Constraint theory: A cognitive, motivational theory of dependence

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Aims: A new theory of substance dependence is presented that models dependence as the absence of cognitive constraints on substance use.

Methods: (1) Critical review of the predominant paradigm that assumes that substance dependence is a pathological state fundamentally caused by the neuropsychopharmacological effects of drugs (NPP paradigm) identified four counter-factual assumptions. Contrary to the NPP paradigm: (I) dependence can occur on atypical substances and other things; (II) dependence is a complex, gradated phenomenon, not a state; (III) heavy protracted substance use can occur without dependence; and (IV) NPP interventions against dependence have not worked other than as drug substitutes. (2) Reconceptualisation of dependence as substance use with few cognitive, behavioural or social constraints. (3) Development of an exhaustive list of constraints on substance use with a panel of experts, achieving theoretical saturation. (4) Modelling of dependence, specifically to explain why socioeconomic deprivation is correlated with substance dependence.

Results: Fifteen common constraints are described, which prevent most substance users becoming dependent. People in more socioeconomically deprived conditions tend to have fewer constraints. Similarities between Constraint Theory and previous sociological and social cognitive theories are discussed.

Conclusions: Constraint theory describes the known nature of substance dependence better than theories from the NPP paradigm. Conceptualising dependence as an absence of constraints shows promise as a theory of addiction and fits with existing knowledge about what works to prevent and treat substance dependence.

Keywords: Addiction, dependence, teliological explanation, cognition and addiction

INTRODUCTION

Many theories of addiction or dependence assume it to be a pathological state that is progressively caused by various factors, primarily the pharmacological properties of drugs and alcohol (Booth et al., 2010; Robbins, Ersche, & Everitt, 2008), and secondarily genetics (Enoch, Gorodetsky, Hodgkinson, Roy, & Goldman, 2011; Wei et al., 2011), psychological processes, both normal (Leventhal & Schmitz, 2006) and pathological (Mills, Teesson, Darke, & Ross, 2007; Schilling, Aseltine, & Gore, 2007) and dysfunctional socioeconomic conditions (Foster, 2000; Seddon, 2000). Addiction research strives to understand how this pathological state of dependence comes about, and how it might be prevented and treated. This article will develop an alternative theory that dependence is caused by a lack of constraints, not a combination of active causal factors.

The dominant conceptual paradigm for understanding dependence has been encapsulated as “Addiction is defined as a chronic, relapsing brain disease” (National Institute on Drug Abuse, 2010). This key document takes the strong reductionist position that drugs change brain function in ways that suffice to explain the adverse effects of drugs on behaviour, without mentioning any of the psychological or social factors long considered important (Zinberg, 1984).
We shall refer to this paradigm as the NPP paradigm, which assumes that (1) addictive drugs (here ‘drugs’ include alcohol unless otherwise stated) alter neuropsychological motivation systems and (2) these alterations (including as yet undiscovered ones) are the sole and sufficient cause of disorders of substance dependence; social and psychological factors can be reduced to NPP. However, (3) these alterations may only occur in some people, in some genetic, developmental, psychological or social conditions. A problem with this third assumption is that it allows an indefinite number of supplementary explanations of dependence, which can arbitrarily cover any eventuality. Finally, (4) the paradigm assumes that the neuropsychological alterations that constitute addiction need to be treated by abstinence, to prevent continued harmful interaction between brain and drug. This assumption is theoretically empty because abstinence is actually an outcome, not an intervention (imagine ‘stay alive’ being heralded as a cure for cancer; but how?). Alternatively, addiction might be treated by reversing or blocking neurological alterations with pharmacology or other neuroscience interventions. Moreover, the purpose of psychological and social interventions against drugs is to facilitate abstinence, for the NPP effects of drugs cannot readily be managed or controlled by the person, at least if susceptible according to assumption 3.

While many addiction researchers, including biological scientists, would question some of these assumptions, the NPP paradigm is culturally sustained and sometimes its assumptions are either assumed implicitly, or challenged less robustly than they could be (Hammersley & Reid, 2002). As will be seen: assumptions 2 and 4 of NPP are not supported by research evidence, while assumption 3 becomes the crux of the entire problem of dependence, as formulated by Constraint Theory.

Constraint Theory of motivation and drug use accepts that drugs affect the brain; often in more durable and subtle ways than addiction research previously envisaged (NPP assumption 1). Drugs have effects that are reinforcing, or that people and animals can learn are reinforcing. There is developing evidence that separate, interacting brain systems underlie (a) mood effects of drugs (b) engaging in previously reinforced behaviours and (c) the ‘incentive salience’ of the reinforcer; i.e. the person or animal’s tendency to attend to it, and think about it (Beckmann, Marusich, Gipson, & Bardo, 2011; Berridge & Robinson, 1998; Wyvell & Berridge, 2000). In humans, the mood system most probably involves the entire cognitive apparatus, rather than mood being formed primarily by physiological changes (Hammersley & Reid, 2009). People indeed take drugs because they can improve mood, learn that drugs are salient to them, and they will exert effort to obtain and consume drugs. As dependence increases these tendencies become stronger and there are more marked brain alterations. These changes describe dependence at the biological level, but they are not its causes.

