What can dependence theories tell us about assessing the emergence of tobacco dependence?

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ABSTRACT

Little is known about the processes that underlie changes in smoking that occur between the first use of a cigarette, subsequent regular use and eventual addictive use. At present, assessments of those critical processes are poorly developed and not strongly informed by contemporary models of drug dependence. The preceding three papers in this special issue address explicitly how modern drug-dependence theories describe the emergence of drug dependence and the implications of those theories for assessment. The papers covered three domains of theories: negative reinforcement, positive reinforcement, and cognitive and social learning. In this paper, we summarize these reviews and extract general themes and issues that emerge across all the articles. These include: (1) the importance of learning processes; (2) limitations of self-report measures; (3) the view of dependence as a process and not a state; (4) the conception of dependence on a continuum in contrast to the conventional perspective of tobacco dependence as a natural category; (5) the ontological status of the dependence concept; (6) limitations of backward extrapolations from adult assessments; (7) the possibility of multiple dimensions or forms of dependence; and (8) the value of a transdisciplinary approach when studying the emergence of tobacco dependence.

KEYWORDS Assessment, cigarette, drug-dependence, tobacco.

OVERVIEW

Our motivation for this special issue was both a recognition of the need for assessment instruments able to capture the emergence of tobacco dependence, as well as the belief that efforts to create such measures would benefit from careful consideration of existing drug dependence theories. The idea that measures are best developed within the context of theoretical considerations is not new in either the general assessment literature (e.g. Loevinger 1957; Wiggens 1973) or the drug-abuse literature (e.g. Tiffany & Drobes 1991; Shiffman *et al.* 2004). Our position is that, whether implicit or explicit, presuppositions about the fundamental nature of the phenomenon being measured inform the content, format and measurement model of assessments.

Wiggens (1973, p. 401) asserted that, when constructing assessments, 'It seems desirable to substitute explicit theory for implicit theory and to sample systematically on the basis of that theory than to sample fortuitously'. Unfortunately, most measures of drug dependence rely on implicit rather than explicit propositions regarding the nature of dependence. Because of this generally loose connection between concept and measurement, data generated from traditional approaches to assessing drug dependence tell us little about the fundamental features of dependence. Such data also fail to offer us much about whether or not a particular instrument accurately represents those implicit concepts that informed the instrument's development. We believe that each of the three papers in this issue provides multiple ideas for how and why contemporary theories of

addiction can be used to generate assessments of the emergence of tobacco dependence.

The authors' efforts to translate theories into assessments will undoubtedly increase the scope and conceptual coherence of dependence measurement. However, this exercise can also work to the benefit of theory evaluation and development. Any attempt to use a theory to create an instrument rapidly exposes the practical value and limitations of that theory. This task is particularly informative when we are interested in the *emergence* of tobacco dependence. Many of the theories reviewed in these papers focus primarily on the end stages of drug dependence, and are surprisingly silent on the question of how dependence develops initially. However, they can be extrapolated to be informative about the early phases as well. Furthermore, several of the theories were created initially to capture alcohol, stimulant or opiate dependence, not necessarily tobacco dependence. Finally, the theories seldom address directly how dependence or the processes posited to underlie it are manifest in measurable ways. Consequently, in many cases the authors were forced to speculate about how key dependence processes posited by a particular theory might be manifest in the early stages of dependence or how those processes could be assessed in the context of tobacco-related behaviors. In essence, the theories had to be extended beyond their original scope. By so doing, the authors give us insights into the core assumptions and conceptual expansion of these theories: insights not immediately evident upon initial theory evaluation. Thus, not only will these papers lead to better assessments, they should also lead to better theories. Before discussing these topics in greater detail, let us summarize briefly each of the papers here.

