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REVIEW ARTICLE

The neuroscience of free will: implications for psychiatry

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Abstract. Belief in free will has been a mainstay in philosophy throughout history, grounded in large part in our intuitive sense that we consciously control our actions and could have done otherwise. However, psychology and psychiatry have long sought to uncover mechanistic explanations for human behavior that challenge the notion of free will. In recent years, neuroscientific discoveries have produced a model of volitional behavior that is at odds with the notion of contra-causal free will and our sense of conscious agency. Volitional behavior instead appears to have antecedents in unconscious brain activity that is localizable to specific neuroanatomical structures. Updating notions of free will in favor of a continuous model of volitional self-control provides a useful paradigm to conceptualize and study some forms of psychopathology such as addiction and impulse control disorders. Similarly, thinking of specific symptoms of schizophrenia as disorders of agency may help to elucidate mechanisms of psychosis. Beyond clinical understanding and etiological research, a neuroscientific model of volitional behavior has the potential to modernize forensic notions of responsibility and criminal punishment in order to inform public policy. Ultimately, moving away from the language of free will towards the language of volitional control may result in an enhanced understanding of the very nature of ourselves.

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A brief history of free will

In the past century, attempts within psychology and psychiatry to elucidate the scientific underpinnings of human behavior have steered academic thinking away from a belief in 'contra-causal' free will, the idea that individuals consciously choose to carry out certain actions and, under the exact same conditions, could have chosen to do otherwise. Such movement took root with Freud's psychic determinism and the primacy of the unconscious, as opposed to conscious deliberation, in governing action, though Freud suggested that freedom in decision making could result from successful psychoanalysis and modern psychoanalytic theory seems to still allow for some variant of free will (Felthous, 2008). The rise of experimental psychology exemplified by Skinnerian behaviorism later shifted causal explanations for behavior from the internal to the external, nearly rendering 'concepts such as free will or conscious choice… no more than quaint holdovers from psychology's philosophical beginnings' (Sappington, 1990). Still, cognitive behavioral therapies are based on the principle that thoughts can be consciously altered to effect behavioral change, while empirical research on self-efficacy and locus of control suggests that believing in the competence to control one's actions and life course may not only make an impact on behavior, but also be vital to mental health (Sappington, 1990; Waller 2004*a*, *b*). As a result, while enhanced psychological understanding of the determinants of human behavior has eroded the concept of free will, it remains possible to adopt at least three distinct philosophical stances on the issue, including hard determinism (the universe is deterministic; there is no free will), libertarianism (the universe is not deterministic; there is free will), and compatibilism (the universe is deterministic; there is free will).

Although age-old debates about whether the universe and human behavior are deterministic are unlikely to be resolved in the near future, neuroscientific discoveries in recent years do warrant an update concerning the role of free will in human decision making and action. Answers to some of the following questions are now well within the grasp of neuroscience: (1) How reliable is our subjective sense of free will? (2) Is volition localizable in the brain? (3) Can some psychiatric disorders be understood as disorders of free will and/or self-control? and (4) How can an enhanced neuroscientific understanding of behavior

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inform morality and the law? The aim of this paper is not to defend any philosophical position about free will *per se*, but rather to step away from ungrounded philosophical speculation and even the use of the term 'free will' altogether in favor of a modernized, evidence-based vocabulary to describe the underpinnings of human behavior and psychopathology alike. Note that while the term 'free will' can have multiple meanings, its use here will refer specifically to contracausal free will.

The neuroscience of free will

Brain activity precedes intention

The existence of free will when making a simple voluntary motor movement was called into question by a study published by Libet *et al.* (1983). In their experiment, electroencephalography (EEG) and electromyography (EMG) were used to monitor brain activity and muscle movement in subjects instructed to make a voluntary movement of their hand. After a designated start time, subjects made a spontaneous movement whenever they felt like doing so while noting the time of the urge to move by simultaneously watching a clock. Although the urge to move preceded the movement, as one would expect, the study found that this intention was itself preceded by cerebral activity, called the 'readiness potential' (RP), detectable several hundred milliseconds before the conscious intention to move. This startling discovery that the time of conscious intention lagged behind the RP suggested that simple voluntary movement does not arise *de novo* by conscious intent or free will, but rather is already set into motion by unconscious neural activity that precedes it. Libet stopped short of concluding that free will was non-existent by proposing that conscious decision making could abort or veto the movement (Libet, 1999), leaving room for a kind of 'free won't' (Obhi & Haggard, 2004).

Libet's results have been extensively critiqued throughout the years, citing both methodological and interpretive problems (Gomes, 1998; Pocket, 2002; Van de Grind, 2002; Danquah *et al.* 2008; Trevana & Miller, 2010; Klemm 2010), not the least of which is the potential unreliability of measuring the precise time of conscious intent[1]†. However, Libet remained a staunch defender of his data (Libet, 2000, 2002, 2003) and the essential finding has been replicated through refined experiments by other investigators (Haggard & Eimer, 1999; Trevana & Miller, 2002; Matsuhashi & Hallett, 2008).

† The notes appear after the main text.

Free will as illusion

Further research using novel versions of the Libet experiment have revealed the presence of the lateralized RP (LRP), a component of the RP that represents specific preparatory activity for movement on the contralateral side of the body (Haggard & Eimer, 1999; Trevana & Miller, 2002). Detection of the LRP suggests that the unconscious neural activity during a simple motor task does not simply reflect general preparation or a decision of when to move, but rather what specific target will be selected long before conscious awareness of that decision. Further technological updates have confirmed that unconscious brain activity occurring before conscious intention is specific to a subject's choice of target in a motor task, to the point of being able to predict the movement prior to conscious intention. For example, Soon *et al.* (2008) used functional magnetic resonance imaging (fMRI) in place of EEG to monitor subjects during a motor task in which they could freely chose to press a left or right button and found that brain activity predictive of that choice (albeit with only about 60% accuracy) preceded the conscious decision by as much as 10 s (Soon *et al.* 2008). Fried *et al.* (2011) likewise replaced EEG with implanted electrodes used to monitor the electrical activity of individual neurons within the brains of patients with epilepsy who were engaged in a variant of the Libet task (Fried *et al.* 2011). They showed that neuronal firing rate and the degree of neuronal recruitment occurring before the conscious decision to move could predict whether the subject would move (with about 80% accuracy), what lateralized movement would be made (about 70% accuracy), as well as when the subsequent sense of conscious intention would occur. These results support a model in which our subjective experience of free will during a voluntary motor act including intention (conscious planning to move) and agency (the subsequent causal ownership of that movement) 'emerges as the culmination of premotor activity starting several hundreds of milliseconds before awareness' (Fried *et al.* 2011).