For, the NPP effects of drugs can be overridden by cognitive effects, and how this occurs to form dependence is described by Constraint Theory. Dependence occurs when a drug is used with few constraints on its use. The neuropsychological changes themselves are neither necessary, nor sufficient causes of dependence: Not necessary because people can exhibit signs of dependence to substances and behaviours without there being specific neuropsychological changes involved; Not sufficient because people can consume enough of a drug to cause the changes without exhibiting signs of dependence. Both these claims are controversial and will be elaborated below.

CONSTRAINT THEORY: DEPENDENCE AS THE ABSENCE OF CONSTRAINTS

Dependence, or problematic substance use, or ‘substance use disorder’ (DSM5.org, 2010) (hereafter ‘dependence’) occurs in the absence of specific constraints on substance use.
As will be seen, these constraints turn out to be well known. NPP theories have problems accounting for four classes of phenomena that constraint theory can explain:

(1) Dependence can occur for substances and activities with weak or entirely generic neuropsychological effects, where NPP theories require ad hoc supplementary explanations. (a) On drugs, such as MDMA (Bruno et al., 2009), which do not seem to cause dependence easily in most users (although they clearly affect neuropsychology). (b) On activities and substances that have no psychopharmacological effects (they may have general neuropsychological effects, such as arousal), including pathological gambling (Raylu & Oei, 2002), problematic internet use (Kittinger, Correia, & Irons, 2012), exercise addiction (Berczik et al., 2012), although this may have dependence relevant neuropsychological effects, and carrots (Kaplan, 1996). (c) On substances that are widely used in benign ways, despite being potentially dangerous and addictive, such as over-the-counter analgesics (Abbott & Fraser, 1998) and prescription opiates prescribed for serious acute pain (Nicholson, 2003).

(2) Constraint theory allows dependence to occur in a gradated manner, rather than as a discrete state that is distinct from normality. Fewer constraints tend to mean more signs of dependence. Moreover, a marked change in one or more constraints on dependence can have dramatic effects on recovery. For example, many people cease to be dependent by simply stopping use at some time (Ditton & Hammersley, 1996; Granfield & Cloud, 1999; Mullen & Hammersley, 2006). This is inexplicable if dependence comprises a major and hard to reverse alteration in the person’s NPP. Often people quit for what appear to be relatively minor reasons, compared to their previous life difficulties that did not lead to protracted cessation (Mullen & Hammersley, 2006; Orford et al., 2002).

(3) It ceases to be necessary to explain why some people can use addictive drugs heavily and protractedly without exhibiting clinically significant signs of dependence. There are data in this regard for heroin (Shewan & Dalgarno, 2005; Warburton, Turnbull, & Hough, 2005), cocaine (Ditton & Hammersley, 1996; Transform, 2009) and alcohol (Orford et al., 2002; Robertson et al., 1987), particularly when ‘unobtrusive’ users are studied who have neither been arrested nor sought treatment. Clinical samples of substance users tend to be biased towards people who have been the least capable of managing their substance use. The causes of drug use are in part as NPP proposes, but the causes of dependence are NPP causes, plus lack of constraints.

(4) Generally, NPP interventions have not worked for drug dependence. Substitute prescribing can be effective in reducing illicit drug use and various detoxification regimes can be helpful, but these are augmented by psychological therapy (Seivewright, 2000) and a detoxified patient is not necessarily free of their drug dependence (Day & Strang, 2011). Other more ingenious interventions, including vaccinations, most recently against cocaine (Wee et al., 2012), electrical stimulation, most recently deep brain stimulation (Chen & Liu, 2012), and blocking opiate receptors with naloxone (Schechter, 1980), now abandoned, have not worked as yet. The basic problem is that people can override such interventions by increasing dose. As with disulfiram (Allen & Litten, 1992) interventions can be overridden if the patient is willing to persist with use regardless. Two neurologically active medications, naloxone and acamprosate, do have modest benefits in treatment for alcohol dependence (Dransitsaris, Selby, & Negrete, 2009; Kranzler & Van Kirk, 2001; Roesner, Leucht, Lehert, & Soyka, 2008), although their mechanisms of action remain uncertain. In understanding how NPP interventions might work, the issue of motivation appears to be important.
WHY ARE ALL USERS NOT DEPENDENT?
A longstanding theme in addiction research and treatment has been to assert the inevitability of addiction given the right biopsychosocial conditions (Hammersley & Reid, 2002), despite evidence that there is no inevitability to, and indeed considerable difficulty predicting, the life course and outcome of substance dependence (Ogborne & Stimson, 1975; Orford et al., 2002; Valliant, 1995). Not only is there considerable variability, but also a reasonably high recovery rate compared to other common mental health problems. Additionally, research is skewed towards the study of drugs and dependence, despite most users not being dependent. To illustrate, Web of Knowledge (webofknowledge.com accessed 10/1/2013) locates approximately 45 000 references with the keyword ‘heroin’. Of these, only 30% (13 000) also do not contain addic* or depend* as keywords. The drugs researched in-depth clearly have pharmacological effects on brain systems involved in motivation. The most definite data comes from animal research with opiates and cocaine (Robbins et al., 2008). However, even for those drugs the specific contingencies of learning and the environment are critical in determining outcome (Alexander, Beyerstein, Hadaway, & Coambs, 1981; Chauvet, Lardeux, Goldberg, Jaber, & Solinas, 2009; Fritz et al., 2011; Gipson, Beckmann, El-Maraghi, Marusich, & Bardo, 2011; Griffin, Lopez, & Becker, 2009; Quadros & Miczek, 2009; Quick, Pyszczynski, Colston, & Shahan, 2011; Solinas, Thiriet, El Rawas, Lardeux, & Jaber, 2009). So, even in animals set and setting matter in determining the behavioural outcomes of substance use, meaning that drugs are not sufficient causes of dependence. This means that it is unlikely to be possible to redefine the behavioural syndrome of dependence into a small number of biological disease entities.