NEGATIVE REINFORCEMENT MODELS

Negative reinforcement models share the premise that tobacco dependence is controlled by a smoker's attempt to avoid or escape aversive states. Eissenberg (2004) reviews four types of negative reinforcement models: basic withdrawal, conditioned withdrawal, self-medication and opponent process. With the exception of the self-medication model, these models hypothesize that aversive states that motivate continued drug use are a consequence of repeated exposure to nicotine. Over the course of continued nicotine use, increasingly more aversive states result when an individual ceases use of the drug, thus avoidance of such aversive states comes to motivate continued use. Self-medication models suggest that aversive conditions such as depression or stress may pre-exist or occur independently of tobacco use, and also posits that smoking or nicotine actually provides relief from aversive conditions.

In each of these models, the tobacco user *learns* that smoking reduces or prevents aversive conditions. Thus, escape or avoidance learning is fundamental to these conceptualizations. Moreover, as this learning strengthens with tobacco experience, dependence is assumed to be an emergent rather than all-or-none phenomenon. Eissenberg suggests further that, in most cases, learning to escape aversive conditions will precede learning to avoid those conditions. This temporal distinction between these two forms of learning may be an important consideration in characterizing the emergence of dependence. For example, a major milestone in the development of dependence may be the transition from smoking marked primarily by escape to smoking controlled predominantly by avoidance of aversive conditions.

According to these models, the emergence of tobacco dependence is a joint function of the magnitude of the aversive state and the strength of escape and/or avoidance learning. Furthermore, with the exception of self-medication models, the patterning of initial tobacco-use episodes should influence strongly the extent to which the user becomes dependent. For example, basic drug-withdrawal models assume that tobacco withdrawal will emerge only if the user is exposed to a sufficient dose and frequency of nicotine. Without that exposure, the user will not experience withdrawal when he or she abstains from tobacco. Thus withdrawal avoidance or relief will not motivate use until the smoker escalates smoking to a point at which dependence starts to emerge.

None of these models is necessarily exclusive of the others; the basic dependence mechanisms specific to each approach may be operative to some degree in all users, or may be present primarily at one or more phases of the dependence trajectory. The conventional view of the motivating influence of withdrawal relief is that these processes are restricted to chronic, 'heavy' smokers. As Eissenberg makes clear, this view does not depict accurately contemporary negative reinforcement models of dependence. The crucial processes presumed by the models are operative at the initial exposures to tobacco.

Although the original incarnations of the theories reviewed by Eissenberg devoted scant attention to the early stages of dependence, each model is readily applicable to those issues. Indeed, as richly detailed by Eissenberg, negative relief models offer a wide array of self-report, behavioral and physiological variables that could be used for assessing the emergence of dependence. Interestingly, self-report measures, which are the most common means of indexing dependence, are not the only, and perhaps not even the most informative, approach. For example, we might believe that an efficient way to determine whether or not someone smokes to avoid withdrawal is to simply ask (e.g. reasons for smoking). However, smokers who have learned to effectively avoid

aversive conditions may not experience those conditions or even be aware of the processes motivating their smoking (see Baker *et al.* 2004), making the assessment of dependence across multiple indices of responding all the more important.

POSITIVE REINFORCEMENT MODELS

Although the models described by Glautier (2004) vary greatly in terms of postulated processes and levels of analyses, all assume that the immediate effects of drug support behaviors that promote continued drug use. Applying this position to cigarette smoking, these models assume that nicotine functions as a primary positive reinforcer that motivates continued smoking behavior over time.

Glautier reviews four subtypes of positive reinforcement models: (1) *operant models* describe dependence in terms of the reinforcing efficacy of nicotine and the control nicotine comes to exert over behavior; (2) *economic models* focus on the interaction between the magnitude of the reinforcer (nicotine dose) and the response requirements, or work, required to obtain the reinforcer; (3) *self-control models* characterize tobacco dependence as a consistent selection of nicotine over other rewards. The choice of nicotine rather than other rewards is a joint function of the relative magnitude of the available reinforcers and the relative delays of those rewards; and (4) *biological models* focus on changes in functioning of the central nervous system to identify the neurophysiological substrates of the positively reinforcing effects of nicotine.