Other experimental strategies have demonstrated the ability to influence decision making for voluntary motor acts through undetected external stimulation. The first such strategy involves 'backward masking' in which a sensory stimulus (e.g. a visual image on a computer screen) is presented followed quickly by a larger one in such a way that the first, smaller stimulus escapes conscious awareness. Despite the masking of the first 'subliminal prime' from consciousness, researchers have demonstrated that such masked stimuli can trigger specific motor responses during forced-choice tasks (e.g. voluntarily moving either the right or left index finger) (Taylor & McCloskey, 1996;

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Eimer & Schlaghecken, 1998; Schlaghecken & Eimer, 2004). A second experimental strategy has featured the application of a transcranial magnetic stimulation (TMS) pulse to relevant motor areas in order to unconsciously influence forced-choice motor response. This effect was demonstrated in two studies (Ammon & Gandevia, 1990; Brasil-Neto *et al.* 1992), but not replicated in a more recent experiment (Sohn *et al.* 2003). As with the Libet-type experiments, such findings indicate that unconscious brain activity that precedes conscious intention can bias the outcome of simple motor acts. Although these motor responses are experienced as acts of free will, it appears that even under the relatively simple mechanics of motor activity, a variety of processes may bias choice and action before and without our conscious awareness. Such evidence has led some to label our subjective sense of free will a mere 'introspection' (Hallett, 2007), a causal inference that is little more than an 'illusion' (Wegner, 2002).

The neuroanatomy of volition

In the 1950s, Wilder Penfield applied electrical stimulation during neurosurgical procedures to different brains regions of epileptic patients that resulted in a variety of responses including involuntary vocalisms and simple motor movements (Penfield, 1958). Subsequent experiments with electrical brain stimulation have provoked a wide range of behaviors including speech arrest, automatisms, re-experienced memories and *déjà-vu*, mood and anxiety responses, sensory experiences and multimodal hallucinations, and cognitive impairment (Selimbeyoglu & Parvizi, 2010). Such research has contributed greatly to our understanding of the functional neuroanatomy of the human brain.

Electrical stimulation applied specifically to the supplementary motor area (SMA) has been shown to produce an irrepressible urge to make a voluntary contralateral movement in the absence of any overt motor activity (Fried *et al.* 1991). In some cases, eliciting the actual motor movement could be achieved by increasing the electrical current applied to that same area. Subsequent studies have suggested that intention and agency may have precedents localizable to the pre-supplementary motor cortex (pre-SMA) (Lau *et al.* 2004; Moore *et al.* 2010) and that this region is the likely origin of Libet's RP (Haggard, 2008). In another recent experiment, electrical stimulation of sites within the inferior posterior parietal cortex produced urges to move specific body parts or to speak (Desmurget *et al.* 2009). When the intensity of that stimulation was increased, subjects reporting having actually moved, despite no EMG evidence of movement. Conversely, stimulation of the premotor cortex could produce actual movements, despite subjects firmly denying having moved. These findings suggest that distinct neuroanatomical regions govern motor intention, awareness and agency, and that these may be separable components of volition. Overall, a model of volitional action emerges in which preparatory activity occurring in the pre-SMA and SMA gives rise to an urge to move that is subsequently carried out into action by the primary motor cortex. In addition, a copy of the 'command' to move is also sent to the parietal cortex, which then generates a sensory representation of the predicted consequences of that movement (Haggard, 2009). In this way, volition in motor movement is better understood as set of processes in specific brain circuits that jointly specify information that determines our actions, such that voluntary action is best characterized as a form of neural decision making (Haggard, 2008).

Free will v. *volition*

The neuroscientific experiments presented thus far provide multiple lines of evidence that suggest that free will may be little more than a 'naïve folk psychological intuition' (Haynes, 2011). But doing away with free will does not mean that human beings are automatons in which our motivations, desires and values do not influence action. On the contrary, neuroscience clearly distinguishes between voluntary behavior and involuntary acts or reflexes. With volitional behavior, organisms have choices and make decisions about whether and when to act, what to do, and what not to do (Haggard, 2008). However, in a neuroscientific model, such choices are made within neural networks rather than any immaterial homunculus and often occur outside conscious awareness and before our subjective sense of intention or agency. Likewise, volition is conceptualized on a continuum, in which variable degrees of control are present across a spectrum of behaviors.

Free will in psychiatry

Simple motor movements offer an attractive experimental subject for studying volition due to the relative ease of observing the results of one's intentions (Kranick & Hallett, 2013). However, such experiments leave open the question of whether something akin to free will might be more pertinent to higher-order, complex human behaviors. One response can be found in social psychology experiments in which priming has been used successfully to bias the outcome of social behaviors. For example, in a now classic study of its kind, subjects primed with words associated with old age were found to walk down the hall more slowly than unprimed subjects (Bargh *et al.* 1996). A subsequent study observed better performance on a test of knowledge among subjects primed with words

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associated with intelligence (Dijksterhuis & van Knippenberg, 1998). Though the underlying mechanisms and replicability of such findings have recently been challenged (Doyen *et al.* 2012; Shanks *et al.* 2013), a substantial body of similar experiments has demonstrated that subliminal primes can activate goals related to a wide variety of higher-order behaviors including social interaction, cognitive performance, moral judgment and decision making (Dijksterhuis & Aarts, 2010; Bargh *et al.* 2012). The ability of unconscious stimuli to unwittingly affect the outcome of voluntary behavior therefore does not appear limited to simple motor movements.

