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Tobacco Dependence: Insights from Investigations of Self-Reported Smoking Motives

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Abstract

The Wisconsin Inventory of Smoking Dependence Motives (WISDM) assesses 13 theoretically-derived dimensions of smoking motivation. These 13 subscales were intended to index comprehensively the severity of tobacco dependence and provide insight into the disorder. Recent studies indicate that four subscales (Automaticity, Craving, Loss of Control, and Tolerance) represent the core features of tobacco dependence and have been dubbed the Primary Dependence Motives (PDM). The remaining nine subscales, the Secondary Dependence Motives (SDM), may be clinically relevant but index less essential features of dependence.

Tobacco dependence (TD) is viewed as the trait or construct that causes smokers to smoke countless cigarettes despite severe health risks, return to smoking shortly after trying to quit, and suffer when they try to stop (i.e., experience withdrawal symptoms). In essence, TD is the construct hypothesized to index the strength of tobacco addiction and explain why some smokers use tobacco so heavily and chronically.

There are practical reasons for assessing TD. For instance, measures of TD could flag individuals who need intensive treatment and could be used to ascertain the prevalence and trends in this disorder. In addition to these practical considerations, assessments are key tools for research designed to deepen our understanding of the nature of TD.

The most commonly used TD assessment is the Fagerström Test for Nicotine Dependence (FTND; Heatherton, et al., 1991). The FTND yields a quasi-continuous score reflecting the degree of dependence. It comprises six questions measuring the heaviness of smoking and an inability to tolerate abstinence. Previous reviews (e.g., Piper et al., 2006) show that the FTND has been moderately successful at satisfying the practical goals of a TD assessment. The FTND does a good job, for instance, of predicting who will be unlikely to quit smoking, and some of its items are useful for assigning smokers to different treatments (e.g., low vs. high-dose nicotine lozenges).

The FTND, and most other TD assessments (e.g., the Diagnostic and Statistical Manual of the American Psychiatric Association; DSM-IV; APA, 1994), however, have been less successful at providing insight into the nature of TD. Simply put, these instruments measure the outcomes of dependence, such as heavy smoking, but not the processes or mechanisms of dependence. An analogy would be if we assessed dementia only on the basis of cognitive decline and did not attempt to isolate the causes or mechanisms of decline (e.g., multi-infarct

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dementia, Lewy bodies, Alzheimer's disease). For many practical or clinical uses, simply indexing the presence and severity of defining end-stage symptoms is all that is needed. However, there is obviously some advantage to trying to "backtrack" from the end-state of the disorder to determine what mechanisms led to it. These deeper insights can enrich the theoretical understanding of the causes of disorder, potentially generating new hypotheses concerning strategies for treatment and prevention.

Another concern with the FTND and the DSM is that they narrowly focus on a small number of TD features. For instance, the FTND was designed primarily to assess the tendency to smoke in response to abstinence distress (Piper et al., 2006). It seems vital, at least initially, to assess a comprehensive set of candidate TD mechanisms. This is necessary to gauge the *relative* validities of different TD features or mechanisms - in other words, to determine which features are, and are not, defining or central features of TD.

In sum, while existing measures of TD do a good job of predicting some important effects of TD such as relapsing back to smoking, we have little understanding of why these measures "work" and we do not know what features of tobacco use are most critical to a person being dependent. The research we have undertaken, reviewed below, is an attempt to elucidate the nature or structure of TD and identify the features of TD that are most related to important criteria (e.g., relapse).

Derivation and Validation of the WISDM

The Wisconsin Inventory of Smoking Dependence Motives (WISDM; Piper, et al., 2004) was intended to elucidate the nature of TD mechanisms by identifying a comprehensive set of motivational processes that might lead a person to use nicotine excessively. A first step was to survey experts to identify the leading theory-based motivations for TD. Thirteen candidate influences were identified that formed the basis of the WISDM subscales (Table 1). Therefore, the WISDM was designed to address two limitations of the DSM and FTND; it targeted mechanisms, and it attempted to do so comprehensively.

Extensive testing with multiple large samples of smokers led to the development of an instrument with highly reliable subscales (Piper, et al., 2004). We next conducted a series of analyses to determine which subscales are central to TD. We hypothesized that, if we found a group of smokers who were highly dependent, based on TD criteria such as relapse vulnerability, but who showed elevations on *only* a subset of WISDM subscales, that subset might reflect the necessary and sufficient motivational influences for TD.