All the models reviewed by Glautier share the assumption that dependence processes are continuous in nature. Also, as these models do not rely on the emergence of withdrawal as a basis for reinforcement, reinforcement and dependence processes may be evident during even early tobacco use. These models offer numerous ideas for assessing the emergence of tobacco dependence. These include measures of the distributions of smoking and non-smoking activities over time, decline in the reinforcing value of non-smoking reinforcers, interactions between reinforcers, tolerance to the reinforcing actions of nicotine, decreasing voluntary control over smoking, increasing stability of smoking patterns, rapidity of relapse following cessation of smoking, control of smoking by stimuli and environments associated with cigarette use and priming effects of nicotine on smoking behavior. To assess these effects, it is notable that these models generally describe reinforcement processes without reference to the subjective or affective manifestations of those processes. Thus nicotine could exert powerful control over behavior, even though its effects may not be as euphoric, exciting or pleasurable as those produced by other drugs of abuse. Therefore, self-report measures might be less important for evaluating the emergence of tobacco dependence than direct assessments of actual smoking behaviors and choices under conditions representative of the natural environments and reinforcement contingencies of the smoker.

COGNITIVE AND SOCIAL LEARNING MODELS

The models described by Brandon *et al.* (2004) derive from social learning theory, cognitive-behavioral traditions and modern cognitive sciences. Given the diversity of concepts invoked by these models, the authors chose to focus on four constructs that appear repeatedly in cognitive and social-learning theories of drug dependence. These are *expectancies*, *self-efficacy*, *coping* and *craving*. The authors selected major contemporary theories to represent each of these concepts and to illustrate a variety of psychological processes with potential relevance to assessment of the emergence of tobacco dependence.

All the models described by Brandon and colleagues assume that major features of smoking dependence can be captured in processes described in cognitive terminology. A theme running throughout most of these models is that expectancy beliefs regarding drug effects and likelihood of drug use or avoidance have a major controlling influence on the development of dependence. Expectancy *beliefs*, not the actual outcomes, drive drug-seeking behavior. A persistent belief that cigarette smoking will relax you will promote cigarette smoking even if the smoking does not have that effect. In addition, the models all assume that cognitive processes critical to dependence develop in a continuous fashion. Thus, none of these models assumes any functional discontinuity between light and heavy smokers.

By and large, cognitive models of drug dependence, particularly those that emphasize expectancy beliefs, suggest that self-report measures provide the best vehicle for uncovering the key processes controlling drug dependence. Brandon *et al.* offer numerous explicit proposals for constructing or refining adolescent-specific self-report questionnaires. Two of the models, however, Goldman *et al.*'s (1999) expectancy theory and Tiffany's (1990) cognitive processing theory, also emphasize the role of automaticity and unconscious cognitive processes in the emergence of drug dependence. That is, with their repeated activation the cognitive processes supporting drug use become increasingly efficient, entrenched and less accessible to verbal reports. From this perspective, self-reports may not be sufficient for mapping the cognitive mechanisms responsible for the development of

tobacco dependence. Here, Brandon and colleagues have described numerous suggestions for tapping processes and structures not available through traditional self-report methods. These include techniques such as implicit memory tasks, fine-grained observations of smoking behavior over time, dual-processing tasks and assessments of memory structure.

COMMON THEMES AND MESSAGES

As the papers' authors note, the diverse theories presented here, although emphasizing different aspects of dependence, are often compatible and even mutually interdependent. Those emphasizing positive reinforcement, for example, do not deny that deprivation leads to aversive states that are relieved by nicotine, promoting the process of negative reinforcement. They simply emphasize positive reinforcement as the more central component driving tobacco use and dependence. Cognitive theories do not deny the fact that nicotine has important neurobiological effects that subserve positive and negative reinforcement—indeed, they rely upon such effects in the theory. Having assumed such effects, the cognitive theories then use cognitive constructs to explain why such reinforcement leads to compelling patterns of use. Thus, although the theories present competing views of dependence and its development, they do not usually present us with contradictory or irreconcilable accounts.

