A Multiple Motives Approach to Tobacco Dependence: The Wisconsin Inventory of Smoking Dependence Motives (WISDM-68)

Megan E. Piper University of Wisconsin Medical School, Madison and University of Wisconsin–Madison Thomas M. Piasecki University of Missouri–Columbia

E. Belle Federman Research Triangle Institute International Daniel M. Bolt University of Wisconsin–Madison

Stevens S. Smith, Michael C. Fiore, and Timothy B. Baker University of Wisconsin Medical School, Madison and University of Wisconsin–Madison

The dependence construct fills an important explanatory role in motivational accounts of smoking and relapse. Frequently used measures of dependence are either atheoretical or grounded in a unidimensional model of physical dependence. This research creates a multidimensional measure of dependence that is based on theoretically grounded motives for drug use and is intended to reflect mechanisms underlying dependence. Data collected from a large sample of smokers (N = 775) indicated that all 13 subscales of the Wisconsin Inventory of Smoking Dependence Motives (WISDM-68) have acceptable internal consistency, are differentially present across levels of smoking heaviness, and have a multidimensional structure. Validity analyses indicated the WISDM-68 subscales are significantly related to dependence criteria such as smoking heaviness and to 4th edition *Diagnostic and Statistical Manual of Mental Disorders* symptoms of dependence and relapse.

Despite the well-publicized dangers of smoking, some 46.5 million American adults currently smoke cigarettes (Centers for Disease Control and Prevention [CDC], 2002). Smoking prevalence rates remain fairly high because once an individual smokes regularly, it is unlikely that he or she will be able to quit easily. In 2000, 15.7 million adult smokers tried to quit smoking, but only 4.7% of smokers who reported daily smoking during the previous year were abstinent for 3–12 months in 2000 (CDC, 2002). It is

Megan E. Piper and Timothy B. Baker, Center for Tobacco Research and Intervention, University of Wisconsin Medical School, Madison, and Department of Psychology, University of Wisconsin–Madison; Thomas M. Piasecki, Department of Psychological Sciences, University of Missouri–Columbia; E. Belle Federman, Research Triangle Institute International, Research Triangle Park, North Carolina; Daniel M. Bolt, Department of Educational Psychology, University of Wisconsin–Madison; Stevens S. Smith and Michael C. Fiore, Center for Tobacco Research and Intervention, University of Wisconsin Medical School, Madison, and Section of General Internal Medicine, Department of Medicine, University of Wisconsin–Madison.

This research was supported in part by Grant P50-CA84724 from the National Cancer Institute. Thomas M. Piasecki was supported in part by a grant from the University of Missouri Research Board. We thank Thomas H. Brandon, David G. Gilbert, Jack Henningfield, John Hughes, Raymond Niaura, and Stephen Tiffany for their assistance in developing and reviewing the 13 motivational domains.

Correspondence concerning this article should be addressed to Megan E. Piper, Center for Tobacco Research and Intervention, 1930 Monroe Street, Suite 200, Madison, WI 53711-2027. E-mail: mep@ctri.medicine.wisc.edu

estimated that, on average, adolescent boys who start smoking now will smoke for approximately 16 years and adolescent girls who start smoking now will smoke for at least 20 years before being able to quit (Pierce & Gilpin, 1996). What is it that makes smoking so refractory despite the high personal costs that are eventually exacted by smoking?

Since the 1980s, broad scientific consensus has developed around the idea that smokers become dependent on nicotine and that tobacco dependence is in fact the primary factor maintaining smoking behavior among adult smokers (U.S. Department of Health and Human Services, 1988). Consequently, tobacco dependence has been assigned a heavy explanatory and clinical burden-it is invoked to account for smoking withdrawal symptoms and relapse, for individual differences in the nature of tobacco motivation, and as a guide to treatment assignment (e.g., Breslau & Johnson, 2000; Fagerström & Schneider, 1989; Killen, Fortmann, Kraemer, Varady, & Newman, 1992; Niaura, Goldstein, & Abrams, 1994). Although dependence is certainly a crucial construct, there is little agreement as to its nature (Fagerström, 1978; Kenford et al., 2002; Robinson & Berridge, 1993). Perhaps if we could develop a clearer understanding of its nature and motivational mechanisms, we might be better positioned to prevent its development in novice smokers and weaken its grip on dependent smokers. Achieving these goals requires a systematic bootstrapping approach to establishing the construct validity of tobacco dependence measures (Cronbach & Meehl, 1955).

Such a research enterprise requires clear theoretical formulations of tobacco dependence; generation of theoretically congruent measure(s); and systematic, iterative tests that determine whether

the measured construct is related to other variables in a theoretically ordained manner. The results of such tests would guide the future refinement of the measure. Where theory-congruent relations are detected empirically, they corroborate both the construct and the measurement tool simultaneously. When these relations are not found, they must inspire reexamination of the dependence construct definition and/or the measurement tools developed to tap dependence.

To date, research in tobacco dependence has not followed this sort of orderly, iterative, bootstrapping approach to construct explication and measure refinement. To a large extent, the field has invoked the dependence construct without a clear specification of its nature or structure. Perhaps owing to this, investigators have, for decades, relied on a handful of relatively blunt dependence measures, even while bemoaning their validity and psychometric properties (e.g., Breslau & Johnson, 2000; Lichtenstein & Mermelstein, 1986). The slow progress of smoking cessation research (e.g., Piasecki & Baker, 2001; Shiffman, 1993a) may, in part, be attributable to the lack of a clear definition of dependence and an inability to measure it sensitively.

Two Traditions in Dependence Assessment

A meaningful measure should arise from careful consideration of the nature of the construct that it is intended to tap and the uses to which the measure is to be put. Disagreement about the meaning of the term *dependence* has plagued tobacco research, and this has almost certainly slowed measurement innovation. There are two broad traditions in tobacco-dependence measurement—one that is based on a medico—psychiatric diagnostic tradition (e.g., American Psychiatric Association, 1994) and one that is inspired by physical-dependence models (e.g., Fagerström, 1978; see Colby, Tiffany, Shiffman, & Niaura, 2000). These traditions differ in their assumptions about the structure of dependence and the explanatory burden they shoulder.

The medico-psychiatric tradition in tobacco dependence is exemplified by diagnostic criteria for tobacco dependence used in recent editions of the Diagnostic and Statistical Manual of Mental Disorders (DSM; American Psychiatric Association, 1980, 1987; 4th ed.; DSM-IV; American Psychiatric Association, 1994). In this tradition, dependence is essentially a binary variable—one is either dependent on nicotine or not. The presence of dependence is inferred (measured) by the display of various diagnostic criteria or features of the prototypic dependence syndrome, as determined by expert consensus (e.g., compulsive use of tobacco, craving or withdrawal symptoms contingent on abstinence). A tobaccodependence diagnosis is essentially a classification, not an explanatory construct. It is designed to identify persons in unselected populations who smoke heavily and persistently. For instance, a dependence diagnosis may be useful in epidemiological research for indentifying rates of dependence, comorbidity of dependence with other disorders, and so on. Tobacco-dependence diagnoses tell us whether someone is or is not addicted to nicotine, but they offer little explanation as to why this is the case and may be insensitive to differences among those labeled dependent.

A second approach has been to generate dependence measures on the basis of particular models of dependence. The widely used Fagerström Test for Nicotine Dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerström, 1991) and the Fagerström Tolerance Questionnaire (FTQ; Fagerström, 1978) represent examples of such measures. In this approach, dependence is construed as a continuously scaled variable—persons are assumed to vary in the degree of their dependence. In contrast to the diagnostic tradition, the Fagerström scales do arise from a particular explanatory model of dependence; the development of these scales was guided by the belief that signs and symptoms of dependence (such as those codified in the *DSM*) arise from a physical dependence—tolerance process (which is thought to motivate compulsive use of tobacco; Fagerström & Schneider, 1989). Thus, the Fagerström scales are designed to assess gradations in dependence, and these gradations are assumed to reflect magnitude of physical dependence—tolerance processes.

However, there are limitations to the Fagerström scales: (a) their assumption that a single tolerance dimension is adequate to capture meaningful individual differences in dependence severity and (b) their psychometric properties. These scales are based on the assumption that the most important and predictive component of dependence is physical dependence (and resulting withdrawal severity). The FTQ and FTND do not attempt to assess directly more specific motivational factors that may affect the dependence construct (e.g., reinforcement value of smoking, magnitude of abstinence-induced urges). Thus, these instruments do not sample the universe of processes that addiction researchers have postulated give rise to dependence outputs. In addition, the Fagerström questionnaires directly assess smoking heaviness (e.g., How many cigarettes do you smoke each day?). When scores from these measures are used to predict common validation criteria that are proxies for smoking heaviness (e.g., breath carbon monoxide [CO]), criterion contamination may inflate the obtained correlations. This criterion contamination may create unfounded beliefs regarding what the Fagerström tests measure and how well they measure it.

Second, two psychometric concerns have dogged the Fager-ström scales: poor internal consistency (e.g., $\alpha s = .51-.55$; Lichtenstein & Mermelstein, 1986) and questions about their structures (Lichtenstein & Mermelstein, 1986). Even when Heatherton et al. (1991) modified the FTQ and created the six-item FTND, the internal consistency was only marginally improved to $\alpha = .61$. Although the FTND is more reliable than the FTQ (Payne, Smith, McCracken, McSherry, & Antony, 1994; Pomerleau, Carton, Lutzke, Flessland, & Pomerleau, 1994), the reliability coefficients are still below traditionally accepted standards for clinical use (Nunnally & Bernstein, 1994). Consistent with the low internal consistency, factor analyses suggest that the Fagerström scales may be multifactorial (e.g., Lichtenstein & Mermelstein, 1986; Payne et al., 1994).

