# **Mini-Review Article:** Towards a Computational Psychiatry of Juvenile Obsessive-Compulsive Disorder

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**Abstract**

Obsessive-Compulsive Disorder (OCD) most often emerges during adolescence, but we know little about the aberrant neural and cognitive developmental mechanisms that underlie its emergence during this critical developmental period. To move towards a computational psychiatry of juvenile OCD, we review studies on the computational, neuropsychological and neural alterations in juvenile OCD and link these findings to the adult OCD and cognitive neuroscience literature. We find consistent difficulties in tasks entailing complex decision making and set shifting, but limited evidence in other areas that are altered in adult OCD, such as habit and confidence formation. Based on these findings, we establish a neurocomputational framework that illustrates how cognition can go awry and lead to symptoms of juvenile OCD. We link these possible aberrant neural processes to neuroimaging findings in juvenile OCD and show that juvenile OCD is mainly characterised by disruptions of complex reasoning systems.

**Keywords:** Juvenile Obsessive-Compulsive Disorder, Adolescence, Neuropsychology, Computational Psychiatry, Neuroimaging

## Introduction

Most mental health conditions emerge early in life. About half of all psychiatric disorders manifest themselves before the age of 14 and three-quarters by the age of 24 (Kessler et al., 2005). This developmental period is also characterised by significant growth and reorganisation of the brain (Whitaker et al., 2016; Ziegler et al., 2019). Protracted maturation, particularly in areas involved in higher-order cognition, characterises this important developmental stage (Giedd et al., 1999; Tamnes et al., 2017). However, the precise nature of these changes and how they might drive and interact with the maturation of cognitive functions is unclear. Likewise, how a derailing of neurocognitive development may lead to psychiatric disorders such as obsessive-compulsive disorder (OCD) remains unknown.

The incidence pattern of OCD has a characteristic developmental trajectory that suggests a close link with ongoing neurocognitive maturation (Kessler et al., 2005). By the age of 14, a quarter of all patients express a manifest disorder and only a few develop OCD later in life (American Psychiatric Association, 2013). This highlights the importance of a developmental perspective in order to understand the inner workings of the disorder.

A thorough investigation of the mechanisms underlying the emergence of OCD is vital for its understanding and treatment. However, current OCD research is facing three key problems. Firstly, despite substantial research on adults with OCD, far fewer studies have investigated neurocognitive deficits in juvenile OCD. Secondly, the link between brain, cognition and OCD symptoms is unclear. Several neuroimaging studies have provided insight into abnormalities in fronto-striatal loops (e.g. Brem et al., 2012; Hauser et al., 2017; Menzies et al., 2008) but while links to cognition and symptoms have been made in the adult literature (e.g. van den Heuvel et al., 2009; Kwon et al., 2003; Mataix-Cols et al., 2004) these links are still underdeveloped in juvenile OCD. It is thus unclear how impairments in these neural circuits drive aberrant cognition and give rise to OCD. Thirdly, inconsistent neuropsychiatric

findings can be observed both in the adult and paediatric literature, as well as between these two fields (Abramovitch et al., 2015; Abramovitch, Abramowitz, & Mittelman, 2013; Abramovitch & Cooperman, 2015).

In this paper, we will address these challenges directly. We will tackle the first problem by projecting well-established findings in adult OCD onto the smaller body of juvenile OCD research, explaining discrepancies and commonalities from a developmental perspective. We will argue that it is possible to close the gap between brain, cognition, and symptoms using methods of computational psychiatry (*cf.* Box 1), thus addressing the second problem. Lastly, we will present a computational framework that illustrates how the heterogeneity and diversity of cognitive deficits (third problem) can be reconciled.

In what follows, we will review areas of research related to computational psychiatry and examine the current state of research on juvenile OCD and how it relates to adult OCD. Lastly, we will present a computational framework summarising current neurocognitive evidence that provides testable hypotheses and can guide future research.

## General Decision Making and Reward Learning

OCD is often cast as a disorder of learning and decision making, but it is unclear whether OCD is characterised by fundamental cognitive biases, or whether underperformance is only manifest in more complex tasks and situations.

The symptomatology of trying to avoid negative outcomes even at high costs (e.g. carrying out compulsions for hours to prevent an unlikely catastrophe) suggests that OCD patients overestimate the likelihood that negative things will happen, or value negative events more negatively (i.e. increased loss/punishment avoidance). While some studies report an increased risk aversion in adult OCD patients (Admon et al., 2012; Sip, Gonzalez, Taylor, & Stern, 2018; Sip, Muratore, & Stern, 2016), other studies, inter alia in juvenile OCD patients,

did not find this (Drechsler et al., 2015; Hauser, Moutoussis, Iannaccone, et al., 2017; Starcke, Tuschen-Caffier, Markowitsch, & Brand, 2010; Zhang et al., 2015). In particular, computational modelling in juvenile and adult OCD patients found no evidence for altered loss and gain processing, but for altered (value independent) choice perseveration in patients (Hauser, Iannaccone, et al., 2017). This suggests these decision-making biases are more complex than simple heightened loss avoidance (Nord, Lawson, Huys, Pilling, & Roiser, 2018). Selective findings showing impaired reward processing may potentially be driven by specific subtypes and/or additional OCD-related psychiatric components such as anhedonia (Abramovitch, Pizzagalli, Reuman, & Wilhelm, 2014). However, overall studies in adult OCD patients show more subtle biases, such as a reduced reward sensitivity (Aranovich, Cavagnaro, Pitt, Myung, & Mathews, 2017), or increased randomness for gains (i.e. a violation of subjective-value maximization resulting from choosing an uncertain over a certain option; Pushkarskaya et al., 2017), with some of them replicating in juvenile OCD patients (Norman et al., 2018). This is in contrast to other disorders such as attention-deficit/hyperactivity disorder (ADHD) and depression that have shown a clearer imbalance in reward and punishment sensitivity (Gotlib et al., 2010; Masunami, Okazaki, & Maekawa, 2009; Tripp & Alsop, 1999).

Many OCD-related clinical behaviours, such as heightened threat perception that induces compulsions, are often based on experienced, rather than explicitly stated stimulus-outcome associations (e.g. compulsions relieving distress). It has thus been speculated whether patients with OCD suffer from a learning impairment, often investigated using reversal learning tasks. In fact, adult patients have been seen to take longer to perform these tasks on a similar performance level as healthy controls (Chamberlain et al., 2008; Remijnse et al., 2009; Valerius, Lumpp, Kuelz, Freyer, & Voderholzer, 2008). In juvenile OCD, however, reversal learning studies are more inconsistent (Gottwald et al., 2018; Hauser, Iannaccone, et al., 2017;

Hybel, Mortensen, Lambek, Thastum, & Thomsen, 2017).

This heterogeneity is not limited to reversal learning findings, but is also present in other studies reporting on procedural learning (Beers et al., 1999; Ornstein, Arnold, Manassis, Mendlowitz, & Schachar, 2010). A key challenge of these tasks is to capture how learning is impaired. Many require complex learning strategies which can go wrong in many different ways. Dissecting the learning mechanisms using computational models may help identify the exact processes that are altered and may help to explain some of the heterogeneity in findings and increase effect sizes.