Similarly, human research finds that chronic cocaine and heroin users can follow pathways other than a trajectory of increasing use and symptoms of dependence (Hammersley, 2011). Broadly – as will be theorised here – most human substance use involves cognitive constraints that make dependence less likely.

INDIVIDUAL DIFFERENCES: DEFICIENCIES AND RISK FACTORS
As not everyone who uses a drug becomes dependent, the NPP paradigm invokes supplementary explanations (Kuhn, 1962), which generally postulate that certain biological, psychological or social characteristics predispose a person to dependence, given access to the drug. Biological characteristics include having various sorts of genetic susceptibility. Genetic variations clearly do affect how people respond to alcohol (Dick & Foroud, 2003), although none of them is a sole and sufficient cause of dependence. There may also be genetic variations in the response to cannabis (Decoster et al., 2011), and it seems likely that the same will be found to apply to heroin (Nielsen et al., 2010) and cocaine (Anon, 2011). Putative psychological ones include personality characteristics and disorders, deficiencies in child-rearing, attachment and socialisation, learning and educational difficulties and problems that result in self-medication or escape coping, whether or not this is beneficial. Social characteristics include lacking social skills, associating with people with drug problems, living in an environment where drugs are widespread and tolerated, and being poor or socially excluded (see, Hammersley, 2008, Chapter 5 for further discussion). Such supplementary explanations postulate that certain forms of deficiency are required for
dependence to develop. The implicit assumption is that people who are ‘sufficient’ will not become dependent. Most commonly, ‘deficiencies’ are called ‘risk factors’.

Constraint theory makes this assumption explicit, and central: People who have sufficient constraints on their substance use will not become dependent. However, a lack of constraints does not imply defectiveness. There are social and psychological conditions where many normal people with access to a drug will become dependent – being a USA soldier in Vietnam remains the canonical example for opiates (Robins & Slobodyan, 2003). Moreover, some of the concepts and attributions that define ‘dependence’ are socially constructed (Davies, 1997; Peele, 1990; Szasz, 1974). For example, the standard diagnostic criteria for dependence from the American Psychiatric Association (1994) and the World Health Organisation (see http://www.who.int/classifications/icd/en/, accessed 8/1/2013) require the clinician to make qualitative judgements about the significance or magnitude of problems, and to attribute those problems to substance use, having excluded other causes.

Explanation in terms of deficiencies or risk factors is problematic, because no single risk factor, or small cluster of risk factors, suffices to predict who will become dependent, or which “high risk” people will not. A related difficulty is that the risk factors approach tends to confuse two different meanings of ‘prediction’; prediction in the sense of forecasting and prediction in the sense of a causal understanding (Morton, 2004). Risk factor models can modestly forecast which people in a population are more likely to become dependent. Typically, such models explain less than a third of the variance in future substance use and often considerably less. Forecasts are not powerful enough to predict, or change, individual behaviour. A major problem is that the best predictor of future substance use is usually current and past substance use, which are confounded with other ‘risk factors’ (Elliott, Huizinga, & Ageton, 1985). So, including drug use as a predictor leaves only a residue of variance for other variables to predict, some of which may only be weakly correlated with use. Excluding drug use as a predictor makes other variables seem to predict drug use at follow up, when they may not. A causal model of how and when effects should occur is required (Cohen, Cohen, West, & Aiken, 2002), but only rarely provided.

Deprivation and dependence
Generally, using social risk factors is particularly problematic because it is unclear how a social factor can cause a supposedly biological condition like dependence, and because social concepts are particularly prone to slippage of meaning. Additionally, superficially independent socially constructed labels can interact. For example, although the definition of a ‘truant’ and a ‘substance user’ are superficially independent, a child found not at school and taking drugs may well be labelled a truant, while one sitting at home unwell in front of the TV, whose alcoholic parent has forgotten to phone the school, may get the benefit of the doubt. When all the definitions are based on self-report then the confounding of different concepts is even more problematic. People who are already stigmatized by drug dependence may be less reluctant to report other bad things than those whose wrongdoings are more concealed.

In contrast, psychological factors may perhaps be reduced to biology, in theory at least. Yet, there is a strong association between socioeconomic status and dependence. Poverty, lack of education, social exclusion and being of minority ethnicity can all greatly increase the prevalence of drug and alcohol dependence. However, drug use is not most common
amongst people in more deprived areas: In England and Wales by postcode area, younger, less settled, people tend to use more than older, more settled ones, while the least and most affluent people tend to use slightly more than those of middle income (Home Office, 2011). Moreover, it is people in rising urban areas, not highly deprived areas, who are most likely to use illegal drugs. Indeed the only common drug where prevalence of use, rather than problems, may be correlated with deprivation is heroin (Neale, 2000) and these data may be biased by underreporting of use of more stigmatised drugs (Patton, 2003). Similarly, alcohol problems are correlated with deprivation, but alcohol use is not (Pollack, Cubbin, Ahn, & Winkleby, 2005).

