Substance Use & Misuse, 43:967–971
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ISSN: 1082-6084 (print); 1532-2491 (online)
DOI: 10.1080/10826080802097389

informa
healthcare

The "F" Words:
Facts, Fictions, Fantasies

Are "Physiological" and "Psychological" Addiction Really Different? Well, No! . . . um, er, Yes?

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The distinction often made between psychological and physiological addiction is a form of mind-body dualism. Therefore, it is a false distinction. However, this does not imply that behavioral and autonomic symptoms of addiction have the same brain substrates. In fact, they likely do not, although there is some overlap.

Keywords psychological withdrawal; physiological withdrawal; habit; drug dependence; symptomatology

The lay public—and some professionals—draws a fundamental distinction between "physiological addiction" and "psychological addiction." In this context, "physiological addiction" apparently refers to addictive behavior that exhibits bodily withdrawal symptoms upon cessation (or administration of an antagonist). These potentially dramatic symptoms include tearing, tremor, piloerection, seizures, nausea, and so forth. In contrast, "psychological addiction" is usually taken to mean compulsive drug use associated with craving and continued use despite obvious adverse consequences, as well as affective discomfort upon cessation. However, it does not exhibit classic "physiological" withdrawal symptoms when a drug's use is terminated or interrupted.

It is easy to dismiss this distinction as mind–body dualism (Arshavsky, 2006). In fact, it probably is. Psychological processes arise from the brain, as do most (but perhaps not all) bodily symptoms of drug use withdrawal. This implies they are manifestations of the same brain functions; they are simply measured in different ways. Psychological addiction is observed or measured by (subjective) self-report or behavioral ratings, whereas physiological addiction can be measured with autonomic recordings or other objective indices.

The difference between subjective ("vague") and objective ("precise") measures is very real to lay people, as is the distinction between psychological (potentially "illusory") and physiological ("real") functions. Recourse to the *Diagnostic and Statistical Manual of Mental Disorders,* Fourth Edition—Text Revision (*DSM-IV-TR*; American Psychiatric

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Association [APA], 1990) is instructive here. The *DSM-IV*'s laundry list of criteria for substance dependence appears to both canonize the psychological versus physiological distinction at the same time that all of the formal criteria for "physiological dependence" are measured typically by self-report! Within the *DSM-IV* substance dependence diagnosis, "Specifiers" make the explicit distinction between dependence *with* versus *without* "Physiological Dependence" (i.e., either chronic tolerance or the withdrawal syndrome characteristic of that particular "drug of abuse"[1]). Even the symptoms of so-called physiological dependence in the *DSM-IV* are worded such that they can be and usually are measured by self-report from the patient rather than by direct observation or autonomic electrophysiology.

It is interesting that "drug craving" (or a synonymous description) does not appear among the *DSM-IV* criteria for either substance abuse or dependence. However, it should be noted that drug "craving" *is* listed as a criterion in the *International Classification of Diseases–Version 10* (WHO, 1993). Given the large body of data about "craving" for drugs, it is likely to emerge as a formal diagnostic criterion for substance abuse and/or dependence in subsequent versions of the *DSM*.

The distinction between psychological and physiological symptoms of substance abuse/dependence remains an important issue for many in the lay public, for individuals who design psychiatric classification of disorders, and for the status of "craving" as a diagnostic criterion. In particular, this issue is likely to dominate changes in substance abuse/dependence criteria from *DSM-IV* to future versions. For example, Langenbucher et al. (2000) developed a "Withdrawal-Gate Model" of alcohol abuse/dependence in which alcohol withdrawal symptoms were proposed as both necessary and sufficient to diagnose alcohol dependence. In this model, symptoms of "'psychological dependence' (e.g., pathological patterns of use)" (p. 807, inner quotation marks are those of the original authors) define alcohol abuse rather than dependence when present without "physiological withdrawal."

The last decade has witnessed tremendous advances in neuroimaging the human brain (and that of other animals). The brain was something of a "black box" in terms of drug addiction at the time that the problematic distinction between physiological and psychological addiction arose in the scientific literature and among the lay public—and even during the development of the *DSV-IV*. This is no longer the case. Although the state of understanding can be overestimated easily, real inroads have been made in mapping the neural substrates of constructs such as "craving" and "withdrawal states." For example, the original neuroimaging study of drug craving (Grant et al., 1996) was followed by much subsequent research that has branched out to craving for many different posited "drugs of abuse." There is now little doubt that drug craving has important regional substrates in the brain, particularly in the prefrontal cortex (Wilson et al., 2004) and mesolimbic areas. Having said that, precise localization of the affected brain regions—and more importantly—how they interact with each other, remain unclear.