Furthermore, despite considerable diversity of concepts and assessment implications derived from the theories, several general themes appear and these have profound implications for research and theory on the emergence of tobacco dependence.

Dependence involves learning

Nearly all modern models of drug dependence start with the assumption that repetitive drug-use is learned behavior. Although the particulars vary from theory to theory, most commonly, theoreticians invoke some form of classical (Pavlovian) conditioning, instrumental conditioning or cognitive-processing approach to describe how stimuli, responses and outcomes combine to generate escalations in drug use across time. According to negative reinforcement models (Eissenberg 2004), smokers learn that tobacco use decreases aversive states. Similarly, all the models reviewed by Glautier (2004) assume that the immediate effects of drugs support the acquisition and maintenance of smoking through learning processes. Finally, the models described by Brandon and colleagues (Brandon *et al.* 2004) share the proposition that transformations in the frequency and extent of smoking from initial episodes to more entrenched patterns of use are propelled and sustained by learning-based changes in cognitive processing.

There are two main reasons that all theories invoke learning processes to help explain dependence. The first is that certain key phenomena of drug abuse and dependence—notably the continued risk of relapse long after primary withdrawal has subsided and the ability of particular environmental stimuli to provoke craving and drug use—are difficult to explain without invoking learning processes. Secondly, because learning generally is strengthened over time and over repeated learning trials, learning processes help implicitly explain the trajectory from use to dependence over time and repeated exposure to the drug.

The assumption that smoking behavior is learned behavior has implications for assessing the emergence of tobacco dependence. Most importantly, the learning processes controlling dependence will be best captured when the conditions of assessment permit the expression of those processes. For example, the compensatory response theory of drug tolerance and dependence (Siegel 1975) posits that smokers will use cigarettes to avoid or escape from conditioned withdrawal syndromes that are triggered by cues previously associated with the direct effects of nicotine. According to this model, assessment of dependence requires testing for withdrawal effects in the presence of evocative cues as well as evaluating the extent to which a person seeks cigarettes when conditioned withdrawal is triggered. Thus, direct assessment of conditioned withdrawal requires that smokers be presented with cues associated presumably with past instances of smoking. As another example, the expectancy model of Goldman *et al.* (1999) hypothesizes that expectancy networks, which are established and differentiated through learning and memory processes, are activated when the current stimulus situation confronting the individual matches the informational template stored in memory. Consequently, expectancies are best accessed when the testing situation activates the drug network. In the case of either of these models (compensatory response or expectancy), the mechanisms responsible for dependence will not be directly evident unless they are assessed in the context of those critical situations that evoke dependence processes.

Self-report measures are limited

Conventional approaches to the assessment of tobacco dependence rely almost exclusively on self-report measures (Colby *et al.* 2000a). The papers by Eisenberg, Glautier, and Brandon (and colleagues) make it evident that widespread use of self-report measures in dependence research appears to be driven more by considerations of

convenience than by attempts to capture processes hypothesized by contemporary models of dependence.

Although self-report measures may provide convenient information about dependence, few of the dependence theories reviewed in these papers indicate that smokers would be able to provide accurate verbal descriptors of the processes that promote smoking behavior. As one example, if smoking escalates because an individual learns to effectively avoid aversive conditions (Eissenberg 2004), that person may not experience withdrawal or other aversive conditions that motivate smoking behavior.

Consequently, smokers successfully avoiding withdrawal would be increasingly less inclined to notice that they smoke to prevent withdrawal symptoms. Similarly, most of the appetitive and positive-reinforcement models reviewed by Glautier (2004) eschew self-report measures. These models emphasize, instead, changes over time in smoking behavior and in choices related to smoking. Many of the cognitive models discussed by Brandon *et al.* (2004) describe core dependence processes as relatively inaccessible via verbal report, as cognitive processes controlling smoking behavior would be no more available to self-reports than those controlling any other complex, well-rehearsed behavior.