Despite the psychometric problems with the FTQ and FTND, they are both frequently used measures of tobacco dependence, perhaps because they are brief and because they measure smoking heaviness and predict efficacy of different doses of nicotine replacement (e.g., Fagerström & Schneider, 1989). However, a recent study by Garvey et al. (2000) showed that 4-mg gum did not significantly improve quit rates for high versus low scorers on the FTND. In addition, although the FTND predicts smoking heaviness quite well, it is unclear that it predicts important dependence criteria such as withdrawal severity and blood cotinine level better than do measures such as the number of cigarettes smoked per day (Breslau & Johnson, 2000; Gilbert, Crauthers, Mooney, McCler-

non, & Jensen, 1999; Hughes & Hatsukami, 1986). This inability to predict important outcomes consistently, above and beyond those predicted by smoking heaviness per se, suggests that the Fagerström measures tap only a narrow aspect of dependence.¹

Dependence as a Multifactorial, Continuous, Explanatory Variable

The current article describes the development of the 68-item Wisconsin Inventory of Smoking Dependence Motives (WISDM-68), a measure intended to redress the shortcomings of previous tobacco-dependence measures. The validation enterprise began with theoretically derived dependence subscales and the implicit assumption that dependence is multidimensional. The current approach attempts to define and measure dependence on the basis of *motivations*, derived from a broad sampling of theoretical domains, which might lead to compulsive or problematic drug (nicotine) use. Thus, the WISDM-68 measures the degree of different motivational forces present, which are intended to illuminate mechanisms underlying compulsive drug use. Motives are not equivalent to dependence, but in this measure, they are serving as indicators of the underlying latent variable of tobacco dependence.

We assessed motives for dependent or addictive tobacco use for several reasons. First, we believe that considerable research shows that dependence (as opposed to physical dependence) is, at heart, a motivational phenomenon. Dependence is not captured by the presence of internal states, behaviors, or external situations alone but by the organism's disposition to respond to such states or phenomena with drug use. This view of dependence accords with numerous observations, such as the fact that medical patients may become physically dependent on opiates but, unless the withdrawal syndrome spurs drug seeking, other manifestations of dependence are not observed (Mansky, 1978). Similarly, states or events such as negative affects or exposure to smoking cues should not index dependence unless they spur drug (tobacco) seeking. Therefore, we posited that the accurate assessment of the level and type of dependence requires inferences about the strength of drug seeking in the context of the motivational prod.

We also believed that the assessment of dependence motives would be more likely to reveal the multidimensional nature of tobacco dependence, especially across the ontogeny of dependence. This is because we believe that although motives for tobacco seeking may differ both across ontogeny and across individuals, chronic tobacco use will inevitably produce a core set of manifestations that do not sensitively reflect these differences. Thus, chronic smoking will likely produce tolerance and personal costs (e.g., ill health) regardless of the motives that instigate use. These residues of dependence motivational processes may reflect the existence of dependence but not important information about its nature.² These residues may be thought of as end states of disease (e.g., dementia) that do not reflect important etiologic information (e.g., normal pressure hydrocephalus, lacunar states).

Previous scales have attempted to assess motives or "reasons" for smoking. Indeed, smoking motivations have been studied for more than 30 years (e.g., Ikard, Green, & Horn, 1969; McKennell, 1970; Russell, Peto, & Patel, 1974). However, it is important to note that the WISDM-68 differs from those earlier scales in some important respects. First, the subscales of the WISDM-68 were not based on smokers' commonly volunteered "reasons for smoking."

Rather, subscales were based on current research and theories of drug motivation. This strategy resulted in assessment of constructs that are distinct from those targeted by earlier "reasons" scales. For instance, the Behavioral Choice–Melioration and Affiliative Attachment subscales are ones that have no counterparts in earlier measures. Thus, the distinct origins of the WISDM-68 subscales result in a broader, more far-ranging measure.

Second, earlier scales were not constructed or validated to optimize discriminant validity with respect to dependence criteria (e.g., withdrawal dimensions, relapse). Therefore, items were not selected, and subscales were not constructed, so as to promote the assessment of dependence. As has been observed previously (e.g., Clark & Watson, 1995), validity does not inhere in instruments. Rather, one validates the *inferences* that one can make on the basis of responses to them. Therefore, one difference between the WISDM-68 and earlier measures is that only the WISDM-68 was constructed and validated to permit inferences about dependence (e.g., its items were generated by theories of dependence and gauged with respect to their relations with dependence criteria).

In addition to conceptual differences, the current measure of dependence is intended to demonstrate psychometric attributes that are superior to those of extant measures, including that the subscales have adequate internal consistency in diverse populations (e.g., ethnic minorities).

A Multiple-Motive Measurement Strategy

Through a careful review of the literature, and after polling experts in the field, we identified 13 separate motives for drug use. These motives arose from research and theory about the motivational bases of compulsive or heavy drug use. The motives, and the developmental processes that led to their assessment, are discussed later. See Appendix A for a more complete description of the 13 domains.

The model guiding the current effort is based on the notion that multiple domains, or different motives for drug use, can be used to infer or assess the construct of tobacco dependence. In other words, smokers may have many different motives for smoking, and each motive may contribute to compulsive drug use, withdrawal, and relapse—the three key criteria of dependence. In theory, dependence may be a function not only of strength of motivation but also of the number and types of motivational

¹ It should be noted that there are new measures of dependence entering the field, such as the Nicotine Dependence Syndrome Scale (Shiffman, Hickcox, Gnys, Paty, & Kassel, 1995; Shiffman & Sayette, 2002) and the Cigarette Dependence Scale (Etter, Le Houezec, & Perneger, 2003). These measures are based heavily on the *DSM* diagnostic criteria. However, we are not aware of articles in peer-reviewed journals on these instruments that have demonstrated their predictive validity with regard to relapse latency and withdrawal severity. For example, the Etter et al. (2003) article assessed relapse likelihood and found that none of the dependence scores yielded by the Etter measures were significantly related to relapse occurrence.

² It is important to note that most researchers studying dependence to other drugs directly assess dependence criteria; that is, the residues of dependence (e.g., in the case of alcohol; Edwards, 1986). Currently, we do not know whether a motivational approach would be effective in assessing these other drug dependencies.

processes involved. These different aspects of dependence may differ not only with respect to their relations with the various dependence criteria but also with respect to course over ontogeny. Eventually, dependence scales will be validated against a broad range of validation criteria (e.g., behavioral economic measures, withdrawal severity). The present report presents data on scale content, internal consistency and structure, and concurrent validity and modest information on predictive validity.

Method

Questionnaire Development

The first step in the development of the WISDM-68 was to identify and define the relevant candidate motive domains and write sample items to assess the proposed domains. Subsequently, various experts in the field were invited to evaluate the identified motives. After receiving the feedback from the expert panel, the motive domains were revised but no new domains were added. Once the domains were finalized, numerous items were generated to assess each domain.

Items were written to ensure adequate sampling of the entirety of each motive (e.g., its frequency, breadth, intensity, and variability). For example, cravings may vary in frequency, intensity, and controllability. Some smokers may have frequent mild cravings, whereas others may have infrequent but very intense cravings. In addition, smokers may differ in the number of smoking cues they encounter, the frequency with which they encounter cues, and the salience of the cues. The total item set used to generate the questionnaire comprised 285 questions assessing the 13 motivational domains. (See Appendix A for a description of the 13 domains.)

The WISDM-68 is based on the supposition that dependence should be related to the following 13 motives for drug use:

Affiliative attachment: Characterized by a strong emotional attachment to smoking and cigarettes;

Automaticity: Characterized by smoking without aware-

ness or intention;

Behavioral choicemelioration:

Characterized by smoking despite constraints on smoking or negative consequences and/or the lack of other options or reinforcers;

Cognitive enhancement: Characterized by smoking to improve

cognitive functioning (e.g., attention);

Craving: Characterized by smoking in response to craving or experiencing intense and/or frequent urges to smoke;

processes:

Cue exposure-associative Characterized by frequent encounters with nonsocial smoking cues or a strong perceived link between cue exposure and the desire or tendency to smoke;

Loss of control: Based on the notion that once dependence becomes ingrained, the dependent person

believes that he or she has lost volitional control over drug use because of any of a variety of factors (e.g., urges, loss of

other reinforcers, automaticity);

Negative reinforcement: Characterized by the tendency or desire to smoke to ameliorate a variety of negative

internal states (e.g., dysphoria, stress,

withdrawal);

Characterized by the desire to smoke to Positive reinforcement:

experience a "buzz" or a "high" or to enhance an already positive feeling or

experience:

Social-environmental

goads:

Characterized by social stimuli or contexts that either model or invite smoking;

Taste and sensory properties:

Characterized by the desire or tendency to smoke in order to experience the orosensory and/or gustatory effects of smoking;

Tolerance: Characterized by the principal need of

individuals to smoke increasing amounts over time in order to experience the desired effects or the ability to smoke large

amounts without acute toxicity;

Weight control: Characterized by the use of cigarettes to

control body weight or appetite.

Participants

A sample of 775 participants (303 men, 454 women, and 18 not identified) from Madison and Milwaukee, Wisconsin were recruited through solicitation of participants from previous smoking cessation experiments, through newspaper and radio advertisements, and from students taking psychology classes at the University of Wisconsin-Madison. Participants were at least 18 years old, and they had to have smoked at least one cigarette within the past 14 days. 638 (82%) participants identified themselves as White, 83 (11%) as African American, 5 (1%) as American Indian, 13 (2%) as Asian/Pacific Islander, 18 (2%) responded with "other," and 18 (2%) did not supply information on race. Hispanics constituted 3% of the sample. See Table 1 for demographic information by smoking status. It should be noted that 2 individuals did not report current smoking status.

Procedure

Participants were invited to attend a large group survey session to complete the questionnaires and provide a breath sample for CO measurement. During the survey session, an overview of the study was provided and participants read and signed the consent form. Participants then completed the research questionnaires. After completing the forms, the participants were given a CO breath test and excused. Participants from the Madison and Milwaukee community received \$30 in exchange for their participation. Students taking psychology classes at the University of Wisconsin-Madison received class credit in exchange for participation.

Measures

WISDM. The preliminary WISDM comprised 285 items designed to assess the 13 different theoretically derived motivational domains listed previously. Each item is answered on a 7-point Likert scale ranging from 1 (not true of me at all) to 7 (extremely true of me).