NPP cannot easily explain the links between deprivation and dependence, particularly as, even in extremely deprived areas, most people do not become dependent, when, if it were biologically determined, they should, given their levels of stress, social exclusion and alienation, lack of alternatives and exposure to substances. Constraint theory reverses the question to ask why many people do not become drug-dependent under conditions when they should be at high risk of doing so?

Next, the paper will explore what can be theorised without assuming the addictive properties of drugs. It will draw upon social attribution theory (Davies, 1997; Reinarman, 2005) and social constructionism (Plumridge & Chetwynd, 1999; Szasz, 1974), but go beyond the unappealing idea that dependence is largely illusory to suggest that it is formed and sustained by cognitive factors, particularly cognitions about interpersonal and social issues, as are many other extreme and distressing mental health problems with biological components including depression (Beck, 2008), psychosis (Tarrier, 2010), obsessive-compulsive disorder (Abramowitz, 1998) and eating disorders (Fairburn, 2008).

A COGNITIVE, TELEOLOGICAL ALTERNATIVE
The importance of cognitive factors has been theorised before (Miller & Rollnick, 2002; Orford et al., 2002; West, 2006), what is novel here is a preliminary exploration of the cognitions that generally prevent dependence, rather than of those that cause it. As will be seen, many of these cognitions are teleological; based on personal deliberate purpose oriented towards the future. People commonly think and do specific things with the purpose and intent of ensuring that they do not use drugs too much or become dependent.

This type of teleological behaviour management is neither remarkable, nor unique to dependence. It is also often inexact and fallible. For example, people budget to ensure that they do not spend too much money, but in the UK mean household debit is about 1.5 times post-tax income (The Economist, 2011). Much of the literatures on topics including debit, work stress and obesity emphasise the importance of external and situational influences on these problems, as well as the importance of people making poor risk decisions based on incomplete information that tends to neglect longer term considerations and often fails to consider properly all the information available at the time of choice (Slovic, 2000). Drug dependence is no different.

Constraints on drug use versus constraints on dependence
From Becker’s (1953) seminal work onwards, sociological and anthropological research has emphasised the role of constraints in governing substance use. Research has tended to conceptualise substance use as deviance, emphasising social and cultural constraints and
norms on substance use, which are socially constructed (Golub, Johnson, & Dunlap, 2005). This tradition of research tended to question, more-or-less strongly, whether dependence or addiction has any biological reality, leading to a large disjuncture between this tradition and health-related research on dependence, and to only a weak distinction between drug use and drug dependence (Reinarman, 2005). There is research on the social processes and conditions of becoming and being a heroin user (Agar, 1973; Johnson et al., 1985; Levy & Anderson, 2005; Weppner, 1981), a cocaine/crack user or dealer (Bourgois, 1995; Reinarman, Murphy, & Waldorf, 1994) and an ‘alcoholic’ or heavy drinker (Alasuutari, 1992; MacAndrew & Edgerton, 1969).

A key theme in this type of work is of the deviant subculture that supports and sustains specific forms of drug use that are regarded as highly problematic by mainstream culture. As substance use has diversified and become more commonplace, even normalised (Aldridge, Measham, & Williams, 2011), the idea that dependence is sustained by a deviant subculture looks weaker because firstly many people use drugs – even purportedly highly addictive ones – but are at most part-time deviants who do not gain their primary identity from drug use. Secondly, polydrug use has become the norm with people not necessarily being affiliated highly with some drugs rather than others. Thirdly, there is increasing recognition of the fact that mainstream cultural values actually promote and support inebriation and intoxication (Hammersley & Dalgarno, 2012), and scientifically no justification for distinguishing alcohol dependence from heroin dependence in terms of how problematic it is (Nutt, King, Phillips, & Independent Sci Comm Drugs, 2010). Fourthly, modern understandings of risk (Beck, 1992; Slovic, 2000) suggest that fear and concern about certain drugs are primarily about fear of the new, which tends to be perceived as alien and uncontrollable, hence dangerous.

While like-minded heavy substance users tend to congregate together, this may be caused by dependence rather than being its cause. They congregate together to pool resources regarding the obtaining and consuming of drugs or alcohol, because their heavy use may be repugnant to others in many ways, including simply being boring, and because they share common interests and values, including less concern about ‘danger’ than others. This lack of concern may be devil-may-care deviance, sensation-seeking, or a lack of the constraints that others have.

It remains true that drug users learn how to use drugs in such ‘deviant’ groups, even if it is a part-time deviance. But people neither arrive at such a group, nor linger, at random. A drug naïve person entering a room of people using a particular drug at a party can leave, sit down and partake but not repeat the experience often, or get involved with enthusiasm. Moreover, nowadays, drug choices are seen more as matters of individual preference that have fewer consequences for friendship and socialisation than they may have had in the past (Aldridge et al., 2011). Additionally, one common pattern of drug use is to restrict extreme use to specific social events and occasions, for instance going clubbing (Hammersley, Khan, & Ditton, 2002), or a skiing holiday (Hammersley, Ditton, Smith, & Short, 1999).

