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# Is the concept of compulsion useful in the explanation or description of addictive behaviour and experience?<sup>☆</sup>



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## ABSTRACT

The concept of compulsion, in which addictive behaviour is said to be carried out against the will, is central to the disease theory of addiction and ubiquitous in modern definitions. The aims of this article are: (i) to describe various meanings of compulsion in the literature; (ii) to compare the part thought to be played by compulsion in addiction with its suggested role in obsessive-compulsive disorder; (iii) to critically examine the place of compulsion in influential neurobiological accounts of addiction; (iv) to summarise the empirical evidence bearing on the usefulness of the compulsion concept, evidence that seems at first sight incompatible with the notion of compulsion. This is followed by a discussion of which possible meanings of compulsion can survive an empirical test and what role they might play in understanding addiction, paying particular attention to a distinction between strong and weak senses of compulsion. A conclusion is that addictive behaviour cannot be considered compulsive *at the time it is carried out*, though other possible meanings of compulsion as an explanation or description of addictive behaviour and experience are discussed. Among other conclusions, it is suggested that, although in some senses of the term it may seem arbitrary whether or not ‘compulsion’ should be retained, its use has important consequences for the public understanding of addiction, and is likely to deter people’s attempts to overcome their addictions and their chances of success.

## 1. Introduction

The concept of compulsion is at the core of the disease view of addiction. In the disease view, it is the compulsive nature of addictive behaviour that distinguishes it from non-addictive behaviour. To say that an addict’s behaviour<sup>1</sup> is compulsive is to say, in respect of their addiction, that they are not free to behave other than they do; they have no choice in the matter or, at least, their ability to choose is severely constrained by the effects of their disease of addiction. In this way compulsive behaviour represents a kind of defect of the will (Wallace, 2003); in some fashion, addictive behaviour is carried out *against the will* of the addicted person. This is in contrast to the behaviour of people who do not suffer from the disease of addiction and whose behaviour is assumed to reflect, in some way, the operation of their free will. In the development of addictive behaviour, the onset of compulsion marks the turning point from normal, recreational drug use to addictive drug use. Thus, in his ‘manifesto’ for the brain disease model of addiction,

Leshner (1997) writes: “Initially, drug use is a voluntary behavior, but when that (metaphorical) switch is thrown, the individual moves into the state of addiction, characterized by compulsive drug seeking and use” (p. 46, parentheses added). In relation to so-called behavioural addictions, to call a behaviour ‘compulsive’ immediately aligns it with substance-related forms of addictive behaviour (e.g., Kraus, Voon, & Potenza, 2016; Maraz, Griffiths, & Demetrovics, 2016).<sup>2</sup> In short, it is compulsion that makes addictive behaviour addictive.

Compulsion also serves an essential socio-political purpose for the disease of addiction. It is because addictive behaviour is compulsive that addicts should not be blamed or punished for the transgression of legal and social norms associated with their addictive behaviour but should instead receive compassion and treatment, when indicated, for their disease. This appeal to compassion and access to treatment is, of course, the basis of longstanding and continuing communications from advocates of the disease theory of addiction to the general public and policy-makers. Indeed, despite the origins of the disease theory of

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<sup>1</sup> I am aware that many people object to the term ‘addict’, agree that those who suffer from what are conventionally known as addictions are individual human beings whose lives cannot be summarised and dismissed by a single term, and apologise for use of the term here. Unfortunately, “people-who-suffer-from-addictions”, or PWSFAs, is just too cumbersome for use, so I ask the reader to keep the foregoing apology in mind.

<sup>2</sup> In this article I shall sometimes make reference to drug addiction or drug use but the reader should bear in mind that, depending on the context, addiction to activities like gambling, aberrant sexual behaviour, internet use, shopping, etc., is often intended too.

addiction at least 200 years ago (Levine, 1978), it is still promoted as a mark of liberal and enlightened opinion to believe that addiction *really* is a disease and that sufferers from it ‘can’t help’ behaving (i.e., are compelled to behave) the way they do. We may note in passing that, despite the appeal of this rationale for a compassionate response to addiction based on the idea of compulsion, it has not generally succeeded in persuading Anglo-American law to withhold criminal responsibility from addicts who break law of the land (Morse, 2017).

Although, as we have seen, crucial for a disease view of addiction and hence for the dispute about whether or not addiction is best viewed as a disease, the notion of compulsion is also accepted by many who reject the disease theory. An influential book by a pioneer of objections to the idea of addiction as a biological disease (Peele, 1985) is subtitled, “Compulsive experience and its interpretation”. A neuroscientist who disagrees that addiction is a disease (Lewis, 2017) nevertheless believes that, in its late stages, addiction is characterised by compulsive urges. Bruce Alexander (2008), who sees addiction as an adaptation to socio-cultural dislocation, refers to it as a compulsive lifestyle. Other examples of non-disease uses of compulsion could be provided. The conclusion is that the concept of compulsion is ubiquitous in modern thinking about the nature of addiction.

### 1.1. Loss of control

In modern writing on addiction as a disease, the idea of compulsion assumes a central place. However, in earlier writings on alcohol addiction, then called ‘alcoholism’, a similar concept was prominent and this should not be forgotten in a discussion of compulsion. This was the concept of ‘loss of control’ over drinking, which had formed the cornerstone of the disease theory of alcoholism from its origins in the early 19th Century to its reformulation by *Alcoholics Anonymous* following the repeal of National Prohibition in the USA in 1933 and subsequent adoption by the medical profession (see Heather, 1991). As in the concept of compulsion, inherent in this concept of loss of control is the idea that alcoholic drinking is ‘against the will’ of the victim of a hypothetical disease process. This is shown most clearly in a famous quotation from Jellinek’s (1960) foundational text for the modern disease concept of alcoholism:

Recovered alcoholics in Alcoholics Anonymous speak of ‘loss of control’ to denote that stage in the development of their drinking history when the ingestion of one alcoholic drink sets up a chain reaction so that they are unable to adhere to their intention to ‘have one or two drinks only’ but continue to ingest more and more – often with quite some difficulty and disgust – *contrary to their volition* (p. 41, italics added).

Loss of control was divided into two kinds of compulsion: ‘inability to abstain’ in which the drinker is unable to refrain from starting to drink after a period of abstinence, and ‘inability to stop’ in which the individual is unable to stop drinking during a single session or keep to limits previously set (Marconi, 1959), as in Jellinek’s example above. The latter kind of loss of control is central to the view of alcohol addiction taken by *Alcoholics Anonymous* (AA), epitomised by the slogan ‘one drink, one drunk’. AA is concerned with the alcoholic’s desire to drink between drinking sessions but refers to this as an ‘obsession’ with drinking rather than loss of control *per se* (see Crowther, 2017).

Following evidence collected during the 1960s and 1970s which cast doubt on the validity of the ‘chain-reaction’ form of loss of control (see below), the concept was watered down by disease theorists by the introduction of an element of unpredictability in the appearance of loss of control. Keller (1972) proposed that alcoholics had not lost control over drinking but could never be sure that, once started, they would be able to stop; Ludwig and Wikler (1974) referred to a *relative* inability to regulate alcohol consumption; and, in the alcohol dependence syndrome, control was seen as “variably or intermittently impaired rather than ‘lost’” (Edwards, 1982, p. 29). Depending on the kind in addiction

in question, the idea of impaired control, as a more refined version of the compulsion concept, can presumably be applied in somewhat different ways to all addictive behaviours.

### 1.2. Aims of this article

Despite extensive references to it in the literature on addiction, it is by no means clear what role compulsion is supposed to play in addiction. As Segal (2017a) has emphasised, different authors mean different things by the term (p. 450). Does it apply to the behaviour itself or to the experience accompanying or preceding addictive behaviour? Does it characterise drug seeking, drug consumption, or both? Is it supposed to be an essential characteristic of addiction or one that applies only to some, presumably more severe forms of it? Above all, is compulsion in some sense of the term required for an adequate explanation of addiction or is its role merely descriptive? If descriptive only, is the description accurate? It is questions of this kind that this article will attempt to answer.

Before proceeding, it may be necessary to make one thing clear. In analysing the concept of compulsion and thus leaving open the possibility that this concept will be found wanting as an explanation or description of addictive behaviour and experience, there is no intention whatever in this article to trivialise addiction. The author recognises that the consequences of severe addiction are devastating and tragic in nearly all spheres of human life. Alcohol addicts drink themselves to death despite the efforts of family, colleagues, neighbours and friends; nicotine addicts continue to smoke despite warnings from their surgeon that limb amputation will be necessary unless they quit; gambling addicts destroy their family’s finances and leave themselves and their loved ones destitute and desperate. In addition to death, disease, mental and social damage, all forms of addiction can lead to terrible feelings of shame and self-loathing (Flanagan, 2013).

The great mystery of addiction is that these consequences normally occur with the full awareness of the addicted individual. So why do addicts persist in their addictive behaviour despite knowing what harm it is doing to their lives and the lives of others? It is not enough to answer this question by saying it is because addicts are compelled to behave that way without attempting to further specify what compulsion means; to fail to do so is merely to restate the central puzzle of addiction. It is also not enough to say that addictive behaviour is ‘against the will’ without further specifying what this means (see Segal, 2017a, 2017b). It is obvious that some addicts do not stop their addictive behaviour before devastating harm has been done. The question is why this happens. Again, it is not enough to say that it is because they ‘can’t stop’ without trying to say why they can’t stop. It is that task of further specification that this article is aimed at assisting.

### 1.3. Structure of the article

The article will be divided into six sections. In the first, in addition to those meanings that have already been noted, various meanings of compulsion will be examined in the literature on definitions of addiction, in classical philosophy, and in modern dictionary definitions of compulsion. This section will conclude by identifying two possible senses of compulsion which, it is claimed, help to clarify how it has been attempted to explain addiction in the literature. Then, the part thought to be played by compulsion in addiction will be compared with its suggested role in obsessive-compulsive disorder. In the third section, the place of compulsion in influential neurobiological accounts of addiction will be critically examined. Next, having previously set out the various possible meanings and theoretical forms compulsion can take, we will examine the behavioural and phenomenological evidence bearing on the usefulness of the compulsion concept. This will be followed by a discussion of which possible meanings of compulsion can survive an empirical test and what role they might play in addiction theory. The article will conclude with an opinion on what part the

compulsion concept can play in the effort to improve the societal response to addictive disorders.

## 2. Meanings of compulsion

### 2.1. Compulsion in definitions or characterisations of addiction

In modern times, addiction (WHO, 1950) or dependence (WHO, 1969) have been defined by the *World Health Organization* (WHO) as compulsion to take drugs. For example, in an oft-quoted passage, WHO (1969) defined dependence,<sup>3</sup> a term with which it wished to replace addiction, as follows:

... a state psychic and sometimes also physical, resulting from the interaction between a living organism and a drug, characterised by behavioural and other responses that *always include a compulsion* to take a drug on a continuous or periodic basis in order to experience its psychic effects, and sometime to avoid the discomfort of its absence (p. 6, italics added).

Of the abundance of other definitions of addiction or dependence in the literature that refer to compulsion as its central feature or as one of them, only a few representative examples will be given. For Leshner (1997) the essence of addiction is “compulsive drug-seeking and use, even in the face of negative health and social consequences” (p. 46). In similar vein, the *Wikipedia* entry for addiction, doubtless viewed by many hundreds of people per day, is: “Addiction is a medical condition characterized by compulsive engagement in rewarding stimuli, despite adverse consequences” (Wikipedia, 2016). These definitions, and the many others in the literature like them, do not themselves give us any idea what kind of thing compulsion is. Also, these typical definitions allow the interpretation that it is possible to have a compulsion without this necessarily being accompanied by harmful or destructive consequences; compulsion here is apparently something more than continuing to engage in a behaviour despite knowledge of harmful consequences.

On the other hand, the *Institute of Medicine* (1996) says that drug addiction consists of “drug seeking behaviour involving compulsion (and) resulting in substantial impairments in health and social functioning” (p. 19), thus making clear that compulsion and harm coincide in addiction. And from a leading psychiatric researcher on addiction and a former Director of the *National Institute of Mental Health* in the USA, we have:

The term compulsion is imprecise, but at a minimum implies diminished ability to control drug use, even in the face of factors (e.g., illness, failure in life roles, loss of job, arrest) that should motivate cessation of drug use in a rational agent willing and able to exert control over behavior” (Hyman, 2007, p. 2).

This ‘minimal’ definition equates compulsion with impaired control over drug use, in whatever form such impaired control is thought to take. We shall return to an evaluation of this minimal characterisation of compulsion towards the end of this essay.

### 2.2. Compulsion in classical philosophy

Stephens and Graham (2009, pp. 206–7) have provided a useful summary of the *locus classicus* for philosophical discussions of compulsion in Aristotle’s *Nicomachean Ethics*. Aristotle says<sup>4</sup>:

<sup>3</sup> In this article I mostly use the terms ‘dependence’ and ‘addiction’ synonymously. However, I prefer ‘addiction’, partly because of the confusion due to ‘physical dependence’, as exemplified in the change made to DSM-5, but mostly because it conveys the sense of enslavement to a substance or activity that is essential to a proper understanding of the disorder in question (see Heather, 2017a).

<sup>4</sup> The quotes to follow are taken from the translation of *The Nicomachean Ethics* of Aristotle by Sir David Ross (1971, Book III, Section 1, pp. 48–52).

“These things, then, are thought involuntary which take place under compulsion or owing to ignorance: and that is compulsory of which the moving principle is outside, being a principle in which nothing is contributed by the person who is acting... [for example] if he were carried somewhere by a wind or by men who had him in their power. ... What sort of acts, then, should be called compulsory? We answer that, without qualification actions are so when the cause is in the external circumstances and the agent contributes nothing.”

Thus, in Aristotle’s sense of the term, compulsion can be said to be present only when the causes of the behaviour are external to the person, the paradigm case being that of someone being blown along by a wind ‘against his will’ and regardless of any internal state that might apply to him at that moment. Stephens and Graham (2009) add that it is in the spirit of Aristotle’s understanding of compulsion, if not the letter, to include as compulsion an ‘internal’ state such as brain damage leading to someone not being able to speak; the person might desire to speak but is compelled by a force outside her desires to be mute. The key point is that compulsion removes control of behaviour from the person’s own motivational states (see Stephens & Graham, 2009). Is addictive behaviour compulsive in this way? Let us call this the *strong version* of the nature of compulsion in addiction.

### 2.3. Dictionary definitions of compulsion

Across a range of dictionaries, there are two kinds of definition of compulsion, one referring to a *want* and the other to a *force*. For example, the *Cambridge English Dictionary* <http://dictionary.cambridge.org/dictionary/english/compulsion> gives: (i) a very strong feeling of wanting to do something repeatedly that is difficult to control, and (ii) a force that makes you do something. The full definition of compulsion from *Miriam Webster* is: 1a) an act of compelling - the state of being compelled; 1b) a force that compels; 2) an irresistible persistent impulse to perform an act ...; also, the act itself.

One of these kinds of definition, that concerning force, is equivalent to the strong sense of compulsion in Aristotle’s writings just reviewed. The other, referring to strong desire, can be found throughout the literature on addiction, beginning with 19th century depictions of ‘habitual drunkenness’ as being due to ‘a burning withering desire for drink’ that was ‘overwhelming’, ‘overpowering’ and ‘irresistible’ (Room, Hellman, & Stenius, 2015). Are addicts’ desires for their substance or activity like this? However strong, irresistible, etc., such desires are thought to be, they refer to the person’s motivational states at the time of acting, and, in contrast to Aristotle’s strong sense of extra-personal compulsion described above, can be called a *weak* version of compulsion in addiction.

### 2.4. The strong and weak senses of compulsion

A modern equivalent of Aristotle’s wind that blows someone along the street, or more accurately the internalised version of that image (Stephens & Graham, 2009), is the concept of *automaticity*, a topic that has been studied intensively by psychologists for the last 40 years (Schneider & Chein, 2003). The basic proposition is that human cognition consists of two different types of information processing – automatic and controlled (or non-automatic). This has led to what is commonly known as the dual process theory of human cognition and the assumption that overt behaviour eventuates from the interplay between automatic and controlled cognitive processes (Evans & Frankish, 2009). An automatic process can be defined as a cognitive-behavioural sequence that “nearly always becomes active in response to a particular input configuration,” and that “is activated automatically without the necessity for active control or attention by the subject” (Schneider & Shiffrin, 1977, p. 2). Automatic processes are the result of highly repetitive learning experiences and are characteristic of most human daily activities, implying that, with sufficient

practice, performance on any task, from tooth brushing to piano playing, can become automatic. In contrast, a controlled process refers to behaviour that is “activated under control of, and through attention by, the subject” (Schneider & Shiffrin, 1977, p. 2–3). Controlled processes are limited in capacity but this is balanced by the fact that they can be easily established and varied in response to novel situations for which automatic sequences of behaviour have never been learned.