Disorders of self-control

Another approach to the question of free will in complex behaviors involves examining pathological states that represent compromised volition or self-control. Thinking about psychiatric illnesses as disorders of volition and free will could result in enhanced neuroscientific models of psychopathology as well as a broader understanding of volitional behavior across a spectrum from disorder to normality.

Addiction is a prime illustrative example. Historically, addictive behaviors have been thought of as deficits in will, suggesting that they represent free choices to be overcome by exercising greater willpower or the surrender of will to a higher power (Chappel, 1992; Committee on Addictions of the Group for the Advancement of Psychiatry, 2002; Gray, 2007; Vohs & Baumeister, 2009). In contrast, the relatively novel disease model of addiction presents such behaviors as consequences of brain chemistry gone awry that result in an impairment of free will. Ironically, for many, the disease model can be difficult to accept because addiction requires the simple motor act of ingesting a psychoactive substance – an action that seems to be under control of free will (Vohs & Baumeister, 2009). And yet, the potential for free will to be illusory in simple motor acts under certain conditions has already been established.

An updated neuroscientific model of addiction abandons antiquated concepts of free will and willpower in favor of a continuum of volitional self-control and loss thereof (Lyvers, 2000; Baler & Volkow, 2006). Cognitive control can be subdivided into quantifiable constructs including response inhibition (the ability to suppress or veto behavior) and impulsive choice (premature selections of behavior without adequately weighing consequences; Moeller, 2001; Hyman, 2007; Perry & Carroll, 2008). In the laboratory, response inhibition is typically measured using the go/no-go or the stop signal tasks. In a simple go/no-go paradigm, subjects are shown visual cues on a computer screen that indicate whether to push a button (e.g. a green 'go' symbol means push). Those with poorer response inhibition will tend to make more frequent errors of commission, pressing the button during 'no-go' cues. Similarly, in a typical stop signal task, subjects press buttons in response to visual cues on a computer screen (e.g. left or right arrows), but are told not to press any buttons when they hear a beep regardless of visual cues. In this paradigm, performance is based on stop signal reaction time. Deficits in response inhibition as measured by such tasks have been demonstrated among those with current substance abuse (Perry & Carroll, 2008) as well as those with acute exposure to alcohol (Marczinski & Fillmore, 2005; Easdon *et al.* 2005; Perry & Carroll, 2008), cocaine (Fillmore & Rush, 2002) and amphetamines (Fillmore *et al.* 2003). Similar effects have been found among those with chronic psychostimulant dependence (Monterosso *et al.* 2005), including one study that demonstrated that greater inhibitory deficits were associated with greater lifetime drug exposure (Colzato *et al.* 2007). These findings indicate that drug use can result in impulsivity that may worsen with chronic dependence, supporting the idea that deficits in volitional control may be progressive during different phases (e.g. acquisition, escalation, abstinence, relapse) of addiction (Perry & Carroll, 2008).

A bidirectional relationship between impulsivity and addiction is supported by observations that inhibitory control deficits detected in childhood are associated with an increased risk of later substance use disorders (Ivanov *et al.* 2008). Such findings highlight the considerable vulnerability of adolescents to drug abuse associated with normal neurodevelopmental changes that mediate impulsivity and risk-taking behavior (Chambers *et al.* 2003). Note that neuroanatomical studies point to a major role of the pre-SMA and SMA in response inhibition (Chambers *et al.* 2009), the same areas that were found to be relevant to volition and agency in the experiments by Libet and others described earlier.

Impulsive choice is measured in the laboratory using the delayed-discounting paradigm in which subjects with greater impulsivity choose smaller immediate rewards over larger delayed rewards. Using this measure, subjects with current alcohol, cocaine and methamphetamine abuse have been found to make more impulsive choices than non-users, though such increased impulsivity was not seen with acute administration of those substances under experimental conditions (Perry & Carroll, 2008). The Iowa Gambling Task (IGT) was developed to measure behavioral choices associated with immediate reward despite significantly negative consequences observed among patients with damage to the ventromedial prefrontal cortex

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(VMPC). The task directs subjects to select cards from four decks with different preordained schedules of immediate and long-term reward and risk. Over the course of the task, normal subjects learn to select from the card decks that generate the most long-term reward, whereas patients with VMPC damage make choices associated with greater immediate reward, but longer-term loss (Bechara, 2005). Similar decision-making deficits have been demonstrated on the IGT among those with alcohol, cannabis, cocaine, methamphetamine and opiate dependence as well as drug-naïve subjects with a family history of substance abuse (Garcia-Verdejo, 2009). These findings, together with evidence of anatomical differences in the VMPC between substance abusers and normal controls, suggest an important role for the VMPC in the neuroanatomy of decision-making and addictive behavior. The 'somatic marker theory of addiction' proposes a neurocircuitry in which the amygdala mediates affective responses to actual substance use (or other immediate external rewards) while the VMPC triggers affective responses to thinking about or recalling previous episodes of drug use including associated negative consequences (Garcia-Verdejo, 2009). Drug choice in the face of negative consequences can result from exaggerating the incentive impact of actual drug use, thinking about drug use, or cravings as well as discounting anticipated negative consequences. Much of this neural decision making occurs unconsciously, with the insular cortex integrating the somatic signals from the amygdala and VMPC in order to produce a subjective feeling of a conscious urge to use drugs that is then implemented into action in the SMA (Naqvi & Bechara, 2008; Li *et al.* 2010). Though it remains unproven (Dunn *et al.* 2006), the somatic marker theory of addiction is supported by a considerable body of evidence and provides an appealing model for understanding the neurocircuitry of complex decision making, both pathological and normal.

