	41	0.1	29	0.1	24	0.1	50	0.1
Reminders_i8	3.1	2.1	0.6	2.0	168	0.3	77	0.2	65	0.1	54	0.1	49	0.1	28	0.1	59	0.1
Feel better_i9	3.7	2.1	0.2	1.8	117	0.2	55	0.1	83	0.2	66	0.1	58	0.1	39	0.1	82	0.2
Decide_i10	4.2	2.2	-0.1	1.6	87	0.2	53	0.1	57	0.1	62	0.1	68	0.1	46	0.1	127	0.3
Friend_i11	3.4	2.3	0.4	1.6	174	0.4	60	0.1	46	0.1	51	0.1	42	0.1	27	0.1	100	0.2
Smells_i12	3.7	2.3	0.2	1.6	143	0.3	61	0.1	45	0.1	59	0.1	55	0.1	39	0.1	98	0.2
Focused_i13	3.0	2.0	0.7	2.2	180	0.4	70	0.1	73	0.2	57	0.1	42	0.1	26	0.1	52	0.1
Thinking_i14	4.1	2.3	0.0	1.5	106	0.2	59	0.1	57	0.1	54	0.1	53	0.1	43	0.1	128	0.3
Taste good_i15	3.9	2.1	0.1	1.7	83	0.2	69	0.1	75	0.2	79	0.2	53	0.1	43	0.1	98	0.2
Rule_i16	3.6	2.4	0.3	1.5	157	0.3	63	0.1	41	0.1	53	0.1	41	0.1	35	0.1	110	0.2
Crave_i17	4.6	2.0	-0.2	1.8	44	0.1	48	0.1	71	0.1	78	0.2	76	0.2	46	0.1	137	0.3
People_i18	3.7	2.3	0.2	1.6	129	0.3	63	0.1	49	0.1	70	0.1	52	0.1	32	0.1	105	0.2
Weight_i19	2.2	1.9	1.5	4.0	321	0.6	42	0.1	33	0.1	35	0.1	17	0.0	6	0.0	46	0.1
Taste great_i20	3.9	2.2	0.1	1.7	98	0.2	69	0.1	58	0.1	81	0.2	52	0.1	44	0.1	98	0.2
Hooked_i21	5.5	1.9	-1.0	2.8	22	0.0	30	0.1	31	0.1	52	0.1	57	0.1	64	0.1	244	0.5
Best friend_i22	3.0	2.2	0.7	2.0	212	0.4	61	0.1	44	0.1	45	0.1	39	0.1	26	0.1	73	0.2
Urges_i23	4.4	2.1	-0.2	1.8	60	0.1	53	0.1	65	0.1	77	0.2	68	0.1	50	0.1	127	0.3
Seeing_i24	4.4	2.2	-0.2	1.7	82	0.2	45	0.1	53	0.1	62	0.1	75	0.2	54	0.1	129	0.3
Reaching_i25	4.2	2.3	-0.1	1.5	111	0.2	51	0.1	48	0.1	45	0.1	65	0.1	45	0.1	135	0.3
Alone_i26	2.9	2.1	0.8	2.2	223	0.5	56	0.1	58	0.1	39	0.1	42	0.1	25	0.1	57	0.1
Family_i27	3.7	2.3	0.2	1.5	135	0.3	71	0.1	43	0.1	57	0.1	48	0.1	36	0.1	110	0.2
Heavy_i28	3.8	2.3	0.2	1.6	118	0.2	62	0.1	63	0.1	63	0.1	43	0.1	48	0.1	103	0.2
Intolerable_i29	3.8	2.1	0.2	1.7	109	0.2	60	0.1	69	0.1	70	0.1	73	0.2	35	0.1	84	0.2
Friends_i30	3.7	2.3	0.2	1.6	134	0.3	63	0.1	55	0.1	60	0.1	45	0.1	33	0.1	110	0.2
Awake30_i31	5.2	2.3	-0.8	2.0	69	0.1	44	0.1	29	0.1	16	0.0	33	0.1	51	0.1	258	0.5
Think_i32	3.0	2.1	0.7	2.2	178	0.4	77	0.2	59	0.1	65	0.1	38	0.1	25	0.1	58	0.1

Feel better_i33	3.3	2.1	0.5	1.8	151	0.3	70	0.1	65	0.1	58	0.1	55	0.1	31	0.1	70	0.1
Overeating_ i34	2.6	2.2	1.0	2.5	262	0.5	61	0.1	35	0.1	28	0.1	33	0.1	20	0.0	61	0.1
Out of control_i35	5.0	2.2	-0.6	1.9	65	0.1	35	0.1	37	0.1	50	0.1	59	0.1	39	0.1	215	0.4
Heavy smoker_i36	4.8	2.3	-0.5	1.7	77	0.2	44	0.1	27	0.1	58	0.1	46	0.1	57	0.1	191	0.4
Feel better_i37	3.6	2.3	0.3	1.6	141	0.3	72	0.1	56	0.1	45	0.1	52	0.1	40	0.1	94	0.2

Note. Columns to the right of the kurtosis column contain the count and proportion of the sample endorsing a particular Likert response (1-7) for a particular item.