Consequently, Constraint Theory does not assume that constraints arise from a deviant subculture. Nor does it assume that dependence is a matter of social definition, so that very similar behaviours involving a different substance, or different people, would be judged and
labelled differently. Rather, it assumes that dependence is what happens to ordinary drug users, when they lack cognitive constraints on use.

Cognitive constraints
Constraints exist at the cognitive, or psychological level, as some form of representation or model that is used to make behaviour choices. The cognitive architecture of this model is a matter for empirical investigation. It is plausible that this might be similar to that of the Extended Theory of Planned Behaviour (McMillan & Conner, 2003; Peters, Kok, & Abraham, 2008) where various cognitions lead to the formation of intentions to act, which in turn lead to actual constraint behaviour. As with other health-related behaviours, constraining substance use is something that people intend more often than they actually do. However, as will be seen, Constraint Theory includes elements that cannot be easily mapped on to TPB.

WHAT MOTIVES DO PEOPLE HAVE FOR NOT BECOMING DEPENDENT?
There is much more research on dependent people seeking to explain their dependence than there is on non-dependent substance users. There is however enough research to be able to give an informed list of the reasons why people who are not dependent stop using or moderate use of specific substances, although the evidence is better for some of these reasons than for others and reasons for not using has almost never been the direct topic of research. Much of the evidence comes indirectly from social science research on drugs. Consequently, the reasons are most available in the knowledge of researchers, so this list was designed by writing a preliminary list of all the types of reason that people could have for stopping or moderating substance use, then in 2009 circulating this to eight colleagues in the UK who research non-dependent substance use and amending the list according to their suggestions, by collapsing reasons that were judged to be essentially the same, and including additional reasons. Finally, the list was circulated to 10 practitioners in the UK who treat and research dependent substance use for further feedback, of whom five provided feedback. This stage of the process showed that dependent people’s motives for stopping or moderating use overlap with nondependent motives, but are not identical in magnitude. The list given here is theoretically saturated (Glaser & Strauss, 1970), in that by the end of the process researchers’ and practitioners’ additions and amendments had ceased to contribute new information. Thus, it is a classification of all the common reasons for stopping or moderating use known to the expert participants. Some of the reasons are well-evidenced in the academic literature, while some are well-known but lack formal evidence. Each constraint is mapped on to Zinberg’s (1984) well-known drug, mind-set, setting typology, although the reasons elicited here do not map fully on to the typology. Where possible, the constraints are also mapped on to the main elements of the extended Theory of Planned Behaviour. Some do not map because they are about drug effects, or about the context or setting of use, which are beyond the scope of TPB.

People stop or moderate their substance use because:
(1) They have explicit and active religious or other moral beliefs that prohibit use (set; attitude/ anticipated regret). There is a well-known relationship between religion and temperate habits (Edlund et al., 2010; Good & Willoughby, 2011), although of course religious adherence is fallible and temperance can lapse with it.
(2) They become jaded of consumerism/materialism (set; attitude/anticipated regret). For drugs, this can include becoming jaded about having to hang about drug users and dealers in order to get drugs (Levy & Anderson, 2005; Mullen & Hammersley, 2006). It can also involve rejecting the values marketed by the alcohol industry or any other relevant industry.

(3) People important to them are strongly opposed to use and that opposition matters to them (set/setting; subjective norm). Or, those people (family, friends, colleagues) consider their use to be escalating to unacceptable levels. The impact of systemic therapies on substance use, which amongst other things get users to recognise the concerns of their families, is one example (Orford et al., 2002).

(4) Opportunities for taking the substance are reduced by life circumstances (setting; perceived behavioural control [PBC]). For instance, closer parental supervision reduces opportunities for adolescent substance use, although substance use makes supervision harder (Clark, Kirisci, Mezzich, & Chung, 2008).

(5) They have other things to do that conflict with use of that particular substance (setting; PBC). ‘Maturation’ often involves responsibilities that make use more difficult. For example, child care whilst intoxicated or hungover can be problematic (Taylor, 2003).

(6) Sympathetic friends to use with are not available (setting; subjective norm/PBC). For example the Vietnam veterans who had used heroin there most likely to inject heroin afterwards were those whose home networks facilitated it. Other users simply quit (Robins & Slobodyan, 2003).

(7) The substance does nothing for them (drug), which applies, not only, to a surprising number of politicians who tried marijuana. Other drug effects are not always blatant, or require learning, or the right setting to appreciate, and can feel ineffective to some (Orford et al., 2002; Shewan & Dalgarno, 2005).

(8) They dislike the effects (drug; anticipated regret), but there is no research on people who try a drug and never use it again.

(9) They lack the stresses and strains that lead to a desire for hedonistic, present oriented escapism (set/setting). There is a well-known link between stress and increased substance use (Jacobsen, Southwick, & Kosten, 2001; Pohorecky, 1981).

