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Review article

The metacontrol hypothesis as diagnostic framework of OCD and ADHD: A dimensional approach based on shared neurobiological vulnerability

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ABSTRACT

Obsessive-compulsive disorder (OCD) and attention-deficit hyperactivity disorder (ADHD) are multi-faceted neuropsychiatric conditions that in many aspects appear to be each other's antipodes. We suggest a dimensional approach, according to which these partially opposing disorders fall onto a continuum that reflects variability regarding alterations of cortico-striato-thalamo-cortical (CSTC) circuits and of the processing of neural noise during cognition. By using theoretical accounts of human cognitive metacontrol, we develop a framework according to which OCD can be characterized by a chronic bias towards exaggerated cognitive persistence, equivalent to a high signal-to-noise ratio (SNR)—which facilitates perseverative behaviour but impairs mental flexibility. In contrast, ADHD is characterized by a chronic bias towards inflated cognitive flexibility, equivalent to a low SNR—which increases behavioural variability but impairs the focusing on one goal and on relevant information. We argue that, when pharmacology is not feasible, novel treatments of these disorders may involve methods to manipulate the signal-to-noise ratio via non-invasive brain stimulation techniques, in order to normalize the situational imbalance between cognitive persistence and cognitive flexibility.

1. Introduction

Obsessive-compulsive disorder (OCD) and attention-deficit hyperactivity disorder (ADHD) are multi-faceted neuropsychiatric conditions that in many aspects, such as in terms of clinical symptoms, cognitive profile, biochemistry, and brain networks, appear to be each other's antipodes (Brem et al., 2014; Carlsson, 2000). OCD is ranked as the fourth most common neuropsychiatric disorder worldwide, with a lifetime prevalence of 2–3% (Flament et al., 1988; Robins et al., 1984; Zohar, 1999). OCD is usually depicted by the presence of recurrent, intrusive, and worrying thoughts (obsessions), which often elicit repetitive behavior (compulsions) carried out with the aim of neutralizing negative feelings caused by the obsessions (American Psychiatric Association, 2013). In young age, ADHD is more common than OCD, with a worldwide prevalence of 5.2% among children and adolescents (Polanczyk et al., 2007) and about 2–3% in adults (Matte et al., 2012). A hallmark of ADHD is a persistent pattern of inattention and/or hyperactivity and impulsivity (American Psychiatric Association, 2013).

Even though a large-scale cross-disorder genome-wide meta-analysis show no genetic correlations between OCD and ADHD suggesting no shared genetic basis between them (Lee et al., 2019), some studies show a familial relationship between ADHD and pediatric OCD (Geller et al., 2007b, 2007b), raising the question whether the two conditions should be regarded as fully separable, which would hardly fit the rather frequently observed co-existence of OCD and ADHD. However, despite this co-existence, almost no studies have compared "pure" ADHD to ADHD with co-existent OCD or "pure" OCD to OCD with co-existent ADHD in terms of cognitive performance. Accordingly, given the few cognitive data available on the co-existent cognitive profiles of OCD and ADHD, we decided to summarize the existing literature related to the "pure" cognitive profiles of OCD and ADHD, even though we agree that from a clinical point of view this clear-cut distinction might appear artificial. We will use this summary as a jumping board to develop a novel cognitive diagnostic framework to explain aspects of the specific cognitive profiles of OCD and ADHD. This framework is dimensional in nature, so to link the two conditions in terms of mechanisms, and it uses

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the "metacontrol hypothesis" (Hommel, 2015; Hommel and Colzato, 2017). Following a dimensional approach, we propose that OCD and ADHD represent the extreme poles of a continuum that is characterized by the abnormal functioning of cortico-striato-thalamo-cortical (CSTC) circuits and by an alteration of the principle of processing of neural noise during cognition. We will conclude by outlining possible new avenues for treating these disorders by targeting the signal-to-noise ratio (SNR) during cognition in order to normalize behaviour and the cognitive profiles in OCD and ADHD. We performed an electronic search for articles with the PubMed database using the following search terms: (OCD OR ADHD OR comorbidity) AND (cognition OR cognitive control). First, we selected articles based on their titles and abstract relevance to OCD and ADHD, and then performed a forward and backward citation search for additional articles. Only articles written in English were included.