A consistent finding in the adult literature is that OCD patients struggle with implicit learning tasks while mastering tasks with explicit instructions (Deckersbach et al., 2002; Rauch et al., 1995; Soref, Liberman, Abramovitch, & Dar, 2018). Implicit learning involves the acquisition and expression of information without awareness 06/10/2020 11:19:00and has been seen to rely on brain circuits typically impaired in OCD (Rauch et al., 1995, 2001). Findings show an overtake of explicit learning systems leading to deficiencies in dual-task paradigms (Deckersbach et al., 2002). Thus, the impairment may result from an imbalance in implicit versus explicit systems.

In line with this, OCD patients seem to consistently struggle with problems of increased complexity without explicit guidelines that require the participant to build a representation of the task structure. Difficulties seem to arise when tasks draw upon certain (learning) processes to master set-shifting and other unpredicted changes in the environment. Such deficits have been found both in adult (Chamberlain, Fineberg, Blackwell, Robbins, & Sahakian, 2006; Gu et al., 2008) and juvenile OCD patients (Britton et al., 2010; Drechsler et al., 2015; Gottwald et al., 2018; Kim et al., 2019; Wolff, Buse, Tost, Roessner, & Beste, 2017; Wolff, Giller, Buse, Roessner, & Beste, 2018), although not ubiquitously (Beers et al., 1999; Hybel et al., 2017; Ornstein et al., 2010). Most recent work showed that planning seems to be a pre-existing trait

marker for paediatric OCD (Negreiros et al., 2020), which underpins the hypothesis that models may not be used or constructed adequately by patients.

Moreover, deficiencies in adult OCD have mainly been found in the most challenging aspects of learning processes (e.g. extra-dimensional, but not intra-dimensional shifts; Chamberlain et al., 2007; Chamberlain, Fineberg, Blackwell, Robbins, & Sahakian, 2006). In contrast, other patient groups, e.g. schizophrenia and depressed patients, show more consistent difficulties in earlier, simpler stages of such tasks (Ceaser et al., 2008; Jazbec et al., 2007; Nord et al., 2018; Purcell, Maruff, Kyrios, & Pantelis, 1997). Recent work has contrasted children with OCD with children with generalized anxiety disorder showing that OCD patients had greater difficulties completing complex planning tasks while patients with general anxiety disorder made more simple reversal learning errors than OCD patients giving insight into the unique difficulties (Kim et al., 2019).

In summary, OCD (primarily juvenile) patients do not express a clear deficiency in simple decision making or learning but seem to show altered behaviours when completing complex learning problems involving the construction or adaptation of mental task spaces. This specificity of alterations further speaks against an explanation on the basis of cognitive capacity models (e.g. Eysenck & Keane, 2005; Kahneman, 1973), which would predict impairments in all highly demanding and complex task.

## Habits

A key domain that is often thought to be aberrant in OCD is (excessive) habitual behaviour. The clinical manifestation of compulsions in OCD are often described as habits (American Psychiatric Association, 2013), behaviours automatically prompted by stimuli and formed through stimulus-action association learning (Andrews, 1903). These can be contrasted

with ‘goal-directed’ behaviour that involves complex reasoning and planning to attain a set goal.

Evidence for a predisposition toward habit formation mainly comes from overtraining studies, that suggest that adult (Gillan, Apergis-Schoute, et al., 2014; Gillan et al., 2011; Gillan, Morein-Zamir, et al., 2014) as well as juvenile (Gottwald et al., 2018) OCD patients’ learned behaviour persists in the face of outcome devaluation. In these paradigms, participants first learn a stimulus-action association, which is then removed after overtraining. The habitual system is thereby assumed to only slowly adapt to such changes and thus stick to the previously learned stimulus-response sequence. However, the ability of these paradigms to induce dominant habitual behaviour in humans has been questioned lately suggesting the explanation of the cited findings might rather be an impaired complex reasoning system (de Wit et al., 2018). Moreover, a different line of research has proposed that instead of an overly dominant habitual or impaired goal-directed system, OCD patients might have difficulties arbitrating between systems (Gruner, Anticevic, Lee, & Pittenger, 2016). This hypothesis has been underpinned by neuroimaging findings showing impaired connectivity in the inferior lateral prefrontal cortex (ilPFC) in OCD, an area that plays an important role in cognitive control and complex decision-making, and alterations in the frontopolar cortex (FPC) and the anterior cingulate cortex (ACC; Anticevic et al., 2014; Fitzgerald et al., 2005; Harrison et al., 2009; Lee, Shimojo, & O’Doherty, 2014).

In the context of reinforcement learning (RL), habitual behaviour is often linked to an excessive model-free learning system, which stands in contrast to a model-based learning system ( *cf.* Box 2; Dolan & Dayan, 2013). While the latter uses a model of the task and is thus able to learn quickly, the former system relies on experienced rewards and learns slower. The impact of these systems has been studied in adult OCD using a task that allows a dissociation between these two systems by introducing a probabilistic transition structure between different

states (Daw, Gershman, Seymour, Dayan, & Dolan, 2011). Adult OCD patients have consistently exhibited a relative deficiency in the model-based system across several studies (Gillan et al., 2011, 2020; Gillan, Kosinski, Whelan, Phelps, & Daw, 2016; Gillan & Robbins, 2014; Voon et al., 2015). Recent work by Gillan and colleagues has further shown that factors such as trait and state anxiety failed to explain the consistently observed goal-directed impairments in OCD (Gillan, Vaghi, et al., 2020; Gillan et al., 2016), suggesting the impairment in these paradigms might be a characteristic of OCD beyond the effects of other important psychiatric dimensions such as anxiety.

While this association appears to be reliable in adult OCD, research on juvenile OCD patients seems to be inexistent. This research gap is of particular significance because of two reasons. Firstly, current research is unclear on whether excessive habit learning is a cause or consequence of OCD. An excessive habitual system may drive the emergence of compulsive symptoms (Robbins, Gillan, Smith, de Wit, & Ersche, 2012), but it could also be that chronic illness and/or a pre-occupation with OCD-related symptoms drains cognitive resources at the expense of a complex model-based reasoning system. Secondly, the model-based system only matures in late adolescence (Decker, Otto, Daw, & Hartley, 2016; Potter, Bryce, & Hartley, 2017). Hence, it is important to understand whether adolescents with OCD show an intact system compared to peers, or whether an impairment is already manifest. Understanding the developmental trajectories of both OCD-symptoms and the model-based system could thus provide great insight into the directionality of the association.

First longitudinal findings by Vaghi et al. (in press) in adolescents show that compulsive symptoms precede and drive the emergence of a model-based system deficiency. This suggests that the reduction in model-basedness in adult OCD might be a consequence of obsessive-compulsive (OC) behaviour rather than a driver. It remains to be determined whether the effect is primarily driven by a disorder-related draining on cognitive resources, as increased

cognitive load from other sources can reduce the degree to which an individual uses model-based decision making (Otto, Gershman, Markman, & Daw, 2013). Alternatively, an overload of symptoms (e.g. obsession) might also lead to a draining of complex control systems and consequently to observed cognitive and behavioural alterations (Abramovitch, Dar, Hermesh, & Schweiger, 2012; for further critical discussion of the habit theory also see Kalanthroff, Abramovitch, Steinman, Abramowitz, & Simpson, 2016). Taken together, these findings suggest that reduced model-based reasoning might be a consequence rather than a cause of altered neurocognitive development in OCD during adolescence.