Dependence is a process, not a state

Most studies of tobacco dependence adopt implicitly the position that dependence is a stable condition that develops following a history of regular smoking. The picture of tobacco dependence that surfaces from the models reviewed in this issue is considerably more dynamic than suggested by those traditional approaches to the assessment of dependence. Some of that dynamic perspective arises as a natural consequence of the authors' use of contemporary theories to guide assessments of change in tobacco dependence over use or, rather, the *emergence* of dependence. Emergence implies transformations in behavior over time and over situations. The theme embraced in these reviews is that dependence is a process that changes over time, not merely a static end-product of regular smoking. Furthermore, the concept of change suggests that research on the configuration of that change over time and situation may be substantially more revealing about the nature of dependence than research using static, one-shot, slice-in-time, group-comparison (dependent versus non-dependent) designs.

Dependence is a continuum

In addition to being thought of traditionally as a state rather than a process, dependence is also often talked about as an all-or-none condition; either you are a dependent smoker or you are not. This categorical model of tobacco dependence assumes that dependence arises when a smoker reaches a threshold level of nicotine exposure sufficient to trigger dependence processes, which are conceptualized implicitly as qualitatively different from 'normal' functioning. Once that threshold is reached the person is dependent on nicotine, and further exposure past that point will serve only to increase the severity of dependence. This view is consistent with the taxonomic approach to dependence espoused by modern diagnostic systems [e.g. *Diagnostic and Statistical Manual* (DSM) and *International Classification of Diseases* (ICD)], well-represented in the methodological foundations of much drug abuse research, and compatible with the beliefs of the general public regarding the nature of drug dependence.

The conception of dependence adopted by most modern theories of drug addiction is substantially divergent from the conventional, categorical view of tobacco dependence. In contrast to the categorical assumption of drug dependence, the theories reviewed in these papers maintain, explicitly or implicitly, that dependence varies on a continuum. That continuum is linked in turn to a continuum or trajectory of smoking behavior, with the basic elements of dependence processes evident even in the early episodes of cigarette use. That is, according to nearly all theories, dependence is fundamentally continuous and dimensional, not categorical, in nature. For example, in the opponent process model of Koob and colleagues (Koob & LeMoal 1997), repeated uses of nicotine alter the neurobiology of reward thresholds such that the cigarette-deprived individual is increasingly anhedonic: that is, less likely to find positive events rewarding. Thus, in this model, dependence reflects a continuous change in reward thresholds over repeated nicotine exposures; the model posits no discontinuity between the dependent and non-dependent user.

Some theories, at first glance, appear to support a categorical conceptualization of dependence. Most notably, withdrawal-based models propose that smokers maintain cigarette use to escape or avoid tobacco withdrawal (Eissenberg 2004). Consequently, it might be argued that the appearance of tobacco withdrawal following repeated use provides the critical transition distinguishing between dependent and non-dependent smokers. However, as noted by Eissenberg, withdrawal builds incrementally over repeated exposure to nicotine. Thus, withdrawal is not all or none; it is a continuous phenomenon. Moreover, there is good reason to believe that the neurobiological processes that produce withdrawal are activated at the first exposure to a drug (Eissenberg 2004). If so, given sufficiently sensitive measurements, withdrawal may be evident, to some extent, in any smoker, regardless of his or her previous smoking experience. Therefore, withdrawal will not provide a qualitative marker of dependence.