Problems with response inhibition, impulsivity and reward–risk decision making have been detected in a wide variety of psychiatric disorders including bipolar disorder, attention deficit hyperactivity disorder, trichtillomania, obsessive–compulsive disorder, borderline personality disorder, conduct disorder, pathological gambling and antisocial personality disorder (Dunn *et al.* 2006; Chamberlain & Sahakian, 2007; Perry & Carroll, 2008; Chambers *et al.* 2009; Garcia-Verdeko, 2009). This has led some to propose a new category of 'volitional disorders' for the Diagnostic and Statistical Manual, fifth edition (DSM-5) in which previously separate categories such as impulse control, obsessive–compulsive spectrum, and addictive disorders might be unified based on common underlying control deficits (Fontenelle *et al.* 2009). Similarly, the National Institute of Mental Health has proposed cognitive control as a construct within its Research Domain Criteria that were formulated to study traits that are present among normal individuals and may be dysfunctional across a variety of different categorical DSM disorders (Insel *et al.* 2010; Sanislow *et al.* 2010). In this way, volitional control represents a more accurate and quantifiable construct than free will that can be used to further elucidate neural mechanisms of control and understand the nature of control across a continuum of normal and pathological states.

Disorders of agency

Although our normal experience of free will may be misleading from a neuroscientific perspective, certain pathological conditions reveal just how disabling loss of agency – the subjective sense of casual ownership of behavior – can be. For example, patients with anarchic or alien hand syndrome experience a limb moving in a semi-purposeful way (e.g. unbuttoning a shirt) without any conscious intent to do so. Conversely, those exhibiting 'utilization behavior' experience a stimulus-driven tendency to use an object despite contextual purposelessness of the action (e.g. putting on a pair of glasses over a pair already being worn). This purposelessness suggests involuntariness, though patients rationalize their behavior as volitional. Both alien hand syndrome and utilization behavior are associated with frontal lobe injury, specifically involving lesions to the SMA and pre-SMA (Frith *et al.* 2000; Blakemore *et al.* 2002; Sumner & Husain, 2008). It is thought that these conditions reflect distinct lesions in a neural circuit that mediates the coordination of movement based upon continuous comparisons between the predicted sensory effects of a movement and its actual sensory effects (Frith *et al.* 2000).

Among psychiatric disorders, certain symptoms of schizophrenia have also been modeled as disorders of agency. Sense of agency can be manipulated under experimental conditions in which normal subjects are tricked into thinking that motor movements seen on a computer screen are made by themselves when in fact they are made by others (Nielsen, 1963; Wegner & Wheatley, 1999). These types of self-attribution agency errors have been found to occur significantly more frequently among patients with schizophrenia (Daprati *et al.* 1997) and prodromal psychosis (Hauser *et al.* 2011) compared with controls, as well as among patients with schizophrenia and delusions of control compared with those without such delusions (Franck *et al.* 2001). It has been proposed that such errors are rooted in deficits whereby the sensory feedback produced by self-movements does not differ, as it should, from feedback produced by the movements

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made by others. For example, two studies reported that patients with schizophrenia do not attenuate tactile sensations arising from self-stimulation as much as normal subjects, suggesting a possible mechanism for somatic passivity experiences (Blakemore *et al.* 2000; Shergill *et al.* 2005). The predictive sensory effects of a volitional movement are thought to arise from an efference copy of that movement generated through a process called corollary discharge. When the predictive sensory effects of the efference copy do not match the actual sensory effects, the movement can become dissociated from agency and instead attributed to an external agent (Syznofzik *et al.* 2008). Several studies support a model in which delusions of control arise from such mismatches caused by deficits involving internal predictions, external sensory feedback, or some difficulty with integration (Syznofzik *et al.* 2010; Voss *et al.* 2010). The application of this comparator model is not limited to motor acts – problems with corollary discharge may also explain thought insertion and hallucinations in which internally generated thoughts or speech are perceived as coming from an external source (Blakemore *et al.* 2000; Ford & Mathalon, 2005; Heinks-Maldonado *et al.* 2007).

Additional studies of agency have focused on a normal phenomenon in which the subjectively perceived timing of a motor act (e.g. pressing a button) is delayed when followed by an effect (e.g. a tone). This intentional and temporal binding of voluntary action and effect is thought to contribute to a sense of agency. In several studies, patients with schizophrenia have demonstrated hyperbinding in which there is excessive linkage between volitional actions and external sensory events. Such studies have revealed that patients with schizophrenia experiencing delusions and hallucinations seem to have particular difficulty with internal predictions about the sensory consequences of their actions, perhaps resulting in overreliance on external cues leading to errors in agency judgments (Syznofzik *et al.* 2010; Voss *et al.* 2010). Modeling psychotic symptoms as disintegrative dysfunction resulting in loss of agency and the ability to distinguish self from others has intuitive appeal given the longstanding notion of schizophrenia as a schism of the mind in which core aspects of self-identity are disrupted (Jardri *et al.* 2011; Syznofzik *et al.* 2013).

Free will, psychiatry and society

Illnesses like schizophrenia highlight how impairing a confused sense of agency can be, but if our normal sense of free will is inaccurate from a neuroscientific perspective, why does it exist and what purpose, if any, might it serve? From an evolutionary perspective, it has been suggested that a belief in free will might be a requisite for the sense of moral responsibility that facilitates social interaction (Wegner, 2002; Cashmore, 2010). Indeed, it appears that our personal intuitions about free will are closely linked to moral reasoning. Evidence from interviews and surveys suggest that from an early age and across different cultures, people are intuitively libertarian, believing in an indeterministic universe that permits free will (Nichols, 2004; Sarkissian *et al.* 2010). If presented with an hypothetical scenario in which an individual commits a crime within a deterministic universe, subjects can reason that the individual is not morally responsible when that crime is minor (e.g. cheating on one's taxes), but tend to revert to a view of free will and moral responsibility when the crime is significant (e.g. rape) (Nichols & Knobe, 2007). These findings suggest that intuitions about free will are biased by emotional assessments about blame.