FTND. The FTND is a six-item scale designed to measure tobacco dependence. Each item has its own individual response scale that varies by item. The FTND is a revision of the original FTQ, and previous research indicates that it has fair internal consistency ($\alpha = .61$; Heatherton et al.,

Kawakami Tobacco Dependence Screener (TDS). The TDS (Kawakami, Takatsuka, Inaba, & Shimizu, 1999) is a self-report measure designed to assess 10 of the DSM-IV criteria for tobacco dependence, each on a dichotomous scale with 0 indicating lack of the symptom and 1 indicating endorsement of the criterion. The sum of symptoms, from 0 to 10, allows for a more continuous measure of dependence that is based on both

Table 1

Demographic Data

Variable	Daily smoker $(n = 443)$	Nondaily smoker $(n = 330)$	All smokers $(N = 775)$
Women (%)	56.3	64.8	60.0
White (%)	78.3	92.0	84.3
African American (%)	17.0	2.6	10.7
High school graduate/GED or greater (%)	89.5	98.8	92.7
Married (%)	18.0	4.0	11.9
Student (%)	27.3	77.9	48.8
Cigarettes per day, M (SD)	16.34 (11.13)	2.96 (2.76)	10.51 (10.81)
CO in ppm, M (SD)	15.97 (10.60)	3.97 (3.31)	10.76 (10.17)

Note. GED = general equivalency diploma; CO = carbon monoxide.

physical aspects of dependence, such as withdrawal and tolerance, as well as on social and behavioral aspects, such as continuing tobacco use despite problems in life or despite other consequences. It has been validated only on Japanese smokers, mainly men. In that research it showed good internal consistency (alphas ranged from .76 to .81 across three studies). It was also significantly correlated with the number of cigarettes smoked per day, years smoking, and CO levels.

Demographics and smoking history. A demographics questionnaire assessed characteristics such as gender, ethnicity, age, marital status, education level, and employment. The smoking history questionnaire included items such as the number of cigarettes smoked per day, age of smoking initiation, smoking status (e.g., daily smoker, occasional smoker), number of quit attempts, longest time abstinent, and other smokers in the household.

CO assessment. Participants provided a breath sample to permit alveolar CO analysis to verify their smoking status and estimate their smoking heaviness. A Bedfont Smokerlyzer (Bedfont Scientific Limited, Kent, United Kingdom) was used to measure the CO in the breath samples. Results were recorded as parts per million of CO.

Preliminary Evaluation of Clinical Validity

We also collected preliminary predictive validity information from a smoking cessation study. In this randomized double-blind placebocontrolled cessation study, conducted in Milwaukee, Wisconsin, smokers were randomly assigned to receive active bupropion SR and active nicotine gum (n = 91), active bupropion SR and placebo gum (n = 86), or placebo bupropion SR and placebo gum (n = 61). Gum was provided for 8 weeks and bupropion was provided for 9 weeks. Participants also received three 10-min counseling sessions to aid in their quit attempt. The WISDM-68, FTND, and TDS were administered at a baseline assessment session. On the basis of the study design, 2-week point prevalence abstinence data were collected at the end of treatment (8 weeks after the quit date) and verified using CO readings of less than 10. The data presented are based on a sample of 238 participants (60.1% women). Of the 238 participants, 78.4% were White, 20.7% were African American, and the remaining 0.8% was Native American and Asian/Pacific Islander. Only 1 participant reported being Hispanic. On average, the participants smoked 22.18 cigarettes per day (SD = 10.07).

Results

Overview of Data Analysis

Data analysis was conducted in four phases: (a) item selection, (b) preliminary modeling, (c) evaluation of differential subscale performance, and (d) preliminary validation. In the item-selection phase, the participants were randomly divided into two groups, a

derivation sample (n = 385) and a validation sample (n = 390). Sixty-eight items were selected using classical item selection criteria with the derivation sample and then verified using the validation sample. After selecting the 68 items of the WISDM-68 from the original pool of 285 items, the next phase of analysis was the assessment of different structural models of the WISDM-68. Using the Mplus software package (Muthén & Muthén, 1998), we conducted confirmatory factor analyses (CFAs) to evaluate the distinguishability of the motives tapped by each subscale. The third set of analyses examined the differential performance of the individual subscales. Using nonlinear regression, we used levels of smoking heaviness to predict scores on the WISDM-68 subscales. After deriving and modeling the internal structure of the WISDM-68 and examining the differential performance of the subscales, we conducted concurrent validation analyses to compare the WISDM-68 with other putative markers of dependence, such as smoking heaviness and DSM-IV criteria, and to examine the discriminant validity of the various subscales. Finally, preliminary data from the validation smoking cessation study were analyzed to determine the ability of the WISDM-68 to predict end-of-treatment relapse.

Item Selection

Using data from the derivation sample only, we selected subscale items on the basis of two criteria: (a) item—whole correlation in the derivation sample and (b) the representativeness of the targeted domain.³ Ideally, each subscale should have a reliability coefficient ≥ .90 to make it an effective clinical tool. Therefore, the initial goal was to create subscales with internal consistencies of approximately .93 or greater in the derivation sample to adjust for the shrinkage expected to occur when the internal consistencies were calculated in the validation sample. We attempted to retain the smallest number of items possible, with the highest item—whole correlations, such that each subscale had an alpha of .93 in the derivation sample. We also surveyed the item content to ensure that there were items to tap the breadth of each domain.

The original subscales ranged from 10 to 38 items. The 13 revised subscales of the WISDM-68 comprised 4 to 7 items each. See Appendix B and Appendix C for the WISDM-68 Questionnaire and scoring key. Each of the revised 13 subscales was

³ We retained items that would tap different dimensions of a motive, such as frequency, timing, and intensity of the motive.

analyzed for internal consistency, using the derivation sample. The results revealed that every original subscale had a Cronbach's alpha > .90, with the exception of the Social–Environmental Goads subscale, which had an alpha of .87. These results indicate that each of the individual subscales exceeded the cutoff of $\alpha =$.80, which has been deemed appropriate for clinical diagnosis (Nunnally & Bernstein, 1994). Then, using the validation sample, we reexamined the internal consistency of each revised subscaleand found all to have a reliability coefficient > .90, with the exception of the Cue Exposure–Associative Processes subscale, which had an internal consistency coefficient of .88.

To ensure that the WISDM-68 subscales are appropriate for various populations, the final step in the item selection analyses was to determine the reliability of the 68 items across six different populations: men, women, daily smokers, nondaily smokers, White smokers, and non-White smokers (83 African American, 5 American Indian, 13 Asian, and 18 other). The results revealed that the WISDM-68 subscales have fair to excellent internal consistency (range: .73–.95) for all six of the groups examined: men, women, daily, nondaily, White, and non-White smokers. The lowest internal consistencies were for nondaily smokers on the Tolerance subscale ($\alpha = .73$), the Cravings subscale ($\alpha = .82$), and the Automaticity subscale ($\alpha = .82$). The internal consistency of the total WISDM-68 in these populations ranged from .97 to .99.⁴

CFA Models

Our hypothesis was that the WISDM-68 would be multidimensional. Therefore, once the subscale items were selected, we examined the dimensional structure of our measure by fitting CFA models. These models addressed the issue of whether dependence, as assessed with the WISDM-68, should be construed as multidimensional. We did not attempt to identify a best-fitting structure of the WISDM-68 at this early point in its development. Nor did we attempt to prune or merge subscales. We believed such steps to be premature given the modest validity data available and the relatively small number of subject samples available for analysis.

The preliminary modeling was done using a second random split of the sample into Sample 1 and Sample 2. Using Sample 1, we tested two different theoretically derived models. These models were then replicated in Sample 2. The first model tested, a singlefactor model, was based on the work of theorists who have suggested that dependence is a unidimensional latent construct (i.e., that either a single factor accounts for the great proportion of variance in indices of addictive drug use or if distinct factors are involved, they become so entrained and coherent that they may be effectively modeled as a single factor; e.g., Edwards & Gross, 1976; Shiffman, 1993a). The single-factor CFA model was created to assess whether the data fit a model in which all of the items reflect a common tobacco-dependence factor. The second model tested the implicit model that guided the generation of the 13 separate subscales; that variance in dependence phenomena can be accounted for by multiple, relatively discrete motives—in this case, the 13 distinct motives tapped by the WISDM-68. Model evaluation involved an examination of the comparative fit index (CFI) and the root-mean-square error of approximation (RMSEA). We considered a model to have adequate fit if the CFI was \geq .90 and the RMSEA was ≤ .08 (Browne & Cudeck, 1993; Newcomb, 1994). The chi-square:degree of freedom ratio is also presented.

Although there are no agreed-on criteria for interpreting this ratio, it is nevertheless a useful criterion for model comparison purposes, with the better model being the one that minimizes the index (e.g., Widaman & Reise, 1997).

Using Sample 1, we found that the single-factor model of dependence had a CFI of .60 and a RMSEA of .113. Neither of these indices indicates a good fit for this model. The model also failed to fit the data from Sample 2. Using Sample 1, we found that the 13-factor model had a CFI of .86 and a RMSEA of .068. These results also fall slightly below common standards for a well-fitting model. However, inspection of modification indices failed to suggest any simple correction that would significantly improve fit, and thus the model was not modified. When fit to Sample 2, the 13-factor model produced a CFI of .90 and a RMSEA of .059, indicating an acceptable fit. In addition, the change in the chisquare:degrees of freedom ratio indicates that the 13-factor model is an improvement on the single-factor model. Thus, despite its more modest fit in Sample 1, the 13-factor model was deemed a reasonably well-fitting model and clearly an improvement over the single-factor model. See Table 2 for a summary of the model-fit indices.

These results indicate that dependence, as measured by the WISDM-68, is not a unitary construct but rather is a diverse collection of distinct motives for drug use. Although these models do not address the potential presence of other latent variables (e.g., a latent variable related to physical dependence, which might include tolerance, craving, and loss of control) or an overarching second-order factor, they do suggest that there may be value in parsing dependence (as reflected in the scales used here) rather than in attempting to model the latent construct of dependence as homogeneous. These results do not mean that the 13-factor model is the best-fitting model possible. Indeed, the strong intercorrelations among some of the WISDM-68 subscales suggest that not all 13 motives are distinct (see Table 3 for the zero-order correlations among the WISDM-68 subscales in daily smokers). However, some subscales (e.g., Social-Environmental Goads, Weight Control) appear to be fairly distinct. It is also clear from the correlations (see Table 3) that although many subscales appear to be strongly related to the FTND, there are certain subscales (e.g., Social-Environmental Goads, Taste and Sensory Properties, Weight Control) that appear to have relatively little overlap.