(10) They like the effects too much, compared to other things. (Drug; PBC) That is, they recognise a risk of immoderate use for them and therefore avoid the drug. This is sometimes given as a reason for not using heroin. People can use this strategy with any drug and those that do sometimes describe themselves as having ‘addictive personalities’ despite research repeatedly failing to find any such construct – i.e. they believe that they are unlikely to make moderate use of something they enjoy, so best avoid it.

(11) They have a health scare, or serious health problems (Drug; Anticipated regret), although what concerns one person may not another (Neale, 2000; Orford et al., 2002).

(12) They recognise immanent dependence (drug; PBC). This phenomenon is less-well researched than it should be, but it is clear that drinkers can cut back or abstain for a while as a moderation strategy (Orford et al., 2002), that cocaine users often take similar steps (Ditton & Hammersley, 1996) and that some long-term heroin users adopt a strategy of carefully controlled use (not every day for example) to prevent dependence (Shewan & Dalgarno, 2005).

(13) They are concerned about the legal risks involved (setting; anticipated regret). This tends to be most evident when users may jeopardize their careers by detected use (Shewan & Dalgarno, 2005).
The substance is not readily available (setting). Dependent people may seek it out, non-dependent, or less heavily dependent, people tend to stop or reduce frequency of use (Weatherburn, Jones, Freeman, & Makkai, 2003).

The substance is unduly expensive relative to other factors (setting; Anticipated regret). This is a common reason for moderating cocaine use (Ditton & Hammersley, 1996).

The practitioner-researchers identified two further reasons for stopping or moderating substance use for people who recognise that they are substance dependent: first, they may recognise their dependence and this suffices to motivate them to moderate or quit. But often they remain ambivalent about their dependence long after it is obvious to others (Oser, McKellar, Moos, & Moos, 2010). Second, they may experience an epiphany that leads to a change in life circumstances that transforms them out of substance use. This sometimes involves participation in a mutual assistance or other treatment or religious regime, but sometimes epiphany is merely that “When the time comes (to quit heroin) I believe you will know it” (Mullen & Hammersley, 2006, p. 81). Interestingly, these moments of epiphany seem inexplicable, instantaneous and not teleological at all. Dependent people have often been thorough many such moments when likely future outcomes could have strongly motivated change, but they did not change.

Given that there are at least 15 types of reason for stopping or moderating substance use, people with none of these reasons may be relatively unusual. Indeed, a range of surveys on a range of substances suggests that only about 1/10 substance users show signs of dependence (Hammersley, 2008), leaving 9/10 users who have reasons to stop or moderate their substance use and therefore do so. Without sufficient constraints, if a person has ad libitum access to a substance that is reinforcing, then he or she will tend to become dependent. While it is unclear that any drug meets strict criteria for being a primary reinforcer, the drugs in wide use have some effects that many people can learn to enjoy.

Constraint Theory may perhaps also be applied to other things that are readily reinforcing, such as sex and gambling. It is not particularly designed to explain apparent dependence on things that most people do not find reinforcing, such as carrots, but it predicts that ‘dependence’ can only occur given that the individual finds an activity reinforcing: In the absence of constraints an obsessive collector will amass. Conversely, there is no need to cognitively constrain a behaviour that the person does not find reinforcing. That is, drug dependence can only develop if the person uses a drug that they learn to enjoy.

Constraints are reasons and they are cognitive. The objective severity of the things that ought to constrain a person’s drug use matters far less than the extent to which they believe those things are severe. This applies even to constraints that initially appear to be easily counted. For example, “lack of sympathetic friends to use with,” reads as if one could get research participants to enumerate their drug using friends. But in reality, people may feel and believe that there is nobody that they can use with, although nearby are many acquaintances with whom they could easily use, if they chose to lower their standards of friendship, or associate with people, or go places that they consider undesirable.

The theory suggests that there is no need to address the question of what leads drug users to dependence: The NPP effects of drugs lead to dependence, but people can constrain and control how they think and behave when experiencing those effects. This, in turn, explains why the search for causes of dependence has been elusive and why people often report that dependence came upon them without awareness, intent or plan. Moreover, it can explain why everyone who uses does not become dependent and why some people who use heavily for long periods of time seem much
less dependent than others; because they have more constraints. The next section will test
constraint theory against the problem of social deprivation being correlated with substance
dependence.

EXPLAINING THE LINK BETWEEN DEPRIVATION AND DRUG DEPENDENCE

For a variety of reasons, socioeconomic deprivation tends to make less likely the constraints that
prevent users becoming dependent. First of all (constraints 1–3 & 13), the mind set of people in
deprieved areas may be more tolerant of intoxicant use, bling and partying, and fewer people who
matter to the person may genuinely be opposed to intoxication, although they may be ferociously
opposed to specific drugs (Alasuutari, 1992; Golub et al., 2005; Hammersley & Dalgarno, 2013; Haw,
2004; Levy & Anderson, 2005). Moreover, there is more likely to be mistrust in and disregard of the
law. Young people learning to use drugs in deprived areas can have a negative view of the police and
have experiences of being hassled or arrested in ways they find unfair. They are unlikely to be
concerned about a criminal record, because they do not imagine a career where this would matter
and sometimes positively aspire to the relatively affluent and prestigious life of a gangster (Golub et
al., 2005; Haw, 2004).