Concerns that moral behavior might erode without a belief in free will have been a timeless feature of free will debates and have been rekindled in recent years in the wake of neuroscientific discovery. At least two studies seem to support this fear. In the first, subjects who read information explaining that neuroscience has determined that free will is an illusion were found to cheat on an examination more than controls that read either neutral statements or statements that supported the notion of free will (Vohs & Schooler, 2008). In the second, subjects who read statements espousing disbelief in free will reported less willingness to help others compared with those who read neutral or pro-free will statements. Likewise, subjects rated with a pre-existing chronic disbelief in free will were found to be less likely to commit to an actual helping paradigm (Baumeister *et al.* 2009).

Psychology experiments of this ilk must be interpreted carefully, examining the way in which questions are worded. For example, it may be that when answering questions about determinism and responsibility, subjects confuse determinism to mean something that does not allow that psychological processes influence behavior (Nahmias, 2006). It is possible that respondents also conflate determinism with fatalism, a resignation that personal actions have no bearing on future outcomes (Miles, 2013*a*). Neither view portrays a neuroscientifically accurate view of volitional behavior. In fact, when presented with a scenario of determinism in which behavior is indeed caused by a chain of internal psychological events as opposed to strictly external physical events, subjects give compatabilist responses that endorse both free will and moral responsibility (Miles, 2013*a*).

A similar potential for confusion arises when trying to understand what it means to be morally responsible.

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If someone commits a crime in a deterministic universe, then it would not be possible to do otherwise. However, though it might be intuitive to think so, the inability to do otherwise – the lack of free will – need not preclude moral responsibility. In fact, Morse has demonstrated that the presence or absence of libertarian free will is not a criterion for criminal responsibility according to current legal standards of *mens rea* and *actus reus*, the M'Naghten Rule, and the American Law Institute Moral Penal Code (Morse, 2007). Others have argued that rather than enhancing moral behavior, the belief in free will and its corollary that individuals chose their fates may actually be associated with neglect of the socially disadvantaged and used to rationalize abusive treatment of prisoners, the accepted supplementary penalty of prison rape, and capital punishment (Miles, 2013*a*, *b*).

Inasmuch as psychiatrists play an important role in forensic assessments of criminal responsibility, psychiatry would seem obliged to take part in revising outmoded conceptions of free will that are not consistent with neuroscience and to reshape the legal system accordingly. While it is increasingly common, if still rarely successful, to seek an insanity defense based on principles of biological causation ('my brain made me do it'), the point here is not that neuroscience should render responsibility null and void (Morse, 2007; Aharoni *et al.* 2008). Instead, Hallett (2007) suggests a revised definition of responsibility that disentangles it from morality: 'If there is no free will as a driving force, are persons responsible for their behavior? This appears to be a difficult question, but it is really not. It is difficult only for the dualist. A person's brain is clearly fully responsible, and always responsible, for the person's behavior' (Hallett, 2007).

In addition, given updated models in which intention and control are at best limited in normal individuals, it has been argued that criminal sentencing should be carried out within a consequentialist rather than a retributivist framework of punishment. A consequentialist approach seeks to promote the welfare of both individuals and society 'rather than meting out just deserts' (Greene & Cohen, 2004). Such an approach has been adopted in Sweden where no one is immune from criminal responsibility, but where criminal 'care' is rooted – in theory if not always in practice – in a philosophy that views punishment as needless cruelty and aims instead to prevent further crime and remedy the roots of criminal behavior (Juth & Lorentzon, 2010). In the USA, recognition that certain populations may have greater levels of impulsivity has resulted in the design of the existing juvenile justice system (Burns & Bechara, 2007), drug courts (Brown, 2010), as well as recently created military veterans' courts (Cartwright, 2011), all of which were founded upon consequentialist principles. It would seem that current neuroscientific models of volitional action support a shift to a more consequentialist approach to criminal justice for all individuals.

Conclusion

Ultimately, unresolved debates about free will and moral responsibility persist due to intuitively dualistic notions of the self. We have the subjective sense that we choose to move and when we learn of evidence that action may be determined by unconscious neural activity that precedes intention, we feel threatened by the loss of free will and worry that moral responsibility will disappear in kind. But modern neuroscientific models of volition are inherently non-dualistic, such that all neural activity, both unconscious and conscious, make up the self. Accepting, as Freud suggested, that conscious intention may not play as significant a role in causing behavior as does an unconscious chain of neural events need not threaten the self. The self – that is, the sum total of our brain activity and its 'flexible and intelligent interaction with current and historical context' (Haggard, 2009) – does indeed engage in intention, planning and decision making, and its ability to select and veto actions is a reality. However, volitional control exists on a continuum with loss of control resulting in features of many mental disorders and complete freedom – in the sense of conscious intention being the ultimate determinant of action – lacking despite a perfectly functioning brain. Replacing the idea of free will with the language of volitional control offers a more accurate and measureable construct for mechanistic explanations of behavior, both pathological and otherwise. Updating models of selfhood and behavior in this fashion has far-reaching implications for psychiatry in terms of enhanced clinical understanding, facilitating etiological research, and informing the role of psychiatry in forensic consultation and public policy.

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Declaration of Interest

None.

Note

[1] In addition to methodological critiques, it could be argued on philosophical grounds that Libet's findings do not, and

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that experiments in general cannot, prove the non-existence of free will, just as one cannot prove the non-existence of God or the soul. While this argument is valid from an epistemological perspective, neuroscience favors a more parsimonious approach to data interpretation.

References

Aharoni E, Funk C, Sinnott-Armstrong W, Gazzaniga M (2008). Can neurological evidence help courts assess criminal responsibility? Lessons from law and neuroscience. *Annals of the New York Academy of Sciences* **1124**, 1145–1160.

Ammon K, Gandevia SC (1990). Transcranial magnetic stimulation can influence the selection of motor programmes. *Journal of Neurology, Neurosurgery, and Psychiatry* **53**, 705–707.