## Indecisiveness

Following insights from clinical observation, several other cognitive symptoms have been investigated, for example indecisiveness (the inability to commit to a decision) which is often reported by OCD patients. One way to experimentally capture indecisiveness is to use tasks in which participants are free to sample as much information as they desire before committing to a choice. In these sequential information-gathering tasks, adult OCD patients have been found to sample more information (Fear & Healy, 1997; Pélissier & O'Connor, 2002; Volans, 1976; Valerie Voon et al., 2017), although not ubiquitously (Chamberlain, Fineberg, Blackwell, et al., 2007; Grassi et al., 2015).

Indecisiveness seems to be a feature present early on in the disorder. Information gathering and thus indecisiveness, was found to be exaggerated in juvenile OCD patients (Erhan et al., 2017; Hauser, Moutoussis, Iannaccone, et al., 2017). Moreover, indecisiveness seems to be a marker of compulsivity as a dimension. Non-clinical subjects with high obsessive-compulsive traits have shown a level of indecisiveness intermediate to the level associated with OCD patients and low compulsive subjects (Hauser, Moutoussis, Dayan, & Dolan, 2017).

The cognitive mechanisms underlying this indecisiveness have recently been investigated using computational modelling. Hauser et al. (2017) found that the excess in information sampling was associated with lower subjective costs for sampling information. These costs can be cast as an urgency signal (Cisek, Puskas, & El-Murr, 2009), which is delayed in high compulsive subjects and juvenile OCD patients (Hauser, Moutoussis, Iannaccone, et al., 2017; Hauser, Moutoussis, Dayan, et al., 2017). This signal modulates a decision threshold and thus explains why OCD patients were less inclined to commit to a decision earlier. Importantly, participants along the compulsivity spectrum were matched on other psychiatric dimensions such as anxiety and depression (Hauser, Moutoussis, Dayan, et al., 2017). Thus, these findings were characteristic for compulsivity independent from alternative psychiatric dimensions, indicating that there may be factors contributing to OCD that go beyond the factors contributing to e.g. anxiety and depression.

It is not entirely clear whether and how a similar indecisiveness mechanism is driving differences observed in perceptual decision making. Several studies showed that adult OCD patients and high compulsive subjects need to accumulate more perceptual evidence to arrive at a decision (Banca et al., 2015; Hauser, Allen, Rees, & Dolan, 2017). Erhan et al. (2017) found that juvenile OCD patients accumulated more sensory evidence for a longer time before making a decision to achieve a certain performance level. This could at least in part be driven by elevated decision thresholds that collapse more slowly (Erhan et al., 2017; Hauser, Moutoussis, Iannaccone, et al., 2017), similar to above-mentioned information gathering findings.

Thus, there is relatively consistent evidence for elevated indecisiveness, both in juvenile and adult OCD patients. This cognitive bias seems to be characteristic of OCD and further differentiates it from other disorders such as schizophrenia, which show the opposite behaviour (Ermakova et al., 2019; Evans, Averbeck, & Furl, 2015).

## Confidence

A separate line of research in adult OCD has focused on confidence. When external feedback is unavailable, the feeling of confidence serves as an internal appraisal signal. Confidence is often operationalised as the evidence strength in favour of a decision (Kiani, Corthell, & Shadlen, 2014). A miscalibration of this signal (i.e. overconfidence or underconfidence) is assumed to contribute to aberrant decision making and has been suggested to drive OCD symptoms, such as repetitive checking (Cuttler, Sirois-Delisle, Alcolado, Radomsky, & Taylor, 2013).

Multiple studies showed lowered confidence ratings in patients with OCD, but to our knowledge only in adult patient populations. Low confidence in OCD has been found across multiple cognitive domains, such as general knowledge (Dar, 2004; Dar, Rish, Hermesh, Taub, & Fux, 2000), memory (Boschen & Vuksanovic, 2007; Moritz et al., 2007; Tuna, Tekcan, & Topçuoğlu, 2005), and perception tasks (Sarig, Dar, & Liberman, 2012), as well as in the evaluation of internal states (Lazarov, Liberman, Hermesh, & Dar, 2014). Impaired confidence processing further seems to be a potential root for specific OCD symptoms (Boschen & Vuksanovic, 2007; Zitterl et al., 2001). For instance, diminishing confidence in a task via false feedback was predictive of doubt and checking urges (Cuttler et al., 2013). In turn, repeated checking reduced confidence but not performance (Radomsky, Dugas, Alcolado, & Lavoie, 2014). This suggests there might be a bidirectional link between low confidence, decision making and OCD symptoms that could constitute a driving force in the development of the disorder.

However, investigating confidence along the compulsivity spectrum, while excluding the factor of anxiety and depression, Rouault, Seow, Gillan and Fleming (2018) as well as Seow and Gillan (2020), showed that high compulsivity was associated with increased

confidence ratings. This is not in contradiction to mentioned findings of lowered confidence, but rather adds new detailed insight suggesting that lowered confidence in OCD might in fact be driven by anxious and depressive symptoms and related comorbidities.

A key function of confidence is to track one's performance and to inform subsequent decision-making processes. It is therefore critical to assess the relationship between performance and confidence. It could well be that OCD patients solely display a lowered average confidence (i.e. confidence bias), but accurately track their performance (i.e. display lower confidence when they are wrong and higher confidence when they are correct). Alternatively, the mapping of performance onto confidence could be corrupted (i.e. metacognitive impairment), which would be detrimental for their decision making (Fleming & Lau, 2014).

To our knowledge, only two studies have investigated metacognition in adult compulsive subjects so far. Both found that subjects with high obsessive-compulsive symptoms had reduced metacognitive ability. This means their confidence was less indicative of their actual performance (Hauser, Allen, et al., 2017; Rouault, Seow, Gillan, & Fleming, 2018). Metacognitive impairments have also been shown in compulsivity independently of anxiety or depression (Hauser et al., 2017) suggesting it might be a characteristic of OCD beyond other psychiatric dimensions. Interestingly, a mismatch between action and confidence was also found during learning in studies with OCD patients (Vaghi et al., 2017) and along the compulsivity dimension (Seow & Gillan, 2020). These findings provide an extended understanding of previously observed under-confidence relative to actual performance compared to controls (e.g. Dar, 2004; Dar et al., 2000) that already indicated a dissociation of subjective beliefs and behaviour.

This observed dissociation of beliefs and actions docks onto a thought and action fusion seen as a common clinical symptom. Thought and action fusion (TAF) entails the belief that

thinking something is equivalent to carrying out that action and that a thought of event is more likely to happen in the future (Shafran & Rachman, 2004; Shafran, Thordarson, & Rachman, 1996). The observed action-confidence misappropriation might be a mechanism related to TAF. Furthermore, 'insight' which is often assessed in clinical settings (e.g. Eisen et al., 1998) and may be understood as the awareness of a patient that their mental experiences are not based on external reality, is a relevant aspect of metacognition. It has been seen to be predictive of therapeutic success in children (e.g. Garcia et al., 2010) and adults with OCD (e.g. Foa, Abramowitz, Franklin, & Kozak, 1999) and thereby underlines the importance of metacognitive evaluations.