The first application of the automatic/non-automatic distinction to addiction was in a classic paper by Tiffany (1990). He begins by rejecting the dominant tradition in theories of addiction up to that time in which craving was seen as its essential defining characteristic and was used to explain the initiation and maintenance of compulsive addictive behaviour, as well as relapse after a period of abstinence. All such theories assume, says Tiffany, that craving and urges are necessary for the production of drug seeking and consumption. However, there is evidence from follow-up studies of addicts who have gone through a treatment programme and have relapsed that drug use is not necessarily preceded by urges or cravings. The alternative hypothesis developed by Tiffany is that psychological processes supporting drug-use behaviour can operate independently of those that control craving and urges and can be derived from the concept of automatic processes. Tiffany writes:

“I contend that drug-use behavior in the addict represents a constellation of specific skills involving drug acquisition and drug consumption. Over a history of repeated practice, the cognitive systems controlling many aspects of drug procurement and consumption take on the character of automatic processes. That is, much of the drug-use behavior of the addict may become largely automatized. Thus, drug-use behaviors tend to be relatively fast and efficient, readily enabled by particular stimulus configurations (i.e., stimulus bound), initiated and completed without intention, difficult to impede in the presence of triggering stimuli, effortless, and enacted in the absence of awareness” (p. 154).

In Tiffany's model, rather than being the proximate causes of addictive behaviour, urges and cravings are supported by non-automatic cognitive processes and activated in parallel with drug-use behaviour. Two kinds of situation are described in which non-automatic craving and urge-responding are invoked: (a) an environmental situation impedes or blocks the drug-use action plan in someone who is not attempting to avoid drug use (abstinence avoidance), or (b) non-automatic cognitive processes are invoked in an attempt to impede or block a drug use action plan (abstinence promotion). The latter kind of situation is relevant to the key issue of relapse and Tiffany clearly states his view that relapse to addictive behaviour can occur in the absence of self-reported urges (i.e., automatically). Abstinent addicts may be particularly prone to relapse when their attention is distracted by some other task, environmental stimuli that trigger the automatic drug-use action plan are present and the drug is readily available. Indeed, Tiffany hypothesises that absent-minded relapses, entailing little or no concurrent activation of non-automatic processes, constitute “a substantial proportion of relapse episodes” (p. 163) and regards this as one of several crucial tests of his model.

For present purposes, Tiffany's (1990) model of the role of automatic processes in addiction represents a clear illustration of the strong version of compulsion. The opposing weak version can be represented, in general terms, by the view of addiction that Tiffany rejects – the one that views addictive behaviour as preceded and caused by craving and drug urges, including importantly the precedents and causes of relapse to drug use after a period of abstinence or non-harmful use. We may think of the first as a strong version of compulsion because the onset of an automatic processes is both necessary and sufficient for relapse to addictive behaviour to occur. In weak version, while non-automatic processes in the form of craving and urges are necessary for relapse, they may not be sufficient depending on whether or not one believes that such craving and urges are literally irresistible.

It was shown above that there are many possible meanings of

**Table 1**  
Meanings of compulsion in the addiction literature.

Addictive behaviour is carried out against the will, or contrary to the volition, of the addicted person (various).
The onset of compulsion marks the turning point from normal, recreational drug use to addictive drug use (Leshner, 1997).
Because of compulsion, sufferers from the disease of addiction ‘can't help’ breaking legal and social norms (various).
In its late stages, addiction is characterised by compulsive urges (Lewis, 2017).
Addiction is a compulsive lifestyle (Alexander, 2008).
A drinker is unable to refrain from starting to drink after a period of abstinence (loss of control 1) (Marconi, 1959).
A drinker is unable to stop drinking during a single session or keep to limits previously set (loss of control 2) (Jellinek, 1960).
Control over drinking is variably or intermittently impaired (impaired control) (Edwards, 1982).
Addiction is characterised by behavioural and other responses that always include a compulsion to take a drug on a continuous or periodic basis in order to experience its psychic effects, and sometime to avoid the discomfort of its absence (WHO, 1969).
Addiction is compulsive drug-seeking and use, even in the face of negative health and social consequences (Leshner, 1997).
Addiction consists of drug seeking behaviour involving compulsion (and) resulting in substantial impairments in health and social functioning (Institute of Medicine, 1996).
Compulsion ... implies diminished ability to control drug use, even in the face of factors ... that should motivate cessation of drug use in a rational agent willing and able to exert control over behaviour (Hyman, 2007).
Compulsion is due to automatic (learned) drug-use action plans to seek and take drugs (Tiffany, 1990).
Compulsion is due to non-automatic and irresistible (or difficult to resist) cravings and urges to seek and take drugs (various).

compulsion and some of these are listed in Table 1. (It is not claimed that this table covers all meanings of compulsion that have appeared in the literature, only some of the more prominent.) Unfortunately, in the absence of additional specification, most of these meanings do not convey any explanatory or even, in some cases, descriptive power. The proposal here is that the strong-weak distinction provides a way of classifying accounts of compulsion in addiction that do have explanatory potential. Strong theories of compulsion are those based on ‘aberrant learning’ while weak theories are those based on strong motivational states of the addict (see Stephens & Graham, 2009). So, in weak versions, compulsion is regarded as the result of abnormal motivation to use or seek drugs; in strong versions, drug-related cues trigger compulsive drug-use and -seeking independently of any motivational states that may be present because the person's normal motivational control over behaviour has been disabled.<sup>5</sup>

### 3. Obsessive-compulsive disorder and addiction

The psychiatric disorder in which compulsion is most obviously implicated is obsessive-compulsive disorder (OCD) and it is important for present purposes to consider similarities and differences between the concept of compulsion as applied to OCD and to addiction. The results of this exercise will hopefully throw light on the nature of compulsion in addiction.

First, compulsive acts in OCD take the form of repetitive or ritualised behaviour, like hand-washing or other cleaning, repeated checking, and excessive and unnecessary orderliness and tidiness (World Health Organisation, 1993; ICD-10, F42.1). The overt behaviour is usually accompanied by anxiety based on a perceived danger to which the individual is either subject or may be the cause of, the ritual act being an ineffective or symbolic attempt to avert the danger. Compulsive acts such as this typically take up many hours of the sufferer's day. Similarly, the drug consumption of addicted persons is

<sup>5</sup> For other discussions of addiction relevant to the distinction between strong and weak senses of compulsion, see Watson (1999), Wallace (2003) and Levy (2006).

highly repetitive, often ritualised and time-consuming.

A possible difference, however, is that, while both addiction and compulsive acts involve reinforcement, in addiction the desire to use a substance or engage in an activity is based on the expectation that the behaviour will be positively reinforced (i.e., result in hedonic reward). In older models of addiction, based mainly on the tolerance and withdrawal found in heroin addiction, addictive behaviour was thought to be maintained by negative reinforcement, i.e., the relief gained from reducing painful withdrawal symptoms and craving (Wikler, 1965). Following the cocaine ‘epidemic’ of the 1980s and for a variety of other reasons, this model was largely replaced by an understanding of addiction emphasising positive reinforcement (Stewart, de Wit, & Eikelboom, 1984). However, it is now still strongly argued that addiction centrally involves both positive and negative reinforcement (e.g., Baker, Piper, McCarthy, Majeskie, & Fiore, 2004) or that negative reinforcement dominates positive reinforcement in later stages of addiction (Koob, 2009). So, while in addiction both positive and negative reinforcement probably play an important part, in OCD it is universally assumed that the sufferer’s expectation is that compulsive behaviour will be negatively reinforced (i.e., result in relief from tension or anxiety).

In the comparison between OCD and addiction, as well as compulsive behaviour we also need to consider compulsion as reported experience. Although they were by no means the first to make the link between addictive drinking and OCD, one of the elements in Edwards and Gross’ (1976) classic delineation of the alcohol dependence syndrome was ‘subjective awareness of compulsion to drink’. The syndrome idea was extremely influential in the history of treatment for alcohol dependence, was later extended to other drugs (Edwards, Arif, & Hodgson, 1982), and was incorporated in DSM-IV (American Psychiatric Association, 1994). The DSM-IV version is often appealed to as a way of demonstrating that harmful forms of a behaviour like gambling can be thought of as addictions (Grant & Odlaug, 2014).

Edwards and Gross (1976) propose subjective awareness of a compulsion to drink as a more accurate representation of the dependent drinker’s phenomenology than descriptions based on of ‘loss of control’ and ‘craving’:

The conventional phrases (loss of control and craving) used to describe the dependent person’s subjective experience are not altogether satisfactory...Perhaps the key experience can best be described as a compulsion to drink, and, though the analogy between alcohol dependence and compulsive disorder has not been considered satisfactory in the past, the subjective experience of dependence may come close to fulfilling the classic conditions for a diagnosis of compulsion. The desire for a further drink is seen as irrational, the desire is resisted, but the further drink is taken...It is the feeling of being in the grip of something foreign, irrational, and unwanted which for severely dependent patients seems to be the private experience which is so difficult to convey (p. 1060).

In a later publication Edwards (1982) describes the compulsion to drink more explicitly as analogous to the “hand washing of a compulsive neurosis” (p. 29). This analogy seems to be in the spirit of the strong sense of compulsion identified above; the experience is of something alien and outside personal wishes and desires. Yet on closer inspection, the similarities between the subjective experience in addiction and in OCD are less obvious. Drawing from the literature on clinical descriptions of OCD, Caetano (1985), in a careful and painstaking analysis, reveals several crucial differences between the two kinds of experience. Caetano concludes:

... while the struggle against a recurring element of consciousness may be experienced by (alcohol) dependent persons, obsessions or compulsions cannot be defined solely by this feature. When the subjective experience of the need to drink is examined in the light of other phenomenological characteristics, it fails to meet the

requirements for diagnosis as an obsession or a compulsion. ... it is not an impediment to effective action; its content cannot be seen as genuinely senseless; it is perceived as expressing a need of the self which is not present in obsessed patients; and the internal resistance it triggers does not lead to defensive compulsive rituals (Caetano, 1985, p. 468, parentheses added).

Edwards’ (1982) analogy between dependent/addictive drinking and compulsive hand-washing may therefore be misplaced. While there may be superficial similarities, compulsion in OCD and addiction arguably reflect quite different phenomenological qualities. This raises the possibility that, *pace* Edwards, the experience of ‘compulsion’ in addiction may be no more than a strong, or exceptionally strong, desire. The question whether the forms compulsion is alleged to take in addiction and in OCD are similar or different will continue to be asked in this essay.

#### 4. Neurobiological theories of addiction

The role of compulsion in addiction has been described in the various neurobiological theories of addiction that have been developed over the last 20 years or so, although this role differs between theories. With the exception of the work of Nora Volkow and her colleagues (see below), these theories are founded mainly on animal models of addiction. Owing to the high prestige of neuroscience in today’s society, accounts of compulsion in these theories are probably the most influential to be found in the scientific literature and deserve careful attention here. In this section we will consider some of the leading theories, and summarise and comment on the way that compulsion is described therein.

##### 4.1. Everitt and Robbins’ aberrant learning theory

In this theory, addiction is seen as the endpoint of a series of transitions from voluntary drug-taking, through habitual use, to compulsive use (Everitt & Robbins, 2005). The theory assumes that there are two kinds of learning relevant to understanding addiction – action-outcome (A-O) learning and stimulus-response (S-R) or habit learning. Initially, voluntary drug use is controlled by expectations users have about the outcomes or consequences, usually hedonic pleasure, of their drug-seeking and drug-taking actions and hence learn to carry out those actions in order to experience those rewards (A-O learning); the user is aware of her expectations and the motivation to seek and use drugs. Eventually, however, over repeated self-administrations, the user learns to associate certain stimuli (e.g., people or settings associated with drug use, drug-using paraphernalia, etc.) with reward and these stimuli become conditioned reinforcers that maintain drug-seeking behaviour (S-R learning). This learning is mostly implicit (i.e., the user is unaware of the connection between drug cues and expectations of reward) and in this way drug-seeking becomes largely ‘automatic’ and independent of the user’s conscious preferences and motivations. Eventually, after yet more drug self-administrations, these over-learned habits become so automatic that they can be called compulsive. It is important to note that both kinds of learning are thought to occur in parallel during the user’s drug-taking career but eventually, in habitual and compulsive drug use, S-R learning comes to dominate behaviour.

These transitions in drug-taking and -seeking behaviour are reflected by a transition at the neural level from control predominantly by the prefrontal cortex to control by the striatum in the forebrain and, at the same time, a progression of control from ventral to more dorsal domains of the striatum, i.e., increased excitation of dopamine neurons in the dorsal sub-region of the striatum. In addition to the effects of repeated self-administrations, these neural transitions may also be the result of changes to cognitive executive functioning brought about directly by the effects of chronic drug ingestion itself.

In theoretical terms, compulsive behaviour is characterised as “a maladaptive stimulus-response habit in which the ultimate goal of the behavior has been devalued so that the behavior is not directly under the control of the goal” (p. 1485). With regard to subjective experience, [Everitt and Robbins \(2005\)](#) write:

Crucial to drug addiction is the persisting quality of these habits, which has been likened to the subjective state of ‘wanting’, but which we would suggest corresponds more obviously to the subjective state of ‘must do!’—although this subjective response could arise *post hoc* as a rationalisation of the ‘out-of-control’ habitual behavior rather than being the driving influence (p. 1485).

The comment here about ‘post hoc rationalisation’ is interesting and will be returned to below. As [Everitt and Robbins](#) themselves say, however, “habitual responding by itself ... does not capture the persistent, indeed, compulsive aspects of ‘out-of-control’ drug bingeing; some additional factor seems to be required.” Although it applies to drug-bingeing and ‘inability to stop’, it is not clear in [Everitt and Robbins’](#) description of their theory what this additional factor is thought to be.

A familiar analogy employed to illustrate the change from conscious, voluntary behaviour to non-conscious, involuntary behaviour is learning to drive a car; the novice is uncomfortably aware of his movements and lack of co-ordination until with practice driving becomes something automatic which he does not need to think about (see, e.g., [Duhigg, 2012](#)). However, in case critics of their theory might think that this is being suggested as an analogy with drug-taking and drug-seeking *per se*, [Everitt and Robbins](#) say that “... it is not an example of a procedural skill, such as playing the piano or tying one’s shoelace—although it is plausible that such skills result from even more extended training. The analogy with drug addiction would be a persistence or constant re-initiation of such activities” (p. 1485). Here it is stressed that addictive behaviour entails the ‘constant re-initiation’ of automatic behaviour similar to playing the piano, tying one’s shoelace or, presumably, driving a car.

In terms of the distinction made earlier between strong and weak versions of the concept of compulsion in addiction, it is clearly the strong version, based on automatic behaviour outside the agent’s motivational control, that marks aberrant learning in this theory. In this respect, [Everitt and Robbins’](#) theory is a continuation of the tradition of thinking about addiction prompted by [Tiffany’s \(1990\)](#) seminal article. Moreover, the key distinction among contemporary theories of addiction is whether compulsion is seen primarily as evidence for a disorder of learning and memory, implying automatic behaviour, or of motivation, implying disordered desires or appetites (see [Berridge & Robinson, 2011](#)).

#### 4.2. *Koob and Le Moal’s allostatic dysregulation theory*

This theory proposes that brain changes occurring during the transition from drug use to addiction account for the addict’s persistent vulnerability to relapse long after drug-taking has ceased ([Koob, Ahmed, Boutrel, et al., 2004](#); [Koob & Le Moal, 2001](#)). Addiction is said to result from a cycle of progressively increasing dysregulation of brain reward systems that eventuate in compulsive use, loss of control over drug-taking and spiralling distress. This dysregulation involves different sources of reinforcement, different neuro-adaptive mechanisms, and different neurochemical changes to the brain reward system but, crucially, alterations in the mesolimbic dopamine system, opioid peptidergic systems, and brain and hormonal stress systems provide the negative emotional state (e.g., dysphoria, anxiety, irritability) when access to the drug is prevented that drives addiction.

