International Journal of Methods in Psychiatric Research
Int. J. Methods Psychiatr. Res. 23(Suppl. 1): 41–57 (2014)
Published online in Wiley Online Library
(wileyonlinelibrary.com) **DOI:** 10.1002/mpr.1410

Dysfunctions of decision-making and cognitive control as transdiagnostic mechanisms of mental disorders: advances, gaps, and needs in current research

THOMAS GOSCHKE

Department of Psychology, Technische Universität Dresden, Dresden, Germany

Key words
cognitive control, volition, decision-making, transdiagnostic mechanisms, large-scale brain systems

Correspondence
Thomas Goschke, Department of Psychology, Technische Universität Dresden, 01062 Dresden, Germany. Telephone (+49) 351-463-34695 Fax (+49) 351-463-33522
Email: goschke@psychologie.tu-dresden.de

Abstract

Disadvantageous decision-making and impaired volitional control over actions, thoughts, and emotions are characteristics of a wide range of mental disorders such as addiction, eating disorders, depression, and anxiety disorders and may reflect transdiagnostic core mechanisms and possibly vulnerability factors. Elucidating the underlying neurocognitive mechanisms is a precondition for moving from symptom-based to mechanism-based disorder classifications and ultimately mechanism-targeted interventions. However, despite substantial advances in basic research on decision-making and cognitive control, there are still profound gaps in our current understanding of dysfunctions of these processes in mental disorders. Central unresolved questions are: (i) to which degree such dysfunctions reflect transdiagnostic mechanisms or disorder-specific patterns of impairment; (ii) how phenotypical features of mental disorders relate to dysfunctional control parameter settings and aberrant interactions between large-scale brain systems involved in habit and reward-based learning, performance monitoring, emotion regulation, and cognitive control; (iii) whether cognitive control impairments are consequences or antecedent vulnerability factors of mental disorders; (iv) whether they reflect generalized competence impairments or context-specific performance failures; (v) whether not only impaired but also chronic over-control contributes to mental disorders. In the light of these gaps, needs for future research are: (i) an increased focus on basic cognitive-affective mechanisms underlying decision and control dysfunctions across disorders; (ii) longitudinal-prospective studies systematically incorporating theory-driven behavioural tasks and neuroimaging protocols to assess decision-making and control dysfunctions and aberrant interactions between underlying large-scale brain systems; (iii) use of latent-variable models of cognitive control rather than single

tasks; (iv) increased focus on the interplay of implicit and explicit cognitive-affective processes; (v) stronger focus on computational models specifying neurocognitive mechanisms underlying phenotypical expressions of mental disorders.

Introduction

Disadvantageous decision-making and impaired volitional control over one's actions, thoughts, or emotions are core characteristics of a wide range of mental disorders such as addiction, eating disorders, depression, and anxiety disorders (Wittchen *et al.*, 2011). Elucidating the underlying psychological and neurobiological mechanisms and pathways of such dysfunctions is a precondition for theory-based improvement of prevention and intervention, as well as for moving from symptom-based classifications towards disorder models based on underlying mechanisms (Maia and Frank, 2011; Morris and Cuthbert, 2012). Although in the past two decades substantial progress has been made in research on the neurocognitive basis of decision-making and cognitive control (for reviews see Goschke, 2013; Hofmann *et al.*, 2012; Mars *et al.*, 2011; O'Reilly *et al.*, 2010), to date this research has had relatively limited impact on mainstream theorizing and classification of mental disorders. At the same time, there is increasing discomfort with classifications of mental disorders based on descriptive phenotypical features, because the resulting disorder categories may not map consistently to the organization of underlying cognitive-affective mechanisms and neural systems. The fact that many disorders are characterized by complex configurations of symptoms that show substantial overlap across diagnostic categories has been taken to suggest that these categories may not depict natural kinds that "carve nature at its joints" in terms of causal mechanisms (cf. Buckholtz and Meyer-Lindenberg, 2012). As a consequence, there has been renewed interest in (i) dimensional approaches to psychopathological classification (Helzer *et al.*, 2008; Robbins *et al.*, 2012; Wittchen *et al.*, 2010), (ii) the development of behavioural tasks and neuroimaging protocols allowing the assessment of cognitive-affective dysfunctions as endophenotypes of mental disorders without exclusive reliance on subjective reports, and (iii) and the search for transdiagnostic core mechanisms that account for shared symptoms across diagnostic categories (Buckholtz and Meyer-Lindenberg, 2012; Morris and Cuthbert, 2012).

In this position paper, I argue that dysfunctions of decision-making, volition, and cognitive control, and aberrant interactions between the underlying large-scale brain systems involved in valuation, performance monitoring, and cognitive control may represent such transdiagnostic mechanisms and possibly constitute vulnerability factors for a wide range of mental disorders. Depending on which processing components are affected (e.g. valuation, cognitive control, salience processing), distinct patterns of cognitive, affective, and motivational dysfunction may result which often cut across diagnostic categories. From this perspective, I will identify central gaps in our current knowledge and specify the most pressing needs for future research on neurocognitive mechanisms of mental disorders.

State-of-the-art: advances in cognitive-affective neuroscience of decision-making, volition, and cognitive control

Human goal-directed action is characterized by a remarkable flexibility and future-directedness, which is evident in our ability to rapidly reconfigure behavioural dispositions in response to changing goals, to flexibly adapt to changing contexts and task demands, and to pursue long-term goals even if this requires delaying rewards, suppressing habitual responses, or resisting immediate temptations. The processes underlying goal-directed action can be classified roughly into *decision-making processes*, which mediate the selection of goals and the formation of intentions, and *volitional* or *cognitive control processes*, which support the realization of chosen intentions, especially when they stand in conflict with competing goals, habits, or motivations. In the past decade, substantial advances have been made in elucidating the cognitive, affective, and neural underpinnings of decision-making and cognitive control by combining behavioural tasks from experimental psychology and decision science with advanced neuroimaging techniques and computational modeling approaches. Importantly, this research has made increasingly clear that the cognitive control of goal-directed action is not mediated by a monolithic "central executive" system exerting top-down control over subordinate sensory and motor systems, but rather emerges from a distributed network of interacting large-scale brain systems (e.g. Banich, 2009; Gruber and Goschke, 2004; O'Reilly *et al.*, 2010). There is

corresponding evidence that cognitive and affective dysfunctions in mental disorders can in most cases not be mapped in a one-to-one manner to dysfunctions in localized brain areas, but reflect aberrant patterns of connectivity and interactions between large-scale brain systems (Buckholtz and Meyer-Lindenberg, 2012; Bühringer *et al.*, 2008; Menon, 2011). While this idea has a long tradition in neuropsychological disconnection models, the non-invasive analyses of adaptive as well as aberrant brain systems interactions have rapidly expanded in recent years due to methodological advances in the analyses of resting-state and task-related functional connectivity, whole-brain multivariate pattern analyses, and graph-theoretical models of anatomical connectivity (for reviews see Buckholtz and Meyer-Lindenberg, 2012; Bullmore and Sporns, 2012; Menon, 2011; Orru *et al.*, 2012). For understanding dysfunctions of decision-making and volitional control in mental disorders, interactions between three large-scale brain systems appear particularly important:[1]

(i) A *valuation and motivation network* which comprises the ventromedial prefrontal cortex (vmPFC), orbitofrontal cortex (OFC), ventral striatum (VS), and amygdala as core nodes, and which mediates the computation of value signals and reward-prediction errors and the assignment of values to states, goals and actions (Peters and Büchel, 2010a).

(ii) A *cognitive control network* which comprises the lateral PFC and parietal cortex (PPC) and mediates the active maintenance of goals and context information, the inhibition of prepotent but unwanted responses, and the top-down modulation of perceptual, emotional, and response processes (Miller and Cohen, 2001).

(iii) A *salience and monitoring network* which includes the anterior insula, anterior cingulate cortex (ACC), and extended amygdala as core nodes (Menon and Uddin, 2010; Sridharan *et al.*, 2008), and is involved in the regulation of vigilance, arousal and negative affect (Shackman *et al.*, 2011), the detection of significant stimuli, the monitoring of response conflicts and the signaling of the demand for enhanced cognitive control (Botvinick *et al.*, 2004; Mansouri *et al.*, 2009).

In the following sections I first summarize key findings on the role of these networks in decision-making and goal-directed behaviour and then discuss how dysfunctions in these networks may contribute to mental disorders. Based on this selective review, I derive gaps in current and major needs for future research.

Decision-making rests on interactions between multiple learning and valuation systems[2]

A wide range of mental disorders is characterized by maladaptive behavioural choices (e.g. when an addicted person continues to consume drugs despite being aware of the adverse long-term consequences; or when an individual decides not to attend an important work meeting because of an overwhelming social fear). Of direct relevance for decision-making dysfunctions in mental disorders, research on reward-based learning and value-based choice has revealed that the assignment of subjective values to goals and actions and the selection of actions based on such values involve multiple learning and memory systems (Dayan and Daw, 2008; Delgado and Dickerson, 2012). One influential distinction has been made between "model-free" and "model-based" learning. *Model-free learning* denotes the incremental trial-and-error learning of action values based on reward and punishment, according to principles specified in reinforcement learning models (Niv, 2009). The associations between cues, actions, and rewards acquired by this system are inflexible and change slowly, as indicated by insensitivity of choices to reinforce devaluation. Model-free learning has been associated with the basal ganglia and midbrain dopamine system, in particular the dorsomedial and dorsolateral striatum involved in action-effect learning and habit formation, respectively, and the ventral striatum involved in the coding of "reward prediction errors" (Maia, 2009; Schultz, 2007).

In contrast, *model-based learning* mediates the formation of internal models of sequential contingencies between stimuli, actions, and their outcomes, and supports choices based on anticipated future consequences (costs and benefits) of possible actions. This system is thought to underlie goal-directed behaviour and supports the rapid reversal and flexible adjustment of behaviour to changing contingencies. It has been related to a brain network including the vmPFC and OFC involved in the computation of model-based value signals (Rangel and Hare, 2010). In addition, brain structures involved in episodic future thinking and anticipation of possible action outcomes (including the hippocampus and lateral PFC; Schacter *et al.*, 2007) play a role in supporting an orientation towards the pursuit of long-term goals (Peters and Büchel, 2010b). Although recent evidence indicates that the distinction between model-based and model-free systems may not be as clear-cut as previously assumed (for review see Doll *et al.*, 2012), it can be concluded that decision-making rests on interactions – and at times competition – between multiple learning, memory, and valuation systems. As

described later, aberrant interactions among these systems are a characteristic of various mental disorders, including substance use disorder, attention deficit hyperactivity disorder (ADHD), eating disorders, and obsessive-compulsive disorder (OCD).