We used an empirical technique called latent profile analysis to identify subgroups of smokers who were similar to one another with respect to their profiles of WISDM subscale scores (Piper, et al., 2008). Across four independent samples, five subgroups of smokers were found in each sample. Four of the groups differed from one another only in terms of their average elevation across *all* subscales. In other words, if they scored highly on one motivational influence they scored highly on all. This is consistent with a view that people differ only in overall level of TD severity and that there are no "special" groups of people who show markedly different elevations on one type of symptom vs. another (e.g., Shiffman, 1993). However, *each* of the four samples revealed a fifth group – a group that scored relatively highly on *only* four subscales: Automaticity, Craving, Loss of Control, and Tolerance. Figure 1 shows the latent profiles that were estimated when all four samples were combined. This figure shows that the ratings of one group of subjects (labeled the "atypical" group) were best estimated by a profile with high scores on only the four subscales mentioned above. The error bands in the figure show that the atypical group's scores were not parallel to those of other smokers across the subscales - - but only about 20% of smokers were in this atypical group - - could this 20% be telling us something of general importance?

We then performed factor analyses on the data from all individuals in these four samples. Factor analysis is used to determine which variables in a set are so strongly interrelated that they can be combined to form a single dimension or factor. Each factor can be thought of as a possible causal process that influences all of the variables forming the factor and that accounts for the observed correlations among these variables. These analyses determined that the four subscales noted above constituted one factor, while all the remaining subscales constituted a separate factor. We labeled the four subscales (Automaticity, Craving, Loss of Control, and Tolerance) the Primary Dependence Motives (PDM) and the other subscales the Secondary Dependence Motives (SDM). Thus, the four PDM subscales form a coherent dimension (potentially reflecting they arise from a common cause) and a group of smokers scores relatively highly on only these subscales. The next step was to determine whether these subscales were especially highly related to important TD criteria. The intent was to investigate whether elevations on just these four scales are enough to consider a person dependent (i.e., the PDM are “sufficient” for producing TD).

Piper et al. (2008) used the PDM and SDM composites to predict TD criteria. When both composites were jointly entered in prediction models, the PDM composite outperformed the SDM composite in predicting most criteria (Table 2). The SDM composite was superior to the PDM composite only in the prediction of the intensity of craving on the first day of a quit attempt. As Table 2 shows, for most outcomes, once variance in the PDM was statistically controlled, the SDM composite had little relation with TD. (The SDM had no significant validity beyond its correlation with the PDM.).

Thus far we had shown that the PDM has relatively strong relations with smoking heaviness as assessed by self-report and carbon monoxide levels. Although encouraging, it was still unclear whether the PDM scales index strong *motivation* to smoke. One concern is the well-known caveat that correlation does not imply causation. Conceivably, high scores on the PDM could be the *result* rather than a *cause* of heavy smoking. For example, a person whose life circumstances allow frequent smoking (e.g., fewer smoking restrictions) might simply notice s/he smokes a lot, infer that his/her smoking must be automatic or out of control, and therefore achieve high scores on some PDM scales. If the PDM really reflects a drive to smoke, this should be apparent when smokers with differing scores are given access to tobacco in a novel, laboratory context. Using a task in which deprived smokers could work to earn individual cigarette puffs, we found smokers with higher PDM scores worked harder and consumed more puffs over the course of a three-hour laboratory session (Piasecki, Piper, & Baker, 2010) than did other smokers. Scores on the FTND were also related to self-administration, but when the PDM, SDM, and FTND were all entered into the prediction models simultaneously, only the PDM was significant in predicting laboratory self-administration. This suggests that the predictive validity of the FTND depends upon the construct measured by the PDM. Other research (e.g., TTURC, 2007) shows especially strong relations between the PDM and the FTND (see also Table 2).

We believed that the SDM subscales assess reasons for nondependent smoking (i.e., when people *choose* or *elect* to smoke), but that such motives do not account for heavy smoking or severe TD (i.e., when people smoke automatically and have lost control or are *compelled* to smoke). This suggested that we would find a particular pattern in the relation of smoking heaviness and the PDM and SDM subscales such that the SDM subscales would tend to account for smoking heaviness at low levels of use while the PDM subscales would account for smoking heaviness at high levels of use. Figure 2 shows the relation between representative PDM and SDM subscales and an index of smoking heaviness (Piper et al., 2004). As expected, up to a certain point, increases in smoking heaviness are tracked sensitively by increases in endorsement of the SDM subscales. However, the trajectories of

the PDM remain relatively flat until higher levels of smoking heaviness are achieved, when the strength of endorsement increases, literally, exponentially.

We also hypothesized that if the PDM score reflects the core of TD, it should show strong associations with genetic factors that permit or promote dependence. Prior research (Weiss, et al., 2008) strongly implicates four chromosome 15 haplotypes (labeled A, B, C, and D) as amongst the genetic foundations of TD, with carriers of Haplotype A being at increased risk of TD. Baker et al. (2009) found that smokers who carried Haplotype A had higher scores on PDM than did smokers with other haplotypes, and that SDM had no association with this haplotype beyond its relation with PDM (the same pattern that emerged in Table 2).