With prolonged consumption of addictive drugs, sensitisation to drug-related stimuli and counter-adaptive processes that are part of a normal homeostatic limitation of reward function, such as the opponent process described by [Solomon and Corbit \(1974\)](#), fail to return to the

normal range and instead form an ‘allostatic’ state. Allostasis is the process of maintaining stability by successive changes in brain reward mechanisms but, with the effects of continued drug-taking and withdrawal, this stability cannot be maintained. Thus, these allostatic brain changes represent a chronic and pathological deviation of reward set-point, fuelled not only by dysregulation of reward circuits but also by the activation of autonomic nervous system and hormonal stress responses induced by the extended amygdala and representing the so-called ‘dark side’ of addiction ([Koob, 2009](#)). The manifestation of this allostatic state as compulsive drug-taking and loss of control over drug-taking is thought to be based on activation of brain circuits such as the cortico-striatal-thalamic loop, similar to the neural mechanism described in [Everitt and Robbins’ \(2005\)](#) theory.

In this way, the Koob and Le Moal theory describes the brain changes hypothesised to result in compulsive drug use, but how is compulsivity itself portrayed? In addition to the usual statements that one of the defining properties of addiction is a compulsion to seek and take drugs, mentions of compulsivity are replete in the writings of Koob and his collaborators and it is variously characterised. For example, “Compulsivity in addiction can derive from multiple sources, including enhanced incentive salience, engagement of habit function, and impairment in executive function. However, underlying each of these sources is a negative emotional state that may strongly impact on compulsivity” ([Koob, 2009](#), p. 27). Consistent with this underlying source, drug addiction is said to involve a progression from impulsivity to compulsivity, with a concomitant shift from positive reinforcement to negative reinforcement driving the motivated behaviour ([Koob & Volkow, 2010](#)). Thus, a negative affective state contributes to compulsivity through negative reinforcement mechanisms (i.e., behaviour reinforced by the termination or reduction of the negative affective state). In later writings, fuller definitions of compulsivity are offered. Compulsivity is:

“...defined as elements of behavior that result in perseveration in responding in the face of adverse consequences, perseveration in responding in the face of incorrect responses in choice situations, or persistent re-initiation of habitual acts ([Everitt & Robbins, 2005](#)). The elements of compulsivity are represented in many of the symptoms outlined in DSM-IV: continued substance use despite knowledge of having had a persistent or recurrent physical or psychological problem and a great deal of time spent in activities necessary to obtain the substance ([American Psychiatric Association, 2000](#))” ([Koob & Volkow, 2010](#), Table 1, p. 218).

The reference to ‘perseveration’ and to [Everitt and Robbins’ \(2005\)](#) paper in this quotation, together with the appeal elsewhere to the idea of the ‘automaticity’ of addictive behaviour, appears to align the Koob and Le Moal understanding of compulsivity in addiction with that of [Everitt and Robbins’](#) aberrant learning theory of addiction (see above). Despite the presentation of the Koob and Le Moal theory as a motivational model of addiction, this suggests the strong version of compulsion in which compulsion exists independently of the agent’s motivational states. On the other hand, the earlier depiction of compulsion as a consequence of the attempt to relieve a negative emotional state, assuming that animal models for this state can be equated with a human desire to feel better, aligns the theory with the weaker version of compulsion identified in this essay. The conclusion is that the allostatic dysregulation theory contains elements of both the strong and weak senses of compulsion.

#### 4.3. *Robinson and Berridge’s incentive-sensitisation theory*

This theory begins with an assumption, derived mainly from neurobiological research on nonhuman animals, that addictive drugs have in common the ability to enhance dopamine transmission in the mesolimbic dopamine system of the brain, a system known to be involved in reward and motivation for natural, appetitive reinforcers. The main

psychological function of this neural system is to attribute 'incentive salience' to the perception and mental representation of a certain class of stimuli or events, where incentive salience is the psychological process that imbues stimuli with salience and makes them attractive and sought-after (see also, e.g., [Robinson & Berridge, 2001](#)). In some individuals, the repeated use of addictive substances results in incremental neuro-adaptations in this system, rendering it increasingly and persistently hypersensitive (i.e., 'sensitised') to drugs and drug-associated stimuli. Although sensitisation is not just a conditioned response, it is triggered by a process of associative (Pavlovian) learning, which causes excessive incentive salience to be attributed to drugs and associated stimuli. Sensitisation of incentive salience is therefore the specific mechanism hypothesised to transform ordinary desires for drug experiences into drug craving. Incentive sensitisation is also responsible for relapse to drug use, even after protracted periods of abstinence and the cessation of withdrawal phenomena. Thus, the theory proposes that "the defining characteristics of addiction (craving and relapse) are due directly to drug-induced changes in those functions normally subserved by a neural system that undergoes sensitisation-related neuro-adaptations" ([Robinson & Berridge, 1993](#), p. 250).

An important part of the theory, and one essential to understanding the role that compulsion plays in it, is the distinction between 'wanting' and 'liking' drugs and drug experiences. The sensitisation of incentive salience is termed drug 'wanting' but the accompanying changes in neural systems can occur independently of changes in other neural systems mediating the subjective, pleasurable (hedonic) effects of drugs, called drug 'liking'. As a consequence, incentive sensitisation can produce compulsive drug-taking and -seeking even when the expectation of drug pleasure (or relief from the aversive properties of withdrawal) is reduced and even in the face of strong disincentives, such as the loss of reputation, job, home, family and other harms of addictive behaviour. Evidence is cited from both human and animal studies against the idea that drug taking is necessarily motivated by the subjective pleasurable effects of drugs, i.e., subjective pleasure is not necessary for the maintenance of drug-seeking and drug-taking behaviour in addiction. Although in normal behaviour, including recreational drug use, 'liking' and 'wanting' coincide, as the development of addiction proceeds they become increasingly dissociated. Moreover, "the attribution of incentive salience is not a conscious process and the introspective experience of 'wanting' or craving is only a person's interpretation of the outcome of that process. Much of the time the attribution of incentive salience may be more implicit than explicit" ([Robinson & Berridge, 1993](#), p. 267). The authors continue:

... the addict can be only subjectively aware of the outcome of excessive incentive salience attribution, craving. The addict may have little insight into the reason for the craving and indeed, may himself be bewildered by its intensity. At a conscious level addicts may recount all of the negative consequences of continued drug use, deplore their situation, even comment that the drug does not continue to give great pleasure - and not understand why their craving persists ([Robinson & Berridge, 1993](#), p. 267).

While in the description of the theory in numerous articles and chapters the role played by compulsion is of the generic kind, roughly equivalent to continued use despite harm, the above quotation gives a more specific meaning to compulsion in the theory - the addict's experience of feeling driven to continue the activity without understanding their motivation for doing so and the sense of bewilderment this experience gives rise to. However, although the addict may not understand their motivation for craving drugs, it is nevertheless excessive motivation to seek and consume drugs brought about by incentive sensitisation that is the basis for describing addiction as compulsive. As [Stephens and Graham \(2009\)](#) assert, it is this aspect of compulsion in the theory that makes it incompatible with the classical meaning of compulsion derived from Aristotle (see above) and with an understanding of compulsion centred on automaticity.

#### 4.4. Goldstein and Volkow's iRISA theory

Nora Volkow, the Director of the *National Institute on Drug Abuse* in the USA, has made many distinctive contributions to neuroscientific research on addiction. One strand of research and theory arises from a collaboration with Rita Z. Goldstein and the development of a model<sup>6</sup> of addiction they call *impaired response inhibition and salience attribution* (iRISA) ([Goldstein & Volkow, 2002, 2011](#)). Unlike the other theories of addiction summarised above, this is based primarily on neuroimaging studies with human subjects.

The starting point for the iRISA theory is that previous theories had focussed primarily on the limbic, subcortical 'reward' centres of the brain but the role of structures in the prefrontal cortex had been neglected. While subcortical circuits may be crucial to the rewarding effects of drugs and the initiation of drug self-administration, the state of addiction also involves disruption of cortical circuits that inhibit or modulate compulsive behaviour and drive. [Goldstein and Volkow \(2002\)](#) propose an integrated model of drug addiction, based on neuroimaging studies, that embraces the addictive cycle of intoxication, bingeing, withdrawal and craving. The orbitofrontal cortex and the anterior cingulate gyrus, which are anatomically connected with limbic structures, are activated during intoxication, craving and bingeing and de-activated during withdrawal. These cortical regions are also involved in higher-order cognitive and motivational functions concerned with the attribution of salience to reinforcers, as influenced by context and expectation, and with the control and inhibition of prepotent responses. In addition, dysfunction of these and other prefrontal regions results in the overvaluing of drug reinforcers, the undervaluing of alternative reinforcers, and deficits in inhibitory control of drug responses. These changes, summarised as iRISA, are hypothesised to enlarge upon the traditional understanding of addiction emphasising limbic-regulated responses to pleasure and reward.

With regard specifically to compulsion, [Goldstein and Volkow \(2002\)](#) write as follows:

... we propose that the behaviours and associated states that are at the core of drug addiction are distinctly the processes of loss of self-directed/willed behaviours to automatic sensory-driven formulas and attribution of primary salience to the drug of abuse at the expense of other available rewarding stimuli. We hypothesise that these states are first evoked in the presence of the drug of abuse or cues conditioned to the drug but then become chronic action tendencies, contributing to relapse/bingeing (behavioural compulsion) and withdrawal/craving (mental compulsion, i.e., obsessiveness), respectively (p. 1643).

In an earlier article, [Volkow and Fowler \(2000\)](#) expand on their view of addiction as 'a disease of drive and compulsive behaviour' and, in a striking passage, stress that pleasure *per se* cannot account for compulsive drug intake:

An analogy that may be useful to explain the dissociation of pleasure from drug intake in the addicted subject could be that occurring during prolonged food deprivation when a subject will eat any food regardless of its taste, even when it is repulsive. Under these circumstances the urge to eat is not driven by the pleasure of the food but by the intense drive from the hunger. It would therefore appear that during addiction the chronic drug administration has resulted in brain changes that are perceived as a state of urgency not dissimilar to states that are observed on (sic) states of severe food or water deprivation. However, different from a state of physiological urgency for which the execution of the behaviour will result in satiety and termination of the behaviour, in the case of the addicted subject the disruption of the orbitofrontal cortex coupled with the

<sup>6</sup> As will be obvious, I make no distinction here between the terms 'theory' and 'model'.



increases in DA elicited by the drug set a pattern of compulsive drug intake that is not terminated by satiety and/or competing stimuli (p. 323).

In a later article, Goldstein and Volkow (2011) present a much more complex and intricate model of addiction and of the role of cortical structures and functions within it. However, there are prominent references to habitual, automatic, stimulus-driven behavioural patterns, involving impairments to self-control mechanisms, attentional bias towards drug-related stimuli and away from other stimuli and reinforcers, and inflexibility in goals to procure the drug (see, e.g., Table 1, p. 654). In this connection it should be noted that other influential articles by Volkow, written with different collaborators, give a somewhat different perspective on compulsion in addiction. For example, Kalivas and Volkow (2005) assert that, “A primary behavioral pathology in drug addiction is the overpowering motivational strength and decreased ability to control the desire to obtain drugs” (p. 1403), i.e., the weak, desire-based, motivational form of compulsion rather than the automatic, amotivational, strong kind that is dominant in Volkow's papers with Goldstein. Volkow and Li (2004) say simply: “Drug addiction manifests as a compulsive drive to take a drug despite serious adverse consequences. This aberrant behaviour has traditionally been viewed as bad ‘choices’ that are made voluntarily by the addict. However, recent studies have shown that repeated drug use leads to long-lasting changes in the brain that undermine voluntary control” (p. 963).

## 5. Evidence causing difficulties for compulsion in addiction

We will now consider a number of types of evidence that cause difficulties for the idea that addictive behaviour and experience is compulsive. Much of this material is familiar to serious students of addiction but reasons for summarising it here are twofold. First, it provides an opportunity to gather together in one place various strands of evidence relevant to compulsion that are scattered throughout the literature on addiction. Secondly, despite what any reasonable person must regard as the direct relevance of this evidence to the issue of compulsion, it seems to have had little effect on contemporary, public-facing and ‘official’ portrayals of addiction. As made clear in the introduction to this essay, it is now standard language to present addiction as a disease characterised by compulsion and, even in non-disease accounts, compulsion is ubiquitous. It is perfectly legitimate, of course, to defend the validity of putting compulsion at the centre of a portrayal of addiction but any defence worthy of respect must take into account the evidence that will be reviewed in this section.

One way to assess the relevance of this evidence to compulsion in addiction is to reflect whether evidence of a similar kind would also cause difficulties for the role of compulsion in a condition like OCD in which compulsion plays an obvious part. This will be done in appropriate places.

### 5.1. Addictive behaviour is operant behaviour

Perhaps the type of evidence that most obviously causes difficulty for compulsion in addiction is the demonstration that addictive behaviour is operant behaviour. This is a term associated originally with the radical behaviourism of B. F. Skinner (1953) and, in a well-known quotation, is defined as behaviour that is “shaped and maintained by its (environmental) consequences” (Skinner, 1972, p. 23, parentheses added); in other words, it refers to behaviour that is influenced by, or is ‘contingent upon’, the events that follow it. Operant behaviour is also called ‘instrumental learning’ and ‘operant conditioning’, the kind of learning identified by Skinner as being distinct from classical or ‘Pavlovian’ conditioning of automatic reflexes. In contrast to classically-conditioned responses, operant behaviour refers for all practical purposes to voluntary and intentional action that is directed towards the

attainment of expected rewards and that is ‘reinforced’, or made more likely to recur, by the receipt of those rewards. If addictive behaviour really is a form of operant behaviour, it is difficult to see, *prima facie*, how it can be called compulsive in any simple or straightforward sense.

#### 5.1.1. Alcohol addiction

A considerable body of experimental evidence put together during the 1960s and 1970s by Mello, Mendelson, and their colleagues (e.g., Mello, McNamee, & Mendelson, 1968; Mello & Mendelson, 1965, 1972; Mendelson & Mello, 1966) showed conclusively that the drinking of even the most chronic and severe alcohol addicts, or ‘alcoholics’, was operant behaviour. These experiments and other relevant evidence were summarised by Heather and Robertson (1983, Chapter 3). We will consider this evidence at some length because it has clear and direct relevance to the place of compulsion in addiction but this relevance seems to have been forgotten in current literature on the topic.

A typical set-up in these experiments is that diagnosed alcoholics were given free access to an operant apparatus at any time during the day or night over periods of about two weeks. Participants could obtain either a single shot of whisky or the money equivalent by pressing a translucent key that changed colour according to a random sequence of reinforcement schedules (Mello & Mendelson, 1965). In another experiment, the key-pressing task was replaced by one more attractive to participants – earning points that could be exchanged for alcohol or money by keeping a model car on the road in a driving machine (Mendelson & Mello, 1966).

In later research by the same team, the emphasis was on the cost of alcohol among alcoholic participants, with cost defined as the amount of work (e.g., the number of consecutive correct key presses) required to obtain a reward (Mello et al., 1968). The main finding here was that the amount of alcohol consumed, inferred from participants' blood alcohol levels, was a predictable function of the degree of effort required to obtain it. Mello and Mendelson (1972) studied alcoholics' drinking behaviour over longer periods than in the previous experiments – 30 or 62 days. A key observation was that, despite the occurrence of partial withdrawal symptoms, participants did not immediately start drinking in the attempt to abolish them but preferred to continue working to amass more tokens that could be exchanged for alcohol. Another observation was that, among those who were studied for 62 days, none of 18 participants drank all the alcohol available despite the absence of any kind of limitation on consumption, and tokens that could have been exchanged for large amounts of alcohol were handed back at the end of the experiment.

Following the precedent set by the Mello and Mendelson team of giving alcohol to alcoholics for research purposes, many other researchers made important contributions during the 1970s to our understanding of the actual drinking behaviour of alcoholics (see Heather & Robertson, 1983, Chapter 3, for review). Especially significant was an ingenious experiment by Miriam Cohen, Liebson, Faillace, and Speers (1971) who were also interested in cost factors. Here, however, the target behaviour was abstinence and the broad aim of the research was to determine what reinforcement contingencies were necessary to ‘buy’ abstinence from male chronic alcoholics. This was done by examining interactions between cost and two other important variables – a priming dose of alcohol and a delay in reinforcement. Participants were allowed to purchase a relatively large quantity of alcohol every third day of the experiment and on subsequent days were offered a certain amount of money to abstain for the entire day. If the participant did not abstain the incentive was increased on the next occasion but if he abstained it was decreased. The immediate result was that abstinence could be bought from each of the four participants for varying amounts ranging from \$7 to \$20.