Volition can be functionally decomposed into a set of cognitive control mechanisms

After a decision has been made and an intention to attain a particular goal has been formed, the subsequent pursuit of the chosen goal often requires the recruitment of cognitive control processes to shield the intention from distracting stimuli, habitual responses, or competing motivational tendencies (e.g. when the commitment to quit smoking is weakened by a cue-elicited craving for a cigarette, or when the intention to attend an important meeting is challenged by an overwhelming social fear). In this context, the term "volition" does not refer to the idea of a causally undetermined "free will", but denotes a set of *cognitive control processes* that serve to configure sensory, affective, motivational, and motor processes according to superordinate goals and support the realization of intentions in the face of conflict from competing responses (Goschke, 2013; Haggard, 2008; Kuhl and Goschke, 1994; Miller and Cohen, 2001). The term volition thus overlaps with related terms such as "executive functions" (Banich, 2009; Hofmann *et al.*, 2012; Miyake *et al.*, 2000) or "self-control" (Cohen and Lieberman, 2009). Examples of cognitive control mechanisms include (i) the active maintenance and shielding of goal representations, (ii) the top-down modulation of perceptual and response systems, (iii) the inhibition of unwanted habitual or impulsive responses, (iv) the rapid updating and flexible switching of goals and behavioural dispositions, (v) the monitoring of conflicts and errors, and (vi) the self-regulation of emotions. The context-sensitive recruitment of such control processes is a precondition for adaptive and flexible goal-directed action and the maintenance of intentions in the face of distractions or temptations (Goschke, 2003; Hassin *et al.*, 2010; Kuhl and Goschke, 1994; Miller and Cohen, 2001). Conversely, as described later, impairments of cognitive control are assumed to increase the risk of self-control failures in both non-pathological cases as well as in mental disorders such as addiction (Bühringer *et al.*, 2008; Goldstein and Volkow, 2011; Heatherton and Wagner, 2011).

The neural basis of cognitive control is assumed to reside in a network of brain systems which includes the lateral PFC and PPC as core nodes. Importantly, this control network and the PFC in particular do not represent a unitary "central executive", but can be functionally fractionated into several subsystems. These include the dorsolateral PFC (dlPFC) involved in goal maintenance, task switching, and top-down modulation of attention; the right inferior frontal cortex involved in response inhibition; the ventrolateral PFC and inferior-frontal junction involved in the retrieval and implementation of response rules (Badre, 2008; Brass *et al.*, 2005; Crone *et al.*, 2006; Miller and Cohen, 2001), and the medial PFC, OFC and subgenual ACC involved in emotion regulation (Gyurak *et al.*, 2011). Accordingly, the once popular (yet homunculus-like) idea of a central executive is increasingly being replaced by explicit computational models specifying information-processing mechanism mediating component processes of cognitive control (O'Reilly *et al.*, 2010). Examples include models of how hierarchical goal structures are represented in prefrontal networks (Badre and D'Esposito, 2009; Botvinick *et al.*, 2009; Koechlin and Summerfield, 2007; Reynolds and O'Reilly, 2009), how cognitive control is recruited in response to conflicts (Botvinick *et al.*, 2004; Brown and Braver, 2005), how the balance between stable maintenance and flexible updating of goals is regulated dynamically (Montague *et al.*, 2004; O'Reilly and Frank, 2006), and how the PFC interacts with subcortical systems involved in reward, emotion, and motivation (Daw *et al.*, 2005; Dayan and Daw, 2008; Frank and Claus, 2006).

Cognitive control is modulated by conflict signals, emotions, and stress

Importantly, the cognitive control network does not simply exert top-down control on "lower-level" systems, but is in turn strongly modulated by brain systems involved in salience processing and conflict monitoring, as well as by systems involved in the processing of emotions, reward, and stress.

First, with respect to systems involved in salience and conflict monitoring, there is evidence that the context-sensitive recruitment of cognitive control processes depends critically on conflict signals assumed to be generated in medial prefrontal regions. According to an influential conflict-monitoring model (Botvinick *et al.*, 2004; Mansouri *et al.*, 2009), the ACC is part of a monitoring system that mediates the detection of potentially significant stimuli and response conflicts at different levels of processing, and that signals the demand for enhanced recruitment of cognitive control to nodes of the cognitive control network, notably the lateral PFC.

Second, there is increasing evidence that both the cognitive control network and the monitoring network

are strongly modulated by emotions, reward, and acute stress. While the pattern of emotional modulations of cognitive control is complex and only partly understood (for reviews see Bolte and Goschke, 2010; Chiew and Braver, 2011; Mars *et al.*, 2011), there is increasing evidence that positive affect modulates the balance between stable goal shielding versus flexible switching (Dreisbach and Goschke, 2004; van Wouwe *et al.*, 2011). Moreover, both positive and negative affect exert distinct influences on the balance between focused versus broadly distributed attention (Rowe *et al.*, 2007; Vermeulen, 2010) and on the monitoring of errors and conflicts (e.g. Wiswede *et al.*, 2009; for review see Dreisbach and Fischer, 2012).

Third, chronic as well as acute stress – in addition to its well established effects on hippocampus-dependent declarative memory – exerts strong effects on cognitive control processes, presumably via influences of stress hormones and catecholamines (noradrenaline, dopamine) on prefrontal neurotransmission (for review see Arnsten, 2009). These stress-related modulations appear to induce a shift of behavioural control from a goal-directed ("top-down") to an affective-habitual ("bottom-up") mode dominated by the amygdale and basal ganglia (Ramos and Arnsten, 2007; Wang *et al.*, 2007; Wingard and Packard, 2008). In addition, recent evidence indicates that acute social stress also shifts the balance between cognitive flexibility and stability towards increased tonic goal shielding and reduced context-sensitive adaptation of cognitive control (Plessow *et al.*, 2011a, 2011b). Stress-induced modulations of cognitive control are likely to play an important role in mental disorders and appear to increase, for instance, proneness to relapse in substance use disorder as well as reinstatement of fear responses after extinction in anxiety disorders.

Global control modes reflect variations in meta-control parameters which can be linked to specific neuromodulatory systems

The described modulatory influences on cognitive control are of direct relevance for the central question of how the mode of operation and the pattern of interaction of large-scale brain systems is regulated and dynamically adapted to changing internal states and external contexts and task-demands. In this context a central unresolved question concerns the factors which determine how the balance between stable maintenance and "shielding" of goals, on the one hand, and flexible goal switching and exploration of alternative options, on the other hand, is dynamically regulated. From a computational perspective one promising approach assumes that the mode of operation and interaction between complementary control systems depend on a limited set of *meta-control parameters*, which determine global properties of neural information-processing (Doya, 2008; Goschke, 2013). Examples of such parameters include:

- the *updating threshold*, which regulates the balance between stable maintenance and flexible updating of goal representations;
- the *attention breadth*, which regulates the balance between focused attention (promoting the selection of task-related information) versus distributed attention (promoting stimulus-driven capture by potentially significant stimuli);
- the *temporal discounting rate*, which determines how steeply delayed reward is discounted;
- the *degree of noise* in neural representations which modulates the balance between the exploitation of learnt knowledge and the (trial-and-error) exploration of novel options;
- the *learning rate*, which determines how rapidly previous knowledge and acquired associations between cues, actions, and rewards are changed by new experiences.

The settings of these parameters determine *global control states* that can be defined as particular configurations and operating modes of large-scale brain systems. Meta-control parameters and the resulting control states are influenced by multiple factors on different time scales (ranging from transient influences of task demands, deliberate strategies, phasic emotions, and acute stress, to more enduring effects of genetic variation, learning history, and personality traits). On a neurobiological level meta-control parameters have been linked to the influence of specific neuromodulatory systems on interactions between brain systems involved in emotion and reward, salience and conflict monitoring, and cognitive control (Doya, 2008; Robbins and Arnsten, 2009; Rogers, 2011). For instance, recent evidence implicates dopaminergic systems in regulating the balance between robust maintenance and flexible updating of working memory representations (e.g. Armbruster *et al.*, 2012; Müller *et al.*, 2007; for reviews see Cools, 2008; Durstewitz and Seamans, 2008; O'Reilly, 2006; van Schouwenburg *et al.*, 2010), whereas serotonergic systems appear to play a role in regulating the delay discounting rate and thus the degree of impulsivity in intertemporal decision-making (Doya, 2008; Schweighofer *et al.*, 2008; Tanaka *et al.*, 2007). Given that dysfunctional changes in neuromodulatory systems have long been implicated in mental disorders, it is an

important question how they contribute to dysfunctional meta-control parameter settings in different disorders.

Implications for research on cognitive-affective mechanisms of mental disorders

Insights gained from research on decision-making and cognitive control have important implications for research on mental disorders. As noted earlier, cognitive-affective dysfunctions in mental disorders can in most cases not be mapped one-to-one to dysfunctions in localized brain areas but involve aberrant interactions and connectivity between valuation, salience, and cognitive control networks (for reviews see Buckholtz and Meyer-Lindenberg, 2012; Bullmore and Sporns, 2012; Menon, 2011; Orru *et al.*, 2012). Such dysfunctions may appear in at least three forms:

(a) *Dysfunctions within a particular system* (e.g. impaired functioning of systems mediating inhibitory control leading to a reduced ability to suppress habitual or impulsive responses and a predominance of stimulus-driven behaviour).
(b) *Dysfunctional connectivity and interaction between systems* (e.g. a hypo-active conflict-monitoring network leading to insufficient recruitment of control in the case of conflict, thereby increasing the likelihood of self-control failures).
(c) *Dysfunctional settings of meta-control parameters* (e.g. a pathologically increased updating threshold leading to reduced cognitive flexibility and perseverative behaviour).

Depending on which networks and meta-control parameters are affected, dysfunctions may show up in different cognitive-affective processing domains:

- Dysfunctions of *valuation and decision-making* may show up in an inappropriate assignment of values to actions, an overweighting of short-term reward at the expense of long-term goals pursuit, and adaptation to changing reward contingencies.
- Dysfunctions of *cognitive control* may show up in a wide range of "executive dysfunctions", including impaired goal maintenance, reduced self-control and insufficient top-down inhibition of habitual or impulsive responses, elevated distractibility, or reduced cognitive flexibility.
- Dysfunctions of *salience signaling and conflict monitoring* may either show up as an insufficient recruitment of cognitive control due to a *hypo*-sensitive conflict-monitoring network, or conversely, as an increased capture of attention by potential threat cues, constant worrying and rumination, and impaired emotion regulation due to a *hyper*-vigilant salience network.