Secondly (constraints 4–6 & 14), deprivation can be associated with a lack of employment or
occupation, meaning that people can spend the effort it takes to use drugs a lot, that drug use is less
likely to conflict with other occupations, and that there are financial and social attractions of use and
drug supply (Forsyth, Hammersley, Lavelle, & Murray, 1992; Haw, 2004). Some research from the
deviance perspective has characterised drug dependence as an occupation or career that can give
meaning to an otherwise underemployed life (Agar, 1973; Golub et al., 2005; Maruna, 2008). This
extends to substance use being a cohesive force in socialisation, although this is only officially
acknowledged for alcohol (Cabinet Office, 2003) and often denied for opiate users who, retroactively,
tend to portray that social life as inauthentic and dishonest (Mullen & Hammersley, 2006). Moreover, drug and alcohol retail outlets are concentrated in deprived areas (Forsyth et al., 1992; Pollack et al., 2005) making them more available.

Thirdly (constraints 7–10), while drug effects do not vary as a function of deprivation, in a deprived
neighbourhood it may be more feasible and acceptable to use another drug instead of one the
person dislikes (cannabis instead of alcohol for instance) and the common drug effects may alleviate
tedium (Dreher, 1983), make mood more positive, and reduce stress, which is a function of the size
of the relative deprivation (Yngwe, Fritzell, Lundberg, Diderichsen, & Burstrom, 2003), although
income also matters (Jones & Wildman, 2008).

Fourth (constraints 11, 12 & 15), the adversity of deprived neighbourhoods can mean that future
personal health can be low priority compared to the cash and glamour of drugs (Golub et al., 2005),
and health improvements are not taken as seriously. For example, there is now a substantial class
differential in tobacco smoking rates. In the absence of other activities substance use can be an
affordable – although not cheap – pastime that is enjoyable and also provides escape from mental,
social and physical adversities, for example amongst the homeless (Thompson, Rew, Barczyk, McCoy, & Mi-Sedhi, 2009). Spelled out like this it is obvious why substance dependence is correlated
with deprivation. Table I summarises some key features of Constraint Theory.

IMPLICATIONS FOR PREVENTION

NPP tries to prevent substance dependence by promoting abstinence, by reducing availability, and
by reducing demand for drugs, which includes as yet speculative attempts to blockade drug effects
in the brain. Working from the assumption that dependence is inevitable given sufficient substance
use, prevention under this paradigm routinely confounds the prevention of substance use and the prevention of substance dependence/problematic use. Substance use and a desire for intoxication are as old as history and it seems highly unlikely that they can be completely prevented (Hammersley & Dalgarno, 2012), while tackling some drugs rather than others is not a scientific, evidence-based activity (Nutt et al., 2010). It may be impossible, and perhaps undesirable, to prevent substance use. NPP has little to offer in terms of permitting some use, but preventing dependence.

In contrast, constraint theory offers a clear and simple account of how to prevent substance dependence (Table I); by maintaining or increasing constraints on use. There are four broad ways of doing this: By addressing social norms about appropriate and inappropriate drug using practices (constraints 1–6); by teaching effective management of less than positive psychological states (7–9); by providing accurate information about drug effects and the course of dependence (10–12); by regulation of drugs (13–15), which is not achieved by complete banning, for this hands ‘regulation’ to the entirely unregulated illegal drugs industry. Effective prevention and harm reduction practices already fit into this framework, although some of them are highly controversial in the addiction paradigm, such as providing honest information about the relative harms of different drugs, or admitting uncertainty about the extent to which a novel drug is dangerous.

**IMPLICATIONS FOR BEHAVIOUR CHANGE**

One way of evaluating a theory of behaviour is to assess the extent to which the theory can be used to implement behaviour change (Michie & Johnston, 2012). Constraint theory and the NPP paradigm both assume that dependence involves a ‘habit’ in its strongest sense: A recurrent, often not fully conscious, pattern of behaviour that has been acquired through frequent repetition. It is also uncontroversial that some drugs are more habit-forming than others, due to their NPP effects (Nutt et al., 2010), but with enough repetition any habit can be learned. Behaviour change occurs by replacing the habit with other behaviours, either by abstinence, or by gradually altering the habit, for instance by using less harmful substitute drugs.

In NPP, the potency and irresistibility of the habit is sometimes emphasised by labelling it a ‘disease’. Psychological interventions are conceptualised as supporting abstinence, rather than as treating dependence. For example, the abstinent person may experience a flood of the negative thoughts and feelings that they had been suppressing with alcohol or a drug, and they need to learn to cope with this. Or this may experience cravings for the drug, although sometimes cravings lead to relapse and sometimes they do not, and sometimes relapse seems to occur without craving (Drummond, Litten, Lowman, & Hunt, 2000).

NPP cannot explain why some people can quit such habits seemingly with little preparation and almost on a whim, while others cannot. The mystery deepens, dependent people can struggle with their habits for years, make unsuccessful repeated, highly planned quit attempts, with complex and expensive professional support, finally to simply stop one day. The mystery deepens further once one accepts the existence of heavy substance users who do not fully fit criteria for drug problems. Apparently, despite having strong habits, some people can quit, and others can avoid dependence. How is this possible?