From this base, the effects of the two experimental manipulations were examined and both showed the same pattern of findings. A delay in reinforcement disrupted abstinence such that the amount of money

previously effective in buying it no longer worked. However, increasing the amount of the reward was able to reinstate abstinence. Similarly, a priming dose of alcohol given on the morning of the day on which payment for abstinence was to be offered disrupted abstinence on that day but increasing the magnitude of monetary reinforcement re-established it. This latter finding is especially noteworthy because it suggests how the idea of loss of control over drinking could have arisen – because a priming dose of alcohol disrupted the choice of abstinence that had previously been made – while suggesting that this apparent loss of control was an illusion – because control over drinking and the choice of abstinence could be reinstated by a sufficiently large incentive.

Bearing this in mind, we may conclude as follows. First, in a situation in which they were able to determine the volume and patterning of their own drinking, none of the participants in these experiments, who would all qualify for a diagnosis of alcohol dependence under DSM-IV criteria or severe alcohol use disorder under DSM-5, attempted to drink themselves into a state of unconsciousness or even drink all the alcohol available to them with little effort on their part. This is not to deny, of course, that drinking to oblivion never occurs in the natural environment but it does show that a *compulsion* to drink to severe intoxication does not predictably occur in alcohol addicts. Secondly, the participants in these studies clearly showed control over their drinking in a range of ways. For example, they drank in order to maintain high but roughly constant blood alcohol concentrations; they did not drink continuously but spontaneously initiated and terminated drinking sessions; some chose to taper-off their drinking in order to avoid or reduce withdrawal symptoms or chose to work to accumulate alcohol rather than drink to abolish withdrawal symptoms. All these observations are inconsistent with a straightforward interpretation of compulsion in the sense of an inability to stop drinking once started or an uncontrollable and irresistible craving to continue drinking until forcibly prevented from doing so. In case these meanings of compulsion are thought to be ‘straw men’, they are both frequently encountered in the literature on the disease concept of alcoholism and on the notion of compulsive drinking as the key feature of alcoholism.

The most general finding from these experiments that appears to go against the validity of compulsion is that the amount of alcohol consumed by alcohol addicts in a free-choice laboratory situation was a direct function of the cost of alcohol, as measured by the degree of effort required to obtain it. One does not have to be a radical behaviourist, and to subscribe to the Skinnerian view that it is only observations of external behaviour that count as science, to realise the significance of this. It shows drinking by alcohol addicts is, as stated, operant behaviour that is largely determined by its consequences. The particular reinforcement contingencies applying to the drinking behaviour of chronic alcohol addicts obviously show marked differences from those applying to non-alcohol addicts – self-evident from the existence of markedly different drinking practices. But what these findings do clearly demonstrate is that, rather than being compelled and qualitatively different, alcohol addicts' drinking behaviour is subject to the same laws that govern normal, goal-directed, voluntary behaviour of any sort.

### 5.1.2. Cocaine addiction

Following the experimental demonstration in the 1960s and 1970s that alcohol addiction consisted of operant behaviour, similar principles were applied to the study of cocaine addiction in the 1990s. During the previous decade, the ‘cocaine epidemic’ had given rise to considerable public and governmental concern, particularly in the USA, and this resulted in a focus of research on cocaine addiction. Stephen Higgins (1997) reviewed evidence available at that time bearing *inter alia* on the following key assumptions: (i) cocaine use is an instance of operant behaviour; (ii) the degree of behavioural control that cocaine exerts as a reinforcer is malleable and dependent on environmental context; and (iii) increasing the availability of alternative, non-drug reinforcers is

one contextual alteration that can significantly disrupt the acquisition and maintenance of cocaine use and abuse. Of particular interest to present concerns, Higgins showed that, in experimental studies in non-humans and humans, in laboratory and clinic settings, with different routes of cocaine administration, and with recreational and addicted human cocaine users, cocaine use can be decreased significantly by increasing the availability of alternative reinforcers. As before, these observations are inconsistent with any straightforward, ‘loss of control’ interpretation of compulsion in addiction.

A demonstration of the ability of alternative reinforcers to modify cocaine use in ‘experienced cocaine smokers’ was carried out by Hart, Haney, Foltin, and Fischman (2000). The six participants in these experiments were volunteers recruited from the community in New York City who were not seeking treatment. However, all reported almost daily use of smoked cocaine, were currently spending between \$100 and \$500 per week on cocaine, had been convicted of cocaine-related crimes in the past and had arranged their lives around their cocaine habit; they would all almost certainly meet DSM criteria for drug dependence or substance use disorder. Following previous work reviewed by Higgins (1997) showing that cocaine self-administration could be modified by the availability of alternative reinforcers (see above), the immediate aim of the experiment was to assess whether money vouchers or merchandise vouchers were more effective as an alternative to cocaine. However, it can serve here as a detailed demonstration of the limitations of the compulsion concept when applied to what was then, and still often is, reputed to be one of the most addictive forms of human drug use ever witnessed (i.e., ‘crack cocaine’).

In an inpatient setting, a six-trial choice procedure was used with sessions consisting of one sample trial, in which participants received the cocaine dose and the alternative reinforcer available that day, and five choice trials where they chose between the available cocaine dose and the alternative reinforcer. There were eight sessions in each of which a dose of cocaine (0, 12, 25, 50 mg) was paired with a money voucher that could be exchanged for \$5 at discharge or a \$5 merchandise voucher. Trials were presented at 14-minute intervals and the beginning of a trial was indicated to the participant by a visual cue (two squares) on a video screen. Each of the two squares served as a discriminative stimulus to indicate the availability of either the stipulated dose of cocaine or the alternative reinforcer. Using a computer manipulandum, participants selected the left (cocaine dose) or the right (alternative reinforcer) option, and fulfilled the response requirement by pressing the keyboard spacebar 200 times. Once the response requirement was satisfied, and assuming that vital signs were within safe criteria for drug administration, a cocaine dose or alternative reinforcer was administered.

The basic finding from this experiment was that money vouchers were a more effective alternative reinforcer than merchandise vouchers among these crack cocaine users. What is more interesting for present purposes however is firstly that, despite the fact that these experienced cocaine smokers chose to self-administer more cocaine doses per session as the cocaine dose increased, they frequently rejected the choice of a lower dose of cocaine in favour of an alternative. Moreover, this often occurred after they had received a ‘taste’ of the drug in the sampling session, which under the conventional disease view of addiction would presumably have elicited an irresistible craving for more. Secondly, the type of alternative reinforcer available differentially influenced cocaine self-administration, i.e., participants chose to self-administer significantly less cocaine when a money voucher was the alternative compared to when a merchandise voucher was available, showing that whether or not cocaine is chosen depends on its specific environmental consequences. What seems remarkable about these findings is that, among these individuals who in the mainstream media and in many conventional scientific and professional circles would be regarded as hopelessly addicted to their drug of choice, a relatively small amount of money (a maximum of \$50 per day if the alternative

reinforcer were consistently chosen) could be effective in persuading them to reject the choice of cocaine.

### 5.1.3. Nicotine addiction

The concept of operant behaviour also applies to nicotine addiction, although the evidence on which this conclusion is based is not experimental but studies of the relationship between smoking and cost. Gallet and List (2003) reviewed economic surveys showing that cigarette smoking responds to monetary contingencies. They concluded that raising prices by 1% reduces consumption by about 0.4% and that, generally speaking, when smokers' income rises they smoke more. Moreover, in these times of widespread bans on indoor smoking, going outdoors to smoke must be more unpleasant in winter than summer and it would therefore be expected that smokers would reduce their smoking during the cold months. Indeed, Momperousse, Delnevo, and Lewis (2007) found that cigarette sales in New Jersey were 37% lower in February than in June. As pointed out by Baumeister and Vonasch (2015), the fact that smoking is sensitive to price changes and other kinds of cost indicates that rational calculations influence behaviour and that impulses to smoke can be resisted. So too, compliance with smoking restrictions is generally high, which could not be the case if urges to smoke were truly irresistible and compulsive.

### 5.1.4. Summary

We have seen in this section that three major classes of addictive behaviour, involving addiction to alcohol, cocaine and nicotine, are examples of operant behaviour in the sense that they are responsive to environmental contingencies; when the consequences of the behaviour change, so does the behaviour. Could the same apply to OCD? While it is possible that the compulsive behaviour of the OCD sufferer could be temporarily changed by altering response contingencies (cf. Lyvers, 2000), it seems very unlikely that the kind of changes seen in the laboratory experiments described above would occur in OCD.

## 5.2. Contingency management programmes

A possible objection to the evidence just reviewed on alcohol and cocaine responses is that it is restricted to the artificial situation of the controlled experiment, with little relevance to the real world and, especially, to the treatment of addictive disorders outside the laboratory. This objection is contradicted by a large amount of evidence that treatment based on the principles of operant conditioning is perhaps the most effective way of helping people to change addictive behaviour.

The treatment in question is known as 'contingency management' (CM). This refers to a form of behaviour therapy in which tangible positive reinforcers are provided to people with substance use disorders contingent on objective evidence, usually in the form of biological specimens, of abstinence from the substance in question. A type of reinforcer widely studied is the provision of vouchers exchangeable for goods and services following evidence of abstinence from cocaine by urinalysis (Higgins, Delaney, Budney, & Bickel, 1991). In programmes of this kind, the value of the reinforcer typically increases with successive abstinence but resets to a minimum value if a positive test occurs. A less expensive kind of CM programme uses prize-based reinforcement (Petry, Martin, Cooney, & Kranzler, 2000), in which patients are permitted to draw from a bowl containing slips of paper with a chance of winning prizes of varying amounts. Alternatively, clinic privileges may be used as reinforcement, where the privilege could be access to a take-home dose in a methadone programme (e.g., Stitzer, Iguchi, & Felch, 1992).

A large number of randomised controlled trials have been carried out to assess the efficacy of CM among addicted individuals. As an example, Silverman et al. (1996) randomised cocaine-addicted patients of a methadone programme to either a voucher CM group or to a yoked-control in which patients received vouchers at the same rate and magnitude as those in the contingent group but regardless of urinalysis

results. Almost half of those in the CM group achieved two or more months of continuous abstinence compared with none in the control group.

CM has been applied and evaluated in relation to all the major substance addictions – cocaine, methamphetamine, opioids, alcohol, marijuana, and nicotine (see Petry, Alessi, & Rash, 2011, p. 233) and is also being applied to problem gambling (Petry, 2010a). At least three independent meta-analyses have found CM to be efficacious in bringing about drug abstinence (Benishek, Dugosh, Kirby, et al., 2014; Lussier, Heil, Mongeon, Badger, & Higgins, 2006; Prendergast, Podus, Finney, Greenwell, & Rolf, 2006) and CM was one of only two psychosocial treatments recommended by the *National Institute for Health and Care Excellence* (NICE) (2007) in the UK for the treatment of problems related to illicit drug use. A review and meta-analysis of psychosocial interventions in general (Dutra et al., 2008) found CM to be the most efficacious type of intervention for substance use disorders.

There have been various criticisms of CM over the years based on ethical, health economic or other practical considerations (see Petry, 2010b). It has also been alleged that the beneficial effects of CM disappear when the provision of reinforcers comes to an end, though there is some evidence of longer-term effects beyond the termination of contingencies (see Petry et al., 2011, p. 235). These objections may have restricted the practical implementation of CM in treatment services across the world (Petry, 2010b) but they are not of immediate concern here. Whatever the ethical or other criticisms and whether or not benefits are only temporary, the evidence on CM strongly supports the conclusion in the previous section that addictive behaviour is operant in nature and not compulsive in the sense of behaviour that is impervious to its consequences. The fact that CM is such an efficacious treatment method gives an indication of the power of reinforcement contingencies, as distinct from the alleged power of compulsive urges, over addictive behaviour.

CM has been used in the treatment of OCD but only for the purpose of increasing compliance with the requirement of treatment, not for addressing the disorder itself (Kircanski, Peris, & Piacentini, 2011). Cognitive-behavioural treatment for OCD is not based on operant principles, the treatment of choice being response prevention.

### 5.2.1. Contingency management in special populations

Even more impressive than the outcomes of CM-based treatment reviewed above are the results of programmes based on CM principles that have been applied to special populations of addicted individuals. As a prime example, *Physicians' Health Programs* (PHPs) that began in the USA over 40 years ago have produced remarkable rates of recovery among physicians addicted to a range of substances who were in danger of losing their licenses to practice (see DuPont, McLellan, White, Merlo, & Gold, 2009). In return for ongoing support, physicians sign five-year contracts agreeing that they will complete treatment, often 12-step based, and submit to intensive random monitoring to ensure they remain abstinent from any use of stipulated psychoactive drugs, including alcohol. If physicians relapse to any use of substances or show other evidence of noncompliance with the programme, there follows an immediate intervention usually consisting of removal from medical practice and placement in extended treatment followed by more intensive monitoring. (For more details of PHPs, see DuPont et al., 2009).

A review of the case-notes of 904 physicians admitted to PHPs, 88% of whom met criteria for a diagnosis of substance dependence, reported that 78% had no positive test for either alcohol or other drugs over the 5-year period of intensive monitoring and that 72% were continuing to practice medicine (Dupont et al., 2009). Such rates of fully successful, long-term treatment outcomes are unheard of in reviews of conventional addiction treatment programmes. A similar case management system has been applied to other professions, including other health-care occupations, commercial airline pilots and lawyers.

An obvious objection to the significance of these high success rates

in PHPs is that physicians are financially well-off and highly educated, with access to the best health insurance; they would therefore be likely to do well in any kind of treatment regime. In response to this objection, [DuPont and Humphreys \(2011\)](#) describe two programmes based on similar CM principles but applied in the criminal justice system (CJS). The CJS population contains some of the heaviest drug users with very poor prognoses, imposing unusually high costs on society. Moreover, while PHPs are expensive, the CJS programmes in question have severe constraints on funding and are similar in this respect to conventional treatment for addiction.

One such programme is *Hawaii's Opportunity Probation with Enforcement (HOPE)* which manages convicted offenders likely to violate their conditions of community supervision. Common drug problems in this population involve smoked crystal methamphetamine and intravenous opioids. In the HOPE programme, probationers are informed by the judge that they will be subject to intensive random drug testing similar to that used by the PHPs. Violations of probation rules, including any drug or alcohol use, missed drug tests and missed appointments, are met with certain, swift and short-term incarceration. Rather than intensive and often prolonged treatment as used in PHPs, in HOPE participants may choose whether or not to attend treatment and only a small proportion do so at the outset. The remainder are monitored without treatment but those who fail monitoring are then referred to treatment. The programme can last up to six years.

The HOPE programme was compared with standard probation in a randomised controlled trial by [Hawken and Kleiman \(2009\)](#). After a year, HOPE probationers were 55% less likely to be arrested for a new crime, 72% less likely to use drugs, 61% less likely to miss appointments with their supervisory officers, and 53% less likely to have their probation revoked than standard probationers (see [DuPont & Humphreys, 2011](#), p. 3).

The other CJS programme based on CM principles is the *24/7 Sobriety Project* in South Dakota. This is aimed at Driving While Intoxicated (DWI) offenders nearly half of whom have three or more DWI convictions and who are therefore likely to include many individuals meeting standard diagnostic criteria for substance dependence. Like the PHP and HOPE programmes, 24/7 Sobriety uses intensive alcohol and drug testing. However, to accurately monitor alcohol use, participants must either undergo twice-daily alcohol breath tests at a local police station or wear continuous transdermal alcohol monitoring bracelets. Participants also undertake regular drug urinalyses or wear drug patches to detect drug use. Any positive test for alcohol or other drugs results in an immediate short-term stay in jail and all missed appointments are immediately followed by the issue of arrest warrants (see [Caulkins & DuPont, 2010](#)).

An evaluation of the 24/7 Sobriety Project found that, among those participants given twice-daily alcohol breath tests, 66.6% were fully compliant with the program requirements, never missing a test or providing a single positive sample; of those subject to transdermal alcohol bracelet monitoring, 78% fully abstained from alcohol use. Recidivism statistics for twice-daily tested offenders showed considerably lower recidivism rates for second time, third time, and fourth time DWI offenders compared to control offenders (see [DuPont & Humphreys, 2011](#)).