Valuation network dysfunction

Valuation network dysfunctions adversely affect the learning and assignment of values to actions, goals, and objects, and can thereby promote disadvantageous or seemingly irrational decisions. There is considerable evidence for aberrant activity and connectivity in brain systems involved in value-based behavioural choice (vmPFC, OFC, VS, ACC) in various mental disorders. On a gross level, valuation network dysfunctions can be grouped into those involving *reduced sensitivity* to long-term outcomes (presumably related to externalizing spectrum disorders such as addiction) and those involving *hyper-sensitivity* to possible future outcomes (e.g. in certain cases of anhedonia, generalized anxiety disorder, and possibly OCD).

Paradigmatic examples of a *reduced* impact of long-term outcomes on behavioural choice are substance use disorder and non-substance related "behavioural addictions" (e.g. pathological gambling), which involve continued maladaptive choices despite the person's awareness of the adverse consequences. It is well established that most drugs of abuse impact on the mesolimbic dopamine system (Volkow *et al.*, 2008). Chronic drug abuse induces enduring changes in reward-based learning systems which lead to the attribution of excessive incentive salience to drugs at the expense of natural reinforcers and which can cause cue-induced "wanting" even after hedonic "liking" of the drug has diminished (for reviews see Hyman, 2005; Loth *et al.*, 2011; Maia and Frank, 2011; Robinson and Berridge, 2003; Volkow *et al.*, 2008). Moreover, a recent meta-analysis of intertemporal choice studies revealed that individuals meeting criteria for an addictive disorder showed significantly greater discounting of delayed rewards (MacKillop *et al.*, 2011). Importantly, given that acute stress appears to influence delay discounting (Stewart, 2008) and shifts the balance of behavioural control from goal-directed to habitual control (Arnsten, 2009), it can increase proneness to relapse even after periods of abstinence.

In line with the earlier discussion of multiple (model-based and model-free) learning and valuation systems, it has been suggested that drug-induced changes in corticostriatal pathways lead to a transition from voluntary goal-directed to habitual and ultimately compulsive drug-seeking and consumption behaviour (Everitt *et al.*, 2008; Everitt and Robbins, 2005). Of note, high impulsivity (presumably reflecting both valuation and cognitive

control network dysfunction) may be a predisposing factor for the transition to compulsive behaviour (Robbins *et al.*, 2012). Consistent with this idea, it has recently been reported that both stimulant-dependent individuals and their non-drug abusing siblings were impaired in a motor inhibition task and showed structural abnormalities in fronto-striatal brain circuits, suggesting that impaired inhibitory control may constitute an endophenotypes for at least some types of drug addiction (Ersche *et al.*, 2012). Interestingly, despite clear differences between substance use disorders, behavioural addictions, OCD, and compulsive eating disorders, there appears to be considerable overlap across these disorders with respect to behavioural and neurocognitive signatures of impulsivity and compulsivity, suggesting that aberrant interactions between valuation and cognitive control networks may give rise to partly shared phenotypical features (Robbins *et al.*, 2012).

It should be noted that not only hypo-sensitivity but also *hyper*-sensitivity to future consequences as indicated by little discounting of delayed rewards appears to be involved in some types of mental disorders. For instance, it has been proposed that chronically reduced responsiveness to immediate reward and hyper-sensitivity to long-term future outcomes may constitute a vulnerability factor for anhedoniain depression (Hasler, 2012; Lempert and Pizzagalli, 2010).

Cognitive control network dysfunction

Dysfunctions of the cognitive control network can increase the risk for mental disorders via a number of mechanisms, including (1) impaired goal maintenance in face of competing desires, (2) a compromised ability to inhibit habitual or impulsive responses, (3) an increased proneness to attentional capture by disorder-related cues (e.g. drug-cues, threat-cues), (4) deficient foresight and meta-cognitive control (e.g. insufficient planning to avoid tempting situations in addiction), (5) reduced ability to regulate stress or negative emotions (e.g. in drug craving), (6) reduced cognitive flexibility. These cognitive control deficits contribute to two main types of impulsivity: (i) impulsive choice (especially in conjunction with valuation network dysfunction) indicated by over-valuation of immediate rewards, poor foresight and planning, and impaired ability to down-regulate current desires or emotional impulses (Kim and Lee, 2011); and (ii) impaired response inhibition, as indicated by an impaired ability to suppress or stop the execution of prepotentor habitual responses (Dalley *et al.*, 2011; Munakata *et al.*, 2011).

Cognitive control deficits and aberrant connectivity between nodes of the cognitive control network (e.g. dlPFC, PPC, ACC) are prominent features of a wide range of both "externalizing" and "internalizing" spectrum disorders, including impulse disorders, substance abuse, behavioural addictions, ADHD (Grant and Potenza, 2012), as well as schizophrenia, major depressive disorder, mania, and anxiety disorders.

Cognitive control impairments in addiction

Addictive disorders are among the most obvious instances of cognitive control network dysfunction (Bühringer *et al.*, 2008; Goldstein and Volkow, 2011; van Holst *et al.*, 2010). In addition to dysfunctions in reward and valuation networks, substance abuse is associated with impaired performance and aberrant brain activity in tasks requiring goal maintenance, response inhibition, or cognitive flexibility (Bechara *et al.*, 2006; Bühringer *et al.*, 2008; Feil *et al.*, 2010; Garavan and Stout, 2005; George and Koob, 2010; Goldstein and Volkow, 2011; Perry *et al.*, 2011; Van den Oever *et al.*, 2010). Increasing evidence indicates that similar cognitive control impairments are present in non-substance related addictive behaviours such as pathological gambling (Clark, 2010; Frascella *et al.*, 2010; Grant *et al.*, 2010; Marazziti *et al.*, 2008; van Holst *et al.*, 2010). While addiction clearly involves multiple distal and proximal vulnerabilities and risk factors (Redish *et al.*, 2008; Wittchen *et al.*, 2008), these findings strongly suggest that addiction involves two core mechanisms: (1) a dysfunctional change of valuation systems leading to the attribution of excessive incentive salience to drugs or addictive behaviours at the expense of reduced incentive value of natural reinforcers; (2) an impaired cognitive control network leading to insufficient top-down regulation of behaviour by long-term goals.

Cognitive control impairments in anxiety disorders

Dysfunctions of the cognitive control network and aberrant connectivity of the ACC, vmPFC, and amygdala have been associated with impaired emotion regulation and dysfunctional expression, inhibition, and extinction of fear responses in anxiety disorders (Etkin *et al.*, 2011). Recent evidence suggests that dysfunctions of the dorsal-caudal ACC and medial PFC may play a role in dysfunctional appraisal and expression of negative emotions and monitoring of emotional conflicts (Etkin *et al.*, 2006), whereas the ventral-rostral medial PFC appears to be involved in the top-down regulation of the generation of emotional responses (Etkin *et al.*, 2011).

Cognitive control impairments in depression

Deficits in cognitive control and impaired inhibition of task-irrelevant and especially negative emotional information have been recognized as a characteristic of depressive disorders, especially in tasks requiring effortful processing (for review see Gotlib and Joormann, 2010). Of note, in contrast to consistent evidence for hyper-vigilance and automatic attention capture by threat-related stimuli in anxiety disorders, there is less clear evidence for increased perceptual bias towards negative material in depression. Rather, depressed individuals tend to engage in extensive elaborative processing of negative material and have difficulty to disengage from negative thoughts once they have gained access to working memory (Goschke, 1996; Gotlib and Joormann, 2010). On a neural level, depression has been associated with reduced dlPFC activation following errors and negative feedback, possibly reflecting insufficient recruitment of cognitive control in situations requiring increased effort. Whereas in control subjects, conflicts, errors, or effortful tasks induce increased recruitment of task-related and control networks (in conjunction with *deactivation* of the default mode network involved in self-related cognition and self-monitoring; Menon, 2011), there is evidence that depressed individuals fail to recruit task-related and control networks to a sufficient degree in such conditions (Pizzagalli, 2011). Moreover, it has been proposed that hyperactivity of ventral PFC and limbic areas increases negative affect and processing of negative emotional stimuli, while hypoactivity of dlPFC and ACC during effortful tasks or following errors reflects insufficient recruitment of cognitive control, which together may contribute to a reduced ability to disengage from or inhibit negative thoughts and emotions (Murrough *et al.*, 2011; Pizzagalli, 2011).

Salience and monitoring network dysfunction

Dysfunctions of salience and monitoring networks can show up either as hyposensitivity or hypersensitivity for salient, motivationally significant, or conflict-related information, and such dysfunctions contribute to mental disorders in particular in combination with impaired cognitive control functions.

Hyposensitivity of the conflict-monitoring network appears to be a characteristic of substance use disorders, as indicated by impaired conflict- and error-monitoring and attenuated error-related ACC activity in opiate addicts (Forman *et al.*, 2004), cannabis users (Hester *et al.*, 2009) and cocaine addicts (Franken *et al.*, 2007). Dysfunctional interactions between ACC and the dlPFC may lead to a vicious circle, wherein impaired monitoring and signaling of conflicts between drug-triggered responses and long-term goals has the consequence that cognitive control is no longer recruited, thus leading to an insufficient modulation of action selection by long-term goals and an impaired ability to inhibit cue-triggered habitual responses (Bühringer *et al.*, 2008; Goldstein and Volkow, 2011).

Conversely, *hypersensitivity* of the salience network – especially in conjunction with cognitive control impairments – likely contributes to symptoms such as worry, generalized anxiety, or obsessive intrusive thoughts. Anxiety disorders are associated with hyper-reactivity to fear cues, and some of them are particularly characterized by more unspecific worry (e.g. generalized anxiety disorder). For both hyperreactivity and worry, meta-analyses of neuroimaging studies have revealed substantial overlap in the neural circuitry involved in specific phobias, social anxiety disorders, and post-traumatic stress disorder (PTSD) (Etkin and Wager, 2007).[3] In particular, there is relatively consistent evidence for hypereactivity in core nodes of the salience network (e.g. amygdala and insula), which presumably reflects increased sensitivity to conditioned fear-cues and elevated interoceptive monitoring of arousal and aversive states (Etkin *et al.*, 2011; LeDoux, 2012; Pessoa and Adolphs, 2010). Of note, aberrant interactions between the salience network and the cognitive control network have also been observed in other disorders, including substance dependence, conduct disorder, major depression, reactive aggression, and schizophrenia. Thus, hypersensitivity of the salience network and aberrant connectivity with the cognitive control network may give rise to impaired processing and dysfunctional regulation of negative emotions across diagnostic categories (Erk *et al.*, 2010; Etkin and Schatzberg, 2011).