Differential Subscale Performance

The third set of analyses focused on the relation of the different subscales to different levels of smoking heaviness and lifetime smoking or cigarette exposure. The variable cigarettes smoked per month was calculated on the basis of the number of cigarettes smoked per day times the number of days smoked (daily smokers were assumed to smoke 30 days per month). Lifetime cigarette exposure was calculated for daily smokers with the following equation: cigarettes smoked per month \times 12 \times years of daily

⁴ It is possible for a multidimensional scale to have a high Cronbach's alpha if it reflects correlated latent variables (see Clark & Watson, 1995).

⁵ We did examine the fit of other multifactorial models comprising merged subscales, and they did show adequate fit. However, because we are interested in the predictive validity of all of the theoretically derived subscales, we are disinclined to merge the subscales before assessing their discriminative validities.

Table 2
Summary of Model Fits

Model	Group	n	χ^2	df	χ^2 : df ratio	CFI	RMSEA
Single-factor model	Sample 1	317	11,223.50	2210	5.08	.60	.113
	Sample 2	393	12,363.85	2210	5.59	.63	.108
13-factor model	Sample 1	317	5,231.94	2132	2.45	.86	.068
	Sample 2	393	5,012.64	2132	2.35	.90	.059

Note. CFI = comparative fit index; RMSEA = root-mean-square error of approximation.

smoking. For nondaily smokers, we used the following equation: cigarettes per month \times 12 \times years since the participant had smoked his or her first cigarette. Because of oversampling of nondaily smokers, we had positively skewed distributions for both cigarettes smoked per month and lifetime nicotine exposure. Therefore, for each variable, we transformed the scores by taking the log of the score plus one.

The relations between these smoking variables and scores on the 13 subscales were studied by fitting nonlinear regression curves to the data. Initially, logistic curve fitting was used to examine the ability of either smoking heaviness or lifetime cigarette exposure to predict the scale scores on the 13 subscales. This analysis allowed us to examine how smoking heaviness predicts different motives. A logistic model was thought to be more appropriate than a linear model because of the presence of floor and ceiling effects in the subscale scores; consistent with this hypothesis, for all 13 subscales, the logistic curves produced a higher multiple correlation squared in predicting subscale variance than a linear regression. For these analyses, the results for both cigarettes smoked per month and lifetime cigarette exposure were similar, so only the results for cigarettes smoked per month are presented and discussed.

Visual inspection of the logistic curves suggested that there may be two different groups of subscales, one with steady growth across all levels of smoking heaviness and one with accelerated growth at higher levels of smoking heaviness (see Figure 1 for examples). To examine this hypothesis further and to characterize better the differences between the 13 curves, we fit a quadratic regression model to each subscale. The results indicated that almost all of the subscales had a significant linear trend and many had a significant quadratic trend, with the exceptions being Social–Environmental Goads, Cue Exposure–Associative Processes, Taste and Sensory Properties, and Weight Control subscales (see Table 4).

To determine the significance of the linear and quadratic terms, F tests were performed using full versus reduced model comparisons. To determine the significance of the linear term, we compared the linear model (full model) against a model with no predictors (reduced model). To determine the significance of the quadratic term, we compared a model with both a linear and quadratic term (full model) against a model with only a linear term (reduced model). Results indicated two distinct groups of motives, those with only a linear component and those with both a linear and a quadratic component (data not shown). The subscales found to lack a significant quadratic component were the same as those identified using the logistic curve-fitting analysis.

There appears to be one group of motives that is influential for both novice and inveterate smokers that might be labeled "early-emergent smoking motives." The early-emergent motives are present for all smokers, regardless of their experience, and appear to include the following: Social–Environmental Goads, Cue Exposure–Associative Processes, and Taste and Sensory Properties. These three motives have significant linear components but no statistically significant quadratic components, implying a consistent rate of increase in relation to smoking heaviness.

Table 3

Zero-Order Correlations of the WISDM-68 Subscales in Daily Smokers

Measures	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Affiliative Attachment	_													
2. Automaticity	.53*	_												
3. Behavioral Choice–Melioration	.82*	.53*	_											
4. Cognitive Enhancement	.65*	.42*	.70*	_										
5. Loss of Control	.63*	.58*	.66*	.49*	_									
6. Craving	.62*	.59*	.71*	.54*	.72*									
7. Cue Exposure–Associative Processes	.51*	.52*	.59*	.51*	.55*	.65*								
8. Negative Reinforcement	.63*	.48*	.71*	.70*	.55*	.70*	.65*							
9. Positive Reinforcement	.70*	.44*	.73*	.68*	.47*	.63*	.59*	.79*						
Social–Environmental Goads	.08	.17	.13*	.12*	.06	.24*	.30*	.25*	.17*	_				
11. Taste and Sensory Properties	.50*	.34*	.50*	.43*	.24*	.43*	.49*	.50*	.69*	.24*				
12. Tolerance	.54*	.61*	.62*	.48*	.71*	.66*	.45*	.50*	.45*	.13*	.34*	_		
13. Weight Control	.36*	.24*	.37*	.33*	.24*	.23*	.32*	.38*	.33*	.06	.16*	.18*	_	
14. FTND	.47*	.48*	.52*	.36*	.58*	.53*	.22*	.33*	.28*	.01	.18*	.78*	.11*	_

Note. WISDM-68 = sixty-eight item Wisconsin Inventory of Smoking Dependence Motives; FTND = Fagerström Test for Nicotine Dependence. *p < .05.

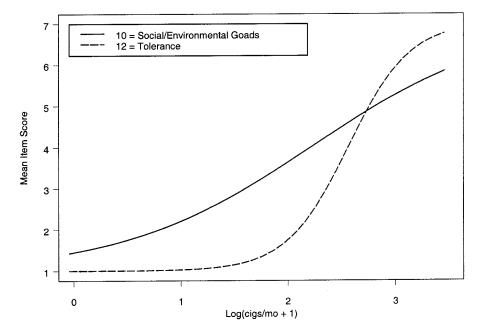


Figure 1. This figure illustrates the different logistic regression curves predicting scores on the Wisconsin Inventory of Smoking Dependence Motives subscales from cigarettes smoked per month for early-emergent motives and late-emergent motives, using Social–Environmental Goads and Tolerance as prototypes of each motive, respectively. The early-emergent motive has a higher intercept at low rates of smoking than the late-emergent motive and has consistent linear growth as smoking rates increase. The late-emergent motive is not endorsed by light smokers, but as smoking rates increase, there is an exponential increase in the rate of endorsement. Cigs = cigarettes; mo = month.

Another group of motives appears to be influential only for individuals who are heavier smokers or who have had a considerable lifetime exposure to nicotine. This group of motives might be labeled as "late-emergent smoking motives." These late-emergent motives, which are present only in individuals who smoke at a moderate daily rate or have at least moderate smoking experience, include the following: Craving, Automaticity, Behavioral Choice—Melioration, Cognitive Enhancement, Affiliative Attachment, and

Tolerance. In the quadratic regression, these subscales have negative linear but positive quadratic components, implying a slower rate of change at low levels of smoking heaviness but a sharper increase at higher levels (see Figure 1).

Loss of Control, Negative Reinforcement, and Positive Reinforcement motives also show strong and significant quadratic components. However, these motives are characterized by nonsignificant linear coefficients. Thus, these motives are more similar to

Table 4
Linear and Quadratic Coefficients Describing the Relation Between the WISDM-68 Subscales and Smoking Heaviness

Outcome	Linear	t	p	Quadratic	t	p
Affiliative Attachment	588	-4.303	.000	1.155	8.456	.000
Automaticity	510	-4.091	.000	1.168	9.360	.000
Behavioral Choice–Melioration	773	-7.180	.000	1.510	14.020	.000
Cognitive Enhancement	470	-3.817	.000	1.140	9.259	.000
Loss of Control	212	-1.522	.128	0.777	5.576	.000
Craving	375	-3.232	.001	1.092	9.422	.000
Cue Exposure–Associative Processes	.619	4.684	.000	0.008	0.063	.950
Negative Reinforcement	.185	1.346	.179	0.403	2.930	.003
Positive Reinforcement	.172	1.190	.234	0.359	2.490	.013
Social-Environmental Goads	.565	3.588	.000	-0.198	-1.258	.209
Taste and Sensory Properties	.663	4.469	.000	-0.182	-1.230	.219
Tolerance	971	-10.698	.000	1.768	19.467	.000
Weight Control	.201	1.310	.190	0.229	1.489	.137

Note. There are approximately 755 dfs for these analyses, making the t statistics nearly equivalent to z statistics. WISDM-68 = sixty-eight item Wisconsin Inventory of Smoking Dependence Motives.

late-emergent motives in that they show steep accelerations at high levels of smoking heaviness. It is interesting to note that whereas the linear components of the Negative and Positive Reinforcement motives are not statistically significant, they are positive, whereas the linear components for all other late-emergent motives are negative.

Validation

Ultimately, the WISDM-68 will be validated against four major criteria: heaviness of smoking–nicotine self-administration, *DSM–IV* criteria for tobacco dependence (e.g., consequences of smoking), severity and duration of withdrawal symptoms, and likelihood or latency to relapse. In this article, we present data on smoking heaviness, which was assessed by self-report of smoking rate and by alveolar CO; *DSM–IV* criteria of tobacco dependence, which was assessed using the TDS; and relapse, assessed using 2-week point prevalence data at the end of treatment.

Concurrent validation. The results from the multiple regression analyses in which the 13 WISDM-68 subscales were entered as predictors indicate that, in the overall population, the 13 WISDM-68 subscales accounted for approximately 58% of the variance in number of cigarettes smoked per day, 53% of the variance in CO level, and 60% of the variance in DSM–IV dependence. Comparatively, the FTND accounted for 62% of the variance in cigarettes smoked per day, 55% of the variance in CO level, and 37% of the variance in DSM–IV dependence.

Regression analyses were also conducted to examine which of the various subscales of the WISDM-68 predicted the most variance in the different dependence indicators. In the derivation sample, the Tolerance subscale best predicted CO level (r=.69, p<.001), but the Craving, Cue Exposure–Associative Processes, and Tolerance subscales were the best predictors of DSM-IV dependence when entered together into a multiple regression equation (R=.79, p<.001). These results were replicated in the validation sample such that Tolerance was correlated at .72 (p<.001) with CO level, and Craving, Cue Exposure–Associative Processes, and Tolerance accounted for almost 55% of the variance in DSM-IV criteria (R=.74, p<.001). See Table 5 for the zero-order correlations among the WISDM-68 subscales and dependence criteria.