According to [Dupont and Humphreys \(2011\)](#), the unique aspects of PHPs and similar programmes, and those responsible for their success, are: (i) they last for years rather than weeks; (ii) they carefully monitor use of alcohol or other drugs; (iii) they employ swift, certain, and meaningful consequences for use and non-use of substances. Such consequences are contrasted with those applying to substance use or other non-compliance in conventional treatment programmes for offenders which are typically unpredictable, slow and harsh. The results from HOPE and 24/7 Sobriety show that CM-inspired programmes can be applied successfully to populations quite different from physicians and with ostensibly much poorer prospects for recovery. The remarkable success rates from all three programmes are incompatible with a

view of addiction that sees addictive behaviour as compulsive and in which relapse is seen as an unavoidable feature of the disorder. The new perspective on addiction treatment based on the CM approach suggests that the key to long-term recovery lies in sustained changes in the environment in which decisions to use and not to use substances are made.

As with the evidence from experimental studies of addictive behaviour, it is very difficult to see how these CM-based programmes could be aimed at the treatment of OCD and achieve anything like the same rates of success; indeed, it would probably and rightly be regarded as unethical to make rewards contingent on the non-appearance of symptoms of OCD.

### 5.3. The flexibility of addictive behaviour

The idea of compulsion implies that addictive behaviour is inflexible, stereotyped, unreflective and unresponsive to changes in personal or environmental circumstances. This is suggested both by ordinary usage of the term 'compulsive' and by theories like that of [Everitt and Robbins \(2005\)](#) that explain addictive behaviour by claiming it to be controlled by the part of the brain responsible for automatic behaviour in the form of S-R habits (see above). But is this depiction of addictive behaviour accurate? We have already seen that evidence on the operant nature of addictive behaviour casts considerable doubt on this depiction of addiction but we can also address the question by examining what addicts tell us about their own behaviour.

[Joanne Neale \(2002\)](#) reported the results from interviews she conducted with 200 drug users, mostly heroin users, in Scotland. The overall picture of addicts' behaviour emerging from this research is very different from its characterisation as inflexible and compulsive. While drug users' lives could reasonably be described as highly structured and narrowly focussed, owing to the demands of obtaining an expensive supply of drugs on a daily basis, the means by which those demands were met were themselves varied and flexible, not to say innovative and highly ingenious on occasion. Rather than helpless victims of forces over which they had little or no control, Neale's respondents were typically self-respecting and self-determining individuals "who actively confronted and purposefully responded to external constraints and life opportunities" (p. 35). This echoes an earlier description of street addicts in New York City as resourceful 'economic entrepreneurs' who remained alert, flexible and resourceful in order successfully to 'take care of business' ([Preble & Casey, 1969](#)).

The idea of compulsion also implies that addicts are obsessively focussed on obtaining and using drugs and that their lives are overwhelmingly dominated by it. However, although most people would consider addicts' daily existence limited in scope and exhausting given the time and effort required to obtain drugs, Neale found that they continued to live ordinary lives in many ways:

Individuals who become addicted to heroin and other opiates do not stop being children, parents, siblings and friends to other members of society. Likewise, some still have business to attend to, hobbies and interests to participate in, and obligations as employees or students to fulfil. Thus, with greater or lesser degrees of success and failure, drug users must juggle the risks and dangers associated with opiate use alongside very ordinary daily behaviour and life roles ([Neale, 2002](#), p. 99).

Neale's description of the realities of drug addiction, based on her meticulous qualitative research, is supported by many ethnographic studies and autobiographical accounts of addiction (see, e.g., [Heyman, 2009](#), Chapter 3). (Ethnographic studies are based on direct, participant observation of behaviour and are not therefore susceptible to any biases that might be present in addicts' self-reports.) However, it most clearly applies to drug-seeking behaviour as opposed to drug-taking. Depending on the kind of addiction in question, the idea of stereotyped and automatic behaviour may apply in some respects to acts of ongoing

drug consumption. ‘Narrowing of the drinking repertoire’, referring to an increasingly restricted selection of places, times and beverages as alcohol addiction proceeds, is an element of the alcohol dependence syndrome delineated by [Edwards and Gross \(1976\)](#); a smoker unthinkingly lighting up another cigarette while one is already burning in an ashtray is a familiar occurrence; and the rituals accompanying intravenous drug injection are well known. If this were all addiction consisted of, then compulsive, stereotyped and automatic might well be appropriate descriptors. Unfortunately for this point of view, it is the *seeking* of drugs or of opportunities for addictive behaviour that is essential to any satisfactory description and explanation of addiction.

It could be that the idea of compulsive addictive behaviour owes much to laboratory studies of non-human animals on which most neurobiological theories of addiction are founded, a point that will be returned to below. As [Stephens and Graham \(2009\)](#) put it: “In the lab, where experimental design restricts the behavioural options for obtaining drugs, it may seem plausible to explain addictive drug-seeking in terms of direct connections between stimuli and stereotyped responses. What constitutes drug-seeking behaviour among human addicts in the wild is seldom so straightforward” (p. 208). [Robinson and Berridge \(2003\)](#), whose own theory of addiction does not require drug-seeking behaviour to be compulsive in the automatic sense, agree:

Many aspects of addictive drug pursuit are flexible and not habitual. Human addicts face a situation different from rats that merely lever-press for drugs. We suspect that if animals were required to forage freely in a complex environment for drugs the picture seen in animal neuroscience might look more like the situation in human addiction, and automatic habit hypotheses would be less tempting. An addict who steals, another who scams, another who has the money and simply must negotiate a drug purchase—all face new and unique challenges with each new victim or negotiation. Instrumental ingenuity and variation are central to addictive drug pursuit in real life... Thus, the formation of S-R habits may explain the rituals addicts display in consuming drugs, but they do not account for the flexible and deliberate behaviors involved in obtaining drugs (p. 34).

Like addicts, the lives of sufferers from OCD are no doubt ordinary in many ways, notwithstanding the interference with daily routines arising from their disorder. But is it realistic to attribute to OCD sufferers the same kind of flexibility and ingenuity in response to the demands imposed on them by their condition, in the same way that heroin users respond to the demands of their addiction? This seems doubtful.

#### 5.4. The natural history of addiction

If addiction entails compulsive behaviour, it must be expected that addicts would take a long time to recover, if they ever did. Linked to the understanding of addiction as a chronic, relapsing disease, compulsion implies that, in medical terms, the prognosis of the condition is bleak, as for other disorders involving compulsion such as OCD. But, since this is presumably an empirical issue, what is the evidence on the natural history of addiction? Does it confirm the postulation of a chronic, compulsive condition with a protracted or lifelong course? Fortunately, the answer to this crucial question is at hand.

Gene Heyman and colleagues have examined data and undertaken re-analyses from four large-scale, longitudinal, psychiatric epidemiological surveys of the general population in the USA carried out at various times since 1980 (see [Heyman, 2009](#), Chapter 4; [Heyman, 2013](#); [Heyman & Mims, 2017](#)). All these surveys used DSM criteria to define substance dependence on licit and illicit drugs and all defined ‘remission’ as an absence of any symptom of dependence during the year before interview. It should also be noted that all these surveys were sponsored by prestigious, government-funded bodies, such as the *National Institute on Drug Abuse* (ironically the most powerful advocate

of the view of addiction as a chronic relapsing brain disease) and were designed and carried out by some of the leading psychiatric epidemiologists in the USA. The evidence gathered by Heyman from this impeccable data-source mostly concerns the description of addiction as a chronic relapsing disease but it is also highly relevant to the related concept of compulsion as the central feature of addiction.

The immediate finding of interest is that, among all participants who had ever met DSM criteria for substance dependence in their lifetime, between 76% and 83% were in remission at the time of the surveys. Further, for the great majority of participants, remission was achieved without the benefit of treatment. It is surely incompatible with the notion of a compulsive, chronic disease that over three-quarters of those who had ever suffered from it no longer did so, and despite never having received treatment.

Heyman then examines possible objections to this finding - reasons that it might be spurious. First, owing to the relapsing nature of addiction, it is possible that many of those who did not show symptoms over the past year would relapse in future, thus reducing the true proportion of those in remission. This is shown to be false simply by the demonstration that relapse rates are roughly stable over time, i.e., as a function of the time elapsed since the first appearance of symptoms of dependence (see [Heyman & Mims, 2017](#), p. 392). It is also possible that rates of remission are biased by the over-inclusion of drugs from dependence on which recovery is thought to be relatively easy, like marijuana. This too is false because, when drug types are separated, remission rates for marijuana dependence were no higher than those for opioids and stimulants. Indeed, a remarkable finding of the analysis is that illicit drugs like opioids and cocaine that are generally reputed to be the most addictive substances available, and therefore the most compulsive, showed significantly earlier remission than alcohol and nicotine which might be conventionally thought to be less addictive. [Heyman and Mims \(2017, p. 391\)](#) calculate that more than half of those who were ever dependent on an illicit drug had remitted by age 30. This is not to deny, of course, that addiction is never long-lasting but it is to assert that protracted histories of addiction are the exception rather than the rule outside the treatment context.

A predictable objection that respondents to the survey were not telling the truth about their dependence status was also contradicted by the existence of relationships between self-report data and other variables that were very unlikely to have occurred if self-reports were invalid. Finally, the possibility that the findings were affected by the existence of a large number of addicts who were missing from the surveys is highly unlikely because, for such a thing to occur, the number of missing addicts would have to have been improbably large (see [Heyman, 2013](#), p. 46).

In view of the clear demonstration by these findings that addiction in the general population is not the chronic, relatively intractable condition it is purported to be, how did such a notion arise? One possibility is that, before the large epidemiological studies Heyman relies on, the majority of statements about the nature and course of addiction were based on treatment samples. Since the seminal work by Robin Room on ‘treatment populations and larger realities’ ([Room, 1980](#)), it is generally accepted by scholars of addiction that addicts in treatment are different in important ways from those in the community. For one thing, they are more likely to suffer from other conditions that increase the chance of a poor outcome. In addition to low personal, social and economic capital ([Storbjoerk & Room, 2008](#)), they are likely to suffer from a range of psychiatric comorbidities, including depression, anxiety states and personality disorders – conditions that are likely to affect the prospects of recovery from addiction and may give the impression that their use of drugs is compulsive, in the sense of impossible to change. On the other hand, addicts in the community seldom seek treatment for addiction. For all these reasons, it is essential to base an understanding of addiction on large, random samples that mirror the demographic and other characteristics of the general population. When this is done, as we have seen, the true

picture of addiction that results is very far from that of a chronic, compulsive disease.

The treatment and non-treatment populations do clearly differ in the extent of comorbidity they show and it is this that Heyman believes is mainly responsible for the poorer outlook among the former (see Heyman, 2009, pp. 82–5). It could still be argued, however, that the longer course of addiction among those in treatment is because they represent the more severe end of the spectrum of severity and are therefore more likely to be compulsive addicts (assuming that there are also non-compulsive addicts). Segal (2017b) has proposed the existence of a subset of addicts, typified by the majority of those who attend *Narcotics Anonymous*, who show more severe levels of addiction than others (p. 452). This subset meets six or seven DSM-IV-TR criteria (American Psychiatric Association, 2000) compared to a less severe subset that meets three to five (Willenbring, Massey, & Gardner, 2009). The drug use of these addicts, claims Segal, is not nearly as responsive to normal incentives as the less severely affected individuals. It is the severe subset that is presumably hypothesised to be represented more heavily in treatment samples and, rather than comorbidity or other factors, accounts for the poorer prognoses of those who receive treatment compared with those who do not. The further implication is that compulsion, in whatever form it is thought to take, applies only or mostly to this subset of addicts. More research on this specific issue would be most welcome but, even if the addiction severity hypothesis were correct, it would not show that compulsion is the essence of addiction, only that it is a feature of its more severe forms. It is even possible that treatment prolongs the course of addiction and delays recovery, as has been suggested in relation to schizophrenia (Whitaker, 2015).

Finally, it could perhaps be argued that, although the course of addiction is much shorter than previously thought, behaviour and experience are still subject to compulsion while it lasts. If so, this would be compulsion of a mild kind since it would not prevent people from recovering from, say, heroin addiction in their late 20s and early 30s.

##### 5.5. How do recoveries from addiction occur?

To consider the validity of the compulsion concept, as well as the natural history and course of addiction, we can consider the nature of recovery – the main factors influencing addicts' changes in behaviour when they give up or radically reduce substance use or other harmful activities. If addiction were compulsive in the sense of being impervious to change, we would expect that, expect possibly for miraculous 'spontaneous' remissions, recoveries would mainly come about as a result of treatment, directed presumably at correcting the neurobiological basis of the compulsion. However, as we have seen, the majority of addicts never receive treatment. In that case, therefore, recovery must be due to extraordinary circumstances involving life events of a sufficiently intense and dramatic quality that the powerful force of compulsive tendencies is able to be overcome.

Is this what we find when we study the factors that predict recovery or when we ask addicts themselves what they believe was responsible for their recovery? When this is done, a quite different story of typical recovery appears. In the alcohol field, it has long been recognised that, while sudden transformations akin to religious conversions do occur, in the majority of cases the correlates of recovery are changes in life circumstances involving marriage, employment, health, and finance (Tuchfeld, 1981). For substance use disorders in general, one of the most common ways in which self-change occurs is by a process described as 'cognitive appraisal' or 'cognitive evaluation', in which the initiation of change is preceded by a process of weighing up the advantages and disadvantages of changing substance use and thereby becoming committed to change (Klingemann, Sobell, Barker, et al., 2001, p. 23). With regard to addiction, Heyman and Mims (2017) summarise the evidence:

In-depth interviews with addicts ... and memoirs ... yield the following correlates of why those who meet the APA criteria for drug dependence stop or greatly reduce drug use as they age: financial pressures, legal pressures, family pressures, hardships associated with pursuing illegal and/or stigmatized activities, drug tolerance, witnessing an overdose, wanting to be a better parent, the desire to make parents proud rather than embarrassed, involvement in a self-help group, an awakened spirituality, a new romantic relationship, the breakup of a romantic relationship, and so on. The material and emotional costs and benefits of everyday life, including existential and value laden self-reflections, are the correlates of remission from addiction.

The question inevitably arises, is the same likely to be true of natural recoveries from OCD? Sufferers from OCD presumably experience the same changes in life circumstances as those described above but this does not appear to improve their poor prognoses.

##### 5.6. The Vietnam veterans follow-up

There is another important body of evidence that, though not relevant to recovery from addiction *per se*, certainly concerns the issue of remission from addiction and how this relates to compulsion. Towards the end of the Vietnam War, the United States government became alarmed about reports that a large proportion of American servicemen in Vietnam were addicted to heroin or other drugs. A team of researchers led by Lee N. Robins was commissioned to interview a large sample of men in Vietnam in order to determine the extent and characteristics of their drug use, and then to follow them up on their return to the US after discharge in 1971, and discover what had happened to them and assess the likely impact on treatment services and American society at large. The results of this investigation by Robins and her colleagues (Robins, Davis, & Goodwin, 1974; Robins, Helzer, & Davis, 1975) were to have profound implications for our understanding of addiction.

In Vietnam, servicemen had easy access to high-quality, inexpensive opiates, with the result that 20% were regular users, with roughly one-sixth of these regular injectors and the remainder either smoking or sniffing. By contrast, only 1% had been regular users of opiates before enlistment. Against all expectations, after their return to the US, the great majority of the sample simply 'gave up' addiction; in the first year after return, only 5% of those who had been addicted in Vietnam were addicted in the US; and despite reports of withdrawal symptoms, fully 88% had not resumed regular use of opiates at a 3-year follow-up point. This did not occur because drugs were unavailable after return home; interviewees reported that they knew how to obtain heroin and some had occasionally, but not regularly, used. The main reasons interviewees gave for this dramatic change in behaviour were that they now considered heroin use 'sordid', high prices, and the fear of arrest. The main predictor of re-addiction was a history of addiction prior to entering the service.

Nor was it the case that treatment after discharge contributed to low rates of re-addiction; only a very small proportion of those addicted in Vietnam entered treatment after returning home and this group had a much higher incidence of relapse than those not in treatment and as high a relapse rate as those in treatment in the civilian population.