Dysfunctional meta-control parameter settings

Apart from dysfunctions of systems involved in decision-making and cognitive control, mental disorders appear also to be associated with chronic dysfunctional settings of *meta-control parameters* (see earlier). It is important to note that particular control parameter settings usually incur complementary costs and benefits. For instance, a high *updating threshold* makes working memory resistant to distraction, but may also incur a cost in terms of reduced cognitive flexibility and perseverative thoughts or behaviours; a narrow *attention breadth* reduces interference from distracting stimuli, but also increases the risk of overlooking potentially significant information; a small *temporal discounting rate* reduces impulsivity and promotes future-oriented decisions, but may also lead to

Int. J. Methods Psychiatr. Res. 23(Suppl. 1): 41–57 (2014). DOI: 10.1002/mpr

over-suppression of current needs or alienation from important implicit motives. As a consequence, a chronic, context-insensitive fixation of control parameters at extreme values is likely to cause dysfunctional behaviour and may constitute a vulnerability factor for specific mental disorders. As an example, it has been proposed (Rolls *et al.*, 2008) that some of the symptoms of OCD reflect overly stable attractor states in prefrontal neural networks (overly high updating threshold), which has the consequence that cognitive states are rendered too stable and the cognitive system remains locked in a given state and cannot easily switch to another state. While this does clearly not provide a comprehensive account of the complex phenomenology of OCD, it may capture a core mechanism contributing to persistent intrusive thoughts (obsessions) and repetitive behaviours (compulsions) (see also Verduzco-Flores *et al.*, 2012). Conversely, positive symptoms of schizophrenia and hypomanic states in bipolar disorder have been related to the opposite control parameter setting, i.e. a pathologically low updating threshold and overly broad scope of attention (Rolls and Deco, 2011). Further examples for dysfunctional control parameters include an overly high delay discounting rate in substance use disorder, pathological gambling, and mania; or an excessive cost valuation and low exploration rate in depression (for further discussion see Hasler, 2012).

Major gaps and unresolved questions

Despite substantial progress, there are still profound gaps in our current understanding of the neurocognitive mechanisms underlying dysfunctions of decision-making and volitional control and their role in mental disorders.

Gap 1: Lack of knowledge about commonalities and differences of neurocognitive mechanisms across disorders

The first gap is our lack of empirical data concerning the question to which degree dysfunctions of decision-making and cognitive control reflect shared neurocognitive mechanisms which cut across mental disorders or whether different component processes (e.g. valuation of future outcomes, response inhibition, goal maintenance) are specifically impaired in different disorders. Moreover, it is an open question how to account for differences in phenotypical symptoms in different disorders despite often shared dysfunctions in overlapping neurocognitive systems (e.g. Lueken *et al.*, 2011). Finally, it is an unresolved issue to which degree non-pathological impairments of cognitive control (e.g. daily self-control failures) and dysfunctions of cognitive control in mental disorders (e.g. addiction) differ qualitatively or lie on a continuum of increasing severity.

Gap 2: Lack of empirical studies on dysfunctional meta-control parameter settings in mental disorders

A second gap concerns the question how specific phenotypical features of mental disorders relate to dysfunctional settings of meta-control parameters. Specifically, little is known about how dysfunctional control parameter settings develop, how they are moderated by genetic variation and learning history, and how they modulated by acute or chronic stress. Moreover, on a neurobiological level we have insufficient knowledge of how dysfunctional parameter settings relate to specific neuromodulatory systems and their influence on interactions between large-scale brain systems involved in valuation, behavioural choice, and cognitive control.

Gap 3: Are cognitive control impairments cause or consequence of mental disorders?

One of the most important gaps is our lack of empirical data on the question whether impaired decision-making and cognitive control are consequences (or byproducts) of mental disorders, or whether these impairments constitute antecedent vulnerability factors or mediating mechanisms increasing the risk for the development of mental disorders. For instance, it is unknown whether impaired cognitive control and conflict-monitoring is a consequence of prolonged substance abuse, or whether pre-morbid control impairments increase the risk for developing addictive behaviours in vulnerable developmental phases, under conditions of acute stress, or in specific social contexts. Likewise, it is unclear whether a hypersensitive salience network is a consequence of impaired emotion regulation in anxiety disorders, or whether it does increase the vulnerability for developing anxiety disorders. Similarly, it is an open question whether impaired cognitive control increases the vulnerability for ruminative negative thoughts in depression, or whether negative emotionality and rumination cause cognitive control deficits.

Gap 4: Generalized competence impairment or context-specific performance failure?

Closely related to gap 3 is the question whether cognitive control functions are *generally* impaired in particular mental disorders or whether control breaks down only in *specific* disorder-related contexts (e.g. in the presence of drug cues or threat cues or under acute stress)? Answering

these questions is of great relevance as it has potentially important implications for treatment (e.g. whether one should attempt to train basic cognitive control skills such as the ability to counteract attentional biases, or whether one should focus rather on motivational or situational factors supporting the context-sensitive mobilization of control functions).

Gap 5: Motivational or volitional dysfunction?

A fifth gap relates to the question whether phenotypical features of mental disorders (e.g. maladaptive decision-making in drug abuse; dysfunctional avoidance in anxiety disorders) reflect *motivational* and *decision-making* deficits (e.g. maladaptive valuation and disadvantageous choice behaviour due to an overly high discounting of future rewards and long-term goals), or whether they reflect genuine deficits in *volitional control* processes (e.g. impaired response inhibition or insufficient goal shielding) despite otherwise intact reasoning and decision-making capabilities. This knowledge is highly relevant for the development of targeted interventions, for instance, with respect to the question whether behavioural change and health-promoting behaviour should better be supported by enhancing *motivational* incentives (e.g. by enhancing the motivational impact of long-term rewards or strengthening the commitment to health goals) or by strengthening *volitional* and *cognitive control* skills (e.g. by training the ability to control attention in order to counteract attentional biases).

Gap 6: Lack of research on detrimental consequences of chronic over-control

Current research is primarily focused on detrimental consequences of impaired or insufficient cognitive control in mental disorders and there is a relative neglect of studies on possible dysfunctional consequences of excessive or chronic *over*-control. Although on a phenotypical level certain disorders seem to be associated with increased levels of effortful cognitive control (e.g. anorexia nervosa), to date relatively few studies have directly investigated whether and under which conditions chronic over-control (e.g. the continuous suppression of emotional responses or reward processes) has detrimental consequences such as recurrent intrusive thoughts, emotional rebound effects, or self-alienation from basic needs and implicit motives.

Gap 7: Lack of computational models

On a theoretical level, arguably the most profound gap in current research on mental disorders is the one between the phenotypical description of mental disorders and computational theories of the underlying information-processing mechanisms. This contrasts sharply with the fact that the field of computational cognitive neuroscience has tremendously expanded in the past two decades, as is evident in an increasing number of sophisticated neural network and dynamical system models, which specify in detail the neurocognitive mechanisms underlying reward learning, value-based decision-making, and cognitive control (Botvinick, 2012; O'Reilly *et al.*, 2010). At the same time, computational approaches are also increasingly integrated in "model-based" neuroimaging studies. In these studies, estimates of control parameters (e.g. learning rate; temporal discounting rate) are derived by fitting a computational model to subjects' behavioural performance and then relating these parameters to signatures of neural processes in order to identify brain systems involved in the respective computations (Doll *et al.*, 2012; Mars *et al.*, 2010; O'Doherty *et al.*, 2007). Computational models have great potential for the analysis of mental disorders and are a precondition for classifications of disorders based on detailed models of underlying neurocognitive mechanisms and for explanatory theories of how phenotypical expressions of mental disorders emerge from dysfunctional meta-control parameter settings and aberrant cognitive-affective systems interactions. However, apart from a few notable exceptions (Maia and Frank, 2011; Maia and McClelland, 2012; Redish *et al.*, 2007; Rolls and Deco, 2011) such models have had to date only fragmented impact on the conceptualization of mental disorders in mainstream clinical psychology and psychiatry (Maia and Frank, 2011; Montague *et al.*, 2012).

Needs for future research

Need 1: Increased research focus on cognitive-affective mechanisms underlying decision making and cognitive control dysfunctions

There is a strong need for intensified research on patterns and mechanisms of decision-making and cognitive control dysfunctions across different mental disorders. In particular, sufficiently powered studies are needed to investigate the pattern of commonalities and differences in cognitive-affective mechanisms across disorders to elucidate to which degree dysfunctions in different component processes (e.g. disadvantageous valuation; impaired response inhibition; attentional bias) reflect shared transdiagnostic mechanisms or constitute disorder-specific impairments. Moreover, there is a strong need for studies examining systematically relations between phenotypical features of mental disorders and dysfunctional constellations of

meta-control parameters. In particular, studies are needed investigating (i) the pattern of dysfunctional control parameter settings (e.g. an overly steep temporal discounting rate in the valuation of future rewards; or an excessively high updating threshold leading to perseveration and cognitive inflexibility) across disorders, (ii) the modulation of control parameters by acute or chronic stress, and (iii) the relation of dysfunctional parameter settings to influences of specific neuromodulatory systems on brain systems involved in learning, valuation and cognitive control. Moreover, studies are needed that systematically investigate to which degree non-pathological impairments of decision-making and cognitive control (e.g. in the case of daily self-control failures) and pathological dysfunctions in clinical conditions (e.g. in addictive behaviour or eating disorders) are characterized by *qualitatively* different mechanisms or by *gradual* differences in the severity of impairment.

Need 2: Reliable behavioural tasks assessing decision-making and cognitive control functions

Directly related to the first need is the requirement for more systematic integration of behavioural tasks and paradigms from experimental psychology and decision science into studies of mental disorders. Although the recent upsurge of neuroimaging studies and the rapid development of advanced methods for the analysis of functional and effective connectivity in mental disorders have yielded a host of important findings, the gain of neuroimaging studies depends critically on the use of behavioural paradigms that allow assessing reliably and validly component processes of decision-making and cognitive control. Thus, there is a pressing need to adapt and validate behavioural tasks and experimental paradigms which are firmly grounded in psychological theory for the study of cognitive control and decision-making dysfunctions in mental disorders. This is a precondition for elucidating how specific patterns of cognitive and affective dysfunctions relate to phenotypical features of mental disorders across diagnostic categories. Moreover, in the longer run such studies are a precondition for the development of *mechanism-targeted training and intervention* aiming to improve specific component skills and strategies as, for instance, training of inhibitory control, voluntary attention focusing, or reflective decision making strategies (for promising effects of attention control on self-controlled choice and craving reduction in smokers see Peters and Büchel, 2010a; Kober *et al.*, 2010; for recent reviews of the effectiveness of attention control training in anxiety disorder see MacLeod and Mathews, 2012).