Table 3. Model Fit Statistics for Confirmatory Factor Analyses

Model	AIC	BIC	χ^2	*df*	RMSEA	[95% CI]	CFI	TLI	SRMR
1-factor	73275.74	73743.56	4130.01	629	0.11	[0.10, 0.11]	0.64	0.62	0.09
11-factor	69960.22	70659.84	1457.92	574	0.06	[0.05, 0.06]	0.91	0.90	0.05
† 11factor+ Covariances.	69494.15	70210.63	1070.15	570	0.04	[0.04, 0.05]	0.95	0.94	0.04
10-factor	62195.43	62802.33	928.63	450	0.05	[0.04, 0.05]	0.94	0.93	0.04
10-factor + Covariances	62110.33	62721.45	856.63	449	0.04	[0.04, 0.05]	0.95	0.94	0.04
10-factor Higher Order	62219.06	62691.1	993.23	482	0.05	[0.04, 0.05]	0.94	0.93	0.05

Note. AIC = Akaike Information Criteria, BIC = Bayesian Information Criteria, df = Degrees of Freedom, RMSEA = Root Mean squared error of approximation, CFI = comparative fit index

† Model yielded out-of-bounds parameter estimate and therefore model results cannot be considered valid.

Table 4. Subscale Descriptive Statistics, Reliability and Latent Variable Correlation Matrix

	Mean (SD)		ω [95% CI]		1	2	3	4	5	6	7	8	9	10
1. AUTO	4.2	(2)	0.9	[0.89, 0.92]	1									
2. CRAV	4.3	(1.7)	0.9	[0.84, 0.88]	0.78	1								
3. LOC	4.4	(1.7)	0.8	[0.79, 0.84]	0.84	0.93	1							
4. COG	3.1	(1.8)	0.8	[0.8, 0.87]	0.57	0.66	0.59	1						
5. CUE	3.7	(1.8)	0.7	[0.69, 0.77]	0.69	0.84	0.75	0.69	1					
6. GOAD	3.7	(2.1)	0.9	[0.89, 0.93]	0.3	0.36	0.33	0.29	0.46	1				
7. TAST	4	(1.9)	0.8	[0.84, 0.89]	0.47	0.62	0.47	0.5	0.53	0.28	1			
8. AFF	3.1	(2)	0.9	[0.83, 0.89]	0.64	0.71	0.67	0.78	0.7	0.29	0.58	1		
9. WGHT	2.5	(1.7)	0.8	[0.75, 0.84]	0.39	0.47	0.38	0.62	0.57	0.19	0.39	0.58	1	
10. AE	3.5	(1.9)	0.8	[0.78, 0.85]	0.67	0.8	0.73	0.89	0.76	0.38	0.65	0.92	0.63	1

Note. All bivariate correlations were statistically significant at $p < .05$.
AUTO = Automaticity; CRAV = Craving; LOC = Loss of control; COG = Cognitive enhancement; CUE= Cue reactivity/associative processes; GOAD = Social/environmental goads; TAST = Taste; AFF = Affiliative attachment; WGHT = Weight control; AE = Affective enhancement

Table 5. Fit Statistics for Factor-Mixture Latent Profile Models

Model	LL	AIC	BIC	aBIC	Entropy	VLMR Test Value	*p*-value	BLRT Test Value	*p*-value[†]
1-Profile	-32385.7	64971.4	65392.9	65075.5	-	-	-	-	-
2-Profile	-31456.9	63135.8	63603.6	63251.3	0.92	1830.8	0.00	1857.6	0.00
3-Profile	-31067.4	62378.9	62893.1	62505.8	0.91	767.7	0.00	778.9	0.00
4-Profile	-30975.1	62216.3	62776.8	62354.7	0.87	181.9	0.15	184.6	0.00
5-Profile	-30874.9	62037.7	62644.6	62187.6	0.89	201.6	0.12[¶]	204.6	0.00
6-Profile	-30821.9	61953.5	62607	62115	0.87	104.5	0.37	106	0.00

Note. LL = log-likelihood; AIC = Akaike Information Criteria; BIC = Bayesian Information Criteria; aBIC = sample size adjusted BIC; VLMR = Vuong-Lo-Mendell-Rubin; BLRT = Bootstrapped Likelihood Ratio Test

[†] Due to estimation difficulties, the *k*-1 log-likelihood could not be replicated, thus the *p*-value for the BLRT may not be accurate.

[¶] Due to estimation difficulties, the *k*-1 log-likelihood could not be replicated, thus the *p*-value for the VLMR for the 5-profile model may not be accurate.