In summary, these findings suggest a relatively consistent confidence impairment in adult OCD patients. This impairment is likely to be at least partially driven by a mismatch between actual, objective performance and the patients' confidence (i.e. metacognitive deficit). However, there is a notable lack of studies on metacognition in juvenile OCD. This might partially be because little is known about the normative maturation of metacognitive skills. First studies indicate that metacognition matures during late adolescence (Fandakova et al., 2017; Weil et al., 2013). An indication that metacognition might already be impaired in juvenile OCD comes from a questionnaire study that showed that adolescents with high OC symptoms have altered metacognitive beliefs (Mather & Cartwright-Hatton, 2004). Whether observed lowered levels of insight in children with OCD (e.g. Lewin, Caporino, Murphy, Geffken, & Storch, 2010) is linked to deficits in metacognitive tasks needs to be determined. It is therefore most critical to better understand aberrant and normative development of metacognition. An early impairment may foster the feeling of internal uncertainty and could contribute to the development of checking compulsions that themselves increase uncertainty.

## Multiple Systems in the Brain

So far, we have discussed several different domains in which (juvenile) OCD patients show altered behaviour. In what follows we will present a schematic framework that brings these findings together and illustrates how deficits may arise from one or multiple aberrant neurodevelopmental mechanisms. This will then be underpinned by simulations of the framework that are freely accessible online (*cf.* supplementary material). Potential relevance for other psychiatric disorders such as ADHD or depression may further result from the frameworks overarching computational and neural foundation.

Here, we rely on the premise that multiple reasoning systems co-exist in the brain (Daw et al., 2011; Dolan & Dayan, 2013). These systems predict the value of specific actions in specific states and differ in their sophistication, i.e. level of complexity. We propose that the simplest models learn about the outcome of specific motor actions (based on simple feedback learning), ignoring other sensory inputs (e.g. type of stimuli on the screen). The most complex models, however, have access to a sophisticated representation (or cognitive model) of the task structure, including various hidden states and transitions between them. These reasoning systems are likely to differ in their demands: a complex system relies on slow and demanding computations, while a simple model is fast and needs only little neural computations. In general, more complex models are likely to make more accurate predictions, unless they are overly complex in which case their performance may be reduced (Gershman & Niv, 2015). It is thus of critical importance that actions are guided by the reasoning system that makes the best predictions with minimal complexity (Friston, 2013).

The majority of current empirical studies reduce this framework to a well-established dichotomous systems theory (*cf.* Box 2; Daw, Niv, & Dayan, 2005). However, recent findings from Shahar et al. (2019) prove the existence of multiple reasoning systems with a motor-spatial system complementing model-based and model-free systems. We propose that these different systems are embedded in separate, parallel feedback loops, referred to as fronto-

striatal loops. These loops are known to play an important role in learning and decision making connecting frontal regions to the striatum in a topographically organised manner (Haber, 2016). It is likely that the functional complexity of the reasoning systems increases from the posterior to the anterior regions of the frontal lobe (Miller & Cohen, 2001), ranging from the motor cortex to regions involved in complex reasoning i.e. the orbitofrontal cortex (OFC; Schuck, Cai, Wilson, & Niv, 2016) and surrounding areas (*cf.* Figure 1; Miller & Cohen, 2001; O'Doherty, Lee, & McNamee, 2015; Schoenbaum, Chiba, & Gallagher, 1998). This assumption is founded in human neuroimaging studies that have shown that anterior regions represent complex, sometimes hidden task-structures (Chan, Niv, & Norman, 2016; Schuck et al., 2016; Schuck & Niv, 2019; Wilson, Takahashi, Schoenbaum, & Niv, 2014), while simpler action-outcome associations are mainly represented in more posterior areas (for review see work by Domenech and Koechlin, 2015). This is also in line with animal literature prescribing goal-directed learning to the prelimbic area (Coutureau & Killcross, 2003) and state representations to the OFC (Baltz, Yalcinbas, Renteria, & Gremel, 2018).

We speculate that such a topographical alignment is also preserved in the basal ganglia following a ventromedial to dorsolateral gradient. Areas of the anterior prefrontal cortex (aPFC) project to medial, and posterior areas to lateral striatal regions via topographically organised fronto-striatal loops (Di Martino et al., 2008; Jarbo & Verstynen, 2015). While ventromedial regions are often associated with complex, 'future-oriented' values (Burton, Nakamura, & Roesch, 2015; Yin, Ostlund, Knowlton, & Balleine, 2005), dorsolateral regions are involved in simpler motor, associative and habitual actions (Burton et al., 2015; Yin, Knowlton, & Balleine, 2004).

If these (partially) independent systems do exist, how do they learn and how does arbitration between them take place? Dopaminergic prediction errors (PEs; Schultz, Dayan, & Montague, 1997; Schultz, 2016) may act as teaching signals for most systems. Recent advances

suggest dopamine does not carry a unified PE signal, but rather multiple distinct PE signals (Dabney et al., 2020; Takahashi et al., 2011, 2017). This is in line with findings showing that dopaminergic activity is area- and circuitry-specific and that separate fronto-striatal loops are associated with distinct dopaminergic signals supporting different value learning mechanisms (Lammel, Ion, Roeper, & Malenka, 2011; Morris et al., 2016). Therefore each loop may be associated with different dopaminergic PEs that update the specific predictions of each reasoning system.

We propose that a meta-controller selects between competing action policies to arbitrate between the systems and determine the best action (Lieder & Griffiths, 2017). A straightforward mechanism to base the arbitration on would be to track each systems' predictive accuracy over time and to form individual systems' confidence signals. Such a 'meta-confidence' (or reliability) could be formed from the (absolute) PEs of each system (Alexander & Brown, 2011; Silvetti, Seurinck, & Verguts, 2011), and subsequently, help to weight the predictions of each system for action selection. The relative difference between these meta-confidences might not only determine the executed action but also feed into the self-report of confidence. However, the meta-controller itself and the arbitration mechanism may not be conscious. How exactly confidence, as well as insight into disorder relate to this framework therefore remains to be explored.

We propose that the dorsal medial prefrontal cortex (dmPFC), encompassing the dorsal anterior cingulate cortex (dACC), could constitute a critical node of the meta-controller. The dmPFC has extensive connections to the striatum, other prefrontal and motor regions (Haber, 2016). It resides in an ideal position to collect, integrate, and select conflicting decision-related signals (Alexander & Brown, 2011). Previous work suggests that the dmPFC integrates multiple, conflicting signals into a single decision output (Alexander & Brown, 2011; Shenhav, Cohen, & Botvinick, 2016). Many functions necessary for a meta-controller involve the dACC.