We also used path analysis, conducted using LISREL software, to test the discriminant validity of the different subscales. An omnibus test was performed to determine whether the predictive relationships between the 13 subscales and these outcomes differed. When the paths for each of the three concurrent validity variables (DSM-IV dependence criteria, CO, and cigarettes smoked per month) were constrained to be equal across the 13 subscales, the model did not fit the data (CFI = 0.96; RMSEA = .12); $\chi^2(36, N = 775) = 432.97$, p < .001. On the basis of the modification indices, we attempted to improve the model by freeing certain paths and allowing them to vary in their relation with the three outcome measures. When the paths from Loss of Control, Craving, Cue Exposure-Associative Processes, and Positive Reinforcement were allowed to vary for DSM-IV dependence (i.e., the TDS), and Loss of Control and Tolerance were allowed to vary for both CO level and cigarettes smoked per month, the model provided a much better fit (CFI = 1.00; RMSEA = .032); $\chi^2(28, N =$ 775) = 49.84, p < .007. This suggests reliable heterogeneity

Table 5
Zero-Order Correlations Between Validation Criteria, the
WISDM-68 Subscales, and the FTND for All Smokers

Measure	TDS	CO (ppm)	Cigarettes per day
Affiliative Attachment	.59	.46	.54
Automaticity	.61	.56	.60
Behavioral Choice–Melioration	.66	.55	.59
Cognitive Enhancement	.56	.42	.49
Loss of Control	.70	.64	.68
Craving	.73	.57	.60
Cue Exposure–Associative			
Processes	.66	.39	.45
Negative Reinforcement	.63	.39	.45
Positive Reinforcement	.53	.34	.42
Social-Environmental Goads	.31	.15	.23
Taste and Sensory Properties	.43	.26	.33
Tolerance	.65	.70	.76
Weight Control	.44	.32	.30
Total WISDM-68	.72	.55	.63
FTND	.61	.74	.79
TDS	_	.49	.54
CO (ppm)	.49	_	.70

Note. WISDM-68 = sixty-eight item Wisconsin Inventory of Smoking Dependence Motives; FTND = Fagerström Test for Nicotine Dependence; TDS = Kawakami Tobacco Dependence Screener; CO = carbon monoxide.

among subscales in terms of their relations with dependence criteria.

Because of the heterogeneity of the smoking population, we examined the predictive power of these questionnaires in different smoker subpopulations. We examined whether the correlations between subscales and the validation criteria (i.e., cigarettes per day, CO level, and DSM-IV dependence) differed between men and women, between daily and nondaily smokers, and between White and non-White smokers. To this end, the LISREL software was used to fit multigroup path models that allowed for a comparison of the path coefficients from individual subscales to the validation criteria across the targeted populations. The results revealed that the models of the WISDM-68 in which the paths were constrained to be equal across groups provided an adequate fit for both men and women (CFI = 1.00; RMSEA = .033), daily and nondaily smokers (CFI = .98; RMSEA = .084), and for White and non-White smokers (CFI = 1.00; RMSEA = .030). This suggests that the assumption of equivalent concurrent validity of the subscales across groups may be reasonable.

Preliminary predictive validity. Using data from the cessation study, we examined predictors of end-of-treatment relapse using hierarchical logistic regression. Results revealed that, after controlling for treatment group in the first step, the total WISDM-68 score was not statistically significant in its prediction of end-of-treatment relapse (B = -0.02, Wald = 2.15, p = .14). However, when each of the individual subscales was examined separately in the second step, after controlling for treatment group, Automaticity (B = -0.22, Wald = 5.71, p = .02), Cognitive Enhancement (B = -0.21, Wald = 5.08, p = .02), Negative Reinforcement (B = -0.20, Wald = 3.68, p = .055) and Social–Environmental Goads (B = -0.20, Wald = 6.69, p = .01) all significantly predicted relapse at the end of treatment. Using the model building strategy

suggested by Hosmer and Lemeshow (2000), a best-fitting model was created, $\chi^2(6, N=238)=33.87, p<.001$, that included Automaticity (B=-0.27, Wald = 6.06, p=.01), Behavioral Choice–Melioration (B=0.72, Wald = 16.63, p<.001), Cognitive Enhancement (B=-0.31, Wald = 5.32, p=.02), and Negative Reinforcement (B=-0.34, Wald = 4.12, p=.04). The FTND did not significantly predict relapse at the end of treatment after controlling for treatment condition (B=-0.08, Wald = 1.49, p=.22).

Discussion

The primary goal of this research was to use construct validation to enhance understanding and measurement of tobacco dependence. The dependence model guiding the development of the WISDM-68 is that dependence is an emergent property of motivational processes that influence compulsive drug use and an inability to quit. Using various theory-based motives, we created the WISDM-68 to assess dependence as a multivariate construct. The assessed motives are intended to reflect both the diversity and strength of drug-use motives. The final common pathways yielded by these motives can be labeled as tobacco dependence, which is associated with outcomes such as heavy smoking, severe withdrawal, and relapse.

Internal Consistency and Structure

The 13 WISDM-68 subscales were found to have acceptable levels of internal consistency in the overall population and in various subpopulations of smokers, such as men, women, daily smokers, nondaily smokers, White, and Non-White smokers. Although the internal consistencies of the subscales are very good, it is clear that some subscales are highly intercorrelated and do not represent wholly distinct constructs. However, the correlation matrix in Table 3 also shows that despite a positive manifold, some intercorrelations are modest (e.g., Loss of Control and Social–Environmental Goads r=.06).

Another important finding is that self-reported motives for smoking appear to be multidimensional. To the extent that these motives reflect dependence, dependence is best modeled as a multidimensional construct. By assessing dependence as a multidimensional construct, researchers will be able to examine the heterogeneity among smokers. In other words, rather than assessing the end states of dependence per se (e.g., chronic use, relapse), this measure allows researchers to examine the different motivational processes in terms of their relations with important criteria.

What is the evidence that dependence motives are multidimensional? First, an analysis of their covariance structures suggests that their interrelations cannot be well modeled as unidimensional.⁶ In addition, their relations with the available criteria suggest nonequivalence among the motives. For instance, the criterion of smoking heaviness (as reflected by CO scores) was predicted consistently by two scales: Tolerance and Loss of Control. These two scales are not only highly intercorrelated (r = .71), but they also have the highest correlations of all scales with cigarettes smoked per day (rs = .76 and .68, respectively; see Table 5). The more multifaceted criterion of DSM symptoms (as measured here by the TDS) was predicted across different samples, by a range of scales, including Craving, Cue Exposure–Associative Processes,

Negative Reinforcement, Tolerance, Behavioral Choice—Melioration, and Affiliative Attachment. Relapse was best predicted by Automaticity, Cognitive Enhancement, Negative Reinforcement, and Social—Environmental Goads. In short, neither the more complex, multifaceted *DSM* criteria nor the distal criteria of relapse could be well predicted by measures that primarily reflect heaviness of smoking. This suggests that criteria such as relapse vulnerability and continuing to smoke despite problems—problems assessed with the *DSM–IV*—are best predicted when measures address affective, interpersonal factors and broad behavioral domains.

Further evidence of the multidimensionality of smoking motives arises from analyses in which measures of smoking heaviness or lifetime exposure to nicotine were used to predict participants' mean subscale scores (see Table 4). These analyses revealed that different subscales bore very different relations with these smoking exposure variables. Some subscales such as Social-Environmental Goads, Taste and Sensory Properties, and Cue Exposure–Associative Processes were linearly related to exposure. Other subscales, such as Tolerance, Craving, Cognitive Enhancement, Behavioral Choice-Melioration, Automaticity, Negative Reinforcement, Positive Reinforcement, and Affiliative Attachment, all shared significant quadratic relations with exposure. Most of these subscales, but not all (i.e., Positive Reinforcement and Negative Reinforcement), had negative linear coefficients. Thus, these subscales showed very little increase at low levels of smoking exposure but rapid acceleration as exposure became heavy.

The above findings suggest that different motives are differentially influential across the ontogeny of smoking. Early-emergent motives, or those that increase in a linear manner across the range of exposure, may be prepotent for lighter and neophyte smokers. However, as nicotine exposure increases, the rate of growth of late-emergent motives overshadows the early motives in terms of smokers' levels of endorsement (see Figure 1). This pattern suggests that the smoking of light or neophyte smokers is influenced primarily by social and nonsocial environmental cues and by sensory and gustatory properties of cigarettes. Although these factors do increase with greater exposure to tobacco, other factors appear to become relatively more important.

One might assume that it is the late-emergent motives that reflect "true dependence." That is, the steplike increase in these motives across exposure levels suggests the passage of individuals into a more discrete taxon that separates mere exposure from addictive use. Although this hypothesis is plausible, dependence may not, in fact, be taxonic. For example, factors that are linearly related with exposure may be more reflective of the graded onset of dependence. Furthermore, it is the case that early-emergent factors such as Cue Exposure–Associative Processes do possess predictive validity with respect to *DSM* criteria. Also, it may be that such factors are more predictive of dependence criteria among those with relatively little tobacco exposure.

⁶ We were not interested so much in identifying a best-fitting model at this point in the scale development process as in determining whether a unidimensional model seemed appropriate. We wish to relate each theoretically derived subscale to the full set of dependence criteria (e.g., smoking heaviness, withdrawal severity, relapse latency) prior to achieving a best-fitting model by scale integration, deletion, or restructuring.

Finally, it is important to examine the nature of the lateemergent scales. The Tolerance subscale comprises questions that address the need of individuals to smoke more to experience tobacco effects. The Loss of Control subscale assesses the participant's sense that he or she has lost control over smoking. Therefore, some of these scales may merely reflect the fact that individuals smoke a lot and may not provide insight into motives that influence smoking (i.e., these are "residues" of other dependence motives). However, some of the late-emerging scales may provide insights into mechanisms and vulnerabilities. For instance, those who have histories of especially heavy tobacco exposures are likely to endorse strongly notions that they have formed emotional attachments to smoking and/or tobacco use, that tobacco use has displaced other sources of reinforcement, that tobacco use allows them to think more clearly and effectively, and that tobacco use reduces cravings and/or urges. It will be important to continue to explore the relation between the different late-onset motives and outcomes such as withdrawal severity and relapse latency to determine whether the quadratic pattern seen is truly informative or merely a reflection of increased use.