Baler RD, Volkow ND (2006). Drug addiction: the neurobiology of disrupted self-control. *Trends in Molecular Medicine* **12**, 559–566.

Bargh JA, Chen M, Burrows L (1996). Automaticity of social behavior: direct effects of trait construct and stereotype-activation on action. *Journal of Personality and Social Psychology* **71**, 230–244.

Bargh JA, Schwader KL, Hailey SE, Dyer RL, Boothby EJ (2012). Automaticity in social–cognitive processes. *Trends in Cognitive Science* **16**, 593–605.

Baumeister R, Masicampo EJ, DeWall CN (2009). Prosocial benefits of feeling free: disbelief in free will increases aggression and reduces helpfulness. *Personality and Social Psychology Bulletin* **35**, 260–268.

Bechara A (2005). Decision making, impulse control and loss of willpower to resist drugs: a neurocognitive perspective. *Nature Neuroscience* **8**, 1458–1463.

Blakemore S, Smith J, Steel R, Johnstone EC, Frith CD (2000). The perception of self-produced sensory stimuli in patients with auditory hallucinations and passivity experiences: evidence for a breakdown in self-monitoring. *Psychological Medicine* **30**, 1131–1139.

Blakemore SJ, Wolpert DM, Frith CD (2002). Abnormalities in awareness of action. *Trends in Cognitive Science* **6**, 237–242.

Brasil-Neto JP, Pascual-Leone A, Valls-Sole J, Cohen LG, Hallett M (1992). Focal transcranial magnetic stimulation and response bias in a forced-choice task. *Journal of Neurology, Neurosurgery, and Psychiatry* **55**, 964–966.

Brown RT (2010). Systematic review of the impact of adult drug-treatment courts. *Translational Research* **155**, 263–274.

Burns K, Bechara A (2007). Decision making and free will: a neuroscience perspective. *Behavioral Science and the Law* **25**, 263–280.

Cartwright T (2011). "To care for him who shall have borne the battle": the recent development of veterans treatment courts in America. *Stanford Law and Policy Review* **22**, 295–316.

Cashmore AR (2010). The Lucretian swerve: the biological basis of human behavior and the criminal justice system. *Proceedings of the National Academy of Sciences USA* **107**, 4499–4504.

Chamberlain SR, Sahakian BJ (2007). The neuropsychiatry of impulsivity. *Current Opinion in Psychiatry* **20**, 255–261.

Chambers CD, Garavan H, Bellgrove MA (2009). Insights into the neural basis of response inhibition from cognitive and clinical neuroscience. *Neuroscience and Biobehavioral Reviews* **33**, 631–646.

Chambers RA, Taylor JR, Potenza MN (2003). Developmental neurocircuitry of motivation in adolescence: a critical period of addiction vulnerability. *American Journal of Psychiatry* **160**, 1041–1052.

Chappel JN (1992). Effective use of Alcoholics Anonymous and Narcotics Anonymous in treating patients. *Psychiatry Annals* **22**, 122–131.

Colzato LS, van den Wildenberg WPM, Hommel B (2007). Impaired inhibitory control in recreational cocaine users. *PLoS ONE* **2**, e1143.

Committee on Addictions of the Group for the Advancement of Psychiatry (2002). Responsibility and choice in addiction. *Psychiatric Services* **53**, 717–713.

Danquah AN, Farrell MJ, O'Boyle DJ (2008). Biases in the subjective timing of perceptual events: Libet *et al.* (1983) revisited. *Consciousness and Cognition* **17**, 616–627.

Daprati E, Franck N, Georgieff N, Proust J, Pacherier E, Dalery J, Jeannerod M (1997). Looking for the agent: an investigation into consciousness of action and self-consciousness in schizophrenic patients. *Cognition* **65**, 71–86.

Desmurget M, Reilly KT, Richard N, Szathmari A, Mottolese C, Sirigu A (2009). Movement intention after parietal cortex stimulation in humans. *Science* **324**, 811–814.

Dijksterhuis A, Aarts H (2010). Goals, attention, and (un) consciousness. *Annual Review of Psychology* **61**, 467–490.

Dijksterhuis A, van Knippenberg A (1998). The relation between perception and behavior, or how to win a game of Trivial Pursuit. *Journal of Personality and Social Psychology* **74**, 865–877.

Doyen S, Klein O, Pichon C, Cleeremans A (2012). Behavioral priming: it's all in the mind, but whose mind? *PLOS ONE* **7**, e29081.

Dunn BD, Dalglieish T, Lawrence AD (2006). The somatic marker hypothesis: a critical evaluation. *Neuroscience and Biobehavioral Reviews* **30**, 239–271.

Easdon C, Izenberg A, Armilio ML, Yu H, Alain C (2005). Alcohol consumption impairs stimulus- and error-related processing during a go/no-go task. *Cognitive Brain Research* **25**, 873–883.

Eimer M, Schlaghecken F (1998). Effects of masked stimuli on motor activation: behavioral and electrophysiological evidence. *Journal of Experimental Psychology: Human Perception and Performance* **24**, 1737–1747.

Felthous AR (2008). The will: from metaphysical freedom to normative functionalism. *Journal of the American Academy of Psychiatry and the Law* **36**, 16–24.

Fillmore MT, Rush CR (2002). Impaired inhibitory control of behavior in chronic cocaine users. *Drug and Alcohol Dependence* **66**, 265–273.

Fillmore MT, Rush CR, Marczinski CA (2003). Effects of *d*-amphetamine on behavioral control in stimulant

abusers: the role of prepotent response tendencies. *Drug and Alcohol Dependence* **71**, 143–152.

Fontenelle LF, Mendlowicz MV, Versiani M (2009). Volitional disorders: a proposal for DSM-V. *World Journal of Biological Psychiatry* **10**, 1016–1029.