The dACC has not only been found relevant for simple error monitoring and behavioural control (Amiez, Joseph, & Procyk, 2005; Brown & Braver, 2005; Heilbronner & Hayden, 2016; van Veen & Carter, 2002), but also seems to encode multiple decision variables (Heilbronner & Hayden, 2016) and control switches between different behavioural strategies (Kerns et al., 2004; Lee et al., 2014). Whether the dmPFC indeed fulfils all functions of a meta-controller needs further investigation.

Given this framework, we can now speculate how the observed cognitive biases in juvenile OCD might arise from a single altered neural process and how multiple deficits might surface in similar symptomologies, but with different underlying mechanisms.

**[Figure 1]**

## Potential Pathomechanisms

Assuming that the multiple-systems framework approximately describes what happens in the brain, one can propose at least three different ways of how such processes can go awry and lead to deficits found in OCD. These potential impairments can guide our research pursuits by taking into account the possibility that distinct OCD subgroups might suffer from different symptomologies. Neurocognitive deficiencies could arise from (i) impaired (complex) reasoning systems, (ii) an impaired formation of meta-confidence for functioning systems, and/or (iii) an impaired arbitration process of the meta-controller.

Complex reasoning systems rely on cognitive models of a task (/the world) that are often high-dimensional and challenging to learn. If these complex mental maps are not constructed or updated adequately, symptoms associated with OCD could arise. A failure in complex systems predicts that tasks necessitating such maps will not be completed adequately. For example, complex extra-dimensional shifts need an expansion of mental models (i.e. taking a previously irrelevant feature dimension into account). If such a model cannot be constructed or exploited, then subjects might be able to perform more simple learning aspects of the task, but no extra-dimensional shifts. The failure of these systems then affects their meta-confidence, which in turn leads to a decreased reliance on these systems. This again can explain why (at least over time) an over-reliance on simpler, model-free systems emerges, leading to habit-like behaviours. Similarly, faulty complex systems can also lead to indecisiveness and corrupt information gathering processes that critically rely on complex systems in order to plan into the future (Fradkin, Adams, Parr, Roiser, & Huppert, 2020). Lastly, if confidence reports primarily rely on complex systems as indicated by recent neuroimaging work (e.g. Bang & Fleming, 2018; Fleming & Dolan, 2012) and simple action exertion on less complex systems, dissociative learning rates in belief and action updating could arise.

Faulty meta-confidence updating can have similar consequences, but the inner workings are different. If meta-confidence is not updated adequately, it can lead to an over-reliance on a not-so-good system, ignoring better systems. Assuming that the brain is developmentally programmed to rely on simpler reasoning systems (*cf.* below), this means that more complex reasoning systems will have less influence in a decision-making process, which in turn can lead to excessive use of simple systems leading to habit-like behaviour. Likewise, this process could also impair the completion of complex tasks such as set-shifting, by ignoring adequate predictions of complex systems.

Lastly, a faulty meta-controller may also account for some of the observed alterations. Concretely, if the controller does not take individual meta-confidence accurately into account, it will base its action selection on suboptimal models and thus make inadequate decisions. Such a deficiency is in line with previous accounts suggesting arbitration difficulties between habitual and goal-directed systems (Gruner et al., 2016). It can further explain both an over-habitual behaviour as well as a failure to solve complex task aspects, such as planning, set-shifting or information gathering. How such an ignorance towards meta-confidence affects confidence reports depends on the concrete implementation, but it is likely that the erratic behaviour of the meta-controller will also interfere with the updating of meta-confidence.

Regardless of which one(s) of the suggested three pathomechanisms may be present, the imbalance between systems may further explain the prominent clinical chain of "obsession- distress- compulsion- relieve". If a simple model dominates behaviour intrusive thoughts may lead to a simple compulsive reaction leading to a momentary relieve of distress. More complex systems may be able to take transition probabilities of events into account and thus acknowledge the unrealistic character of these thoughts and the ineffectiveness of compulsions. However, if these complex systems do not exert any power over executed behaviours and

thought patterns the named chain of symptoms is likely to prevail and potentially even worsen the systems' imbalance.

Our algorithmic framework can be expanded to an implementation level. Previous models prescribe OCD-symptoms to an imbalance of the direct (excitatory) and indirect (inhibitory) cortico-striatal pathways (Maia & Frank, 2011; Saxena, Bota, & Brody, 2001). Such an imbalance can explain why a single system can go awry and become unable to adapt. In fact, previous work has highlighted the role of different fronto-striatal networks attributing OCD-like symptoms to impairments in different circuits (Maia, Cooney, & Peterson, 2008; Rolls, Loh, & Deco, 2008). This is in line with our framework assigning concrete cognitive functions to the different fronto-striatal loops. Furthermore, all three pathomechanisms could explain observed heightened dACC PEs (Hauser et al., 2017) as well as error-related negativity (ERN; Hajcak, Franklin, Foa, & Simons, 2008). A wrong model of the world resulting in inaccurate predictions and/or a faulty behaviour-selection mechanism leading to actions that are not in line with the original predictions could explain faulty predicted values of rewards, which in turn modulates and determines behavioural output. It could further lead to an increase in uncertainty mirrored in increased anxiety and lowered confidence shown in OCD. This is in line with work suggesting that increased ERNs may be a potential marker for anxiety-related phenotypes (e.g. Gillan et al., 2017) and may constitute an important marker in OCD-related anxiety. Moreover, anxiety and the cognitive load that comes with it may further foster an imbalance between systems and/or a misallocation of behavioural influence by the meta-controller.

In summary, we propose three potential mechanisms that can go awry and lead to underperformances consistently observed in OCD patients. Whilst several of these mechanistic disruptions suggested here make similar predictions about the behavioural consequences, their neural implementation is likely to differ. Therefore, they could be dissociated using

computational modelling and neural probing. In what follows, we will thus review the known neural alterations in OCD, and end by highlighting why a developmental perspective on these is essential.

## Neural Alterations in OCD

Evidence for structural and functional differences in juvenile OCD has been accumulated for more than two decades, revealing effects primarily in the frontal-striatal circuitry (Brem et al., 2012; Hauser et al., 2017; Maia et al., 2008; Marzuki et al., 2020; Menzies et al., 2008).

A key area altered in (juvenile) OCD is the dmPFC, including the dACC (Maia et al., 2008). Grey matter (Szeszko et al., 2008) as well as functional activity in the ACC (e.g. Carrasco et al., 2013; Hajcak, Franklin, Foa, & Simons, 2008) has been seen to be increased in juvenile OCD patients. Recently, heightened PEs were found in the dACC in juvenile patients (Hauser et al., 2017), which supports the idea that processing of a meta-controller could be compromised. Interestingly, this was replicated in adult OCD, and it was shown that this effect could be normalized by altering dopamine functioning (Murray et al., 2019). Similar evidence comes from electrophysiological studies showing that ERN in this area is more pronounced in juvenile OCD patients (Carrasco et al., 2013; Hajcak et al., 2008). As part of the action monitoring system, altered ACC activity has further been associated with symptom severity in adult OCD (e.g. Fitzgerald et al., 2005; Ursu, Stenger, Shear, Jones, & Carter, 2003). It should be noted that the direction of an altered activation (i.e. hyper- vs hypo-activity), may depend on the specific task used, and may flip in resting-state studies (He, 2013).