Need 3: Systematic use of latent-variable models of cognitive control functions and dysfunctions

A third need is the more systematic use of latent-variable models of cognitive control functions in studies of mental disorders. Behavioural tasks used to measure cognitive control are in most cases not "process-pure" but task performance reflects several component processes, which is particularly true for many of the complex tasks that have traditionally been used to assess executive functions (cf. Miyake *et al.*, 2000). Moreover, tasks assessing cognitive control often show only moderate retest reliability (Strauss *et al.*, 2006). To alleviate the task-impurity and reliability problem, studies investigating the predictive validity of cognitive control impairments for phenotypic expressions of mental disorders should assess cognitive control functions with more than one task in order to derive latent variable scores. This would allow analysing relations between cognitive control impairments and mental disorders on the level of latent constructs (e.g. response inhibition, set shifting) rather than in terms of performance in individual tasks (Bollen and Noble, 2011; Miyake *et al.*, 2000).

Need 4: Systematic integration of behavioural tasks and neuroimaging measures into longitudinal-prospective studies

There is a strong need for studies with sufficiently powered longitudinal-prospective designs which systematically incorporate comprehensive sets of behavioural tasks assessing component processes of cognitive control and decision-making functions alongside with neuroimaging measures and theory-driven assessments of genetic variation. Such studies would allow relating behavioural measures (preferably on the level of latent variable scores), associated patterns of brain activity and genetic variation in neurotransmitter systems linked to the regulation of meta-control parameters to phenotypical expressions of mental disorders (preferably derived both from clinical assessments and experience sampling methods). The high cost of such large-scale studies appears justified in light of promising recent findings indicating that laboratory measures of decision-making and cognitive control as well as associated brain activation patterns are indeed predictive of real-life decision-making and behavioural choice. For instance, measures of inhibitory control and delay discounting have been shown to predict daily self-control failures in smokers attempting to quit (Berkman *et al.*, 2011), relapse and potentially onset of addictive behaviours (George and Koob, 2010), initiation of smoking behaviour in adolescents (Audrain-McGovern *et al.*, 2009), relapse in adolescent smokers after a smoking cessation programme (Krishnan-

Sarin *et al.*, 2007). Moreover and quite remarkably, self-control assessed in preschoolers predicted cognitive and social competence and stress tolerance a decade later (Eigsti *et al.*, 2006; Mischel *et al.*, 1988, 2011).

Need 5: From dual systems models to theories of large-scale brain systems interactions

It is becoming increasingly clear that simple dichotomies (e.g. between an "impulsive" and a "reflective" system) – notwithstanding their unquestionable heuristic value – fall short of doing justice to the complexity of the neural circuits and component processes underlying adaptive as well as maladaptive behavioural control. We thus need studies using advanced methods for assessing functional and effective connectivity to investigate how multiple learning, valuation and cognitive control systems cooperate or compete in determining value signals and choice behaviour, and how these interactions are modulated by acute stress or emotional states. Importantly, such studies should examine whether phenotypically similar behavioural manifestations of decision-making or control dysfunctions are mediated by the same or distinct processing pathways. For instance, recent evidence indicates that self-controlled decisions may not always involve the direct inhibition of an "impulsive" valuation system by a "reflective" control system (McClure *et al.*, 2004), but rather rests on the modulation of a common value signal in the vmPFC by anticipated long-term consequences of a behaviour (Hare *et al.*, 2009). We thus not only need to take the idea of cooperative and competitive interactions between large-scale brain networks seriously, but also the possibility of multiple mediating pathways towards cognitive-affective dysfunctions in mental disorders. For instance, using addiction as an example, studies are needed that systematically investigate under which conditions impaired cognitive control in addictive behaviours reflects: (a) impaired top-down control of cue-triggered habitual or impulsive responses; (b) deficient anticipation of one's own *future desires* leading to insufficient strategic avoidance of tempting situations; or (c) impaired emotion regulation leading to an impaired ability to cope with stress or negative emotions during craving and thereby increasing the risk of relapse.

Need 6: Increased focus on implicit cognitive-affective processes

As suggested by the earlier discussion of multiple learning and valuation systems, there is a need for more systematic research on interactions between explicit and implicit goals, valuations, and motives in mental disorders. While it is still common practice in research on mental disorders to rely mainly or even exclusively on self-report measures assessing introspectively accessible beliefs, feelings, and goals, there is strong evidence that especially apparently "irrational" choices as in addiction or anxiety disorders cannot be explained solely by conscious beliefs and goals, but depend on implicit attitudes and motives (Gawronski and Payne, 2010; Hassin *et al.*, 2005; Stacy and Wiers, 2010). For instance, a recent meta-analysis (Rooke *et al.*, 2008) revealed that *indirect* tests supposed to assess implicit attitudes and valuations reliably predicted addictive behaviour. Importantly, recent experimental evidence indicates that the influence of implicit attitudes and automatic affective reactions on actual choice behaviour is systematically moderated by cognitive control competencies as assessed by measures of executive attention, response inhibition, and emotion regulation (Friese *et al.*, 2010; Hofmann *et al.*, 2009; Nederkoorn *et al.*, 2010; Wiers *et al.*, 2010). Thus, more studies are needed which simultaneously obtain measures of conscious goals and attitudes, cognitive control competencies, and measures of implicit affective attitudes and automatic valuations, in order to investigate how dysfunctional interactions between these processes relate to disadvantageous choices in mental disorders.

Need 7: From symptoms to mechanisms: integrating computational modeling into psychopathology

Last but not least, to make progress at a theoretical level a most pressing need is to put more emphasis on the integration of computational modeling approaches into research on mental disorders. Especially in light of the earlier described recent advances in computational cognitive neuroscience, the time appears ripe to apply neural network and dynamical system models to questions of clinical relevance (cf. Maia and Frank, 2011; Maia and McClelland, 2012; Montague *et al.*, 2012; Redish *et al.*, 2007; Rolls and Deco, 2011). In particular, closer integration of computational models into psychopathology provides an avenue towards explanations of how phenotypical manifestations of mental disorders such as impaired cognitive control may emerge from non-linear interactions among underlying neurocognitive mechanisms, dysfunctional meta-control parameter settings, and aberrant systems interactions on different levels of analysis. Moreover, computational models generate novel testable hypotheses about common mediating mechanism across disorders. As case in point, connectionist models have been shown to account for reinstatement of learnt behaviours after extinction across diagnostic categories (substance use disorder, problem gambling, and fear extinction) in terms of the same underlying mechanism

(Redish *et al.*, 2007). Computational models thus may substantially contribute to refined classifications of disorders based on explicit neurocognitive mechanisms.

Conclusion

To conclude, the study of decision-making and cognitive control has made impressive progress on both psychological and neurobiological levels of analysis, thanks to the integration of behavioural tasks derived from experimental psychology, advanced neuroimaging methods for analysing large-scale brain systems interactions, and computational modeling approaches. Applying this multidisciplinary approach more systematically to research on mental disorders carries not only the promise to elucidate how phenotypical characteristics of mental disorders emerge from transdiagnostic dysfunctions of core cognitive-affective mechanisms, but may in the long run help to develop mechanism-based rather than symptom-based classifications and treatments of mental disorders.

Acknowledgements

Preparation of this article was partly supported by the German Science Foundation (SFB 940/1 2013).

This article was generated as part of the activities of the group of leading European experts on psychological research and intervention, in order to provide an assessment of the state-of-the-art of research in different domains, identifying major advances and promising methods and pointing out gaps and problems which ought to be addressed in future research. A similar critical appraisal with partly similar conclusions is concurrently provided elsewhere (Schumann *et al.*, 2013) by the ROAMER work group "Biomedical research". Experts in both work groups have been selected for their academic excellence and for their competence in the different units of analysis needed to comprehensively characterize particular symptom domains. Their contributions do not aim to be systematic reviews of the field but rather provide a well-informed opinion of the authors involved. They also do not represent official statements of the ROAMER consortium, but are meant to inform the discussion on psychological research and intervention in mental disorders among interested stakeholders, including researchers, clinicians and funding bodies. Recommendations made in this issue will undergo a discussion and selection process within the ROAMER consortium, and contribute to a final roadmap, which integrates all aspects of mental health research. We thus hope to provide an informed and comprehensive overview of the current state of psychological research in mental health, as well as the challenges and advances ahead of us.

Declaration of interest statement

The author has no competing interests.

Endnotes

1. Due to space limitations I will not discuss the role of the so-called "default mode network" in mental disorders (cf. Buckholtz and Meyer-Lindenberg, 2012; Menon, 2011).
2. Here I exclusively focus on the role of learning systems for decision-making. Learning processes do, of course, play a much broader role in mental disorders (e.g. with respect to emotional-associative learning in anxiety disorders), which is beyond the scope of this paper.
3. The present focus on transdiagnostic commonalities is not meant to deny that there are most likely also important differences in the mediating mechanisms of different anxiety disorders.

References

Arnsten A.F.T. (2009) Stress signalling pathways that impair prefrontal cortex structure and function. *Nature Reviews Neuroscience*, **10**(6), 410–422.

Armbruster D.J., Ueltzhöffer K., Basten U., Fiebach C.J. (2012) Prefrontal cortical mechanisms underlying individual differences in cognitive flexibility and stability. *Journal of Cognitive Neuroscience*, **24**(12), 2385–2399.

Audrain-McGovern J., Rodriguez D., Epstein L.H., Cuevas J., Rodgers K., Wileyto, E.P. (2009) Does delay discounting play an etiological role in smoking or is it a consequence of smoking? *Drug and Alcohol Dependence*, **103**(3), 99–106.

Badre D. (2008) Cognitive control, hierarchy, and the rostro-caudal organization of the frontal lobes. *Trends in Cognitive Sciences*, **12**(5), 193–200.

Badre D., D'Esposito M. (2009) Is the rostro-caudal axis of the frontal lobe hierarchical? *Nature Reviews Neuroscience*, **10**(9), 659–669.