Remission did not necessarily involve abstinence. Although nearly half the men addicted in Vietnam tried opiates again after return, only 6% overall became re-addicted. On this point, Robins (1993) comments: "This surprising rate of recovery even when re-exposed to narcotic drugs ran counter to the conventional wisdom that heroin is a drug which causes addicts to suffer intolerable craving that rapidly leads to re-addiction if re-exposed to the drug" (p. 1046).

In case it is thought that the majority of these young men were not really addicted to opiates in Vietnam but were merely casual or 'recreational' users, it should be noted that a subsample who had failed a

well-publicised urine test at departure (see [Robins, 1993](#)), and who were therefore likely to have been frankly addicted, showed re-addiction rates at follow-up of only 14%. Further, as stated, roughly 17% were regular injectors in Vietnam. The fact that these groups showed the same phenomenon of large-scale remission on return home confirms that these findings are relevant to addiction at any level of severity.

There are, of course, several ways in which these remarkable findings might be explained. The most obvious candidate is that high rates of heroin use and addiction in Vietnam had to do with the stress of combat and the hardship of service conditions; once this stress was removed, the escapist attractions of drug effects were removed too. Alternatively, from an economic perspective, the cheap and easy availability of drugs in Vietnam could have been the main factor, and high prices was given by respondents at follow-up as one of the reasons for cessation or marked reduction of use. Or it could have been the absence of cues associated with drug use in the home environment, including the absence of peer example and pressure, that was mainly responsible for change. Whatever the favoured explanation, the findings of the Vietnam veterans follow-up clearly present grave difficulties for any account of addiction with compulsion at its centre and particularly for those accounts in which long-term neurobiological changes are alleged to give rise to compulsive behaviour or experience. If compulsion is an essential characteristic of addiction, why did it disappear, and apparently so easily, when addicted servicemen returned home? Can it be imagined that someone suffering from OCD in Vietnam would recover merely by returning to the US?

### 5.7. Rat Park

In his popular book presenting a radically new perspective on addiction to the general reader, [Hari \(2015\)](#) claims that two studies during the 20th Century have changed, or should have changed, our current view of addiction. The first of these is the Vietnam veterans follow-up which we have just considered; the second is a series of laboratory experiments on rats carried out by Bruce Alexander and his colleagues during the late 1970s ([Alexander, Coombs, & Hadaway, 1978](#); [Alexander et al., 1985](#)), which we will consider now. Largely ignored at the time of their publication, the significance of Alexander's findings is recently becoming widely appreciated.

It is first essential to understand the context of animal research on addiction against which the significance of this work should be seen. Following the ground-breaking report by [Olds and Milner \(1954\)](#) that rats will press a lever, to the point of exhaustion and sometimes death, to obtain electrical stimulation in the so-called 'reward centre' of the brain, [Bozarth and Wise \(1981\)](#), in another famous experiment, showed that rats will show similarly extreme and 'compulsive' lever-pressing to obtain injections of morphine in the same brain region. This was taken by many to be a striking demonstration of the compulsivity of addiction. More generally, the escalation of self-administration of various substances by isolated, caged rats in preference to food and water and, again, sometimes to the point of death, was seen as a neat animal model of compulsive human drug-taking.

What Alexander and his coworkers did in a typical experiment was simply to take so-called morphine-addicted rats out of their isolated cages and put them in a spacious, naturalistic setting, complete with wheels and coloured balls, where they could play, have sex, nest and reproduce. In this situation, the rats mostly preferred plain water to morphine-laced water, even when they visibly experienced withdrawal symptoms and even when the morphine-laced water was sweetened to make it more palatable to them ([Hari, 2015](#)).

There has subsequently been much research, typically conducted under the more controlled conditions favoured by experimenters, that confirms Alexander's main findings. For example, several studies have shown that concurrently available, non-drug alternative reinforcers can significantly decrease cocaine-maintained behaviour in laboratory

animals (e.g., [Carroll, Lac, & Nygaard, 1989](#); [Nader & Woolverton, 1992](#)). [Ahmed \(2010\)](#) has called the fact that, in standard drug self-administration settings, animals have no choice but drug use a 'validation crisis' in animal models of addiction. It will also be obvious that these findings from animal research are perfectly in line with studies of operant behaviour in humans and of contingency management programmes reviewed above. They confirm the basic principle of behavioural economic theory that emitted behaviour is a function of the alternative behavioural options (see [Vuchinich & Heather, 2003](#)).

It is tempting to see the Rat Park experiments as a metaphor for the relationship between rates of human addiction and social and economic deprivation; there is good evidence that addiction, together with mental health problems, is associated with low socioeconomic status (see [Pickard & Ahmed, 2017](#)). For marginalised people from underprivileged backgrounds the benefits of drug use may seem to exceed the costs on a realistic evaluation of life circumstances and the limited alternatives available to them. While evidence clearly shows that, in the natural environment, the majority of those addicted to illicit drugs mature out in their early 30s (see above), more chronic addiction is shown mainly by those who also suffer from comorbid psychopathology and who lack personal, social and economic capital. Be that as it may, the evidence from the Rat Park and other experiments shows that the idea of compulsion in addiction is extremely problematic even in nonhuman animals.

### 5.8. The nature of addictive relapse

There would be wide agreement among those involved in treating addiction that the main difficulty lies not in initiating the desired behaviour change but in preventing relapse after the client's change in behaviour has been made ([Marlatt & Gordon, 1985](#)). At first sight, the statement that addiction is a relapsing condition might seem to conflict with data summarised above on high rates of natural recovery and 'maturing out' in the common addictions (see [Heyman, 2009, 2013](#); [Heyman & Mims, 2017](#)). This problem is more apparent than real. Though rates of natural recovery are much higher than would be expected from a 'chronic brain disease' perspective and though many addicts do mature out at relatively early ages, they may still experience great difficulty and a number of failures before doing so. With regard to smoking, for example, [Borland, Parros, Yong, Cummings, and Hyland \(2012\)](#) found that about 40% of smokers in four English-speaking countries reported making a quit attempt each year and over two attempts on average. Their data suggest that, by the age of 40, the average smoker may have made 25 failed quit attempts (see [Borland, 2014](#), p. 41). It is part of the folk wisdom on addiction that, while many eventually succeed, smoking and other addictive behaviours are very difficult habits to break.

For theoretical purposes, craving and relapse are often considered to be the two cardinal features of addiction that require explanation (e.g., [Robinson & Berridge, 1993](#)). It therefore behoves any theoretical account of addiction, such as the proposal that compulsion is the key pathology in addiction, that it can contribute to an explanation of relapse. We can ask, then, whether it makes sense to think of relapse to addictive behaviour after a substantial period of abstinence or controlled use in terms of a compulsion to re-engage in the behaviour.

The first difficulty such an account faces is evidence that some relapses are planned, in the sense of a decision being made to resume use at some specified time or event in the future. The most obvious example of this is the well-documented phenomenon of someone deliberately stopping intake of disulfiram some time before they have decided to resume drinking ([Liskow, Nickel, Tunley, Powell, & Penick, 1990](#)). (Drinking within a week of stopping disulfiram is likely to result in the highly unpleasant disulfiram ethanol reaction [DER] and it is presumably awareness of this that led these individuals to plan their relapse for when the chance of the DER had receded.) In addition to this example, [Saunders and Allsop \(1989, p. 18\)](#) mention other instances of

planned relapse and also reports of relapse in which people chose not to deploy skills that were in their repertoire and which they had previously employed successfully to cope with high-risk relapse situations.

While these examples cause problems for a compulsion account of addictive relapse, they clearly represent only a minority of relapses. Unfortunately, there seems to be little research or analysis in the literature that could be used to estimate the relative contributions of non-conscious and automatic influences on the one hand and conscious decision-making on the other to the relapse process. However, here is a quote from a leading researcher on nicotine addiction:

For smoking at least, most relapses are to some degree deliberative (executive choices). A small percentage start out with automated smoking but even here the decision to not recover is a deliberative one. In most cases it is a choice made after a struggle, but in others the person feels worn down and decides it is not worth the effort, so decides to give up or to put themselves in a position where they think they will slip (to provide an excuse). This is because for nearly all of us persistent self-control in the face of challenge is just too hard (Borland, 2016).

Certainly, in the most influential model of the relapse process and relapse prevention treatment by Marlatt and Gordon (1985), while it is possible that a lapse (i.e., the initial use of a substance after abstinence or moderation) may be partly precipitated by an automatic response to conditioned cues, the transition from lapse to relapse involves cognitive processes in which the decision to continue use is mediated by the individual's outcome expectancies for the effects of the substance and the 'abstinence violation effect'. The main role for non-conscious processes in relapse may be the possible existence of 'apparently irrelevant decisions' - those decisions made without conscious awareness that the resulting behaviour renders the occurrence of a high-risk situation more likely and a relapse more probable (Marlatt & Gordon, 1985, p. 49 *et seq*). Unfortunately again, though based presumably on clinical insights, no research appears to have tested this hypothesis.

An influential reconceptualisation of the relapse process by Witkiewitz and Marlatt (2004), concluding that it is more complex and unpredictable than previously thought, could be interpreted as allowing a more prominent role for automatic processes but, once more, subsequent theory and research have failed to clarify this issue. Another possibility is the existence of 'absent-minded relapses' postulated by Tiffany (1990) (see above) in which relapse occurs automatically without the conscious awareness or intention of the person concerned. Again, however, there seems to have been no research that might throw light on this possibility. The conclusion can only be that evidence on the nature of relapse causes difficulties for the validity of the compulsion concept in addiction, at least for the strong version of the concept appealing to automatic processes.

### 5.9. Addicts' self-reports

Much of the evidence taken to support the existence of compulsion in addiction is from the self-reports of addicts who say that they can't help behaving the way they do, can't resist the temptation to use drugs or engage in other addictive behaviours, etc. This evidence is often used by proponents of the compulsion concept to bolster their case for its essential role in addiction. For example, in referring to the effects of damage to the orbitofrontal cortex in laboratory animals, leading to perseveration and resistance to extinction of reward-associated behaviours, Volkow and Fowler (2000) say, "This is reminiscent of what happens to drug addicts who frequently claim that once they start taking the drug they cannot stop even when the drug is no longer pleasurable" (p. 319). As Pickard (2016) has pointed out, however, 'can't' can have multiple meanings:

When addicts say they "can't" resist, they may not be claiming that it

is literally *impossible for them to resist* but rather that abstinence is very difficult, and that the costs of foregoing drugs are high, and the benefits of using drugs are many ... the testimony of addicts, like other forms of self-report, is a complicated form of evidence for use in theorizing (p. 457, italics original).

In similar vein and as an illustration of this point, Heather (1998) writes:

If you ask someone to a party and they reply that they can't come, they presumably mean that other commitments, duties, obligations etc. prevent them from coming. They do not mean that they are physically unable to attend or, with any useful meaning, that they experience a compulsion to do something else. They mean that, given the prevailing circumstances they feel obliged to choose, perhaps very reluctantly, not to come. This is surely what drug users mean when they say they can't resist taking drugs. However painful or difficult it may be to choose not to take them, this remains nevertheless a possible option and one which addicts do sometimes choose and maintain (p. 6).<sup>7</sup>

An ingenious experiment by Davies and Baker (1987) is relevant to this issue. They gave a sample of 20 adult male heroin users in Glasgow two parallel forms of a questionnaire separated by 10 to 14 days. The questionnaires were typical of those used in studies of drug-taking behaviour and included items on drug consumption, attitudes towards drug-taking, and reasons for drug use. The first interview was carried out by a 26-year-old locally-known male heroin user who presented himself as having been recruited by a nearby university to help carry out interviews; the second was conducted by a 40-year-old 'straight' (non-drug-using) interviewer who presented himself as a researcher from the university. Subjects received a different version of the questionnaire on each occasion and the two forms were presented as belonging to two separate studies with no connection with each other.

The results of this experiment showed that regular heroin users consistently presented themselves as heavier users and as being more addicted when interviewed by the 'straight' interviewer than when interviewed by the known heroin user. In the latter case, they gave the impression of having more choice and greater control over their drug use. The differences between the two sets of interview responses were substantial and clearly showed that people who use heroin will present themselves very differently depending on the person interviewing them and the circumstances in which they are interviewed. This not to imply that these drug users were telling lies to one interviewer and not to the other but it is to assert that, "People are able to construct their explanations on the basis of their knowledge and experience of the attributions that others are likely to make about them; and when this occurs, attribution may be said to have a strategic component" (Davies, 1997, p. 122).<sup>8</sup> The main point for present purposes is simply that self-reports of addicts that they are or feel compelled to engage in addictive behaviour should be taken with a sizeable pinch of salt. More research involving deeper investigation of the meaning of addicts' self-reports and further conceptual analysis of such reports would be useful. Meanwhile, it would be instructive to carry out a similar experiment with OCD patients, with interviews conducted by a psychiatrist in a white coat or by a fellow sufferer. Although some differences in reported behaviour and experiences might emerge, it is surely highly unlikely that they would differ sufficiently in a way that would have implications for how the condition should be described and understood.

<sup>7</sup> I am grateful to John B. Davies for suggesting this example to me.

<sup>8</sup> Other research by Davies and his colleagues supports the view that what people say about drug use very much depends on whom they are talking to and in what context rather than reflecting any underlying objective state, neurophysiological, socio-psychological, or otherwise. See Heather & Segal (2015, p.79).



## 6. Discussion

There will now be an attempt to integrate the material in the various sections of this article - to put together the lessons that can be derived from various definitions and characterisations of compulsion, from the comparison of compulsion in addiction and in OCD, from the roles compulsion plays in neurobiological theories of addiction, and from the various strands of evidence that seem *prima facie* to cause difficulties for the idea that compulsion is an essential feature of addiction.

### 6.1. Yawning gaps in the literature on addiction

The first and unavoidable conclusion from this exercise is there exists a huge gap between, on the one hand, accounts of compulsion in the medical literature on addiction as a disease and in neurobiological theories of addiction and, on the other hand, evidence based on direct observation of addicts' behaviour, both experimental and naturalistic, participant observation and addicts' self-reports of their addictive behaviour and recoveries, epidemiological evidence on the course of addiction, and other lines of evidence with similar implications. This large and varied body of evidence shows that addictive behaviour cannot reasonably be described as compulsive *at the time it is carried out*. And it is this evidence that seems to be entirely neglected in the disease and neurobiological literatures. This is the 'simple and straightforward sense' of compulsion that was referred to above as being contradicted by the evidence reviewed. This is not to say that it would necessarily be impossible to accommodate such evidence in some kind of psychiatric or neurobiological account of compulsion, only that it must be a continuing obstacle to progress in understanding addiction from a disease/neurobiological perspective that the evidence in question should continue to be ignored.

A related gap is the familiar one between accounts of addiction based on nonhuman animals and those based on human participants. There are good reasons to believe that depictions of compulsion in addiction derived from the behaviour of rats in confined and isolated cages with lever-pressing as the only response option available to them bear little resemblance to human addictive behaviour in the real world. The same can even be said of the behaviour of rats themselves when they are freed from those confining conditions and placed in environments more like those their fellow creatures inhabit in the wild, as demonstrated by the rightly celebrated experiments by Bruce Alexander and his colleagues (Alexander et al., 1978, 1985). Again, no depiction of compulsion in addiction worthy of serious consideration can afford to brush these contrasts aside.

Yet another gap in the literature lies between accounts of compulsion founded on the presumed functions of areas and circuits in the human brain, whether based on neuroimaging or otherwise, and compulsion as behaviour or experience. It must always be remembered that compulsion is a behavioural and/or experiential phenomenon, not some aspect of brain activity. Arguments for compulsion in addiction in neurobiological theories, although differing considerably as we have seen between different theories, are all based on changes in brain function after chronic ingestion of psychoactive drugs (or perhaps longstanding engagement in some non-drug activities like gambling) but that is not enough to warrant the term compulsion being applied to those changes (cf. Stephens & Graham, 2009). On this point, Heyman and Mims (2017) write:

The proper question is not whether drugs change the brain, but whether they change the brain so that drug use is no longer voluntary. This is a behavioural question that can only be answered by studying the natural history of drug use. To determine whether drug addicts are compulsive drug users, we need to know what influences drug use in those who meet agreed-upon criteria for addiction. If the factors are similar to those that affect voluntary actions, then drug use in addicts remains voluntary, albeit irrational and self-

destructive (p. 389).

One would only add that other types of evidence are relevant to this issue besides natural history and that addicts' reports of their experience as well as their behaviour must be taken into account in evaluating the utility of the compulsion concept.