Similarly important for these cognitive functions, other fronto-striatal areas besides the dmPFC also express functional and structural abnormalities in juvenile OCD. These alterations are focused on striatal as well as orbitofrontal and adjacent regions (Hauser et al., 2017;

Woolley et al., 2008; Norman et al., 2017; Jayarajan et al., 2015; Fitzgerald et al., 2011) and have been linked to cognitive alterations such as limited inhibitory control (Woolley et al., 2008) and indecisiveness (Hauser et al., 2017).

All of these areas are known to undergo pronounced maturation during adolescence (Blakemore & Choudhury, 2006; Giedd et al., 1999). It is therefore critical to understand when and how these deficits in juvenile OCD arise. A recent longitudinal study showed that adolescents exhibit widespread myelin-related growth across the entire prefrontal cortex (Ziegler et al., 2019). However, this growth was substantially reduced in ACC and striatum in adolescents with high OCD symptoms. Further support for such development-dependent alterations was seen in cross-sectional patient studies. For instance, while grey matter in the ACC has been found to increase in healthy adolescents, this increase was absent in juvenile OCD patients, who in turn exhibited larger baseline volumes (Rosenberg & Keshavan, 1998; Ziegler et al., 2019). Likewise, Fitzgerald et al. (2011) found reduced connectivity in fronto-striatal loops encompassing the dACC and striatum that was specific to children with OCD, while excessive connectivity of dorsal striatum regions to the ventromedial PFC (vmPFC) was present in all age groups. How myelin-related growth integrates with functional connectivity during development, however, remains unclear. Together, these findings suggest that different fronto-striatal loops follow distinct (aberrant) developmental trajectories.

Due to the prominence of anxiety in OCD, the amygdala has also been proposed to play an important role in OCD. Its exact involvement has however been a matter of debate and past research in adult OCD has shown under-, hyper- as well as normal amygdala activity (Milad & Rauch, 2012). While some studies in the adult OCD literature suggested an alteration in the fronto-amygdala connectivity (Subirà et al., 2016) but other studies could not find changes in amygdala activity during threat learning (Apergis-Schoute et al., 2017). Szeszko et al. (2004) found (asymmetric) volume differences in juvenile patients and Britton et al. (2010) an

amygdala under-activation in response to facial expressions. However, a recent meta-analysis on emotional processing recently pointed out the small number of studies on paediatric OCD (Thorsen et al., 2018). The same study further showed increased amygdala activity during emotional processing in adult OCD and indicated that uncontrolled variables such as medication status, comorbidities, and symptom severity seem to have led to inconsistencies in the field. Thus, while the amygdala is likely to play an influential role in OCD the current literature on amygdala activity in (juvenile) OCD is scarce and inconsistent. It further remains to explore whether other OCD-like alterations might drive certain amygdala responses, or the other way around or whether these changes come about in a more synchronic matter.

In summary, it is critical to investigate neural alterations of OCD in the context of neurocognitive development. Understanding how and when a specific neural alteration arises and how it relates to cognitive impairments and OCD symptoms helps us trace the derailing that promotes the emergence of the disorder (*cf.* Box 3 on open questions).

## A Derailed Development in OCD

We have proposed that the emergence of OCD can be understood in the context of a meta-controller framework and that areas implicated in this framework show specific developmental trajectories in OCD patients.

A key developmental contribution is that different reasoning systems appear to develop at different times. Structural brain development studies show that areas where complex reasoning systems are likely to reside mature last in adolescence (Toga, Thompson, & Sowell, 2006). This is mirrored by a late development in model-based reasoning during adolescence (Decker et al., 2016). The consequence of this slow maturation is that earlier in life, we are more likely to rely on simpler reasoning systems (Vaghi et al., in press). This could even be reflected in the meta-confidence of complex systems. As long as a complex system is

immature, it may make faulty predictions and thus have low meta-confidence. Only once the predictions become more accurate, meta-confidence will increase, and adolescents will rely more on these systems. However, if complex systems never fully mature, one will never rely on these complex reasoning systems. The above findings of altered orbitofrontal/vmPFC regions and connectivity support a notion of impaired development of complex reasoning systems in OCD.

It is also important to note that the emergence of the meta-controller itself may be perturbed in juvenile OCD. In particular, the findings on altered structure and function in the dmPFC in juvenile OCD support this notion. The altered maturation of the dmPFC could impact the arbitration between the reasoning systems, perhaps even prevent the complex system from fully maturing.

The proposed pathomechanisms may further account for the observed bi-phasic onset seen in OCD (see Hauser, 2020 for more details). A misled development of complex systems may lead to a so-called 'early-onset' (i.e., onset around puberty) of OCD which is also associated with higher heritability (Chabane et al., 2005; Taylor, 2011). If an entire complex system or meta-controller fails to mature however, a later onset in early adulthood ('late-onset'; Taylor, 2011) could result. Adult OCD may similarly be explained by the present framework. Described pathomechanisms may not only lay the foundation at disorder onset but also exacerbate over time. Resulting neural alterations may look different in adult OCD as a simple consequence of the investigation taking place at different stages of development. Symptom and cognitive portrayals may further differ as a consequence of longer exposure to the disorder which could also lead to e.g. even higher differences in meta-confidence across systems.

It is also important to note that effect sizes seem to differ between the paediatric and adult OCD literature. While meta-analyses on neuropsychological functioning have on average shown medium to large effect sizes in adults (Abramovitch & Cooperman, 2015; Shin et al.,

2008; Snyder, Kaiser, Warren, & Heller, 2015), the only meta-analysis paediatric OCD literature has found small effects (Abramovitch et al., 2015). One reason for this could be that effect sizes increase as a disorder becomes more chronic. Differences may get larger with further neurodevelopmental derailing and a longer disease duration. Alternatively, smaller effect sizes in children may be indicative of their long-term disease progress, so that patients that will remit show fewer impairments (e.g. Abramovitch, 2017). Research showing that pre-treatment executive functioning was predictive of treatment success is in line with this hypothesis (D'Alcante et al., 2012). These essential questions need to be addressed in longitudinal developmental investigations.

## Limitations and future research

In the present paper, we brought together the current state of research on juvenile OCD and considered the possible computational mechanisms that could drive observed difficulties. Our computational framework proposes how computations could go awry and lead to the observed problems. Despite having established face validity of our framework, it remains speculative at the current stage. Further computational and neuroimaging research is now needed to test and examine it in more detail, based on the specific predictions that our framework makes (also see Box 3. Outstanding questions).

Our review further highlights a relative lack of developmental and especially longitudinal studies in juvenile OCD. While there is a growing body of research on adult OCD, our inferences about juvenile OCD are still limited and concerted efforts are needed to further deepen our computational understanding of juvenile OCD. Using longitudinal and computational studies will enable us to investigate how brain and cognitive functions dynamically change over development, and help to identify the mechanisms that underlie the emergence of OCD symptoms during development.

## Conclusion

In conclusion, we provided a review of the cognitive and neural alterations in (juvenile) OCD. We highlighted the commonalities, differences and research gaps between adult and juvenile OCD. Based on these findings we formulated a meta-controller framework and showed how different impairments can give rise to OCD symptoms. It is now critical to verify this framework, by examining the normative development of its components and to assess when and how it goes awry in OCD.