Table 6. Model Implied Descriptive Statistics for the 3-Profile Model

	Low PDM		Medium PDM		High PDM	
Female, % (SE)	47.2	(0.04)	56.7	(0.04)	51.9	(0.05)
Age initiated smoking, mn (SE)	19.51	(0.51)	18.84[b]	(0.47)	17.37[c]	(0.42)
Light smoker, % (SE)	72.7[a]	(0.04)	47.8[b]	(0.04)	28.6[c]	(0.04)
Menthol	*	*	*	*	*	*
PROMIS, mn (SE)	4.65[a]	(0.24)	8.65[b]	(0.23)	11.55[c]	(0.30)
PSS-4, mn (SE)	3.14[a]	(0.23)	4.49	(0.22)	4.92[c]	(0.32)

[a] Low vs. Medium PDM, $p < .05$; [b] Medium vs. High PDM, $p < .05$; [c] Low vs. High PDM, $p < .05$.
Note. SE = Standard Error; *PDM* = Primary Dependence Motives
* Because of low numbers of non-menthol smokers in this sample, the estimates for the Menthol variable could not be computed with precision and are thus not reported.

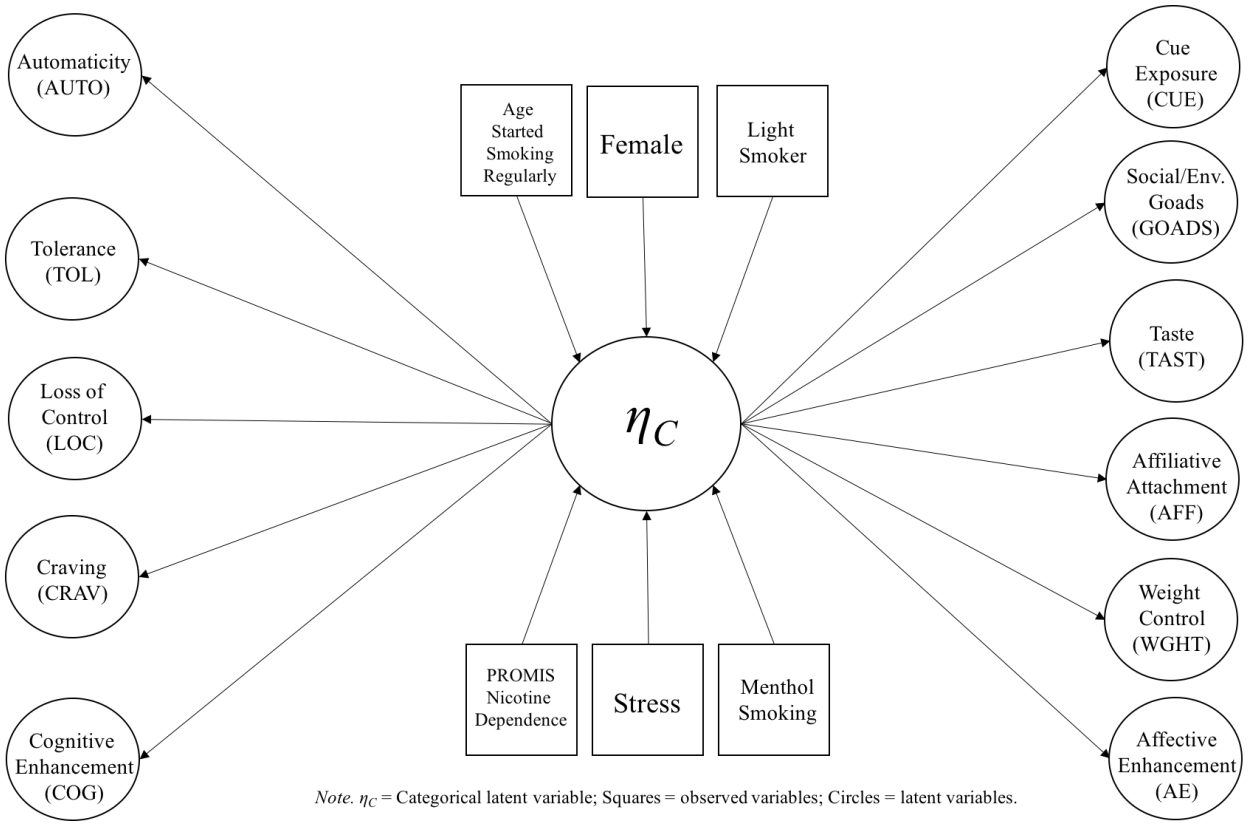
Table 7. Predictors of Profile Membership