Another example is Volkow et al.'s (2007) recent study using positron emission tomography and [^{11}C]raclopride binding among detoxified alcoholic patients. These individuals showed profoundly decreased dopamine D_2/D_3 receptor activation in response to

[1]The journal's style utilizes the category *substance abuse* as a diagnostic category. Substances are used or misused; living organisms are and can be *abused*. In addition the often used nosology "drugs of abuse" is both unscientific and misleading in that it mystifies and empowers selected active chemicals into a category whose underpinnings are neither theoretically anchored nor evidence informed and which is based on "principles of faith" held and transmitted by a range of stakeholders representing a myriad of agendas and goals. Editor's note.

methylphenidate (Ritalin) in the ventral striatum, a deep brain structure that has been implicated strongly in "reward" processes and addiction (Di Chiara and Imperato, 1988).

It is not surprising to biological reductionists that processes normally measured using self-report and objective psychological measures have brain substrates. This is the essence of rejecting mind–body dualism. It was noted above that when going beyond the notion of psychological versus physiological addiction, one might conclude these are identical manifestations of the same underlying brain process—but simply measured differently. This assumption is likely to be invalid.

As one possible example, our own recent theorizing (Newlin and Regalia, in preparation; Newlin and Strubler, 2007) about drug addiction belies this notion. First, we introduced the construct of a Habit Capture System that entrains and entraps behavior that is likely to be adaptive into behavioral habits that can become intractable. The pharmacological actions of "drugs of abuse" have the unfortunate property of being experienced by their users of acting as if they were adaptive to the user when they are not (Nesse and Berridge, 1997; Newlin, 2002, 2007). Nesse and Berridge argued that these drugs "create a signal in the brain that indicates, falsely, the arrival of a huge [Darwinian] fitness benefit . . . that displaces adaptive behaviors (p. 64)." This brain signal may be experienced as a sense of personal empowerment or social agency (related to survival fitness) or to enhanced personal and sexual attractiveness (related to reproductive fitness; Newlin, 2002, 2007). In addition to the Habit Capture System, which is hypothesized to control drug-seeking behavior, we hypothesize a Habit Maintenance System that efficiently maintains these habits such that they have considerable behavioral inertia. We (Newlin and Regalia, in preparation; Newlin and Strubler, 2007) view these two systems as fundamentally psychobiological. That is, each system has subjective, behavioral, and physiological aspects that are somewhat different from the other system.

We use the metaphor of "passing the baton" from drug-seeking and experimental/intermittent drug use that is controlled by prefrontal and ventral striatal brain mechanisms—Habit Capture—to the Habit Maintenance System. The latter represents activity of the dorsolateral striatum. The Habit Maintenance System supports and promotes chronic, compulsive drug use by responding automatically to internal (e.g., falling brain drug levels) and external cues (e.g., drug-associated stimuli or triggers). These ideas build on those of Everitt and Robbins (2005), Yin and Knowlton (2006), and Tiffany (1990). We (Newlin and Regalia) used control theory (linear dynamical systems) to model several existing homeostatic (such as opponent process theory and respondent drug conditioning) and nonhomeostatic (such as instrumental learning and incentive sensitization) theories of addiction. We then presented a control theory implementation and mathematical simulations of our Habit Capture/Maintenance model. One value of control theory is that it effectively integrates into one psychobiological model constructs that are quite different. So, for example, control theory allows one to build a model that includes affect, neurotransmitter levels, conditioning (learning), and internal cognitive maps in the same system; the mathematical relations among control elements are more important than the measurement domain or the physical manifestation of the constructs.

Our baton-passing metaphor emphasizes that different brain systems may correspond to successive phases of the addiction cycle and to different psychological processes. Therefore, there is a sense in which the psychological *DSM-IV* symptoms of drug dependence relate, at least in part, to different brain substrates than do the physiological symptoms of tolerance and withdrawal. This is hardly an unqualified "Yes" to the question of whether there really is a difference between psychological and physiological addiction. However, it does demonstrate that modern neuroscience has advanced far beyond the "black box" stage.

Although virtually all drug dependence symptoms are both psychological and physiological, they may map somewhat differently within the brain.

This discussion leads to the following conclusions:

1. The way the central question is phrased [Are psychological and physiological addiction different?] succumbs to the fallacy of mind–body dualism.
2. This does *not* imply that the compulsive drug-use-related symptoms described in the *DSM-IV* and those concerning chronic tolerance and the characteristic withdrawal syndrome arise from the same neural systems within the brain. They likely do not.
3. A truly psychoneurobiological approach to defining, diagnosing, and treating addictive behavior is needed that does justice to *both* realms of study, integrating them to the fullest degree possible. This may be a distant and lofty goal, but addiction is a difficult, important problem that may require this integrative approach.