Consistent with this conception of dependence as a quantitative continuum, many measures of tobacco dependence yield continuous estimates (e.g. Heatherton *et al.* 1991; Shiffman *et al.* 2004). However, in practice many analyses dichotomize such measures to classify individuals as either dependent or not (Fagerström & Schneider 1989). Moreover, the medical–psychiatric tradition of diagnosis conceptualizes dependence as a pathological condition that is either present or absent (Robins & Helzer 1986). Similar conceptions are implicit in epidemiological research that estimates the prevalence of dependence (Anthony *et al.* 1994).

The issue of the fundamental latent structure of tobacco dependence is particularly relevant for studies of the development of dependence and has profound implications for dependence assessment. Ruscio & Ruscio (2002) note that it is important to match the measurement approach used to assess a construct to the latent structure of that construct. If, for example, tobacco dependence is a dimensional (or continuous) construct, it would be statistically and conceptually disadvantageous to adopt a measurement model that categorizes individuals as either dependent or non-dependent. On the other hand, if dependence is categorical, the use of a dimensional model to assess smokers (for example, locating a smoker along a dimension of dependence) would increase measurement error and decrease the validities of the assessment.

Interestingly, despite the apparent disconnection between predominantly dimensional theories of dependence and conventional categorical assumptions about the fundamental nature of dependence, there has been little discussion or programmatic research on this issue in the literature on addiction. We believe that resolution of this issue is critical as a prerequisite to systematic advances in our understanding of the development of tobacco dependence. Fortunately, there are now methods available to reveal the latent structure of the dependence construct, methods that require no a priori endorsement of any particular theory of tobacco dependence. These methods, which were developed originally by Paul Meehl and colleagues (Meehl 1973, 1995, 1999; Golden & Meehl 1979; Meehl & Yonce 1994; Waller & Meehl 1998), involve a family of taxometric procedures for identifying qualitative boundaries that demarcate latent groups or taxa. These procedures have been used by several researchers to investigate the latent structure of a variety of psychopathological conditions including schizophrenia, personality disorders, mood disorders, anxiety disorders, eating disorders and dissociative disorders (see review by Haslam & Kim 2002). Not only can these approaches help assess whether or not a given disorder is best conceptualized as either taxonic or dimensional, they provide evidence as to the base rate of any identified taxon as well as the optimal cut-point on variables used to define the taxon. At present, these taxometric procedures have not been used in any published studies of drug dependence. However, these procedures have considerable potential for major advances in our understanding of the nature of tobacco dependence.

Dependence consists of multiple dimensions and can take multiple forms

Aside from the question of whether or not dependence is continuous or categorical, our discussion thus far has conveyed the impression that tobacco dependence is either a unidimensional or a unicategorical phenomenon. There are reasons to believe that it is not. Many of the consultants who worked with the authors as well as the authors themselves advised that it would be overly simplistic to believe that any one process or mechanism might account for the diversity of behaviors that constitute tobacco dependence. As Eissenberg (2004) stated, 'Thus, readers should keep in mind that coverage of these mechanisms in separate papers is a matter of convenience, and the inclusion of these papers into one volume highlights the notion that all are relevant to understanding drug use in general and etiology of tobacco dependence specifically'.

Within an individual, initiation, escalation and maintenance of tobacco use may be controlled by multiple motivating influences. Across individuals there may be several variations of tobacco dependence, with each dominated by a particular class of motivating processes. For example, some people may smoke because of withdrawal relief or self-medication, whereas others may smoke because their behavior is captured by the positively reinforcing effects of tobacco. Still others may smoke because they have formed strong expectations that smoking will provide positive outcomes that far outweigh the negative consequences of tobacco use. Each of the theories described in these papers in effect paints a slightly different portrait of dependence, and different subgroups of smokers may best fit each of the different portraits. Or, multiple processes might be operative to some extent in all smokers, with one or a limited set of those processes having the greatest influence on smoking behavior in given individuals. Although particular theories emphasize particular mechanisms, many of the theories are mutually compatible, and seem simply to be 'grasping different parts of the elephant'. It seems likely that several of the theoretically postulated mechanisms may contribute to dependence.