	OR	*b*	SE	*p*-value
Low PDM vs. High PDM*				
Female	0.69	-0.37	0.37	0.31
Age of Initiated Smoking	1.06	0.06	0.04	0.11
Light Smoking	2.44	0.89	0.38	0.02
Menthol	1.15	0.14	0.50	0.78
PROMIS-ND	0.51	-0.67	0.08	0.00
PSS4	0.83	-0.18	0.07	0.01
Medium PDM vs. High PDM*				
Female	1.21	0.19	0.27	0.48
Age of Initiated Smoking	1.04	0.04	0.03	0.10
Light Smoking	1.36	0.31	0.30	0.30
Menthol	0.95	-0.05	0.38	0.89
PROMIS-ND	0.75	-0.29	0.05	0.00
PSS4	0.96	-0.04	0.04	0.31

Note. PROMIS-ND = PROMIS Nicotine Dependence; PSS4 = Perceived Stress Scale-4; *OR = odds ratio; b = coefficient on logit scale; SE = standard error*

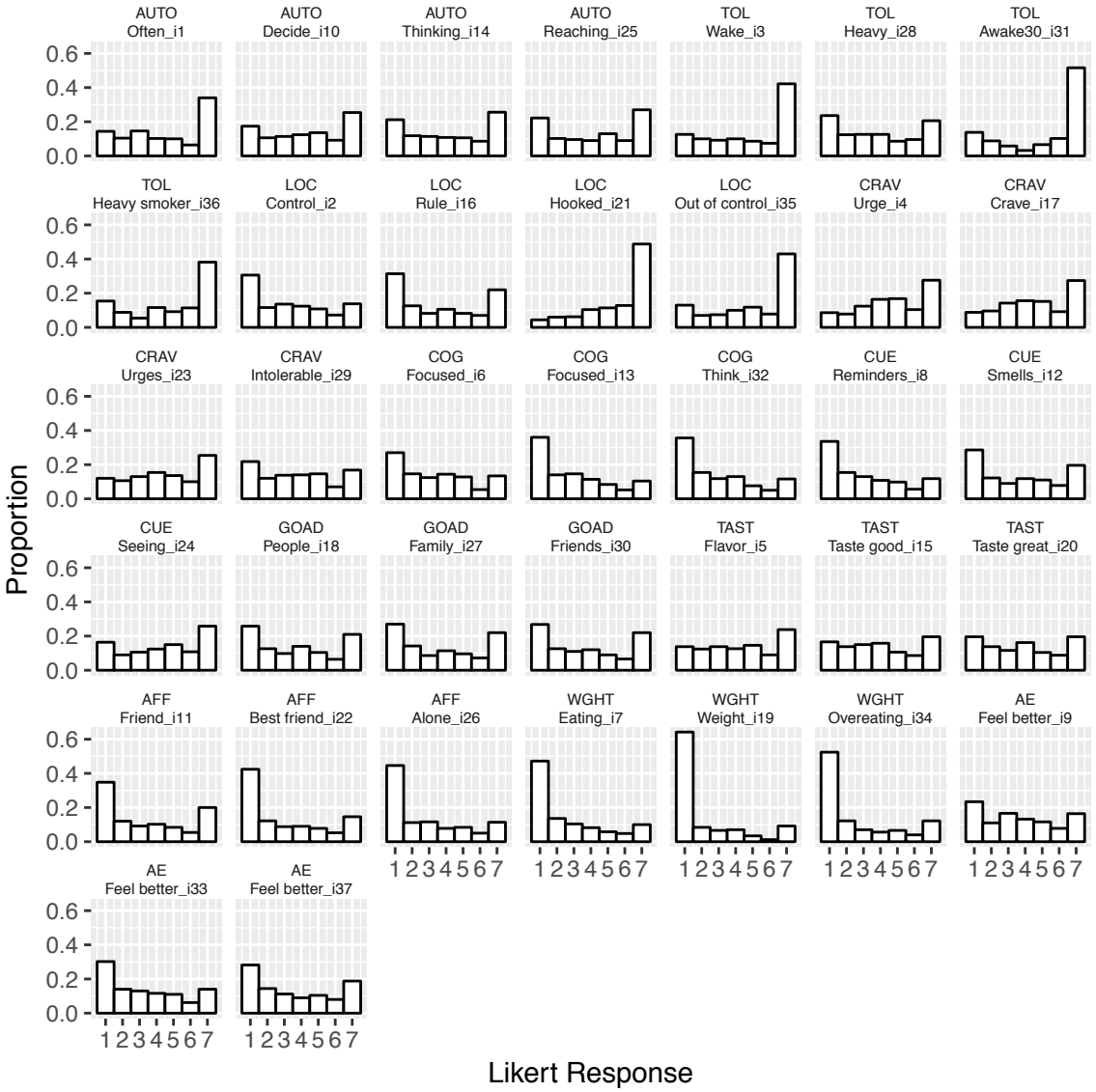
*For each multinomial logistic regression model, the High PDM profile is the reference group.