Banich M.T. (2009) Executive function. The search for an integrated account. *Current Directions in Psychological Science*, **18**(2), 89–94.

Bechara A., Noel X., Crone E.A. (2006) Loss of willpower: abnormal neural mechanisms of impulse control and decision making in addiction. In Wiers R.W., Stacy A.W. (eds) *Handbook of implicit cognition and addiction*, pp. 215–232, Thousand Oaks, CA, Sage Publications.

Berkman E.T., Falk E.B., Lieberman M.D. (2011) In the trenches of real-world self-control: neural correlates of breaking the link between craving and smoking. *Psychological Science*, **22**(4), 498–506.

Bollen K.A., Noble M.D. (2011) Structural equation models and the quantification of behavior. *Proceedings of the National Academy of Sciences of the United States of America*, **108**(Suppl. 3), 15639–15646.

Bolte A., Goschke T. (2010) Thinking and emotion: affective modulation of cognitive processing modes. In Glatzeder B., Goel V., Müller A.v. (eds) *On Thinking, Vol. II: Towards a Theory of Thinking*, Heidelberg, Springer.

Botvinick M.M. (2012) Hierarchical reinforcement learning and decision making. *Current Opinion in Neurobiology*, **22**(6), 956–962.

Botvinick M.M., Cohen J.D., Carter C.S. (2004) Conflict monitoring and anterior cingulate cortex: an update. *Trends in Cognitive Sciences*, **8**(12), 539–546.

Botvinick M.M., Niv Y., Barto A.C. (2009) Hierarchically organized behavior and its neural

foundations: a reinforcement learning perspective. *Cognition*, **113**(3), 262–280.

Brass M., Derrfuss J., Forstmann B., von Cramon D.Y. (2005) The role of the inferior frontal junction area in cognitive control. *Trends in Cognitive Sciences*, **9**(7), 314–316.

Brown J.W., Braver T.S. (2005) Learned predictions of error likelihood in the anterior cingulate cortex. *Science*, **307**(5712), 1118–1121.

Buckholtz J.W., Meyer-Lindenberg A. (2012) Psychopathology and the human connectome: toward a transdiagnostic model of risk for mental illness. *Neuron*, **74**(6), 990–1004.

Bühringer G., Wittchen H.-U., Gottlebe K., Kufeld C., Goschke, T. (2008) Why people change? The role of cognitive-control processes in the onset and cessation of substance abuse disorders. *International Journal of Methods in Psychiatric Research*, **17**(Suppl. 1), S4–S15.

Bullmore E., Sporns O. (2012) The economy of brain network organization. *Nature Reviews Neuroscience*, **13**(5), 336–349.

Chiew K.S., Braver T.S. (2011) Positive affect versus reward: emotional and motivational influences on cognitive control. *Frontiers in Psychology*, **2**, 279.

Clark L. (2010) Decision-making during gambling: an integration of cognitive and psychobiological approaches. *Philosophical Transactions of the Royal Society of London, Series B: Biological Sciences*, **365**(1538), 319–330.

Cohen J.R., Lieberman M.D. (2009) The common neural basis of exerting self-control in multiple domains. In Hassin R.R., Ochsner K., Trope Y. (eds) *Self control in society, mind, and brain*, pp. 141–160, Oxford, Oxford University Press.

Cools R. (2008) Role of dopamine in the motivational and cognitive control of behavior. *The Neuroscientist*, **14**(4), 381–395.

Crone E.A., Wendelken C., Donohue S.E., Bunge S. A. (2006) Neural evidence for dissociable components of task-switching. *Cerebral Cortex*, **16**(4), 475–486.

Dalley J.W., Everitt B.J., Robbins T.W. (2011) Impulsivity, compulsivity, and top-down cognitive control. *Neuron*, **69**(4), 680–694.

Daw N.D., Niv Y., Dayan P. (2005) Uncertainty-based competition between prefrontal and dorsolateral striatal systems for behavioral control. *Nature Neuroscience*, **8**(12), 1704–1711.

Dayan P., Daw N.D. (2008) Decision theory, reinforcement learning, and the brain. *Cognitive, Affective, & Behavioral Neuroscience*, **8**(4), 429–453.

Delgado M.R., Dickerson K.C. (2012) Reward-related learning via multiple memory systems. *Biological Psychiatry*, **72**(2), 134–141.

Doll B.B., Simon D.A., Daw N.D. (2012) The ubiquity of model-based reinforcement learning. *Current Opinion in Neurobiology*, **22**(6), 1075–1081.

Doya K. (2008) Modulators of decision making. *Nature Neuroscience*, **11**(4), 410–416.

Dreisbach G., Fischer R. (2012) The role of affect and reward in the conflict-triggered adjustment of cognitive control. *Frontiers in Human Neuroscience*, **6**, 342, DOI: 10.3389/fnhum. 2012.00342

Dreisbach G., Goschke T. (2004) How positive affect modulates cognitive control: reduced perseveration at the cost of increased distractibility. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, **30**(2), 343–353.

Durstewitz D., Seamans J.K. (2008) The dual-state theory of prefrontal cortex dopamine function with relevance to catechol-o-methyltransferase genotypes and schizophrenia. *Biological Psychiatry*, **64**(9), 739–749.

Eigsti I.-M., Zayas V., Mischel W., Shoda Y., Ayduk O., Dadlani M.B., Davidson M.C., Lawrence Aber J., Casey B.J. (2006) Predicting cognitive control from preschool to late adolescence and young adulthood. *Psychological Science*, **17**(6), 478–484.

Erk S., Mikschl A., Stier S., Ciaramidaro A., Gapp V., Weber B., Walter H. (2010) Acute and sustained effects of cognitive emotion regulation in major depression. *Journal of Neuroscience*, **30**(47), 15726–15734.

Ersche K.D., Simon Jones P., Williams G.B., Turton A.J., Robbins T.W., Bullmore E.T. (2012) Abnormal brain structure implicated in stimulant drug addiction. *Science*, **335**(6068), 601–604.

Etkin A., Egner T., Kalisch R. (2011) Emotional processing in anterior cingulate and medial prefrontal cortex. *Trends in Cognitive Sciences*, **15**(2), 85–93.

Etkin A., Egner T., Peraza D.M., Kandel E.R., Hirsch J. (2006) Resolving emotional conflict: a role for the rostral anterior cingulate cortex in modulating activity in the amygdala. *Neuron*, **51**(6), 871–882.

Etkin A., Schatzberg A.F. (2011) Common abnormalities and disorder-specific compensation during implicit regulation of emotional processing in generalized anxiety and major depressive disorders. *American Journal of Psychiatry*, **168**(9), 968–978.

Etkin A., Wager T.D. (2007) Functional neuroimaging of anxiety: a meta-analysis of emotional processing in PTSD, social anxiety disorder, and specific phobia. *American Journal of Psychiatry*, **164**(10), 1476–1488.

Everitt B.J., Belin D., Economidou D., Pelloux Y., Dalley J.W., Robbins T.W. (2008) Review. Neural mechanisms underlying the vulnerability to develop compulsive drug-seeking habits and addiction. *Philosophical Transactions of the Royal Society of London, Series B: Biological Sciences*, **363**(1507), 3125–3135.

Everitt B.J., Robbins T.W. (2005) Neural systems of reinforcement for drug addiction: from actions to habits to compulsion. *Nature Neuroscience*, **8**(11), 1481–1489.

Feil J., Sheppard D., Fitzgerald P.B., Yucel M., Lubman D.I., Bradshaw J.L. (2010) Addiction, compulsive drug seeking, and the role of frontostriatal mechanisms in regulating inhibitory control. *Neuroscience & Biobehavioral Reviews*, **35**(2), 248–275.

Forman S.D., Dougherty G.G., Casey B.J., Siegle G. J., Braver T.S., Barch D.M., Stenger V.A., Wick-Hull C., Pisarov L.A., Lorensen E. (2004) Opiate addicts lack error-dependent activation of rostral anterior cingulate. *Biological Psychiatry*, **55**(5), 531–537.

Frank M.J., Claus E.D. (2006) Anatomy of a decision: striato-orbitofrontal interactions in reinforcement learning, decision making, and reversal. *Psychological Review*, **113**(2), 300–326.

Franken I.H., van Strien J.W., Franzek E.J., van de Wetering B.J. (2007) Error-processing deficits in patients with cocaine dependence. *Biological Psychology*, **75**(1), 45–51.

Frascella J., Potenza M.N., Brown L.L., Childress A. R. (2010) Shared brain vulnerabilities open the way for nonsubstance addictions: carving addiction at a new joint? *Annals of the New York Academy of Sciences*, **1187**(Feb), 294–315.

Friese M., Bargas-Avila J., Hofmann W., Wiers R. W. (2010) Here's looking at you, Bud: alcohol-related memory structures predict eye movements for social drinkers with low executive control. *Social Psychological and Personality Science*, **1**(2), 143–151.

Garavan H., Stout J.C. (2005) Neurocognitive insights into substance abuse. *Trends in Cognitive Sciences*, **9**(4), 195–201.

Gawronski B., Payne B.K. (eds). (2010) *Handbook of implicit social cognition: measurement, theory, and applications*, New York, Guilford Press.

George O., Koob G.F. (2010) Individual differences in prefrontal cortex function and the

transition from drug use to drug dependence. *Neuroscience & Biobehavioral Reviews*, **35**(2), 232–247.

Goldstein R.Z., Volkow N.D. (2011) Dysfunction of the prefrontal cortex in addiction: neuroimaging findings and clinical implications. *Nature Reviews Neuroscience*, **12**(11), 652–669.

Goschke T. (1996) Gedächtnis und emotion: affektive bedingungen des einprägens, behaltens und vergessens. In Albert D., Stapf K.-H. (eds) *Enzyklopädie der Psychologie Serie II, Band 4: Gedächtnis*, pp. 605–694, Göttingen, Hogrefe.

Goschke T. (2003) Voluntary action and cognitive control from a cognitive neuroscience perspective. In Maasen S., Prinz W., Roth G. (eds) *Voluntary action: brains, minds, and sociality*, pp. 49–85, New York, Oxford University Press.

Goschke T. (2013) Volition in action: intentions, control dilemmas and the dynamic regulation of intentional control. In Prinz W., Beisert A., Herwig A. (eds) *Action Science: Foundations of an Emerging Discipline*, Cambridge, MA, MIT Press.

Gotlib I. H., Joormann J. (2010) Cognition and depression: current status and future directions. *Annual Review of Clinical Psychology*, **6**, 285–312.