The prospect that several processes may contribute to the formation of dependence is particularly important when considering the emergence of dependence. Different processes may be more or less active or more or less

important at different stages of development. For example, expectancies could be particularly important early in the process, particularly before the smoker has had much experience with smoking and nicotine. Positive reinforcement may play a particularly important role during the early exposures to smoking, when withdrawal is not yet fully developed. The role of negative reinforcement in smoking may grow as smokers discover its anxiolytic properties or find that deprivation leads to aversive states that are relieved by smoking (e.g. Herman 1974). An alternative developmental model suggests that different processes or dimensions emerge in a particular fixed sequence. For example, it may be precisely the repeated use for positive reinforcement that leads to the dysregulation of reward systems and thus the emergence of aversive states when nicotine is removed (Koob & LeMoal 1997). These considerations indicate that assessments targeting the initial stages of tobacco smoking will have to be multi-dimensional in scope (e.g. Shiffman *et al.* in press), capturing a variety of processes that may be at play over the course of emerging dependence.

A more complex possibility is that the dimensional structure of dependence itself may change as dependence develops. Aspects of dependence that initially are only loosely linked may coalesce into a unified whole, leading to a more unified dependence syndrome—i.e. to a reduction in the dimensionality of dependence. Conversely, dependence may become more articulated with increasing drug experience and increasing dependence, as has been observed for drug expectancies (Brandon *et al.* 2004).

Backward extrapolation from adult assessments is limited for the assessment of emergence

The foregoing implies that early manifestations of dependence may not simply be weaker, child-sized versions of full-blown or mature dependence. When developing assessments of the early stages of tobacco dependence, it is tempting to create instruments derived directly from those used for adult smokers. For example, the Fagerström Tolerance Questionnaire (Fagerström 1978), which was designed to assess tobacco dependence in adult smokers, has been used in several studies with adolescent smokers to evaluate dependence (see review by Colby *et al.* 2000a). These instruments are employed with the assumption that the manifestations of dependence assessed in the adult end-stage smoker are fully applicable to the adolescent or young adult in the early stages of smoking. The validity of this assumption is suspect on several grounds. First, experiences of tobacco dependence in young smokers might be qualitatively different from those of adults (Colby *et al.* 2000a). This possibility is particularly true when assessing adolescent smokers who may assign lower (or higher) salience to certain aspects of their smoking behavior than would adults. For example, adults might find that their concentration at work is diminished severely during abstinence from cigarettes; an adolescent might also have diminished concentration when abstinent from cigarettes but not be concerned about or acutely aware of that effect. Secondly, adolescents might experience the same configuration of dependence effects endorsed by adult smokers but use different specific terms or qualifiers to express those effects. Thirdly, and most importantly, the profile of behaviors that constitute markers of dependence may shift over the course of smoking, such that what might be most relevant for assessing dependence in a highly experienced smoker does not capture the essence of dependence or its precursors in less experienced smokers. That is, instruments developed and validated on adults displaying the 'mature' dependence syndrome, which arguably are limited in the assessment of adult dependence, may not be adequate for measuring the emergence of dependence. Indeed, we may find that assessments designed explicitly to capture the early stages of dependence will yield insights that substantially improve our understanding of dependence in its mature forms.

Theory and definition of dependence are intertwined

Many of the models described in these review papers are described explicitly as theories of dependence. On the other hand, some theoreticians assert that chronic drug use, motivated by the processes described in their model, may lead to dependence, but they offer no definition of dependence *per se*. Still other theories do not purport to be theories of dependence *per se*, but simply accounts of drug use, explaining why drug use may become frequent and seem uncontrolled. In that sense, dependence might be seen as a condition somehow distinct from the motivational mechanisms addressed by the theory. We believe that all the theories described in these papers are, in fact, statements about the fundamental nature of dependence. The theories each necessarily imply a definition of dependence and, conversely, definitions of dependence necessarily rely on theory. Even formal diagnostic systems, such as DSM or ICD, which have been portrayed as purely descriptive and atheoretical, are actually theories of dependence with assertions about central and accessory features, necessary and sufficient conditions for dependence diagnosis, expectations regarding prognosis and the categorical nature of the dependence construct.