Figure 1. Latent Profile Model Diagram



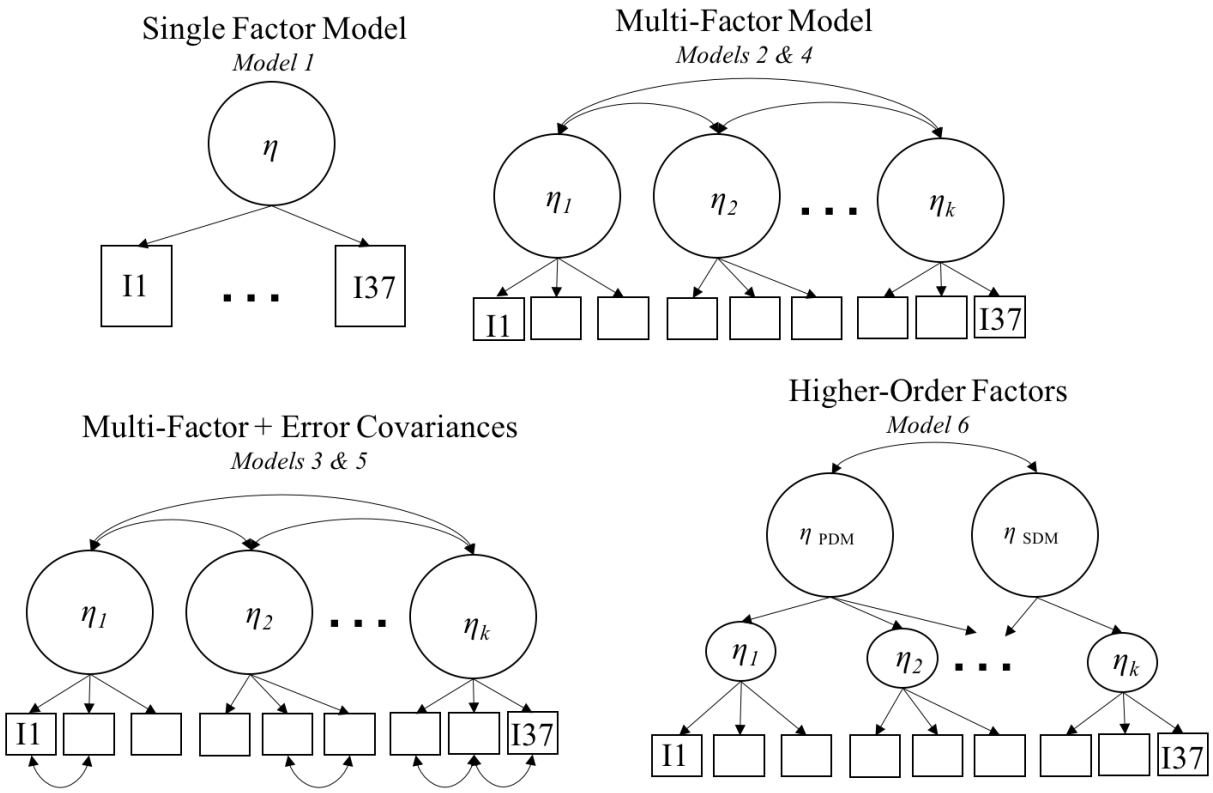
Note. η_C = Categorical latent variable; Squares = observed variables; Circles = latent variables.

Figure 2. *Brief WISDM* Item Response Distributions



Note. The heading in all capital letters for each histogram corresponds to the B-WISDM subscale that each item belongs to. The other heading refers to the core theme of each item and the number of the item.