### 6.2. Many meanings of compulsion

Another obvious lesson from the material covered is that there are many meanings of compulsion in the literature and that Segal (2017a) is correct to say that different authors mean different things by the term. This is shown by differences, even within the same neurobiological theory, between the strong sense of compulsion, referring to an automatic force outside the agent's motivational resources, and what we have termed here the weak sense, referring to the effects of powerful desires and temptations. This is a distinction based in ancient philosophical writings and replicated by definitions in modern dictionaries; it represents an important conceptual clarification of uses of the term, compulsion for scientific purposes (Stephens & Graham, 2009; Wallace, 2003; Watson, 1999) but one that has yet to be taken fully on board by scientists and scholars interested in addiction. (It is possible, of course, that both kinds of compulsion apply simultaneously to addiction but, if so, this should be clearly stated.)

Beyond strong and weak versions, this article has revealed many other possible meanings of 'compulsion' and 'compulsive' as applied to addiction and some of them will be examined below. There are also meanings that have not yet been mentioned and some of these will be briefly discussed in what follows. Perhaps even worse than confusion and inconsistency in what compulsion means is a failure to specify what it means at all. This charge can be levelled at some authors, both in the disease and non-disease camps, who seem to employ the term only because it is a conventional way of characterising addiction but without any further meaning than that. The clear conclusion from all this is that scientists and scholars who apply the term compulsion to addiction, either as an explanation or a description (see below), should clearly specify what they mean by reference to some property of addictive behaviour or experience. This applies to both disease and non-disease accounts of compulsion.

### 6.3. Compulsion in addiction and in OCD

A conclusion from the material covered in this article is that, whatever meaning can justifiably be given to compulsion in addiction, it should bear little resemblance to compulsion in OCD. The observations made on this issue by Caetano (1985) over 30 years ago have been substantially confirmed by the review of evidence here relevant to the question whether compulsion is a property of addictive behaviour and experience and the further question whether this evidence could be said to apply to compulsion in OCD, i.e., whether sufferers from OCD would be likely to show the same apparently voluntary behaviour, or changes in behaviour, in response to experimental or naturalistic conditions as addicted individuals do. The answer is clearly no. This is important because it is sometimes asserted, without much evidence or argument to support it, that compulsion in addiction and in OCD are manifestations of the same underlying biological or psychological process. We have encountered very little support for this hypothesis and many reasons to doubt it.

### 6.4. So is addiction just an ordinary choice?

A great deal of evidence of different kinds was reviewed above to show that addictive behaviour cannot reasonably be described as compulsive at the time it is carried out. A possible deduction from this is simply that, rather than being compelled, so-called addictive behaviour represents a free choice just like other, ordinary, everyday

choices; indeed, this deduction has been made (e.g., Schaler, 2000). This view would be based directly on the copious evidence that addictive behaviour was operant behaviour and therefore voluntary, that treatment based on the principles of contingency management was probably the most successful ever documented and, perhaps more indirectly, on the other types of evidence summarised. It could even be further deduced that, since what is called addictive behaviour is not compelled but voluntary, addiction itself is a myth (Davies, 1997).

This does not follow from the evidence presented and is certainly not the position taken in this essay. Given the long history of the topic of addiction and the countless reports people give of problems they experience in regulating their behaviour in relation to the consumption of substances or engagement in activities, it is inconceivable that addictive choices are just ordinary choices and that no puzzle exists to warrant a special term like addiction or dependence. Ordinary choices people make are sometimes, perhaps often, irrational but addictive behaviour can be so extremely irrational and so self-destructive that it is difficult to believe that the choices leading to it are ordinary ones. Anyone who has worked in services aimed at helping people with addictive disorders knows that their clients have typically resolved on numerous occasions to desist from an unwanted behaviour but have failed time and again to do so (see Heather, 2017a). This simple observation alone is sufficient to show that addictive behaviour exists and that addictive choices are not ordinary choices.

Now, how this brief justification can be reconciled with the evidence presented above demonstrating the voluntary nature of addictive behaviour at the time it is carried out is another question and one which is beyond the remit of the present article fully to attempt to answer. Suffice to say that this is the major issue facing addiction studies at the present time and one to the possible solution of which a recent collection has been dedicated (Heather & Segal, 2017). The truth about addiction lies somewhere between the extremes of free choice and no choice but where exactly that place is, and how it should be described, is the crucial issue. To provide one sketchy possibility, it could be that addicts respond to incentives and so are free to choose to use or not to use at any one time, but that their autonomy is impaired when their pattern of choices is considered over time. From this perspective, addiction is marked by dynamic inconsistency and is a disorder of choice in the sense of a failure to make consistent choices over time (see Heather, 2017b, p. 465; Levy, 2006).

Another interesting conjecture, and one with similarities to the above, has recently been proposed by Noggle (2016). This begins with the idea of ‘ego depletion’ (Baumeister, Bratlavsky, Muraven, & Tice, 1998), according to which willpower is a limited resource that becomes depleted by use; we can resist temptation for a while but not indefinitely. Applied to addiction, the implication is that addictive craving and urges persist long enough to deplete the addict’s willpower, leading to relapse (cf. Levy, 2006). Thus drug cravings are irresistible, not in the obvious sense that they cannot be resisted at any one time but in the weaker sense that the ability to resist is worn down and eventually fails over time (Taylor, 2005). Noggle’s original contribution is to suggest that the persistence of the desire to consume drugs among addicts is a significant form of dysfunction in its own right and one that makes an important contribution to compulsivity. More specifically, addiction involves dysfunction in a mechanism that normally prevents a person from being tempted to consume drugs “in situations where such consumption poses a grave, obvious, and imminent danger to things that they care about—things like their careers, their family, their health, their freedom, their self-respect” (p. 218). When desire to consume drugs in such situations occurs in a non-addict, a ‘quashing mechanism’ usually either blocks the formation of the motivation to consume or eliminates it soon after it forms. By contrast, the addict’s motivation to consume drugs often persists even when it is obvious that consumption invites dangerous or repugnant consequences. Noggle summarises his hypothesis as follows:

... the apparent compulsivity of addiction is a product of three main facts: First, due to the effect of chronic drug use on the dopamine-based reward system, the addict experiences a motivation to consume drugs that is abnormally strong, though not so strong as to be strongly irresistible. Second, these abnormally strong motivations to consume often fail to be quashed, so that the addict continues to be tempted to consume even in situations where doing so poses a high risk of disaster. Third, the ability to resist a persistent motivation—especially a strong one—is limited (p. 222).

Noggle’s (2016) ideas are of interest here because they exemplify an account of compulsivity that concedes that drug use in the addict can be voluntary at the time it is carried out, and that drug craving need not be irresistible, while at the same time suggesting a mechanism by which addictive behaviour, and relapse, becomes more likely over time. If validated by further analysis and research, it may be a sense of compulsion with use in the understanding of addiction.

### 6.5. The minimal version of compulsion in addiction

In the Introduction, we encountered a definition of compulsion that relied only on the fact that addicts continue to show diminished ability to control drug use even in the face of adverse consequences that should motivate cessation “in a rational agent willing and able to exert control over behavior” (Hyman, 2007, p. 2). This was therefore dubbed a minimal version of compulsion in addiction. In similar vein, Everitt (2014) writes:

It is indeed compulsive drug use that is widely seen as a core aspect of addiction, and by that is meant repeated, persistent use, despite placing an individual in danger, compromising their health, family and social lives (p. 6).

This minimal definition seems to be a restatement in other language of the central puzzle of addiction that people continue to behave in ways that are bad for them or, more pertinently, in ways they *know* are bad for them. It does not get us any further in understanding or *explaining* this puzzle, or even in approaching an understanding of it, merely to call the behaviour in question compulsive.

Even as a description of compulsion, this minimal definition seems no advance on the traditional and *more* descriptive term ‘loss of control’ or, at least, the more refined version, ‘impaired control’, including Edwards’ (1982) control that is “variably or intermittently impaired rather than lost”. Further, as Heyman and Mims (2017) point out: “Modern, research-based choice theory predicts semi-stable suboptimal outcomes. Thus, it is possible to explain why addiction is self-destructive, yet significantly correlated with its consequences” (p. 403). The fact of self-destructive consequences of drug use is not inconsistent with the assumption that addicts make choices; after all, we speak naturally of ‘self-destructive choices’. Choices need not be rational but the task is to explain *why* addicts’ choices are self-destructive, even variably or intermittently so. In short, in their definitions above, Hyman (explicitly) and Everitt (implicitly) confuse compulsion with irrationality. The conclusion is that, to be useful, the concept of compulsion in addiction needs to refer to something more than the fact of self-destructive addictive behaviour.

### 6.6. Could compulsion apply to some addicts and not to others?

In a section on the nature of recovery from addiction above, we encountered the suggestion that there may be a subset of individuals labelled as suffering from addiction to whom the concept of compulsion was applicable and another subset to whom it was not or, at least, in whom it was present in a milder form. The principal difference between these subsets was thought to be the severity of their addiction, assessed for example by the number of DSM-IV-TR criteria they met (Segal, 2017a). If valid, such a distinction might explain the difference between

the low rates of recovery shown by addicts in treatment compared with much higher rates in those in the natural environment who had not received or sought treatment. This hypothesis would have to be compared with an alternative that invoked the higher level of psychiatric comorbidity in the treatment than in the non-treatments populations (Heyman, 2009). Lastly, the existence of the less severe subset could explain why diagnosed addicts appeared responsive to normal incentives in laboratory experiments, the assumption being that such addicts did not show the severe form of addiction that involved compulsion. With regard to the success of contingency management programmes, Noggle (2016) has pointed out that, while 80–90% of the professionals treated in the PHP and similar programmes successfully abstained, 10–20% did not “even though they knew that consuming threatened careers in which they had made great personal investment” (p. 214). It is possible that this minority typified the more severe kind of addict for whom compulsion to use rendered incentives for abstinence less effective.

It should be remarked that the hypothesis of a more severe subset of addicts who show compulsion contradicts the well-known WHO (1969) definition in which compulsion is said to be *always* present. This raises the possibility that the less severe subset without compulsion or with a less destructive form of it, if it exists, should be labelled as suffering from some milder disorder than addiction. The hypothesis might also have difficulty with other kinds of evidence reviewed in this article, particularly that from the follow-up of Vietnam veterans. However, the main point in bringing these observations forward here is simply to suggest that research is needed into these important speculations.

### 6.7. Compulsion as an explanation of addiction

We turn now to those definitions and meanings of compulsion in which it signifies something more than continued use despite negative consequences, i.e., *compulsive* use despite negative consequences. In particular, we shall focus on the two senses of compulsion that have been identified in this essay – the strong version in which compulsion is seen as an example of automatic, involuntary behaviour following repeated learning experiences and the weak, motivationally-based version in which it is seen as resulting from a failure to resist abnormally strong desires to engage in addictive behaviour. Further, we shall consider each version of compulsion both as an explanation and as a description of addiction, beginning with explanation here.<sup>9</sup> This will be applied both to addictive behaviour and addictive experience. Lastly, in each case we will evaluate the usefulness of both the strong and weak versions of compulsion. The intersections of explanation or description, behaviour or experience, and strong or weak kinds of compulsion are shown in Table 2, together with a rough verdict in each of the eight cells.

#### 6.7.1. Explanations of addictive behaviour: strong version

Recall that the strong version of compulsion in addiction refers to a force outside the person's motivational states, like Aristotle's wind that blows someone along the street or like being thrown bodily out of a night club by a bouncer (cf. Watson, 1999). As Stephens and Graham (2009) say, we should think of an internal state analogous to such forces as what is sometimes proposed as compulsion in addiction – something pictured “as bypassing the will altogether, causing us to do things that do not satisfy the minimal conditions of voluntariness” (Wallace, 2003, p. 424).

The outstanding example of such an explanation is by Everitt and Robbins (2005), summarised above, in which addiction is said to

<sup>9</sup> At the risk of stating the obvious, an explanation in science differs from a description in that it claims to demonstrate a cause for the phenomenon in question, while a description makes no such claim.

**Table 2**  
Verdicts on usefulness of concepts of compulsion as explanations or descriptions of addictive behaviour and experience in strong and weak versions (see text).

	Explanation		Description	
	Strong version	Weak version	Strong version	Weak version
Behaviour	Could perhaps explain the nature of drug consumption but is contradicted as an explanation of drug-seeking and relapse by evidence on the reality of addictive behaviour.	More plausible but in need of modification in the light of evidence on addictive behaviour and recovery, after which it would not be clear whether ‘compulsion’ was still a useful term.	Is descriptive of aspects of drug consumption and engagement in addictive activities by addicts but contradicted by evidence as a description of drug seeking and relapse.	Not applicable.
Experience	Contradicted by evidence.	If the theory is valid, it could explain the addict's feeling of being driven and the associated bewilderment.	A feeling as if compelled could arise as a <i>post hoc</i> rationalisation of non-rational behaviour.	Abnormal urges, desires, cravings, etc. to engage in addictive behaviour are often very difficult for addicts to resist but never impossible.

consist of non-conscious, involuntary behaviour formed by overlearned S-R habit connections and under the control of the part of the brain (the dorsal striatum) that regulates automatic behaviour like car-driving by an experienced driver. An analogy is with persistent or constant re-initiation of skills like playing the piano or tying one's shoelace.

This explanation is flatly contradicted by the evidence on addicts' actual, as opposed to stereotyped and imagined, behaviour examined above. This is most obviously true from accounts by Neale (2002) and many others of the flexibility, ingenuity and sheer ordinariness of addicts' behaviour in the real world. It can also be inferred from the flexibility of behaviour shown by the alcohol addicts in the laboratory experiments by Mendelson, Mello and others and, overall, by the large volume of research of different kinds showing that addictive behaviour and recovery is responsive to environmental conditions. As conceded above, repetitive and seemingly 'automatic' behaviour might apply to acts of consuming substances or engaging in addictive activities but even that depiction may have been exaggerated and, in any event, it is easy for someone to become aware of such habitual behaviour and change it if necessary (Wallace, 2003). Lastly, relapse to addictive behaviour after periods of abstinence or moderation, which is the main obstacle to permanent behaviour change, is unlikely to be due to automatic processes. Thus, this theory cannot account for drug-seeking or relapse in addictive behaviour. The same inadequacies apply to other theories, such as those of Koob and Le Moal (2001) and Goldstein and Volkow (2002), that propose similar kinds of automaticity as explanations of drug-seeking behaviour and relapse.

#### 6.7.2. Explanations of addictive experience: strong version

Since the strong version of compulsion has just been shown to be inadequate as an explanation of addictive behaviour, it follows that it is inadequate as an explanation of addictive experience too. It makes no sense to imagine an extrapersonal and non-conscious mechanism that does not affect one's behaviour but somehow affects one's experience. However, the strong version of compulsion may have some relevance to the description of addictive experience (see below).

#### 6.7.3. Explanations of addictive behaviour: weak version

To recap, the weak version of compulsion in addiction refers to the effects of strong desires or impulses etc. to which the addict is subject. As an explanation, therefore, the proposal is that powerful desires, urges, cravings or other motivating feelings or sensations cause addicts compulsively to carry out addictive behaviour contrary to their better judgement, against their considered evaluations or, in short, against their wills. To distinguish it from the non-motivational kind of compulsion we have been considering above, Watson (1999) calls this simply 'motivational compulsion'.

The outstanding example of a theory that invokes this kind of compulsion to explain addictive behaviour is Robinson and Berridge's (1993) incentive-sensitisation theory. As we saw above, a neuro-adaptive process known as incentive sensitisation is hypothesised to transform ordinary desires for drug experiences into drug craving and is also responsible for relapse to drug use after abstinence. This 'drug wanting', as opposed to 'drug liking', results in compulsive drug-taking and -seeking even when the expectation of drug pleasure is reduced and even in the face of strong disincentives. This hypothesised process clearly fits the requirements of the motivational kind of compulsion described in the philosophical literature (e.g., Stephens & Graham, 2009). There is a slight complication here in that incentive sensitisation itself can be implicit, outside the addict's awareness, and it might therefore be thought to be an automatic process similar to that described by Everitt and Robbins (2005). This would be a mistake, however, because, although often non-conscious, incentive sensitisation is part of the person's motivational inclinations and not linked to learning and habit-formation.