The scientific and clinical value of a given theory of dependence rests on the extent to which it accurately describes behavior, provides compelling explanations for relationships between and among dependence-related behaviors, predicts future behaviors, suggests how

relevant behaviors might be modified and aids in the precise assessment of behaviors. The DSM and ICD diagnostic systems, because they were established to diagnose dependence in what has been considered its fully established form, simply do not provide a conceptual or assessment framework adequate for capturing the emergence of dependence. In contrast, the theories of dependence described in these reviews provide some guidance for the assessment of smoking behavior and its motivations during the early phases of tobacco use.

A transdisciplinary approach is needed to address dependence

At present, the limited research on assessing the emergence of dependence has been informed largely by traditional diagnostic approaches using self-report measures adapted from adult assessments. For reasons enumerated here and elsewhere (Colby *et al.* 2000a,b), this approach appears limited in concept and method. In contrast, the theories described by Eissenberg, Glautier and Brandon *et al.* represent a wide range of scientific disciplines and advance a multitude of methods for assessing the emergence of dependence. The large number of disciplines and methods with potential contributions to our understanding of the emergence of dependence suggests that the greatest advances will be made by adopting a transdisciplinary perspective. Presumably, a complete picture of the emergence of tobacco dependence will represent an integration of biological, behavioral, psychological, sociocultural and economic perspectives on the processes that moderate trajectories of dependence. Although we realize that no single research project or research program will meld easily all these perspectives into a coordinated whole, tobacco use is far too complex to expect that a research agenda guided by any single, isolated disciplinary perspective will capture the multi-faceted nature of the emergence of tobacco dependence (Clayton *et al.* 2000).

CONCLUSIONS

The authors of the review papers contained in this issue were charged with describing the implications of contemporary theories of drug dependence for developing assessments of the emergence of tobacco dependence. Each paper provides an excellent summary of the core features of major models and, on that basis alone, these papers will serve to more bring out the theoretical underpinnings of drug dependence research and treatment. In addition, the authors discussed the implications of these theories for understanding tobacco dependence in its emergent forms. Their exploration of how these theories might conceptualize the early stages of tobacco use illustrates further the heuristic range and, in some cases, limitations of the theories. Finally, the authors give specific suggestions of how theoretically derived concepts and methods might be used to inform the development of new assessments of the emergence of tobacco dependence. The view of dependence emerging from these reviews is substantially more dynamic, multi-dimensional and multi-determined than the conventional perspective on tobacco dependence. The ideas generated from these papers can provide the conceptual foundations for a new generation of research on the etiology of tobacco dependence, research that explicitly integrates modern theories into the assessment of tobacco dependence.

In the end, this series leads us to consider several important issues facing contemporary researchers in their quest to assess and understand nicotine dependence. If dependence is continuous and grows over time, it is critical that our measurements have the dynamic range and sensitivity to assess key variables over the history of a person's smoking. We need much better measures of consumption patterns in general and 'acquisition' patterns specifically in 'real' time in order to understand not only how dependence emerges, but why it does so in some and not in others. Furthermore, if dependence is multi-dimensional, we need a much better understanding of what those dimensions are and how they change over time and in response to consumption or other events, not only independently but also in interaction with one another. Lastly, if dependence is continuous and multi-dimensional and different across developmental stages of life and trajectories of tobacco use, then we need much better (more efficient and effective) ways of capturing the complexity of the behavior and its concomitant effects. We believe that development of more articulated conceptual models of dependence and, accordingly, of more sophisticated and refined methods of assessing dependence are essential to progress in understanding and combating nicotine dependence and tobacco use.

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