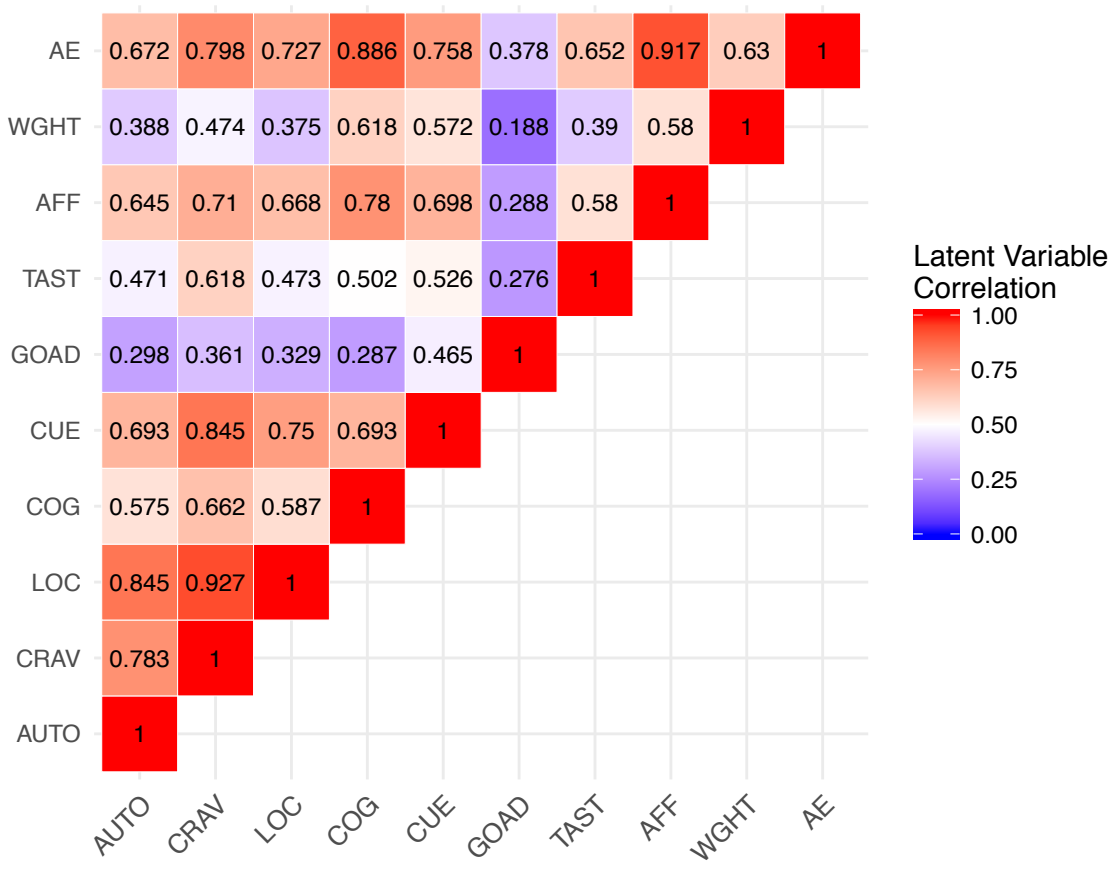
Figure 3. Schematic Diagrams of Confirmatory Factory Analytic Models



Note. Circles denote latent variables (or factors) and rectangles denote items. Single headed arrows from the circles to rectangles denote factor loadings. Double-headed arrows between circles represent latent variable correlations. Double-head arrows between rectangles denote correlated item residual variances, or error covariances.

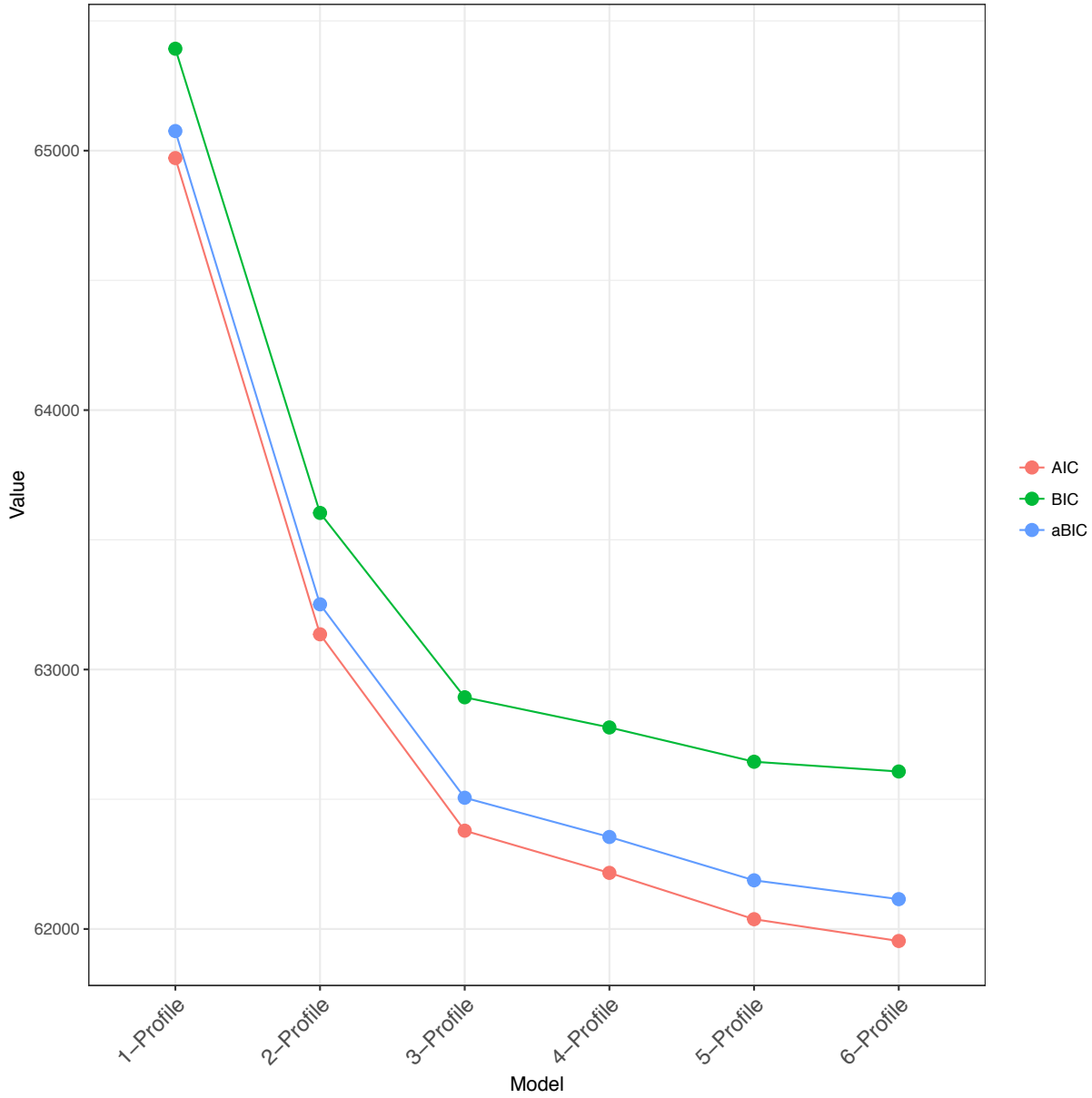
The figure visually depicts the factor structures implied by the CFA models reported in Table 4. The names of the models that correspond to the models described in Table 4 are in italics. Of note, what differentiates Model 2 from 4 and Model 3 from 5 is the number of latent variables included in each model. This is why the subscript *k* is used and not the number of factors, or subscales, implied by the Brief WISDM.