In summary, what has been demonstrated is: 1) certain dependence motivational influences ("Primary" motives) were highlighted because a subgroup of dependent smokers scored highly on only these influences; 2) these motives form a homogeneous factor distinct from other dependence influences; 3) these PDM influences are especially highly related to core dependence features (smoking heaviness, other valid dependence scales, laboratory self-administration, genetic risk, and relapse likelihood) and once variance due to the PDM subscales is statistically controlled, the SDM subscales tend not to be related to such features; 4) the PDM subscales appear to account for much of the predictive validity of the leading clinical measure of TD, the FTND; and 5) while the SDM subscales reflect differences in amount smoked in lighter smokers, the PDM subscales seem to reflect differences in amount smoked amongst heavier smokers. These findings are consistent with the notion that the PDM subscales may index necessary and sufficient features of TD that arise with especially heavy tobacco use.

Meaning and Implications?

There are some important caveats to our accepting the above findings at face value. For instance, it is unclear whether self-report instruments like the WISDM accurately reflect the underlying biology or information processing mechanisms targeted by some of the scales. But, there is evidence that some of its subscales do tap the targeted mechanisms. For instance, high scores on the Taste/Sensory Processes subscale accurately predict PTC ("taster") genotypes (Cannon et al., 2005) and the Tolerance subscale strongly predicts self-administration (Piasecki et al., 2010). However, some relations between particular WISDM subscales and theoretically linked real-world events are weak (Japuntich et al., 2009; Piasecki et al., 2007). This is an important topic for future research. However, to the extent that the WISDM results are telling us something about TD, what do they suggest?

Considerable evidence from animal models suggests *habit learning* accounts for addictive drug self-administration. This research indicates that, with practice, drug self-administration behaviors are elicited by the stimuli that *signal* drug, rather than being sustained by the *effects* of drug (Everitt & Robbins, 2005). Another important insight is that over many drug use occasions, the self-administration response becomes automatic (i.e., it occurs effortlessly, without much intention or even awareness, and it takes effort to stop it; Tiffany, 1990). So, for a heavy user, the innumerable drug cues present in daily life powerfully elicit the drug self-administration response which tends to occur effortlessly and without planning -- as if it has a life of its own.

This picture seems consistent with the nature of TD as it is portrayed by the PDM. The atypical smokers may be telling us that, contrary to some models of drug dependence and motivation, they do not smoke because of the consequences of smoking - - they do not smoke to gain relief from withdrawal or dysphoria, to experience a high, pleasure, or a "rush," or because of taste or the allure of enhanced cognition. Rather, they smoke because the act of smoking has escaped their control and now occurs automatically - - without

planning, effort, or awareness. Moreover, they may be telling us that craving is part of this package. Why would this be? When automatic behaviors are prevented or short circuited, a person suddenly becomes aware of this and must, with considerable effort, use cognitive control resources to decide how to act without the tried-and-true automatic response (Tiffany, 1990). In essence, craving is awareness of not smoking - - not smoking where smoking has occurred innumerable times before.

What relevance do our results have for smokers and for treatment? First, they suggest why some treatments are effective (e.g., those that break-up the contingencies between cues and smoking). The results also encourage extensive practice of nonsmoking responses in typical smoking settings prior to the quit attempt to sap the power of smoking cues to influence behavior. Further they suggest new treatments might be developed for those who differ greatly in endorsement of PDM vs. SDM motives. Individuals smoking for primary motives might be helped by interventions that change environmental cues, while the latter might be better helped by treatments that affect the consequences of smoking and smoking deprivation, such as the treatment of withdrawal.

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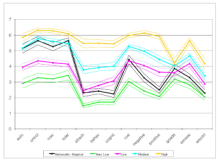

Figure 1. Latent profile curves for the 13 WISDM subscales using four independent samples

Results of a latent profile analysis of WISDM subscale scores using data pooled from 5 samples of smokers (N=2,257). Each line represents the estimated subscale score means for each empirically-identified group (possible scale scores ranged from 1–7). The 95% confidence intervals around each profile are also shown. The "Automatic Atypical" group (16.9% of the sample) is characterized by relative elevations on the Automaticity, Loss of Control, Craving, and Tolerance. A group showing a similar pattern was recovered from each of the individual samples contributing to the pooled analysis. The remaining groups of smokers ("Very Low," 14.0% of the sample; "Low," 24.8%; "Medium," 30.2%; "High," 14.0%) tended to be distinguished by differences in the elevation of the profile rather than the pattern of scores. The "Automatic Atypical" group may represent a "pure group" of smokers who show the core features of tobacco dependence without the secondary features. These kinds of observations led us to designate Automaticity, Loss of Control, Craving, and Tolerance the Primary Dependence Motives. Figure reprinted with permission from: Piper, M.E., Bolt, D.M., Kim, S., Japuntich, S.J., Smith, S.S., Niederdeppe, J., Cannon, D.S. & Baker, T.B. (2008). Refining the tobacco dependence phenotype using the Wisconsin Inventory of Smoking Dependence Motives. *Journal of Abnormal Psychology*, *117*, 747–761.