Validation

At this time, the validation of the WISDM-68 is a work "in progress." Until we relate the WISDM-68 to additional validation criteria, such as inability to stop smoking or relapse vulnerability at 12 months, and to withdrawal severity and persistence (e.g., Piasecki, Jorenby, Smith, Fiore, & Baker, 2003a, 2003b), the construct validity of the instrument must be considered unsubstantiated.

Examination of the available validity criteria, however, is promising. For instance, one of the subscales (i.e., Tolerance) is essentially equivalent to the FTND in terms of its relation with cigarettes smoked per day and CO level. Moreover, the Tolerance subscale has a considerably stronger relation with these criteria than do the *DSM–IV* criteria (data not shown). This pattern of findings is encouraging in that the FTND directly queries cigarettes smoked per day. In addition, several of the WISDM-68 subscales show correlations with *DSM* symptoms that are as high, or higher, than is the correlation between the FTND and these symptoms. Finally, the preliminary predictive validity analyses revealed that four of the WISDM-68 subscales, Automaticity, Cognitive Enhancement, Negative Reinforcement, and Social–Environmental Goads, predicted relapse at the end of treatment, whereas the FTND did not.

The best-fitting model for predicting relapse, the model that included Automaticity, Behavioral Choice—Melioration, Cognitive Enhancement, and Negative Reinforcement, suggests that four different mechanisms may be related to relapse by the end of treatment. Smokers reporting automatic smoking motives (e.g., smoking without thinking about it) were more likely to relapse as were individuals who reported smoking to enhance their cognitive abilities. In addition, smokers reporting high levels of smoking to alleviate negative states were also more likely to relapse. However, if an individual endorsed high levels of behavioral choice—melioration motives (e.g., "it would take a pretty serious medical problem to get me to quit" or "smoking is the best thing I do all day") but was motivated enough to participate in the cessation study, then he or she was less likely to relapse. One possible

interpretation for the strong negative relation between the Behavioral Choice—Melioration subscale and relapse is that it is a measure of overall motivation to quit. If an individual is motivated enough to participate in a smoking cessation program, despite believing that smoking is the best thing he or she does all day, then the scale becomes a proxy for strong motives to quit. Future research must be done to characterize further how the Behavioral Choice—Melioration subscale relates to cessation/relapse—especially among general smoker populations not self-selected for high levels of motivation to quit.

Future research must not only address the relations among WIDSM-68 subscales and dependence criteria but also carefully assess its validity relative to alternative instruments such as the Cigarette Dependence Scale (Etter et al., 2003) and the Nicotine Dependence Syndrome Scale (Shiffman et al., 1995). Its future use depends, of course, on its ability to predict a range of dependence criteria across the broad population of smokers. Thus far, however, our results support the use of the WISDM-68 subscales for research purposes: The subscales are internally consistent, and different subsets of subscales predict major dependence criteria, providing an opportunity to examine heterogeneous processes among smokers.

References

American Psychiatric Association. (1980). Diagnostic and statistical manual of mental disorders (3rd ed.). Washington, DC: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical man*ual of mental disorders (3rd ed., rev.). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical man*ual of mental disorders (4th ed.). Washington, DC: Author.

Baker, T. B., & Brandon, T. H. (1990). Validity of self-reports in basic research. *Behavioral Assessment*, 12, 33–51.

Baker, T. B., Morse, E., & Sherman, J. E. (1987). The motivation to use drugs: A psychobiological analysis. In C. Rivers (Ed.), *Nebraska Sym*posium on Motivation (Vol. 34, pp. 257–323). Lincoln: University of Nebraska Press.

Bell, S. L., Taylor, R. C., Singleton, E. G., Henningfield, J. E., & Heishman, S. J. (1999). Smoking after nicotine deprivation enhances cognitive performance and decreases tobacco craving in drug abusers. *Nicotine & Tobacco Research*, 1, 45–52.

Brandon, T. H., & Baker, T. B. (1991). The Smoking Consequences Questionnaire: The subjective expected utility of smoking in college students. *Psychological Assessment*, 3, 484–491.

Breslau, N., & Johnson, E. O. (2000). Predicting smoking cessation and major depression in nicotine-dependent smokers. *American Journal of Public Health*, 90, 1122–1127.

Browne, M. & Cudeck, R. (1993). Alternative ways of assessing model fit. In K. A. Bollen & J. S. Long (Eds.), *Testing structural equation models* (pp. 136–162). Newbury Park, CA: Sage.

Centers for Disease Control and Prevention. (2002). Cigarette smoking among adults—United States, 2000. Morbidity and Mortality Weekly Report, 51, 642–645.

Clark, L. A., & Watson, D. (1995). Constructing validity: Basic issues in objective scale development. *Psychological Assessment*, 7, 309–319.

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 and Alcohol Dependence*, 59, 523– 539.

Cronbach, L. J., & Meehl, P. E. (1955). Construct validity in psychological tests. *Psychological Bulletin*, 52, 281–302.

Edwards, G. (1986). The Alcohol Dependence Syndrome: A concept as stimulus to enquiry. *British Journal of Addiction*, 81, 171–183.

Edwards, G., & Gross, M. M. (1976). Alcohol dependence: Provisional description of a clinical syndrome. *British Medical Journal*, 1, 1058– 1061.

- Etter, J. F., Le Houezec, J., & Perneger, T. V. (2003). A self-administered questionnaire to measure addiction to cigarettes: The Cigarette Dependence Scale. *Neuropsychopharmacology*, 28, 359–370.
- Fagerström, K. O. (1978). Measuring degree of physical dependence to tobacco smoking with reference to individualization of treatment. Addictive Behaviors, 3, 235–241.
- Fagerström, K. O., & Schneider, N. G. (1989). Measuring nicotine dependence: A review of the Fagerström Tolerance Questionnaire. *Journal of Behavioral Medicine*, 12, 159–181.
- Garvey, A. J., Kinnunen, T., Nordstrom, B. L., Utman, C. H., Doherty, D., Rosner, B., & Vokonas, P. S. (2000). Effects of nicotine gum dose by level of nicotine dependence. *Nicotine & Tobacco Research*, 2, 53–63.
- Gilbert, D. G., Crauthers, D. M., Mooney, D. K., McClernon, F. J., & Jensen, R. A. (1999). Effects of monetary contingencies on smoking relapse: Influences of trait depression, personality, and habitual nicotine intake. *Experimental & Clinical Psychopharmacology*, 7, 174–181.
- Heatherton, T. F., Kozlowski, L. T., Frecker, R. C., & Fagerström, K. O. (1991). The Fagerström Test for Nicotine Dependence: A revision of the Fagerström Tolerance Questionnaire. *British Journal of Addiction*, 86, 1119–1127.
- Heyman, G. M. (1996). Resolving the contradictions of addiction. Behavioral and Brain Sciences, 19, 561–610.
- Hosmer, D. W., & Lemeshow, S. (2000). Applied logistic regression (2nd ed.). New York: Wiley.
- Hughes, J. R., & Hatsukami, D. K. (1986). Signs and symptoms of tobacco withdrawal. Archives of General Psychiatry, 43, 289–294.
- Ikard, F. F., Green, D., & Horn, D. (1969). A scale to differentiate between types of smoking as related to the management of affect. *International Journal of the Addictions*, 4, 649–659.
- Kawakami, N., Takatsuka, N., Inaba, S., & Shimizu, H. (1999). Development of a screening questionnaire for tobacco/nicotine dependence according to ICD-10, DSM-III-R, and DSM-IV. Addictive Behaviors, 24, 155–166.
- Kenford, S. L., Smith, S. S., Wetter, D. W., Jorenby, D. E., Fiore, M. C., & Baker, T. B. (2002). Predicting relapse back to smoking: Contrasting affective and physical models of dependence. *Journal of Consulting and Clinical Psychology*, 70, 216–227.
- Killen, J. D., Fortmann, S. P., Kraemer, H. C., Varady, A., & Newman, B. (1992). Who will relapse? Symptoms of nicotine dependence predict long-term relapse after smoking cessation. *Journal of Consulting and Clinical Psychology*, 60, 797–801.
- Lichtenstein, E., & Mermelstein, R. J. (1986). Some methodological cautions in the use of the Tolerance Questionnaire. *Addictive Behaviors*, 11, 439–442.
- Mansky, P. A. (1978). Opiates: Human psychopharmacology. In L. L. Iversen, S. D. Iversen, & S. H. Snyder (Eds.), Handbook of psychopharmacology (pp. 95–184). New York: Plenum.
- McKennell, A. C. (1970). Smoking motivation factors. British Journal of Social and Clinical Psychology, 9, 8–22.
- Muthén, L. K. & Muthén, B. O. (1998). *Mplus user's guide*. Los Angeles: Author.
- Newcomb, M. D. (1994). Drug use and intimate relationships among women and men: Separating specific from general effects in prospective data using structural equation models. *Journal of Consulting and Clinical Psychology*, 62, 463–473.
- Niaura, R., Goldstein, M. G., & Abrams, D. B. (1994). Matching high- and low-dependence smokers to self-help treatment with or without nicotine replacement. *Preventive Medicine*, 23, 70–77.
- Nunnally, J. C., & Bernstein, I. H. (1994). Psychometric theory (3rd ed.). New York: McGraw-Hill.