This is not the place to attempt an overall evaluation of the scientific validity of Robinson and Berridge's theory but a few remarks can be

made about the way it handles the issue of compulsion. The theory seems to explain well some of the more curious aspects of addictive behaviour, notably the excessive preoccupation with the object of addiction, the fact that the behaviour is continued when the addict no longer gains much pleasure from it, and why relapse sometimes occurs after many years of abstinence. Most importantly, we know that addicts do experience intense desires for the effects of drugs or activities, although whether such desires can coherently be said to cause addictive behaviour is uncertain (see Wallace, 2003). Nevertheless, some variant of the motivational basis for compulsion seems far more plausible as an explanation than the strong, automatic version. However, the theory is still embarrassed, like the other neurobiological theories, by the evidence reviewed in this article, particularly the demonstration of preferences for alternative reinforcers, including those that convey apparently quite small rewards, over drug consumption and the much shorter course of most histories of addiction in the natural environment than would be predicted by semi-permanent brain changes brought about by incentive sensitisation.

Whether or not the theory can be modified to take account of this evidence is hard to say. At the very least, it should recognise that the causal link between drug wanting/craving and use/relapse is far from inevitable and depends on environmental and situational variables to an extent not yet captured by the theory. If such changes to the theory were made, it may not be useful to continue to use the term 'compulsion' to describe the cause of addictive behaviour.

A more general version of the idea that the weak form of compulsion causes addictive behaviour is based on the well-attested phenomenon of conditioned craving, first studied by Wikler (1965). Stimuli associated with persistent drug use, such as the setting in which use occurs, the people associated with it, drug-using paraphernalia or internal mood states preceding use, acquire conditioned properties and the re-occurrence of such stimuli then motivates drug-taking. O'Brien, Childress, Ehrman, and Robbins (1998) maintain that the chief significance of conditioned craving for compulsive behaviour is its role in relapse after withdrawal symptoms have subsided. More recently, Childress, Ehrman, Wang, et al. (2008) published the first evidence that brain reward circuitry responds to conditioned drug cues presented outside awareness and believe that this "may represent a potential vulnerability in disorders (e.g., the addictions) for whom poorly-controlled appetitive motivation is a central feature" (p. 1506).

There is no question that conditioned craving occurs and that it plays a part in the process of relapse. However, as we have seen, there is evidence that relapse also involves a deliberative choice. The occurrence of conditioned craving does not lead inevitably or on its own to relapse and should not therefore be construed as a form of compulsion.

#### 6.7.4. Explanations of addictive experience: weak version

If the Robinson and Berridge (1993) theory is a valid explanation of addictive behaviour, then incentive sensitisation and the resulting drug wanting could cause the addict's experience of feeling driven to continue drug use or other addictive behaviour without understanding their motivation for doing so and with an accompanying sense of bewilderment. It is also possible that conditioned cues occurring outside awareness could contribute to the same effect. Again, however, it would have to be further explained why the feeling of being driven did not always, depending on the circumstances, result in the addictive behaviour being carried out.

#### 6.8. Compulsion as a description of addiction

Accurate descriptions are important because they form the data on which explanations of phenomena are built. So, if addiction is proposed as a disorder of compulsion, as opposed to a disorder of choice, descriptions of the compulsive nature of addictive behaviour and experience must be veridical.

### 6.8.1. Descriptions of addictive behaviour: strong version

The verdict here is the same as for explanations of addictive behaviour from the strong version of compulsion and for its representation in neurobiological theories of addiction. Automaticity is descriptive of the ritualised and ‘habitual’ aspects of the behaviour of drug addicts and those addicted to non-drug activities. However, it is clearly contradicted by observations of addictive seeking behaviour under laboratory and naturalistic conditions and by addicts’ self-reports of their behaviour and recoveries. It is further inconsistent with the natural history of addiction inferred from random surveys of the general population and from follow-ups of drugs users such as the Vietnam veterans follow-up.

### 6.8.2. Descriptions of addictive experience: strong version

We saw above that [Everitt and Robbins \(2005\)](#) likened the persisting quality of automatic S-R habits to a subjective state of ‘must do!’ and went on to say that “this subjective response could arise *post hoc* as a rationalisation of the ‘out-of-control’ habitual behavior rather than being the driving influence” (p. 1485). This raises an interesting possibility. In a discussion of the work of the American philosopher, Donald Davidson and of the idea that addiction can be fruitfully seen as a form of *akrasia* (i.e., weakness of will), [Heather and Segal \(2015\)](#) end by repeating Davidson’s conclusion that the *akrates* (and therefore the addict) does not have a reason for preferring the akratic action to action based on an all-things-considered judgement about what it would be better to do; she cannot therefore tell us or herself why she broke her resolution to refrain from behaviour she knows to be ill-advised. Heather and Segal continue:

This (not having a reason for the breakdown of a resolution) may also represent a sense in which the addict reports feeling compelled to engage in addictive behaviour—the subjective sense of not being able to understand one’s past behaviour and therefore feeling that one must have been driven by some extrapersonal force to carry it out. If one cannot understand why one has repeatedly done something one would rather not have done and, on the basis of past evidence, is likely to continue doing, then one is likely to feel that one was and is compelled to keep doing it (p. 81, parentheses added).

The suggestion is that the addict’s attempt to make sense of her predicament results in her feeling *as if* she had been compelled by some extrapersonal influence to behave additively. This might be called a sort of *post hoc* rationalisation of irrational, or more precisely, non-rational behaviour but, as Everitt and Robbins say, it has no causal force.

A similar kind of feeling *as if* compelled could arise from the addict’s realisation that he has ceased to gain much or any pleasure from his addictive behaviour ([Kennett, Matthews, & Snoek, 2013](#)). If one keeps doing something without gaining any pleasure from it, one will be inclined to think that one is compelled to do it.

### 6.8.3. Descriptions of addictive behaviour: weak version

As a description, compulsion arising from the existence of strong desires, cravings, temptations etc. is not relevant to behaviour but only to experience and this will be dealt with next.

### 6.8.4. Descriptions of addictive experience: weak version

We come at last to what is probably the most relevant sense of compulsion to addiction or, at least, to a description of addictive experience. Since the very beginnings of the disease concept, desires to consume substances or engage in behaviours have been described as ‘irresistible’ temptations or as ‘overpowering’ any resistance the person might put up. We saw above that, in the light of the accumulated evidence on the nature of addictive behaviour, this ‘weak’ version of compulsion was inadequate as an explanation of addictive behaviour and had only limited and speculative relevance to an explanation of addictive experience. We shall consider here how it fares as a

description of addictive experience.

One of the most commonly cited passages in the literature on addiction is the following anecdote from Dr. Benjamin Rush, the so-called father of American psychiatry, reporting the words of one of his patients: “Were a keg of rum in one corner of a room, and were a cannon constantly discharging balls between me and it, I could not refrain from passing before that cannon, in order to get at the rum” ([Rush, 1812](#), p. 266).<sup>10</sup> Other 19th Century anecdotes about inebriety include a man who, while in treatment, secretly drank the alcohol from six jars containing morbid specimens ([Watson, 1999](#), p. 23) and another who reputedly chopped his hand off in order to be given brandy ([Holton & Berridge, 2017](#), p. 153). Such tales are reminiscent of [Volkow and Fowler’s \(2000\)](#) comparison with the hunger of starvation.

Do these anecdotes and analogies give a realistic portrayal of addictive desires? [Watson \(1999\)](#) is sceptical and so is the present author. The 19th Century anecdotes seem fine examples of Victorian melodrama and the more recent analogies appear similarly exaggerated. This is not to deny that addictive desires, particularly after periods of unwanted deprivation, are sometimes powerful, highly distressing if unfulfilled and deserve the label ‘craving’. But such desires cannot be literally irresistible or overpowering because, if they were, “it would be an utter mystery how people ever succeed in overcoming their addictive conditions by exercising strength of will, and yet this seems to happen all the time” ([Wallace, 2003](#), p. 425). It should hardly be necessary to add that the notion of irresistible and overpowering desires is inconsistent with the literature on the realities of addictive behaviour and experience that was reviewed earlier in this article.

From the point of view of their ego-depletion theory, [Baumeister and Vonasch \(2015\)](#) put it this way:

We think the idea of being overwhelmed by irresistible urges is inaccurate in most cases. Instead, there are frequent but weak impulses. Many of these are resisted successfully, but some may eventually be enacted. Frequent or chronic resistance can deplete one’s willpower, so that the addict yields to a weak impulse that he or she would otherwise resist. In those cases, the lapse is caused not by an overwhelmingly powerful impulse but by a temporarily low level of willpower. The well documented patterns of increased relapse during stress or dysphoria could also reflect the fact that the person’s resources have been depleted by coping with the stress or problem (p. 7)

The only point of disagreement here is that, while urges to use may be mild during ongoing addictive behaviour, they are sometimes, depending on the context, much stronger and dangerous for relapse after a period of abstinence, when to use the vernacular the addict is ‘strung-out’. This is especially true if the opportunity to use is perceived as possible ([Wertz & Sayette, 2001](#)). Nevertheless, the main point is agreed that, although desires, impulses, urges, cravings, etc. to engage in addictive behaviour are sometimes extremely difficult and painful to resist, resistance never becomes impossible in principle. It is also agreed with [Spada, Albery, and Moss \(2015\)](#) that “the exercise of volition is not lost to the automaticity and irresistibility of addictive responses, but rather ... can be employed to decide whether to comply and satisfy, or to deny and abstain” (p. 7). These comments are consistent with a dual process theory of addiction (see [Heather, 2017b; Henden, 2017](#)) but a discussion of that is beyond the scope of the present article.

<sup>10</sup> Some authors give the source of this quotation as William James (1890, p. 543). Since the wording is identical, James must have borrowed the quotation from [Rush’s \(1812\)](#) book, curiously without acknowledgment. Harry Levine (1978) was responsible for popularising the quote and its true origins among modern students of alcohol addiction.

### 6.9. The public understanding of addiction

Before ending this exploration of the concept of compulsion in addiction, it is necessary to offer a few remarks on implications for the public understanding of addiction. We saw at the outset of this article that the idea of compulsion served a socio-political function for the disease concept of addiction by persuading policy-makers, opinion-formers and the general public that addicts were not responsible for their troublesome behaviour and should therefore be offered compassion and treatment rather than disapproval and punishment. The public understanding is also important, not only for influencing the extent to which public funds should be allocated to research on and treatment of addiction, but also for the sense family members make of their loved one's otherwise incomprehensible behaviour and for the sense addicts themselves make of their own difficulties.

The philosopher of science, Ian Hacking (1986) has described the existence of 'human kinds' that come about through the classifications made by scientists of human affairs, for example psychiatric classifications. What distinguishes these human kinds from natural kinds is that they have 'looping effects' that change the people who are thus classified. For present purposes and in simple terms, addicts come to believe what is said about them in 'official' pronouncements and then behave accordingly. More generally, everything we know about the effects of 'expectancy' on human behaviour means that it is very difficult to underestimate how behaviour, say addictive behaviour, is shaped by the cultural milieu in which it is embedded and how behaviour can change dramatically when those culturally-based expectations change.

For many years, the social psychologist, Stanton Peele has argued that the disease concept of addiction and the treatment based on it have the effect of reducing addicts' chances of recovery by telling them that they are powerless to change without special help (see Peele, 1985, 2017). Indeed, he believes that treatment founded on the idea that addiction is a chronic, relapsing brain disease, implying thereby the conceptual and treatment goal of eliminating choice in addiction and recovery, is "not only futile, but iatrogenic" (Peele, 2017). Peele sees the future of treatment for addiction as centred on the opposite goal of encouraging choice and empowering people to change:

That people's belief in their capacity to change is the single greatest factor in psychological and behavioural change is one of the underlying principles of the modern cognitive behavioural therapy movement, which has repeatedly been shown to be the most empirically well supported therapy model. Self-empowering addiction treatment is also in line with the modern drug policy reform movement represented by harm reduction. As a movement, harm reduction applies the values of choice, self-respect, and humanity to the millions of people who confront the wide range of addictive proclivities we all face in one way or another (Peele, 2016).

Many contemporary behavioural and social scientists, counsellors and therapists would agree with Peele and there is much theory and research to support his argument. Efficacy expectancies, our belief in our ability to master a specific change in behaviour, are the most important determinant of successful therapeutic and self-initiated change according to the dominant theory of behaviour change during the second half of the 20th Century (Bandura, 1997); increased self-efficacy is a *sine qua non* in the most influential model of relapse prevention (Marlatt & Gordon, 1985); and, more generally, there is a large amount of evidence that theories people have about themselves in which their abilities are seen as malleable and capable of improvement are associated with 'mastery' responses and greater levels of success in life than self-theories in which abilities are seen as fixed and unchangeable, and associated with maladaptive 'helpless' responses (Dweck & Leggett, 1988).

In the present discussion of evidence bearing on the concept of compulsion in addiction, it emerged that, in some meanings of the term,

compulsion could make sense of aspects of addiction and it might therefore seem arbitrary whether or not the term should be retained. Such a conclusion would ignore the impact of the public understanding of addiction and, in particular, the effects of the public-facing language used to describe addiction on addicts' self-concepts and their confidence in their ability to overcome their problems. It seems very likely that the language of compulsion, of automatic responses or of overpowering and irresistible cravings is a strong disincentive to self-change and to the success of treatment aimed at helping people change.

As Heyman and Mims (2017) have recommended, we need to bring about a radical transformation in the public's understanding of addiction, one in which they are persuaded to believe that breaking free from addiction is possible and told what we know about how this can be successfully accomplished.

## 7. Conclusions

The following conclusions from this review and discussion of the usefulness of the concept of compulsion in addiction seem justified:

- (i) There exists a large body of evidence of different kinds – laboratory experiments with human and nonhuman participants, evaluations of treatment methods, randomised population surveys, follow-up studies of addicted persons, qualitative and ethnographic research, studies of relapse, analyses of addicts' self-reports – appearing to show that, at the time it is carried out, addictive behaviour is voluntary behaviour and not 'against the will' of the addicted individual.
- (ii) This body of evidence should not be ignored by scientists and scholars who wish to defend the idea that compulsion is the essential, or even an important, characteristic of addiction.
- (iii) Many different meanings of compulsion in addiction exist in the literature and, while some interpretations of compulsion may have some validity in the explanation or description of addiction, those who use the concept should clearly specify what they mean by it.
- (iv) Evidence and analysis clearly suggest that compulsion in addiction, in whatever form it is thought to take, is different from its manifestation in obsessive-compulsive disorder.
- (v) Although inconsistent on the whole with addiction as a disorder of compulsion, the evidence should not be interpreted as showing that 'addictive' choices are ordinary choices just like any others. The evidence can be accommodated within a theory of addiction as a *disorder* of choice.
- (vi) Philosophical analysis and modern dictionary definitions suggest two different versions of the compulsion concept that have potential explanatory power: a *strong* version in which it takes the form of an automatic, involuntary force outside the person's motivational states, and a *weak* version in which it refers to the effects of abnormally strong desires, craving, or urges that are difficult or impossible to resist.
- (vii) As an explanation of addiction in neurobiological theories (e.g., Everitt & Robbins, 2005), the strong version of compulsion, referring to involuntary, automatic, habitual learned behaviour, might have some relevance to drug consumption but is contradicted by evidence as an explanation of drug seeking and relapse.
- (viii) As an explanation of addiction, the weak version of compulsion referring to the effects of strong desires (e.g., Robinson & Berridge, 1993) seems more plausible but needs modification to account fully for the evidence on addictive behaviour and recovery.
- (ix) Neurobiological theories incorporating either versions of compulsion might be able to explain the addict's feeling of being driven to use drugs, the associated bewilderment and a *post hoc* rationalisation as if compelled to take drugs.
- (x) As a description of addictive experience, the weak version of

compulsion, involving literally ‘irresistible’ temptation and ‘overpowering’ desires, has been exaggerated. Although cravings in addiction are sometimes powerful and very difficult to resist, they are never impossible to resist in principle. The fact that some addicts *do not* ever change their addictive behaviour is not evidence that they *cannot* ever change it.

- (xi) Although in some senses of the term it may seem arbitrary whether ‘compulsion’ should be retained, its use has important implications for the public understanding of addiction and is likely to deter people’s attempts to overcome their problems and their chances of success.
- (xii) A transformation of public-facing communications on addiction is needed in which people are persuaded to believe that breaking free from addiction is possible and given the best available information on how it can be done.

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#### Conflict of interest

None.

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