**Box 1. From Neuropsychology to Developmental Computational Psychiatry**

Computational psychiatry uses models from machine learning to understand how aberrant brain processes can lead to mental illness (Adams, Huys, & Roiser, 2016; Huys, Maia, & Frank, 2016). These models describe how a task can be solved algorithmically and which cognitive processes may occur. The processes are often formalised using RL (*cf.* Box 2) and/or Bayes' rule, principles of which many have been found to be implemented in the brain (Parr, Rees, & Friston, 2018; W. Schultz et al., 1997).

A key advantage over descriptive, psychological concepts (e.g. cognitive flexibility) is the generative nature of these computational models. This means they make concrete, testable predictions about cognitive processes and behaviour. Using simulations and model fitting, one can predict behaviour and cognitive patterns, which in turn can be used to understand the neurocomputational groundings of mental illness.

The computational models help to link aberrant neural processes to cognitive biases and psychiatric symptoms (cf. Figure 2). After determining the specific computations of brain areas and whether they are altered in patients, we can use the models to investigate how a derailing of such processes may alter cognition and drive psychiatric symptoms.

The emerging field of developmental computational psychiatry (Hauser, Will, Dubois, & Dolan, 2019) extends the current efforts and focuses on how computational capacity develops during youth. It investigates how deviation from canonical developmental trajectories can lead to imminent or protracted neurocognitive impairments.

**[Figure 2]**

**Box 2. Reinforcement Learning as a tool for Computational Psychiatry**

RL formalises how human and non-human agents take actions to maximise rewarding outcomes (Sutton & Barto, 1988). The agent thereby finds itself in a specific **state**, in a physical or virtual world that is associated with distinct properties (e.g. what was before and what will come next) and possibilities to act (e.g. going left or right). The agent aims to transition to states associated with the highest **rewards**. To do so, it can act upon the world by executing an **action**, which might lead it to another state at the following time step. The optimal action to take is thereby determined by the maximisation of future outcomes, formalised as **state-action values**. Consequently, the agent develops a policy that guides its behaviour.

A wealth of evidence has shown that humans and other animals exhibit similar behaviours to the ones predicted by RL models (Daw et al., 2011; O'Doherty, Dayan, Friston, Critchley, & Dolan, 2003; Yin et al., 2004, 2005). This makes the algorithms well suited to study (aberrant) decision making and learning in humans. Moreover, brain activity has been found to align well with predictions from RL. The most prominent example is the encoding of reward PEs in firing patterns of the dopaminergic midbrain (Schultz et al., 1997).

Here, we use RL to describe and understand cognitive and behavioural differences found in OCD and to speculate about their neurobiological underpinnings, i.e. how and where these processes are embedded.

**Model-based and Model-free Reinforcement Learning.** In the present paper, we heavily rely on the premise that the brain incorporates multiple, parallel reasoning systems. These systems' representation of the world (e.g. a task structure), span a wide range of complexity, from simple motor-outcome associations to complex cognitive models with many unobservable states and state transition.

Most previous work has been focused on distinguishing only two of these RL systems: **model-based** and **model-free** RL (*cf.* Figure 3) (Daw et al., 2005). Model-based RL

incorporates a (complex) model of the world, which allows to learn and guide actions most accurately, at the expense of high computing/energy costs. Model-free RL has a very limited model of the world and forms predictions based on experienced outcomes. It caches encountered rewards and slowly builds up expectations about actions and outcomes. While it demands only little computational and memory resources, it is inflexible, generalises poorly and its ability to react to sudden changes in the environment is limited. These two systems can be regarded as extreme prototypes, while recent evidence shows that it is likely that many intermediary systems are embedded in the brain and contribute to our behaviour (Russek, Momennejad, Botvinick, Gershman, & Daw, 2017; Shahar et al., 2019).

**[Figure 3]**

**Box 3. Outstanding questions**

Our knowledge about the neural and cognitive mechanisms that underlie juvenile OCD is still limited. It is thus critical to conduct longitudinal studies that track the development of cognitive skills and brain maturation. Such approaches can help to get an understanding of the neural and cognitive factors that contribute to the emergence of OCD:

- How does the development of OCD symptoms relate to the development of cognitive markers (e.g. indecisiveness)? Does cognition precede symptoms or vice versa?
- When do different fronto-striatal loops mature and how are they related to cognition and symptoms?
- Do juvenile OCD and other psychiatric disorders express similar or distinct neurocognitive developmental trajectories?
- Can we identify different at-risk stages before the development of OCD (Fineberg et al., 2019; similar to schizophrenia) and if so, what are their cognitive and neural markers?

## Supplementary Material

More details about the computational framework including equations and simulations are provided at www.github.com/DevComPsy/MetaController.

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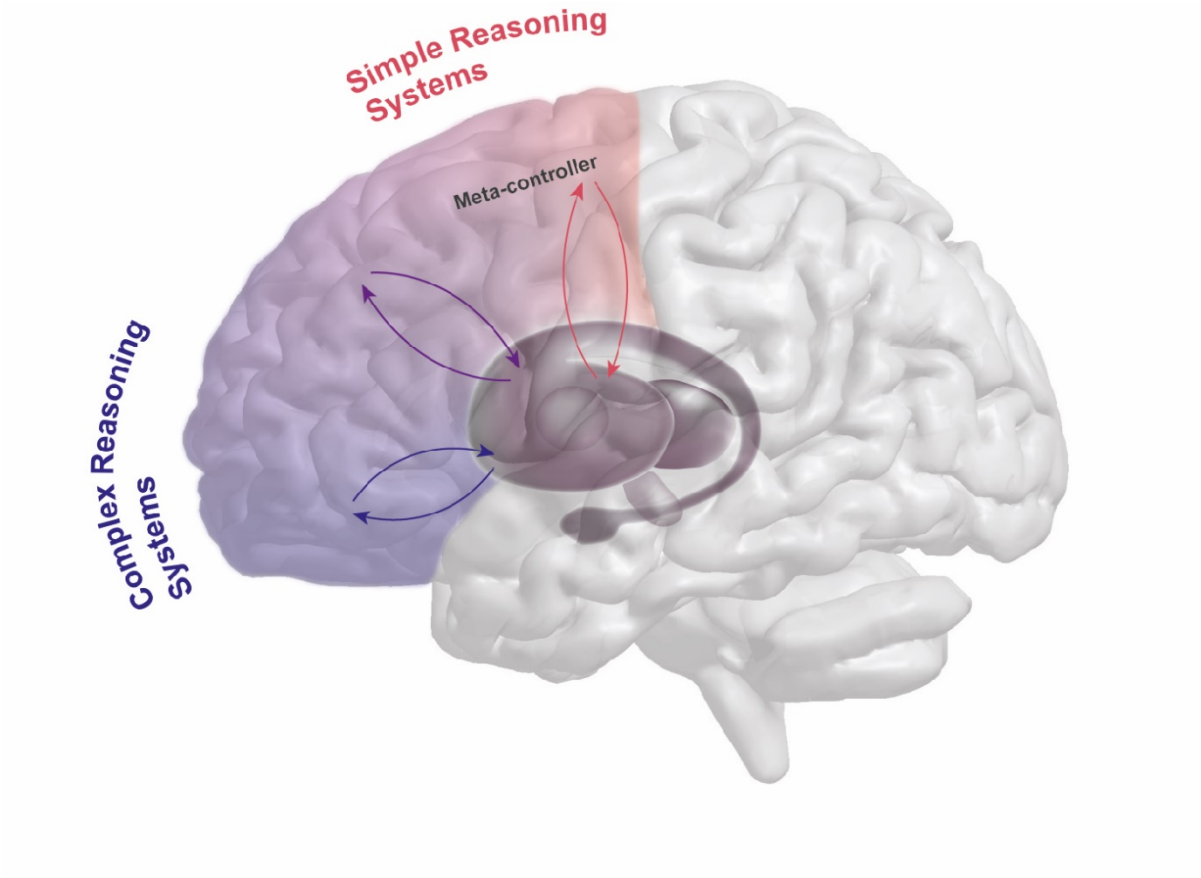