Figure 4. Latent Variable Bivariate Correlation Heatmap Plot



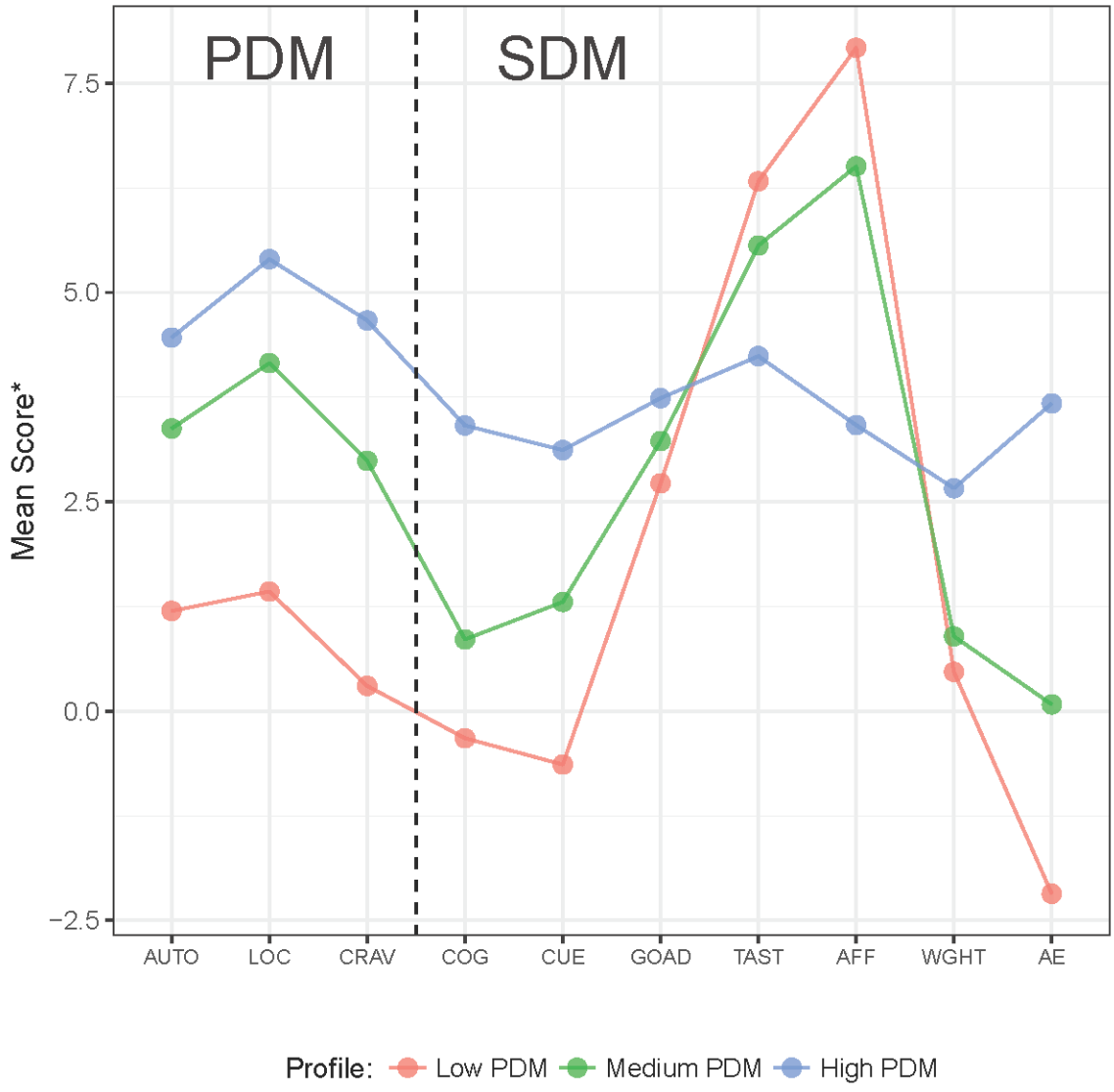
Note. Auto = Automaticity; LOC = Loss of control; CRAV = craving COG = Cognitive enhancement; CUE = Cue Exposure/Associative Processes; GOAD = Social goads; TAST = Taste; AFF = Affiliative Attachment; WGHT = Weight control; AE = Affective Enhancement. All correlations are statistically significant at $p < 0.05$.