Grant J.E., Potenza M.N. (eds). (2012) *The Oxford Handbook of Impulse Control Disorders*, Oxford, Oxford University Press.

Grant J.E., Potenza M.N., Weinstein A., Gorelick D.A. (2010) Introduction to behavioral addictions. *American Journal of Drug and Alcohol Abuse*, **36**(5), 233–241.

Gruber O., Goschke T. (2004) Executive control emerging from dynamic interactions between brain systems mediating language, working memory and attentional processes. *Acta Psychologica*, **115**(2), 105–121.

Gyurak A., Gross J.J., Etkin A. (2011) Explicit and implicit emotion regulation: a dual-process framework. *Cognitive Emotion*, **25**(3), 400–412.

Haggard P. (2008) Human volition: towards a neuroscience of will. *Nature Reviews Neuroscience*, **9**(12), 934–946.

Hare T.A., Camerer C.F., Rangel A. (2009) Self-control in decision-making involves modulation of the vmPFC valuation system. *Science*, **324**(5927), 646–648.

Hasler G. (2012) Can the neuroeconomics revolution revolutionize psychiatry? *Neuroscience & Biobehavioral Reviews*, **36**(1), 64–78.

Hassin R.R., Ochsner K., Trope Y. (eds). (2010) *Self control in society, mind, and brain*, Oxford, Oxford University Press.

Hassin R.R., Uleman J.S., Bargh J.A. (2005) *The New Unconscious*, New York, Oxford University Press.

Heatherton T.F., Wagner D.D. (2011) Cognitive neuroscience of self-regulation failure. *Trends in Cognitive Sciences*, **15**(3), 132–139.

Helzer J.E., Kraemer H.C., Krueger R.F., Wittchen H.-U., Sirovatka P.J., Regier D.A. (2008) *Dimensional Approaches in Diagnostic Classification: Refining the Research Agenda for DSM-V*, Washington, DC, American Psychiatric Publishing.

Hester R., Nestor L., Garavan H. (2009) Impaired error awareness and anterior cingulate cortex hypoactivity in chronic cannabis users. *Neuropsychopharmacology*, **34**(11), 2450–2458.

Hofmann W., Friese M., Strack F. (2009) Impulse and self-control from a dual-systems perspective. *Psychological Science*, **4**(2), 162–176.

Hofmann W., Schmeichel B.J., Baddeley A.D. (2012) Executive functions and self-regulation. *Trends in Cognitive Sciences*, **16**(3), 174–180.

Hyman S.E. (2005) Addiction: a disease of learning and memory. *American Journal of Psychiatry*, **162**(8), 1414–1422.

Kim S., Lee D. (2011) Prefrontal cortex and impulsive decision making. *Biological Psychiatry*, **69**(12), 1140–1146.

Kober H., Kross E.F., Mischel W., Hart C.L., Ochsner K.N. (2010) Regulation of craving by cognitive strategies in cigarette smokers. *Drug and Alcohol Dependence*, **106**(1), 52–55.

Koechlin E., Summerfield C. (2007) An information theoretical approach to prefrontal executive function. *Trends in Cognitive Sciences*, **11**(6), 229–235.

Krishnan-Sarin S., Reynolds B., Duhig A.M., Smith A., Liss T., McFetridge A., Cavallo D.A., Carroll K.M., Potenza M.N. (2007) Behavioral impulsivity predicts treatment outcome in a smoking cessation program for adolescent smokers. *Drug and Alcohol Dependence*, **88**(1), 79–82.

Kuhl J., Goschke T. (1994) A theory of action control: mental subsystems, modes of control, and volitional conflict-resolution strategies. In Kuhl J., Beckmann J. (eds) *Volition and personality: action versus state orientation*. Göttingen/Toronto, Hogrefe.

LeDoux J. (2012) Rethinking the emotional brain. *Neuron*, **73**(4), 653–676.

Lempert K.M., Pizzagalli D.A. (2010) Delay discounting and future-directed thinking in anhedonic individuals. *Journal of Behavior Therapy and Experimental Psychiatry*, **41**(3), 258–264.

Loth E., Carvalho F., Schumann G. (2011) The contribution of imaging genetics to the development of predictive markers for addictions. *Trends in Cognitive Sciences*, **15**(9), 436–446.

Lueken U., Kruschwitz J.D., Muehlhan M., Siegert J., Hoyer J., Wittchen H.U. (2011) How specific is specific phobia? Different neural response patterns in two subtypes of specific phobia. *NeuroImage*, **56**(1), 363–372.

MacKillop J., Amlung M.T., Few L.R., Ray L.A., Sweet L.H., Munafo M.R. (2011) Delayed reward discounting and addictive behavior: a meta-analysis. *Psychopharmacology (Berlin)*, **216**(3), 305–321.

MacLeod C., Mathews A. (2012) Cognitive bias modification approaches to anxiety. *Annual Review of Clinical Psychology*, **8**(1), 189–217.

Maia T.V. (2009) Reinforcement learning, conditioning, and the brain: successes and challenges. *Cognitive, Affective, & Behavioral Neuroscience*, **9**(4), 343–364.

Maia T.V., Frank M.J. (2011) From reinforcement learning models to psychiatric and neurological disorders. *Nature Neuroscience*, **14**(2), 154–162.

Maia T.V., McClelland J.L. (2012) A neurocomputational approach to obsessive-compulsive disorder. *Trends in Cognitive Sciences*, **16**(1), 14–15.

Mansouri F.A., Tanaka K., Buckley M.J. (2009) Conflict-induced behavioural adjustment: a clue to the executive functions of the prefrontal cortex. *Nature Reviews Neuroscience*, **10**(2), 141–152.

Marazziti D., Catena Dell'Osso M., Conversano C., Consoli G., Vivarelli L., Mungai F., Di Nasso E., Golia F. (2008) Executive function abnormalities in pathological gamblers. *Clinical Practice and Epidemiology in Mental Health*, **4**(1), 7.

Mars R.B., Sallet J., Rushworth M., Yeung N. (2011) *Neural basis of motivational and cognitive control*, Cambrdige, MA, MIT Press.

Mars R.B., Shea N.J., Kolling N., Rushworth M.F. (2010) Model-based analyses: promises, pitfalls, and example applications to the study of cognitive control. *Quarterly Journal of Experimental Psychology*, **65**(2), 1–16.

McClure S.M., Laibson D.I., Loewenstein G., Cohen J.D. (2004) Separate neural systems value immediate and delayed monetary rewards. *Science*, **306**(5695), 503–507.

Menon V. (2011) Large-scale brain networks and psychopathology: a unifying triple network model. *Trends in Cognitive Sciences*, **15**(10), 483–506.

Menon V., Uddin L.Q. (2010) Saliency, switching, attention and control: a network model of insula function. *Brain Structure and Function*, **214**(5–6), 655–667.

Miller E.K., Cohen J.D. (2001) An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience*, **24**, 167–202.

Mischel W., Ayduk O., Berman M.G., Casey B.J., Gotlib I.H., Jonides J., Kross E., Teslovich T., Wilson N.L., Zayas V., Shoda Y. (2011) 'Willpower' over the life span: decomposing self-regulation. *Social Cognitive and Affective Neuroscience*, **6**(2), 252–256, DOI: 10.1093/scan/nsq081

Mischel W., Shoda Y., Peake P.K. (1988) The nature of adolescent competencies predicted by preschool delay of gratification. *Journal of Personality and Social Psychology*, **54**(4), 687–696.

Miyake A., Friedman N.P., Emerson M.J., Witzki A.H., Howerter A., Wager T.D. (2000) The unity and diversity of executive functions and their contributions to complex 'frontal lobe' tasks: a latent variable analysis. *Cognitive Psychology*, **41**(1), 49–100.

Montague P.R., Dolan R.J., Friston K.J., Dayan P. (2012) Computational psychiatry. *Trends in Cognitive Sciences*, **16**(1), 72–80.

Montague P.R., Hyman S.E., Cohen J.D. (2004) Computational roles for dopamine in behavioural control. *Nature*, **431**(7010), 760–767.

Morris S.E., Cuthbert B.N. (2012) Research domain criteria: cognitive systems, neural circuits, and dimensions of behavior. *Dialogues in Clinical Neuroscience*, **14**(1), 29–37.

Müller J., Dreisbach G., Brocke B., Lesch K.-P., Strobel A., Goschke T. (2007) Dopamine and cognitive control: The prospect of monetary gains influences the balance between flexibility and stability in a set-shifting paradigm. *European Journal of Neuroscience*, **26**(12), 3661–3668.

Munakata Y., Herd S.A., Chatham C.H., Depue B. E., Banich M.T., O'Reilly R.C. (2011) A unified framework for inhibitory control. *Trends in Cognitive Sciences*, **15**(10), 453–459.

Murrough J.W., Iacoviello B., Neumeister A., Charney D.S., Iosifescu D.V. (2011) Cognitive dysfunction in depression: neurocircuitry and new therapeutic strategies. *Neurobiology of Learning and Memory*, **96**(4), 553–563.

Nederkoorn C., Houben K., Hofmann W., Roefs A., Jansen A. (2010) Control yourself or just eat what you like? Weight gain over a year is predicted by an interactive effect of response inhibition and implicit preference for snack foods. *Health Psychology*, **29**(4), 389–393.

Niv Y. (2009) Reinforcement learning in the brain. *Journal of Mathematical Psychology*, **53**(3), 139–154.

O'Doherty J.P., Hampton A., Kim H. (2007) Model-Based fMRI and its application to reward learning and decision making. *Annals of the New York Academy of Sciences*, **1104**(1), 35–53.

O'Reilly R.C. (2006) Biologically based computational models of high-level cognition. *Science*, **314**(5796), 91–94.

O'Reilly R.C., Frank M.J. (2006) Making working memory work: a computational model of learning in the prefrontal cortex and basal ganglia. *Neural Computation*, **18**(2), 283–328.

O'Reilly R.C., Herd S.A., Pauli W.M. (2010) Computational models of cognitive control. *Current Opinion in Neurobiology*, **20**(2), 257–261.

Orru G., Pettersson-Yeo W., Marquand A.F., Sartori G., Mechelli A. (2012) Using support vector machine to identify imaging biomarkers of neurological and psychiatric disease: a critical review. *Neuroscience & Biobehavioral Reviews*, **36**(4), 1140–1152.

Perry J.L., Joseph J.E., Jiang Y., Zimmerman R.S., Kelly T.H., Darna M., Huettl P., Dwoskin L. P., Bardo M.T. (2011) Prefrontal cortex and drug abuse vulnerability: translation to prevention and treatment interventions. *Brain Research Reviews*, **65**(2), 124–149.