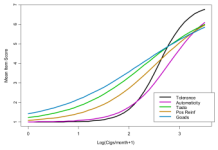

Figure 2. Curves comparing PDM and SDM subscale performance compared to smoking heaviness

This figure illustrates how the endorsement of representative Primary Dependence Motives (PDM) and Secondary Dependence Motives (SDM) change as a function of smoking heaviness. The PDM subscales (Tolerance and Automaticity) are not highly endorsed at low levels of smoking heaviness, but their scores increase exponentially once smoking heaviness reaches a certain level. Conversely, SDM subscales (Positive Reinforcement [‘Pos Reinf’], Social/Environmental Goads [‘Goads’] and Taste/Sensory Properties [‘Taste’]) continue to increase in a linear fashion across the range of smoking heaviness. Figure reprinted with permission from: Piper, M. E., Piasecki, T. M., Federman, E. B., Bolt, D. M., Smith, S. S., Fiore, M. C., & Baker, T. B. (2004). A multiple motives approach to tobacco dependence: The Wisconsin Inventory of Smoking Dependence Motives (WISDM-68). *Journal of Consulting and Clinical Psychology*, *72*, 139–154.

Table 1

Content of the WISDM-68 Subscales

Subscale	Target Construct
Primary Dependence Motives[a]	
Automaticity	Smoking without awareness or intention
Craving	Smoking in response to craving or experiencing intense or frequent urges to smoke
Loss of Control	Believing one has lost volitional control over smoking
Tolerance	Needing to smoke increasing amounts over time to experience the desired effects, or smoking large amounts without acute toxicity
Secondary Dependence Motives[a]	
Affiliative Attachment	Having a strong emotional attachment to smoking and cigarettes
Behavioral Choice/Melioration	Smoking despite constraints on smoking or negative consequences and/or the lack of other options or reinforcers
Cognitive Enhancement	Smoking to improve cognitive functioning
Cue Exposure/Associative Processes	Frequently encountering nonsocial smoking cues or experiencing a strong link between cue exposure and a desire or tendency to smoke
Negative Reinforcement	Smoking to ameliorate negative internal states
Positive Reinforcement	Smoking to experience a “buzz” or “high” or to enhance an already positive feeling or experience
Social/Environmental Goads	Having social stimuli or contexts either model or invite smoking
Taste/Sensory Processes	Smoking to experience the orosensory/gustatory effects of smoking
Weight Control	Smoking to control body weight or appetite

[a] Classification of motives based on analyses reported by Piper, et al. (2008); see text.

Table 2

Linear (continuous outcomes) and logistic regressions (binary outcomes) when both PDMs and SDMs are simultaneously entered to predict markers of tobacco dependence

Dependent variable	Analysis	Predictor	B	SE	B	t
FTND	Linear	PDM	.21	.01	.66	25.21**
		SDM	−.04	.01	−.10	−3.74**
Cigarettes per day	Linear	PDM	3.97	.26	.46	15.46**
		SDM	−1.02	.28	−.11	−3.58**
Breath carbon monoxide	Linear	PDM	3.41	.31	.35	11.07**
		SDM	−1.76	.34	−.16	−5.19**
Quit day increase in craving	Linear	PDM	.32	.11	.10	2.88**
		SDM	.92	.12	.26	7.45**
Age of smoking initiation	Linear	PDM	−.28	.11	−.08	−2.53*
		SDM	−.04	.12	−.01	−.29
Age began smoking daily	Linear	PDM	−.43	.11	−.12	−3.83**
		SDM	.10	.12	.03	.85
			B	**SE**	**B**	**OR**
Relapse at 1 week (no = 0)	Logistic	PDM	.20	.07	9.54	1.23**
		SDM	.02	.07	.06	.80
Relapse at 6 months (no =0)	Logistic	PDM	.25	.08	10.31	1.28**
		SDM	−.07	.09	.63	.94

* $p \leq .05$

** $p < .01$

Note: Concentration of carbon monoxide in a breath sample is a sensitive biochemical indicator of the heaviness of recent smoking. Higher absolute values of B and β indicate stronger relations between the predictor and the dependent measure. Odds ratio (OR) estimates higher than 1.0 indicate the predictor is associated with an increased odds of relapse. SE = standard error.