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Glossary

Physiological addiction: Theories whose constructs and mechanisms are physiological, usually without direct reference to subjective or experiential aspects.

Psychological addiction: Theories whose constructs and mechanisms are cognitive, affective, or behavioral, and which usually do not specify possible biological substrates.

Homeostatic: A system that "seeks" or tracks a neutral state; it returns to equilibrium after being perturbed, usually through compensatory mechanisms

Nonhomeostatic: A system that "seeks" a non-neutral state; therefore, it is driven to new levels when perturbed and does not automatically return to homeostasis.

Craving: A term used to describe the strong desire for something, in this case drug self-administration; often called drug "urges" or "drug-seeking"

Mind–body dualism: The belief that there are many properties of the mind that are not reducible to biochemical or bioelectric mechanisms of the brain; the alternative is biological reductionism, or the belief that all aspects of the mind can and will be described eventually in terms of functions of the brain.

Specifiers: In the *DSM-IV,* these are characteristics of diagnosis that are particularly important to classification, but are not part of the Axis I or II classification of the disorder.

Control theory: A branch of mathematical engineering in which systems are described conceptually, diagrammatically, and mathematically that control mechanical devices, electronic circuits, or biological or chemical processes.

Opponent process theory: A theory of opposing psychobiological functions that acting together to determine the response to intense affective stimuli, such as psychotropic drugs.

Respondent drug conditioning: Pavlovian learning in which cues that predict an affective stimulus produce reflexive anticipatory responses such as salivation.

Incentive sensitization: systematic alterations in the incentive properties of stimuli such that the organism shows larger responses to them after repeated presentations.

References

American Psychiatric Association. (1994). *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision*. Washington, DC: American Psychiatric Association.

Arshavsky, Y. I. (2006). "Scientific roots" of dualism in neuroscience. *Progress in Neurobiology* 79:190–204.

Di Chiara, G., Imperato, A. (1988). Drugs abused by humans preferentially increate synaptic dopamine concentrations in the mesolimbic system of freely moving rats. *Proceedings of the National Academy of Sciences of the United States of America* 85:5274–5278.

Everitt, B. J., Robbins, T. W. (2005). Neural systems of reinforcement for drug addiction: from actions to habits to compulsion. *Nature Neuroscience* 8:1481–1489.

Grant, S., London, E. D., Newlin, D. B., Villemagne, V. L., Liu, X., Contorreggi, C., et al. (1996). Activation of memory circuits during cue-elicited cocaine craving. *Proceedings of the National Academy of Sciences* 93:12040–12045.

Langenbucher, J., Martin, C. S., Labouvie, E., Sanjuan, P. M., Bavly, L., Pollock, N. K. (2000). Toward the DSM-V: The Withdrawal-Gate Model versus the DSM-IV in the diagnosis of alcohol abuse and dependence. *Journal of Consulting and Clinical Psychology* 68:799–809.

Nesse, R. M., Berridge, K. C. (1997) Psychoactive drug use in evolutionary perspective. *Science* 278(5335): 63–66.

Newlin, D. B. (2002). The self-perceived survival ability and reproductive fitness (SPFit) theory of substance use disorders. *Addiction* 97:427–446.

Newlin, D. B. (2007). Self-perceived survival and reproductive fitness (SPFit) theory: Substance use disorders, evolutionary game theory, and the brain. In S. Platek, J. P. Keenan, & T. Shackelford (Eds.), *Evolutionary cognitive neuroscience* (pp. 285–326). Cambridge, MA: MIT Press.

Newlin, D. B., Regalia, P. (in preparation). Control theory and addiction: Systems, synthesis, and simulations.

Newlin, D. B., Strubler, K. A. (2007). The habitual brain: An "adapted habit" theory of substance use disorders. *Substance Use and Misuse* 42:503–526.

Tiffany, S. T. (1990). A cognitive model of drug urges and drug-use behavior: Role of automatic and nonautomatic processes.*Psychological Review* 97(2):147–168.

Volkow, N. D., Wang, G. J., Telang, F., Fowler, J. S., Logan, J., Jayne, M., et al. (2007). Profound decreases in dopamine release in striatum in detoxified alcoholics: Possible orbitofrontal involvement. *Journal of Neuroscience* 27:12700–12706.

Wilson, S. J., Sayette, M. A., Fiez, J. A. (2004). Prefrontal responses to drug cues: a neurocognitive analysis. *Nature Neuroscience* 7:211–214.

World Health Organization. (1993). *The ICD-10 Classification of Mental and Behavioural Disorders: Diagnostic Criteria for Research* (pp. 36–40). Geneva, Switzerland: World Health Organization.

Yin, H. H., Knowlton, B. J. (2006). The role of the basal ganglia in habit formation. *Nature Reviews Neuroscience* 7 (6):464–476.

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