Pessoa L., Adolphs R. (2010) Emotion processing and the amygdala: from a 'low road' to 'many roads' of evaluating biological significance. *Nature Reviews Neuroscience*, **11**(11), 773–782.

Peters J., Büchel C. (2010a) Neural representations of subjective reward value. *Behavioral and Brain Research*, **213**(2), 135–141.

Peters J., Büchel C. (2010b) Episodic future thinking reduces reward delay discounting through an enhancement of prefrontal-mediotemporal interactions. *Neuron*, **66**(1), 138–148.

Pizzagalli D.A. (2011) Frontocingulate dysfunction in depression: toward biomarkers of treatment response. *Neuropsychopharmacology*, **36**(1), 183–206.

Plessow F., Fischer R., Kirschbaum C., Goschke T. (2011a) Inflexibly focused under stress: acute psychosocial stress increases shielding of action goals at the expense of reduced cognitive flexibility with increasing time lag to the stressor. *Journal of Cognitive Neuroscience*, **23**(11), 3218–3227.

Plessow F., Kiesel A., Kirschbaum C. (2011b) The stressed prefrontal cortex and goal-directed behaviour: acute psychosocial stress impairs the flexible implementation of task goals. *Experimental Brain Research*, DOI: 10.1007/s00221-011-2943-1

Ramos B.P., Arnsten A.F.T. (2007) Adrenergic pharmacology and cognition: focus on the prefrontal cortex. *Pharmacology & Therapeutics*, **113**(3), 523–536.

Rangel A., Hare T. (2010) Neural computations associated with goal-directed choice. *Current Opinion in Neurobiology*, **20**(2), 262–270.

Redish A.D., Jensen S., Johnson A. (2008) A unified framework for addiction: vulnerabilities in the decision process. *Behavioral and Brain Sciences*, **31**(4), 415–437.

Redish A.D., Jensen S., Johnson A., Kurth-Nelson Z. (2007) Reconciling reinforcement learning models with behavioral extinction and renewal: implications for addiction, relapse, and problem gambling. *Psychology Review*, **114**(3), 784–805.

Reynolds J.R., O'Reilly R.C. (2009) Developing PFC representations using reinforcement learning. *Cognition*, **113**(3), 281–292.

Robbins T.W., Arnsten A.F.T. (2009) The neuropsychopharmacology of fronto-executive function: monoaminergic modulation. *Annual Review of Neuroscience*, **32**, 267–287.

Robbins T.W., Gillan C.M., Smith D.G., de Wit S., Ersche K.D. (2012) Neurocognitive endophenotypes of impulsivity and compulsivity: towards dimensional psychiatry. *Trends in Cognitive Sciences*, **16**(1), 81–91.

Robinson T.E., Berridge K.C. (2003) Addiction. *Annual Review of Psychology*, **54**, 25–53.

Rogers R.D. (2011) The roles of dopamine and serotonin in decision making: evidence from pharmacological experiments in humans. *Neuropsychopharmacology*, **36**(1), 114–132.

Rolls E.T., Deco G. (2011) A computational neuroscience approach to schizophrenia and its onset. *Neuroscience & Biobehavioral Reviews*, **35**(8), 1644–1653.

Rolls E.T., Loh M., Deco G. (2008) An attractor hypothesis of obsessive-compulsive disorder. *European Journal of Neuroscience*, **28**(4), 782–793, DOI: 10.1111/j.1460-9568.2008.06379.x

Rooke S.E., Hine D.W., Thorsteinsson E.B. (2008) Implicit cognition and substance use: a meta-analysis. *Addictive Behaviors*, **33**(10), 1314–1328.

Rowe G., Hirsh J.B., Anderson A.K. (2007) Positive affect increases the breadth of attentional selection. *Proceedings of the National Academy of Sciences of the United States of America*, **104**(1), 383–388.

Schacter D.L., Addis D.R., Buckner R.L. (2007) Remembering the past to imagine the future: the prospective brain. *Nature Reviews Neuroscience*, **8**(9), 657–661.

Schultz W. (2007) Behavioral dopamine signals. *Trends in Neuroscience*, **30**(5), 203–210.

Schumann G., Binder E., Holte A., de Kloet E.R., Odegaard K.J., Robbins T.W., Walker-Tilley T.R., Brown V.J., Buitelaar J., Ciccocioppo R., Cools R., Escera C., Fleischhacker W., Flor H., Frith C.D., Heinz A., Johnsen E., Kirschbaum C., Klingberg T., Lesch K.-P., Lewis S., Maier W., Mann K., Martinot J.-L., Meyer-Lindenberg A., Müller C.P., Müller W. E., Nutt D.J., Persico A., Perugi G., Pessiglione M., Sandi C., Stephan K.E., Undurraga J., Vieta E., van der Wee N., Preuss U.W., Roiser J., Wykes T., Haro J.M., Wittchen H.-U., Rossini P.M., Rybakowski J.K. (2013) Stratified medicine for mental disorders. *European Neuropsychopharmacology*, DOI: 10.1016/j.euroneuro.2013.09.010

Schweighofer N., Bertin M., Shishida K., Okamoto Y., Tanaka S.C., Yamawaki S., Doya K. (2008) Low-serotonin levels increase delayed reward discounting in humans. *Journal of Neuroscience*, **28**(17), 4528–4532.

Shackman A.J., Salomons T.V., Slagter H.A., Fox A. S., Winter J.J., Davidson R.J. (2011) The integration of negative affect, pain and cognitive control in the cingulate cortex. *Nature Reviews Neuroscience*, **12**(3), 154–167.

Sridharan D., Levitin D.J., Menon V. (2008) A critical role for the right fronto-insular cortex in switching between central-executive and default-mode networks. *Proceedings of the National Academy of Sciences of the United States of America*, **105**(34), 12569–12574.

Stacy A.W., Wiers R.W. (2010) Implicit cognition and addiction: a tool for explaining paradoxical behavior. *Annual Review of Clinical Psychology*, **6**, 551–575.

Stewart J. (2008) Review. Psychological and neural mechanisms of relapse. *Philosophical Transactions of the Royal Society of London, Series B: Biological Sciences*, **363**(1507), 3147–3158.

Strauss E., Sherman E.M.S., Spreen O. (2006) *A Compendium of Neuropsychological Tests. Administration, Norms, and Commentary*, 3rd edition, Oxford, Oxford University Press.

Tanaka S.C., Schweighofer N., Asahi S., Shishida K., Okamoto Y., Yamawaki S., Doya K. (2007) Serotonin differentially regulates short- and long-term prediction of rewards in the ventral and dorsal striatum. *PLoS ONE*, **2**(12), e1333.

Van den Oever M.C., Spijker S., Smit A.B., De Vries T.J. (2010) Prefrontal cortex plasticity mechanisms in drug seeking and relapse. *Neuroscience & Biobehavioral Reviews*, **35**(2), 276–284.

van Holst R.J., van den Brink W., Veltman D.J., Goudriaan A.E. (2010) Why gamblers fail to win: a review of cognitive and neuroimaging findings in pathological gambling. *Neuroscience & Biobehavioral Reviews*, **34**(1), 87–107.

van Schouwenburg M.R., Aarts E., Cools R. (2010) Dopaminergic modulation of cognitive control: distinct roles for the prefrontal cortex and basal ganglia. *Current Pharmaceutical Design*, **16**(18), 2026–2032.

van Wouwe N.C., Band G.P.H., Ridderinkhof K.R. (2011) Positive affect modulates flexibility and evaluative control. *Journal of Cognitive Neuroscience*, **23**(3), 524–539.

Verduzco-Flores S., Ermentrout B., Bodner M. (2012) Modeling neuropathologies as disruption of normal sequence generation in working memory networks. *Neural Networks*, **27** (Mar), 21–31.

Vermeulen N. (2010) Current positive and negative affective states modulate attention: an attentional blink study. *Personality and Individual Differences*, **49**(5), 542–545.

Volkow N.D., Fowler J.S., Wang G.J., Baler R., Telang F. (2008) Imaging dopamine's role in drug abuse and addiction. *Neuropharmacology*, **56**(Suppl. 1), 3–8, DOI: 10.1016/j.neuropharm.2008.05.022

Wang M., Ramos B.P., Paspalas C.D., Shu Y., Simen A., Duque A., Vijayraghavan S., Brennan A., Dudley A., Nou E., Mazer J.A., McCormick D.A., Arnsten A.F. (2007) Alpha2A-adrenoceptors strengthen working memory networks by inhibiting cAMP-HCN channel signaling in prefrontal cortex. *Cell*, **129**(2), 397–410.

Wiers R.W., Ames S.L., Hofmann W., Krank M., Stacy A.W. (2010) Impulsivity, impulsive and reflective processes and the development of alcohol use and misuse in adolescents and young adults. *Frontiers in Psychology*, **1**(Sep), 144.

Wingard J.C., Packard M.G. (2008) The amygdala and emotional modulation of competition between cognitive and habit memory. *Behavioral Brain Research*, **193**(1), 126–131.

Wiswede D., Münte T.F., Goschke T., Rüsseler J. (2009) Modulation of the error-related negativity by induction of short-term negative affect. *Neuropsychologia*, **47**(1), 83–90.

Wittchen H.-U., Behrendt S., Höfler M., Perkonigg A., Lieb R., Bühringer G., Beesdo K. (2008) What are the high risk periods for incident substance use and transitions to abuse and dependence? Implications for early intervention and prevention. *International Journal of Methods in Psychiatric Research*, **17**, 16–29.

Wittchen H.-U., Höfler M., Gloster A.T., Craske M.G., Beesdo-Baum K. (2010) Options and dilemmas of dimensional measures for DSM-V: which types of measures fare best in predicting course and outcome? In Regier D.A., Narrow W.E., Kuhl E.A., Kupfer D.J. (eds) *The Conceptual Evolution of DSM-5*, pp. 119–143, Washington, DC, American Psychiatric Publishing.

Wittchen H.-U., Jacobi F., Rehm J., Gustavsson A., Svensson M., Jönsson B., Olesen J., Allgulander C., Alonso J., Faravelli C., Fratiglioni L., Jennum P., Lieb R., Maercker A., van Os J., Preisig M., Salvador-Carulla L., Simon R., Steinhausen H.C. (2011) The size and burden of mental disorders and other disorders of the brain in Europe 2010. *European Neuropsychopharmacology*, **21**(9), 655–679.