Ford JM, Mathalon DH (2005). Corollary discharge dysfunction in schizophrenia: can it explain auditory hallucinations? *International Journal of Psychophysiology* **58**, 179–189.

Franck N, Farrer C, Gerogieff N, Marie-Cardine M, Dalery J, d'Amato T (2001). Defective recognition of one's own actions in patients with schizophrenia. *American Journal of Psychiatry* **158**, 454–459.

Fried I, Katz A, McCarthy G, Sass KJ, Williamson P, Spencer SS, Spencer DD (1991). Functional organization of human supplementary cortex studied by electrical stimulation. *Journal of Neuroscience* **11**, 3656–3666.

Fried I, Mukamel R, Kreiman G (2011). Internally generated preactivation of single neurons in human medial frontal cortex predicts volition. *Neuron* **69**, 548–562.

Frith CD, Blakemore S, Wolpert DM (2000). Abnormalities in the awareness and control of action. *Philosophical Transactions of the Royal Society London B Biological Sciences* **355**, 1711–1788.

Garcia-Verdejo A (2009). A somatic marker theory of addiction. *Neuropharmacology* **56**, 48–62.

Gomes G (1998). The timing of conscious experience: a critical review and reinterpretation of Libet's research. *Consciousness and Cognition* **7**, 559–595.

Gray MT (2007). Freedom and resistance: the phenomenal will in addiction. *Nursing Philosophy* **8**, 3–15.

Greene J, Cohen J (2004). For the law, neuroscience changes nothing and everything. *Philosophical Transactions of the Royal Society London B Biological Sciences* **359**, 1775–1785.

Haggard P (2008). Human volition: towards a neuroscience of will. *Nature Reviews. Neuroscience* **9**, 934–946.

Haggard P (2009). The sources of human volition. *Science* **324**, 731–733.

Haggard P, Eimer M (1999). On the relation between brain potentials and the awareness of voluntary movements. *Experimental Brain Research* **126**, 128–133.

Hallett M (2007). Volitional control of movement: the physiology of free will. *Clinical Neurophysiology* **118**, 1179–1192.

Hauser M, Knoblich G, Repp BH, Lautenschlager M, Gallinat J, Heinz A, Voss M (2011). Altered sense of agency in schizophrenia and the putative psychosis prodrome. *Psychiatry Research* **186**, 170–176.

Haynes J (2011). Decoding and predicting intentions. *Annals of the New York Academy of Sciences* **1224**, 9–21.

Heinks-Maldonado TH, Mathalon DH, Houde JF, Gray M, Faustman WO, Ford J (2007). Relationship of imprecise corollary discharge in schizophrenia to auditory hallucinations. *Archives of General Psychiatry* **64**, 286–296.

Hyman SE (2007). The neurobiology of addiction: implications for voluntary control of behavior. *American Journal of Bioethics* **7**, 8–11.

Insel T, Cuthbert B, Garvey M, Heinssen R, Pine DS, Quinn K, Sanislow C, Wang P (2010). Research Domain Criteria (RDoC): toward a new classification framework for research on mental disorders. *American Journal of Psychiatry* **167**, 748–750.

Ivanov I, Schulz KP, London ED, Newcorn JH (2008). Inhibitory control deficits in childhood and risk for substance abuse disorders: a review. *American Journal of Drug and Alcohol Abuse* **34**, 239–258.

Jardri R, Pins D, Lafargue G, Very E, Ameller A, Delamire C, Thomas P (2011). Increased overlap between the brain areas involved in self–other distinction in schizophrenia. *PLoS ONE* **6**, e17500.

Juth N, Lorentzon F (2010). The concept of free will and forensic psychiatry. *International Journal of Law and Psychiatry* **33**, 1–6.

Klemm WR (2010). Free will debates: simple experiments are not so simple. *Advances in Cognitive Psychology* **30**, 47–65.

Kranick SM, Hallett M (2013). Neurology of volition. *Experimental Brain Research* **229**, 313–327.

Lau HC, Rogers RD, Haggard P, Passingham RE (2004). Attention to intention. *Science* **303**, 1208–1210.

Li X, Lu Z, D'Argembeau A, Ng M, Bechara A (2010). The Iowa Gambling Task in fMRI images. *Human Brain Mapping* **31**, 410–423.

Libet B (1999). Do we have free will? *Journal of Consciousness Studies* **6**, 47–57.

Libet B (2000). Time factors in conscious processes: reply to Gilberto Gomes. *Consciousness and Cognition* **9**, 1–12.

Libet B (2002). The timing of mental events: Libet's experimental findings and their implications. *Consciousness and Cognition* **11**, 291–299.

Libet B (2003). Timing of conscious experience: reply to the 2002 commentaries on Libet's findings. *Consciousness and Cognition* **12**, 321–331.

Libet B, Gleason CA, Wright EW, Pearl DK (1983). Time of conscious intention to act in relation to onset of cerebral activity (readiness-potential). The unconscious initiation of a freely voluntary act. *Brain* **106**, 623–642.

Lyvers M (2000). "Loss of control" in alcoholism and drug addiction: a neuroscientific interpretation. *Experimental and Clinical Psychopharmacology* **8**, 225–249.

Marczinski CA, Fillmore MT (2005). Compensating for alcohol-induced impairment of control: effects on inhibition and activation of behavior. *Psychopharmacology* **181**, 337–346.

Matsuhashi M, Hallett M (2008). The timing of the conscious intention to move. *European Journal of Neuroscience* **28**, 2344–2351.

Miles JB (2013*a*). 'Irresponsible and a disservice': the integrity of social psychology turns on the free will dilemma. *British Journal of Social Psychology* **52**, 205–218.

Miles JB (2013*b*). The integrity of social psychology turns on the free will dilemma: reply to Baumesiter, Vonasch, and Bargh. *British Journal of Social Psychology* **52**, 231–237.

Moeller FG, Barratt ES, Dougherty DM, Schmitz JM, Swann AC (2001). Psychiatric aspects of impulsivity. *American Journal of Psychiatry* **158**, 1783–1793.