According to constraint theory, habits can stay strong, but the constraints on them can change. Sometimes these constraints change gradually, but sometimes they can change quickly or even in a onetrial manner. Table I shows which constraints may be able to change quickly. When a constraint changes quickly then the person may stop or moderate their habit. For example, some people
change their ways because they suddenly have custody of children. Some people give up a drug because they are prosecuted for possessing it. Different people value different constraints differently, but when there is a rapid change in the strength of constraints on drug use, then dependent people may moderate or quit, not because they did not really have a habit after all, but because the constraints have changed. For example, after protracted use of morphine for pain relief in hospital, a patient may show little or no behavioural or psychological dependence once free of pain and leaving hospital. Constraint theory predicts that seemingly spontaneous or inexplicable quit attempts occur when constraints change suddenly. Constraints are cognitive because they are how people think and feel about things, not how they objectively are. This means that people may not be retrospectively aware of how their cognitions changed, and the process may seem mysterious (Ericsson & Simon, 1984).

Gradual changes in constraints are better understood. Effective interventions focus on facilitating personal change in the client by changing behaviours and cognitions, including motives. Such interventions include cognitive behavioural therapy (Beck, Wright, Newman, & Liese, 1993) and motivational interviewing (Miller & Rollnick, 2002). It appears that interventions that more endorse NPP, such as those using Minnesota model 12-steps approaches, actually use similar methods, but conceptualised as dealing with relapse from abstinence, rather than as achieving abstinence.

Constraints against drug dependence can often be weakened by people having other psychological and social problems, because they have less to lose by heavy drug use. Moreover, most popular drugs impair cognition and may prevent constraints from being fully exercised, so chronic intoxication can cause a downwards spiral of dysfunctionality.

Table II summarises the theory as a typology of dependence, combining two continua, habit strength and the extent of constraints on use. Globally, responses to drug use have tended to involve increasing external constraints on use, by, often harsh, legal sanctions and attempts to restrict supply. According to the scheme of Table II, this will be of some benefit for drugs that have a high potential for habit formation, such as the opiates, but of little or no benefit for drugs with less habit formation potential.

PREDICTIONS AND DEVELOPMENTS

A potential difficulty with Constraint Theory is that as yet there is no way of quantifying the strength of different constraints. Also, the role of individual differences needs to be established, as does the relative power of the different constraints and their architecture. For example, when and how can a person’s strong personal values against substance use override absence of all other constraints on use? Additionally, because constraints are fundamentally cognitive, their logical representation and the processes underlying them are matters for empirical research. For example, how do different constraints combine to produce behaviour? As discussed above, the TPB framework may be extendable to incorporate constraints.

Nonetheless, the theory makes some predictions that are open to empirical test. Focussing first on predictions that appear to be unique to the theory:

(1) The conventional account is that dependence often involves heavy use despite huge constraints on use. This should only be so if one considers ‘constraints’ to be objective facts. According to Constraint Theory, the more cognitive constraints a person has on use of a particular drug, the less likely they will be to become dependent on it. Conversely, people who score highly on measures of dependence will have few constraints on use of that drug.
Constraints are cognitive so people’s constraint cognitions should be better predictors of their dependence status than is their objective situation. For example, amongst high risk populations, such as the homeless, people who are much less dependent should exhibit higher levels of cognitive constraints. If constraints are more social and situational, then the theory is little advance on previous more sociological theories of drug use.

It is not the absolute strength of individual cognitive constraints that affect behaviour, but their relative strength compared to each other and to other motivators of behaviour. For instance, severe lack of money by itself is often not a good constraint on alcohol or drug dependence. For this reason, there shall not necessarily be a strong correlation between the strength of any particular constraint and the severity of dependence.

When a constraint changes suddenly then dependence may suddenly increase, or decrease. For example, the loss of family who care about the person’s drug use can escalate dependence. The immediate likelihood of a potentially fatal health problem can reduce dependence. However, as discussed above, how constraints interact may be more important than the strength of specific constraints, so a sudden change in any given constraint may lead to behavioural change in one person, but not in another.

Dependence can occur on any activity that has become highly reinforcing for the person, under conditions were there are few constraints upon the activity. This can include eccentric hobbies, collections and obsessions, as well as socially valued but extreme activities such as elite sports, and excessive interactions with electronic media.

CONCLUSIONS
A limitation of this theory is that the expert knowledge used to form it came entirely from the UK. However, should people’s common motives for not becoming dependent differ markedly cross-culturally, then this would hardly support the addiction paradigm, which predicts a relatively uniform pattern of dependence everywhere. People’s attitudes to drugs and addiction do differ markedly across cultures, but this can be accommodated within constraint theory. For example, in a culture that is extremely temperate and concerned about addiction more people avoid drug use, mainly for reasons 1–3.

Reversing the problem of dependence solves some thorny problems in addiction research. If dependence is caused by a lack of common constraints on substance use, then there is no need to work out a complex set of causes of dependence. Additionally, the theory can explain how and why social deprivation tends to be correlated with dependence. Finally, the theory offers some clear implications for prevention and treatment, including a theoretical understanding of why techniques known to be effective work, and a promising typology of types of dependent people. There is also a need for more research on the psychological and social, rather than biological, causes of dependence.

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