Figure 1. Schematic framework illustrating how cognitive deficits in OCD may arise from aberrant neurodevelopmental mechanisms. Different reasoning systems may be embedded in separate fronto-striatal loops (arrows). Functional complexity of the systems is likely to increase from the posterior to the anterior regions of the frontal lobe. A meta-controller in the dmPFC/dACC may arbitrate and select between competing actions. The cortex was visualised using the BrainNet Viewer (http://www.nitrc.org/projects/bnv/; Xia, Wang, & He, 2013).

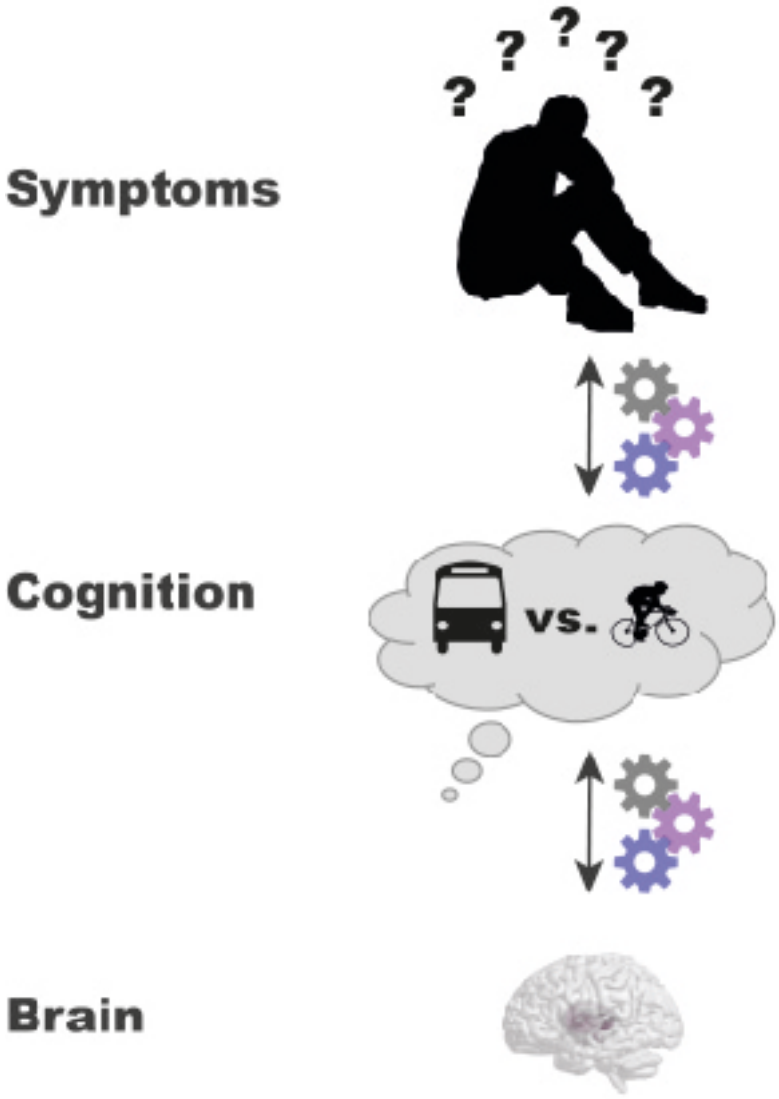


Figure 2. Computational psychiatry allows us to infer mechanisms which generate brain activity, cognition and behaviour in psychiatric patients. It helps us to bridge the gaps between neural implementation (e.g. altered neural systems), cognition (e.g. decision making) and symptoms (e.g. indecisiveness).

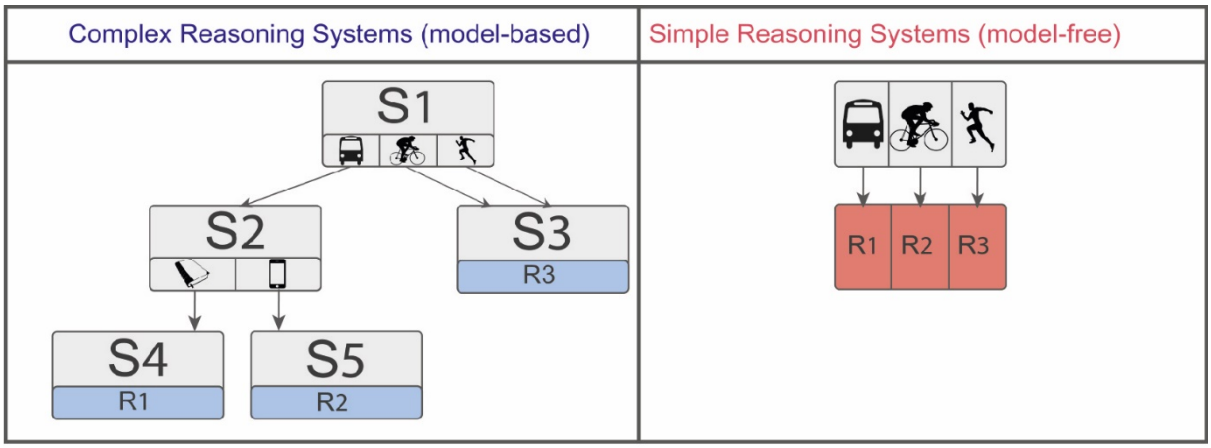


Figure 3. Complex (model-based) and simple (model-free) reasoning system. The systems represent the world as states (S1-5), actions (taking the bus, cycling, running, looking at the phone, and reading a book) and rewards (R1-3). **The complex reasoning system** (left) incorporates a complex model of the world, which represents the transitions between states and the actions causing the transitions. For a known current state, the system can calculate the likely outcome (R1-3 on the left) of simulated actions in each state. **The simple reasoning system** (right) reduces the representational and computational demands, mirroring a cache rather than a tree system. It only represents the expected value as a scalar (R1-3 on the right) for each action in each state. It does not take into account the actual transition structure (i.e. action-state sequence) leading to the outcome.