Figure 5. Plot of Latent Profile Model Fit Statistics



Note. AIC = Akaike Information Criteria; BIC = Bayesian Information Criteria; aBIC = sample size adjusted BIC

Figure 6. Profile Plot for 3-Profile Model



Note. Auto = Automaticity; LOC = Loss of control; CRAV = craving COG = Cognitive enhancement; CUE = Cue Exposure/Associative Processes; GOAD = Social goads; TAST = Taste; AFF = Affiliative Attachment; WGHT = Weight control; AE = Affective Enhancement; PDM =Primary dependence motives; SDM = Secondary dependence motives. Model implied posterior probabilities of profile membership were 34% for the Low PDM profile, 43.5% for the Medium PDM profile and 22.5% for the High PDM profile.

*The scale for the y-axis is arbitrary due to latent variables serving as indicators fo r the LPA rather than observed indicators. The scale was determined by setting the latent variable means for profile 3 to the latent means for each latent variable from the overall sample.

Appendix

Brief *WISDM* Subscales and Descriptions

Subscale	Description	Example Item
	Primary Dependence Motives (PDM)	
Automaticity (AUTO)	This scale is informed by Tiffany's (1990) Automaticity Theory, which posits that over time drug use becomes an overlearned and largely automatic behavior. In this theory, use is not governed by craving, rather subjective craving occurs only when automatic use is blocked.	"I often smoke without thinking about it."
Tolerance (TOL)	Tolerance refers to the upregulation of receptors in response to increasing doses of a drug, results in the ability to tolerate greater doses of a drug without toxic effects and the requirement of greater amounts of a drug to achieve a desired effect. This subscale overlaps with the FTND and incorporates TTFC and indexes withdrawal-induced motivation to smoke and habitual smoking,	"I smoke within the first 30 minutes of awakening in the morning."
Loss of Control (LOC)	Although not exactly a motivation to smoke, the subjective loss of control over smoking is considered to be indicative of compulsive drug use (DiFranza et al., 2002).	"Cigarettes control me."
Craving (CRAV)	This subscale refers to smoking in order to alleviate a craving or urge to smoke.	"It's hard to ignore an urge to smoke."
	Secondary Dependence Motives (SDM)	
Cognitive Enhancement (COG)	Smoking enhances concentration, as indicated by cognitive testing and self-report. This subscale indexes the motivation to smoke in order to improve concentration.	"I frequently smoke to keep my mind focused."
Cue Exposure/Associative Processes (CUE)	Because smoking and withdrawal occur within myriad cue rich environments, associative learning is a formidable mechanism underlying nicotine dependence. This subscale reflects the motivation to smoke in relation to cues.	"My life is full of reminders to smoke."
Social/Environmental Goads (GOAD)	During the maintenance of smoking, social environments contribute to persistent smoking through the provision of cues and permissive beliefs regarding smoking.	"Most of the people I spend time with are smokers."
Taste (TAST)	The taste of cigarette is a reinforcing stimuli component of smoking for uninitiated and initiated smokers alike. This subscale assesses the extent to which smokers are motivated to smoke by the taste of cigarettes.	"The flavor a cigarette is pleasing."
Affiliative Attachment (AFF)	Nicotine activates neural systems related to affective responses to social stimuli. The items of this subscale reflect the tendency for smoking to be motivated by the social satisfaction of smoking itself.	"Cigarettes keep me company, like a close friend."
Weight Control (WGHT)	Smokers frequently smoke to control weight and appetite. This subscale reflects this motivation to smoke.	"I rely upon smoking to control my hunger and eating."
Affective Enhancement (AE)	Smokers report smoking to enhance and affect and/or alleviate dysphoria resulting from withdrawal. This subscale reflects this motivation to smoke.	"Smoking helps me feel better in seconds."

Sources: Piper et al., 2004, p. 151; Smith et al., 2010