- Panksepp, J., Siviy, S. M., & Normansell, L. A. (1985). Brain opioids and social emotions. In M. Reit & T. Field (Eds.), *The psychology of attachment and separation* (pp. 3–49). New York: Academic Press.
- Payne, T. J., Smith, P. O., McCracken, L. M., McSherry, W. C., & Antony, M. M. (1994). Assessing nicotine dependence: A comparison of the Fagerström Tolerance Questionnaire (FTQ) with the Fagerström Test for Nicotine Dependence (FTND) in a clinical sample. Addictive Behaviors, 19, 307–317
- Piasecki, T. M., & Baker, T. B. (2001). Any further progress in smoking cessation treatment? *Nicotine & Tobacco Research*, 3, 311–323.
- Piasecki, T. M., Jorenby, D. E., Smith, S. S., Fiore, M. C., & Baker, T. B. (2003a). Smoking withdrawal dynamics: I. Abstinence distress in lapsers and abstainers. *Journal of Abnormal Psychology*, 112, 3–13.
- Piasecki, T. M., Jorenby, D. E., Smith, S. S., Fiore, M. C., & Baker, T. B. (2003b). Smoking withdrawal dynamics: II. Improved tests of withdrawal–relapse relations. *Journal of Abnormal Psychology*, 112, 14–27.
- Pierce, J. P., & Gilpin, E. (1996). How long will today's new adolescent smoker be addicted to cigarettes? *American Journal of Public Health*, 86, 253–256.
- Pomerleau, C. S., Carton, S. M., Lutzke, M. L., Flessland, K. A., & Pomerleau, O. F. (1994). Reliability of the Fagerström Tolerance Questionnaire and the Fagerström Test for Nicotine Dependence. *Addictive Behaviors*, 19, 33–39.
- Robinson, T. E., & Berridge, K. C. (1993). The neural basis of drug craving: An incentive-sensitization theory of addiction. *Brain Research Reviews*, 18, 247–291.
- Russell, M. A. H., Peto, J., & Patel, U. A. (1974). The classification of smoking by factorial structure of motives. *Journal of the Royal Statistical Society*, 137, 313–346.
- Shiffman, S. (1993a). Assessing smoking patterns and motives. *Journal of Consulting and Clinical Psychology*, 61, 732–742.
- Shiffman, S. (1993b). Smoking cessation treatment: Any progress? *Journal of Consulting and Clinical Psychology*, 61, 718–722.
- Shiffman, S., Hickcox, M., Gnys, M., Paty, J. A., & Kassel, J. D. (1995, February). The Nicotine Dependence Syndrome Scale: Development of a new measure. Poster presented at the annual meeting of the Society for Research on Nicotine and Tobacco, San Diego.
- Shiffman, S., & Sayette, M. A. (2002, February). Validation of the Nicotine Dependence Syndrome Scale (NDSS): Differences between and within chippers and heavy smokers. Poster presented at the Eighth Annual Meeting of the Society for Research on Nicotine and Tobacco, Savannah, GA.
- Tiffany, S. T. (1990). A cognitive model of drug urges and drug-use behavior: Role of automatic and nonautomatic processes. *Psychological Review*, 97, 147–168.
- U.S. Department of Health and Human Services. (1988). The Health Consequences of Smoking: Nicotine Addiction, a Report of the Surgeon General (DHHS Publication No. CDC 88–8406). Washington, DC: GPO.
- Vuchinich, R. E., & Tucker, J. A. (1988). Contributions from behavioral theories of choice to an analysis of alcohol abuse. *Journal of Abnormal Psychology*, 97, 181–195.
- Widaman, K. F., & Reise, S. P. (1997). Exploring the measurement invariance of psychological instruments: Applications in the substance dependence domain. In K. J. Bryant & M. Windle (Eds.), The science of prevention: Methodological advances from alcohol and substance abuse research (pp. 281–324). Washington, DC: American Psychological Association.
- Wise, R. A. (1988). The neurobiology of craving: Implications for the understanding and treatment of addiction. *Journal of Abnormal Psychol*ogy, 97, 118–132.

Appendix A

The 13 Identified Motives for Drug Use

Affiliative Attachment

This motive arises from evidence that psychomotor stimulants like nicotine activate neural systems involved in the motivational impact of social cues (e.g., Panksepp, Siviy, & Normansell, 1985; Wise, 1988). These neuropharmacologic data are supported by self-report data from addicted smokers that report that cigarettes come to share many of the same affective and motivational properties as attractive social stimuli (i.e., "friends") and that withdrawal from tobacco is tantamount to social loss or mourning (Baker, Morse, & Sherman, 1987). The more attached a smoker is to his or her cigarettes, the harder cessation will be, suggesting stronger dependence. Some examples of questionnaire items follow: "Cigarettes keep me company, like a good friend" or "Sometimes I feel like cigarettes are my best friends."

Automaticity

Tiffany (1990) suggested that there is insufficient evidence that self-reports of urges are strongly linked to either physiological measures or drug consumption, indicating that, "Urges may not be necessary for the initiation or maintenance of drug-use behavior" (p. 151). The automaticity theory proposed by Tiffany posits that, like any activity an individual practices, smoking eventually becomes automatic and is controlled by automatic processes. Subsequently, urges, or subjective awareness of wanting to smoke, result when the automatic ritual of smoking is blocked, resulting in nonautomatic cognitive processes. For example, if a smoker automatically reaches for a pack of cigarettes, only to find that there are none left, the smoker experiences a craving or an urge to smoke. Smokers with highly automatic smoking processes find it harder to quit because of either a stronger dependence or an inability to counter their automatic behavior. Examples of questionnaire items tapping automaticity follow: "I often smoke without thinking about it" and "I smoke without deciding to."

Behavioral Choice-Melioration

Behavioral theories of choice (Vuchinich & Tucker, 1988) suggest that drug use is inversely proportional both to constraints on the access to drug and to the availability of other reinforcers. It has been suggested that the latter may play a more important role in drug behavior (Vuchinich & Tucker, 1988). According to this theory, smokers who are more dependent are more likely to smoke even in the presence of constraints on cigarettes and when other reinforcers are available. In addition, more dependent smokers may also have fewer reinforcers available to them.

Melioration theory (Heyman, 1996), based on Herrnstein's matching law, refers to the use of a "local bookkeeping strategy" for deciding among competing reinforcers that emphasizes the current value of each. Therefore, rather than maximizing the long-term reinforcement by construing the choice problem in terms of competing groups of reinforcers, combining rather than comparing options, the person focuses only on the immediate value of the different options. This results in a discounting of future reinforcers. Therefore, smokers who are more dependent report being unwilling to give up cigarettes even when confronted with negative consequences such as cigarette taxes or illness. Two sample items that tap the behavioral choice—melioration motive follow: "Very few things give me pleasure each day like cigarettes" and "Few things would be able to replace smoking in my life."

Cognitive Enhancement

Studies have shown that nicotine can improve attention and vigilance (Bell, Taylor, Singleton, Henningfield, & Heishman, 1999). Therefore,

smokers may be smoking to increase their cognitive abilities either above their baseline ability or to restore their cognitive abilities after nicotine deprivation. Questions that address this motive ask about the perceived cognitive impact of smoking. Smokers were asked to rate how much they agree or disagree with sentences such as "I smoke when I really need to concentrate" and "I frequently smoke to keep my mind focused."

Craving

This is a very traditional theoretical motive for drug use. Craving is an aversive state that motivates relapse and self-administration of drug. Although this motive reflects negative reinforcement, it is such a crucial motive for drug use that we attempt to assess it separately from the negative reinforcement motive. The stronger the dependence, the stronger the cravings may be. An item that assesses craving frequency is, "I frequently crave cigarettes." An item such as, "When I haven't been able to smoke for a few hours, the craving gets intolerable," assesses craving intensity. Finally, craving controllability was assessed using items such as, "It's hard to ignore an urge to smoke."

Cue Exposure-Associative Processes

This motive reflects basic associative learning processes. The smoker learns to associate certain cues with smoking or withdrawal, and these cues gain the capacity to elicit smoking behavior either by increasing the perceived motivation for a cigarette or by automatizing self-administration. Smokers who are more dependent are exposed to more salient cues more frequently than less dependent smokers. Some sample items from this domain include the following: "My life is full of reminders to smoke" and "There are particular sights and smells that trigger strong urges to smoke."

Loss of Control

Loss of control is not a motive to smoke per se; however it does provide an important measure of how addicted a smoker feels, how compulsive the smoker feels his or her drug use is. The assessment of control may provide important information regarding the smoker's ability to quit smoking. If a smoker endorses significant loss of control, it is hypothesized that he or she will be less likely to quit smoking than will the smoker who feels that he or she still has some control over his or her drug use. Sample items to assess level of control include: "Cigarettes control me" and "Sometimes I feel like cigarettes rule my life."

Negative Reinforcement

This motive for drug use is based on the operant conditioning learning theory that operant behaviors that alleviate an aversive physical or psychological state are reinforcing and increase the probability that those behaviors will be repeated. Within this domain, the aversive state may be due to life events such as stress or they may be due to withdrawal symptoms. Smokers may differ in the severity or frequency of their aversive states, such that more dependent smokers will have more severe and/or more frequent aversive states because of either withdrawal symptoms or negative life events and stress, causing them to use cigarettes more frequently to alleviate these states. Smokers have also been shown to differ on their expectancies that smoking will alleviate their distress (e.g., Brandon & Baker, 1991). This suggests that the operant conditioning model may be paralleled by subjective awareness. Therefore, questionnaire items attempted to assess these individual differences within the negative rein-

forcement domain. Some sample items include: "Smoking a cigarette improves my mood" and "Smoking helps me deal with stress."

Positive Reinforcement

This motive is based on Thorndike's law of effect, which states that behaviors followed by positive outcomes are strengthened and more likely to reoccur. Therefore, behaviors that result in positive experiences, such as a high or a buzz, are reinforcing and are more likely to be repeated. Items address the following: the perceived intensity of appetitive effects, the reliability between smoking and appetitive effects, and the nature of appetitive effects. It is important to note that items were written so as to preserve the distinction between the appetitive positive reinforcement and negative reinforcement models. It is predicted that more dependent smokers will report more positive reinforcement motives for smoking than will less dependent smokers. Examples of sample items designed to tap positive reinforcement motivation include: "Smoking makes a good mood better" and "Smoking makes me feel content."

Social-Environmental Goads

This motive also plays an important role in motivating drug use, and it may be especially important in the initiation as well as the maintenance of drug behavior. Social learning theory, proposed by Bandura, posits that individuals can learn by observing the behavior of others. Not only may modeled smoking behavior influence initiation, but a lack of modeled abstinence behavior may make it very difficult for smokers to quit. Additionally, social or occupational environments may promote smoking, making cessation difficult. Therefore, smokers with more smokers in their environment or smokers who interact with other smokers who do not value cessation will have a harder time quitting. Examples of questions to assess social and environmental goads include: "I'm around smokers much of the time" and "Most of the people I spend time with are smokers."