Monterosso JR, Aron AR, Cordova X, Xu J, London ED (2005). Deficits in response inhibition associated with

chronic methamphetamine abuse. *Drug and Alcohol Dependence* **79**, 273–277.

Moore JW, Ruge D, Wenke D, Rothwell J, Haggard P (2010). Disrupting the experience of control in the human brain: pre-supplementary motor area contributes to the sense of agency. *Proceedings of the Royal Society B* **277**, 2503–2509.

Morse SJ (2007). The non-problem of free will in forensic psychiatry and psychology. *Behavioral Science and the Law* **25**, 203–220.

Nahmias E (2006). Folk fears about freedom and responsibility: determinism vs. reductionism. *Journal of Cognition and Culture* **6**, 215–237.

Naqvi NH, Bechara A (2008). The hidden island of addiction: the insula. *Trends in Neuroscience* **32**, 56–67.

Nichols S (2004). The folk psychology of free will: fits and starts. *Mind and Language* **19**, 473–502.

Nichols S, Knobe J (2007). Moral responsibility and determinism: the cognitive science of folk intuitions. *Nous* **41**, 663–685.

Nielsen T (1963). Volition: a new experimental approach. *Scandinavian Journal of Psychology* **4**, 225–230.

Obhi SS, Haggard P (2004). Free will and free won't. *American Scientist* **92**, 358–365.

Penfield W (1958). Some mechanisms of consciousness discovered during electrical stimulation of the brain. *Proceedings of the National Academy of Sciences USA* **44**, 51–66.

Perry JL, Carroll ME (2008). The role of impulsive behavior in drug abuse. *Psychopharmacology* **200**, 1–26.

Pockett S (2002). On subjective back-referral and how long it takes to become conscious of a stimulus: a reinterpretation of Libet's data. *Consciousness and Cognition* **11**, 144–161.

Sanislow CA, Pine DS, Quinn KJ, Kozak MJ, Garvey MA, Heinssen RK, Wang PS, Cuthbert BN (2010). Developing constructs for psychopathology research: research domain criteria. *Journal of Abnormal Psychology* **199**, 631–639.

Sappington AA (1990). Recent psychological approaches to the free will *versus* determinism issue. *Psychological Bulletin* **108**, 19–29.

Sarkissian H, Chattterjee A, de Brigard F, Knobe J, Nichols S, Sirker S (2010). Is belief in free will a cultural universal? *Mind and Language* **25**, 346–358.

Schlaghecken F, Eimer M (2004). Masked prime stimuli can bias "free" choices between response alternatives. *Psychonomic Bulletin and Review* **11**, 463–468.

Selimbeyoglu A, Parvizi J (2010). Electrical stimulation of the human brain: perceptual and behavioral phenomena reported in the old and new literature. *Frontiers in Human Neuroscience* **4**, 46.

Shanks DR, Newell BR, Lee EH, Balakrishnan , Elelund L, Cenac Z, Kavvadia F, Moore C (2013). Priming intelligent behavior: an elusive phenomenon. *PLOS ONE* **8**, e56515.

Shergill SS, Samson G, Bays PM, Frith CD, Wolpert DM (2005). Evidence for sensory prediction deficits in schizophrenia. *American Journal of Psychiatry* **162**, 2384–2386.

Sohn YH, Kaelin-Lang A, Hallett M (2003). The effect of transcranial magnetic stimulation on movement selection. *Journal of Neurology, Neurosurgery, and Psychiatry* **74**, 985–987.

Soon CS, Brass M, Heinze H, Haynes J (2008). Unconscious determinants of free decisions in the brain. *Nature Neuroscience* **11**, 543–545.

Sumner P, Husain M (2008). At the edge of consciousness: automatic motor activation and voluntary control. *Neuroscientist* **14**, 474–486.

Syznofzik M, Their P, Leube DT, Schlotterbeck P, Linder A (2010). Misattributions of agency in schizophrenia are based on imprecise predictions about the sensory consequences of one's actions. *Brain* **133**, 262–271.

Syznofzik M, Vosgerau G, Newen A (2008). Beyond the comparator model: a multifactorial two-step account of agency. *Consciousness and Cognition* **17**, 219–239.

Syznofzik M, Vosgerau G, Voss M (2013). The experience of agency: an interplay between prediction and postdiction. *Frontiers in Psychology* **4**, 127.

Taylor JL, McCloskey DI (1996). Selection of motor responses on the basis of unperceived stimuli. *Experimental Brain Research* **110**, 62–66.

Trevena J, Miller J (2010). Brain preparation before a voluntary action: evidence against unconscious movement initiation. *Consciousness and Cognition* **19**, 447–456.

Trevena JA, Miller J (2002). Cortical movement preparation before and after a conscious decision to move. *Consciousness and Cognition* **11**, 162–190.

Van de Grind W (2002). Physical, neural, and mental timing. *Consciousness and Cognition* **11**, 241–264.

Vohs K, Baumeister RF (2009). Addiction and free will. *Addiction Research and Theory* **17**, 231–235.

Vohs K, Schooler JW (2008). The value of believing in free will: encouraging a belief in determinism increases cheating. *Psychological Science* **19**, 49–54.

Voss M, Moore J, Hauser M, Gallinat J, Heinz A, Haggard P (2010). Altered awareness of action in schizophrenia: a specific deficit in predicting action consequences. *Brain* **133**, 3104–3112.

Waller BN (2004*a*). Neglected psychological elements of free will. *Philosophy, Psychiatry, and Psychology* **11**, 111–118.

Waller BN (2004*b*). Comparing psychoanalytic and cognitive–behavioral perspectives on control. *Philosophy, Psychiatry, and Psychology* **11**, 125–128.

Wegner DM (2002). *The Illusion of Conscious Will*. MIT Press: Cambridge, MA.

Wegner DM, Wheatley T (1999). Apparent mental causation: sources of the experience of will. *American Psychology* **54**, 480–492.