Taste and Sensory Properties

The taste and sensory properties of smoking are being considered as separate motives from positive reinforcement, although the law of effect and operant-conditioning principles are relevant to these motives as well. The more positive the experience of smoking a cigarette is, the more that behavior is strengthened. Therefore, even if the taste and sensory properties were not reinforcing initially, every time a smoker has a cigarette and enjoys the taste, smell, and so forth, these positive sensations increase the likelihood that the smoking will be repeated. Smokers who are more dependent will have a stronger appreciation for the taste and sensory properties of smoking. Some examples of questions to assess this domain are "I enjoy the taste of cigarettes most of the time" and "I love the feel of inhaling the smoke into my mouth."

Tolerance

This motive is a necessary component of dependence and is frequently considered to be one of the defining characteristics of dependence. Tolerance theory is based on the idea that homeostatic adaptations to the presence of drug in the body oppose the drug effect, rendering the tissues less sensitive to the drug. This enables the individual to tolerate higher doses of drug without suffering its toxic effects, and individuals require

higher doses of the drug to achieve the same subjective high. Tolerance is a very physiological construct and is probably best measured using some sort of physiological assay. However, this questionnaire can tap into some aspects of tolerance that are available through self-report. Smokers who are dependent no longer experience the toxic effects of nicotine, such as nausea, that novice smokers experience. Additionally, more dependent smokers will report smoking more now than they used to. Some sample items include "I usually want to smoke right after I wake up" and "I can only go a couple hours between cigarettes."

Weight Control

Finally, smokers may be motivated to continue using the drug for the purpose of controlling their weight. Cigarettes do appear to increase metabolism and serve as an appetite suppressant. This weight control motive may occur in response to weight loss that occurred after smoking was initiated or it may be driven by a fear of gaining weight once the smoker quits. People who are concerned about their weight or concerned about controlling and/or suppressing their hunger may have more trouble quitting smoking. Items that tap into the weight control motive include "Smoking keeps me from gaining weight" and "I rely upon smoking to control my hunger and eating."

It is important to acknowledge the limitations of this assessment strategy. First, there are many general limitations to using a self-report measure to assess motives (Baker & Brandon, 1990). It is possible that the items written to tap specific constructs may activate different or altered constructs in participants. Item wording may present problems in the consistency of interpretation across participants. There is a certain amount of response reactivity inherent in self-report measures. Self-report measures are likely to increase attentional focus on the construct being measured, which, in turn, influences that construct. Specifically for smoking, answering questions regarding smoking motivations may activate certain motivations and make those motivations more salient than they would be if the participant was not answering questions regarding his or her smoking. Individual differences, such as personality variables or social desirability, also influence participants' responses to self-report measures. In addition to these more global limitations to self-report assessment, there is one additional caveat specific to this proposed questionnaire. It is possible that the constructs we are asking participants to report on may not be accessible to self-awareness (e.g., Automaticity); however, we have done our best to ask questions that tap conscious processes but that also assess the latent motive of interest. Despite these limitations, given the necessities of clinical research and past research indicating that self-report measures can produce valid and reliable results, we believe that a self-report measure is appropriate (Baker & Brandon, 1990).

In each domain, the presence of the motive was assessed, as were characteristics of the motive. For example, cravings may vary along a variety of dimensions, such as frequency, intensity, and controllability of cravings. Some smokers may have frequent mild cravings, whereas others may have very intense cravings less often. It is important to examine each of these dimensions in relation to drug motivation and dependence. In addition, smokers may differ in the number of cues they encounter, the frequency with which they encounter smoking cues, and the salience of these cues. Therefore, individual characteristics of each motive were assessed along with the presence of the motive itself.

Appendix B

The Wisconsin Inventory of Smoking Dependence Motives (WISDM-68)

Below are a series of statements about cigarette smoking. Please rate your level of agreement for each using the following scale:

	1 Not true of me at all	2	3	4	5		6		7	Extremo of	ely true me	
1.	I enjoy the taste of	of cigarette	s most of the ti	me.		1	2	3	4	5	6	7
2.	Smoking keeps m	e from gai	ning weight.			1	2	3	4	5	6	7
3.	Smoking makes a	good moo	d better.			1	2	3	4	5	6	7
4.	If I always smoke	e in a certa	in place it is ha	ard to be there	and not	1	2	3	4	5	6	7
	smoke.											
	I often smoke wit		ing about it.			1	2	3	4	5	6	7
	Cigarettes control					1	2	3	4	5	6	7
	Smoking a cigare					1	2	3	4	5	6	7
	Smoking makes r					1	2	3	4	5	6	7
	I usually want to					1	2	3	4	5	6	7
	Very few things g			like cigarettes.		1	2 2	3	4	5	6	7 7
	It's hard to ignore	_				1 1	2	3	4 4	5 5	6 6	7
	The flavor of a ci		_			1	2	3	4	5	6	7
	I can only go a co	•		ettes		1	2	3	4	5	6	7
	I frequently smok		_			1	2	3	4	5	6	7
	I rely upon smoki		•			1	2	3	4	5	6	7
	My life is full of			and carring.		1	2	3	4	5	6	7
	Smoking helps m					1	2	3	4	5	6	7
	I smoke without					1	2	3	4	5	6	7
	Cigarettes keep m	_		riend.		1	2	3	4	5	6	7
	Few things would					1	2	3	4	5	6	7
22.	I'm around smoke	ers much o	f the time.			1	2	3	4	5	6	7
23.	There are particul	ar sights a	nd smells that t	rigger strong u	rges to	1	2	3	4	5	6	7
	smoke.											
24.	Smoking helps m	e stay focu	ssed.			1	2	3	4	5	6	7
	Smoking helps m					1	2	3	4	5	6	7
	I frequently light	-	,	g about it.		1	2	3	4	5	6	7
	Most of my daily	-	-			1	2	3	4	5	6	7
	Sometimes I feel	_	•	е.		1	2	3	4	5	6	7
	I frequently crave	-		1		1	2	3	4	5	6	7
	Most of the people					1	2 2	3	4	5	6	7
	Weight control is			oke.		1 1	2	3	4 4	5 5	6	7 7
	I usually feel muc		_			1	2	3	4	5	6 6	7
	Some of the cigar I'm really hooked		-			1	2	3	4	5	6	7
	Smoking is the fa	_		lf		1	2	3	4	5	6	7
	Sometimes I feel		•			1	2	3	4	5	6	7
	My urges to smol					1	2	3	4	5	6	7
	I would continue					1	2	3	4	5	6	7
	time on my hobbi	_										
39.	My concentration			g a cigarette.		1	2	3	4	5	6	7
40.	Seeing someone s	moke mak	es me really wa	ant a cigarette.		1	2	3	4	5	6	7
41.	I find myself read	ching for ci	garettes withou	t thinking abou	t it.	1	2	3	4	5	6	7
42.	I crave cigarettes	at certain t	times of day.			1	2	3	4	5	6	7
	I would feel alone					1	2	3	4	5	6	7
	A lot of my friend					1	2	3	4	5	6	7
	Smoking brings n					1	2	3	4	5	6	7
	Cigarettes are aboneed it.				when I	1	2	3	4	5	6	7
	Other smokers we		•	smoker.		1	2	3	4	5	6	7
	I feel a strong bo					1	2	3	4	5	6	7
49.	It would take a prosmoking.	retty seriou	s medical prob	lem to make m	e quit	1	2	3	4	5	6	7
50.	When I haven't b gets intolerable.	een able to	smoke for a fe	ew hours, the c	raving	1	2	3	4	5	6	7
51.	When I do certain	n things I k	now I'm going	to smoke.		1	2	3	4	5	6	7
	Most of my friend	_				1	2	3	4	5	6	7

(Appendixes continue)

Appendix B (continued)

53. I love the feel of inhaling the smoke into my mouth.	1	2	3	4	5	6	7
54. I smoke within the first 30 minutes of awakening in the morning.	1	2	3	4	5	6	7
55. Sometimes I'm not aware that I'm smoking.	1	2	3	4	5	6	7
56. I'm worried that if I quit smoking I'll gain weight.	1	2	3	4	5	6	7
57. Smoking helps me think better.	1	2	3	4	5	6	7
58. Smoking really helps me feel better if I've been feeling down.	1	2	3	4	5	6	7
59. Some things are very hard to do without smoking.	1	2	3	4	5	6	7
60. Smoking makes me feel good.	1	2	3	4	5	6	7
61. Smoking keeps me from overeating.	1	2	3	4	5	6	7
62. My smoking is out of control.	1	2	3	4	5	6	7
63. I consider myself a heavy smoker.	1	2	3	4	5	6	7
64. Even when I feel good, smoking helps me feel better.	1	2	3	4	5	6	7
65. I reach for cigarettes when I feel irritable.	1	2	3	4	5	6	7
66. I enjoy the sensations of a long, slow exhalation of smoke.	1	2	3	4	5	6	7
67. Giving up cigarettes would be like losing a good friend.	1	2	3	4	5	6	7
68. Smoking is the easiest way to give myself a lift.	1	2	3	4	5	6	7

Appendix C

WISDM-68 Scoring Key

Item numbers	Motive assessed
20, 36, 43, 48, 67	Affiliative Attachment
5, 19, 26, 41, 55	Automaticity
6, 28, 34, 62	Loss of Control
10, 21, 35, 38, 46, 49, 68	Behavioral Choice-Melioration
13, 15, 24, 39, 57	Cognitive Enhancement
11, 29, 37, 50	Craving
4, 17, 23, 40, 42, 51, 59	Cue Exposure-Associative Processes
7, 18, 25, 32, 58, 65	Negative Reinforcement
3, 8, 45, 60, 64	Positive Reinforcement
22, 30, 44, 52	Social-Environmental Goads
1, 12, 27, 33, 53, 66	Taste and Sensory Processes
9, 14, 47, 54, 63	Tolerance
2, 16, 31, 56, 61	Weight Control

Note. To calculate the scores of the subscales, take the mean of the items that load onto each subscale. The total scale score is the sum of all of the subscale scores, or a sum of the means for each subscale.

Received October 20, 2002
Revision received March 22, 2003
Accepted June 27, 2003