2. Co-existence of OCD and ADHD

In line with the idea of a shared neurobiological vulnerability, there is evidence that co-existence of OCD and ADHD in children forecasts a higher severity of OCD and a higher grade of perseverance of OC symptoms in follow-up assessment (Walitza et al., 2008). In patients with OCD, co-existence of ADHD has been found in 17.1% (Masi et al., 2010) and 25.5% (Masi et al., 2006) of the patients, while the rate of co-occurrence of OCD in children with ADHD is around 8.6% (Geller et al., 2000). Besides the fact that higher co-existence rates have been described for ADHD in OCD samples than for OCD in ADHD samples, lower co-existence rates have been detected in adults as compared to children (Abramovitch, Dar et al., 2015). Recent studies showed that OCD-ADHD co-existence in adults is linked to an earlier onset of OCD (Blanco-Vieira et al., 2019; Mersin Kilic et al., 2020) and that a history of ADHD symptoms during childhood is frequent (40.9%) in adult OCD patients who have never received a diagnosis of ADHD (Tan et al., 2016). Yet, it is important to keep in mind that clinic-based studies might undergo the so-called Berkson's bias (Berkson, 2014): it is more probable that clinical samples of OCD co-exist with ADHD and, consequently, might reveal higher rates of co-existence than the general population. In any case, few investigations free from this potential bias, such as studies carried out in community samples like national surveys (Kessler et al., 2006; Ruscio et al., 2010), confirmed the co-existence between OCD and ADHD. One study reported that 2.7% of their ADHD sample suffered from OCD (Kessler et al., 2006), whereas another 19% of the OCD sample suffered from ADHD (Ruscio et al., 2010). As pointed out by Rothenberger and Roessner (2019), co-existence is an important issue because overlapping disorders have different courses and might necessitate different options of treatment. Another important question regarding OCD and ADHD for clinical practice is the eventual co-existence of tic disorders, the presence of which might impair psychosocial functioning even more (Banaschewski et al., 2007; Roessner et al., 2007; Rothenberger et al., 2007, 2010; Schlander et al., 2011; Wanderer et al., 2012). Albeit recent evidence from cross-disorder genetics revealed no significant genetic relationship between OCD and ADHD (Lee et al., 2019), some studies revealed familial relationship between them, likely involving tic disorders as well (Geller et al., 2007a, 2007b; Roessner et al., 2016). Hence, the high co-existence of OCD and ADHD supports the idea of a shared neurobiological vulnerability between these conditions.

Shared neurobiological vulnerability might explain aspects of the specific cognitive profiles associated with OCD and ADHD. In particular, we suggest that OCD and ADHD may not be two independent conditions, but in many aspects rather two poles of a common dimension. We are not the first to consider this possibility, as other authors have already suggested that OCD and ADHD might fall onto an impulsive–compulsive continuum (Allen et al., 2003). This continuum has been characterized in terms of harm avoidance at the compulsive pole and as risk seeking at the impulsive pole (Allen et al., 2003). However, the terms compulsivity and impulsivity refer to classes of empirical observations, and are therefore descriptive in nature, rather than explanatory (e.g., Berlin and Hollander, 2014). Accordingly, considering a compulsive-impulsive dimension might provide a system to categorize observations, but does not point to the underlying mechanisms (Grant and Kim, 2014). The persistence-flexibility dimension, in contrast, has been described in terms of the computational consequences of biases towards persistence or flexibility, the neural and neurochemical underpinnings thereof, and the performance profiles associated with such biases (Cools, 2016; Durstewitz and Seamans, 2008; Goschke, 2000; Hommel, 2015; Hommel and Colzato, 2017). Moreover, whereas the functional implications of a bias towards persistence are exactly opposite to those of a bias towards flexibility, compulsivity and impulsivity are often related to very similar situational behavior, suggesting that these latter terms are unlikely to refer to different poles of a common dimension. Indeed, research guided by compulsivity-impulsivity concepts were so far not particularly successful in identifying the neural underpinnings of OCD and ADHD, nor did they stimulate broadly successful therapeutic interventions.

Hence, while we do not exclude that the compulsivity-impulsivity description captures aspects that the metacontrol approach does not, or not sufficiently address (an issue we will come back to below), we prefer the conceptually and mechanistically more transparent metacontrol model (Hommel, 2015; Hommel and Colzato, 2017) as a conceptual framework to elucidate cognitive functioning in OCD and ADHD in most instances. However, it is important to keep in mind that both OCD and ADHD are very broad, multifaceted categories that render it unlikely to account for all possible subtypes by means of one single factor. For instance, the meta-analysis of Willcutt and colleagues (2005) provided evidence for a considerable connection between executive control functions and ADHD, but effect sizes that were too low to suggest that executive control issues are the only factor in ADHD. A closer look revealed that one subtype (the hyperactive-impulsive type) had particularly low association with executive control functions, suggesting that with this particular subtype, other factors will be more important. While our metacontrol approach is more comprehensive (e.g., in considering both the classical executive-control concept and flexibility—an ability that the classical concept ignores altogether), we also do not consider it a realistic goal to account for 100% of the within-category variability. After all, ADHD and OCD are historically grown, mainly a-theoretical observational categories, and there is no reason to believe that the mere existence of a common label necessarily indicates the existence of just one underlying mechanistic cause for each category (Hommel, 2020). However, we do aim to account for a substantial portion of the variability, and for the key characteristics underlying OCD and ADHD, and their mutual relationship in particular.

3. The metacontrol hypothesis as a cognitive diagnosis model bridging OCD and ADHD: cognitive persistence (OCD phenotype) vs. cognitive flexibility (ADHD phenotype)

According to the metacontrol model (Hommel, 2015; Hommel and Colzato, 2017), people can vary in their cognitive processing style, on a dimension with the extreme poles "persistence" and "flexibility". A high degree of persistence is characterized by a strong focus on one goal and on the information related to that goal, which facilitates concentration, while a high degree of flexibility is characterized by a more integrative, less selective and exclusive processing style, which facilitates switching between tasks, ideas, and actions, and taking into consideration a broader range of possibilities. As pointed out by Hommel, 2015; Hommel and Colzato (2017), metacontrol can be described as the ability to keep an appropriate balance between cognitive persistence and cognitive flexibility in various kinds of decision-making under various circumstances. Following Bogacz (2007), decision-making is competitive (winner-takes-all) in nature and is biased according to currently active goals. It is competitive in the sense that a stronger preference for, or activation of one alternative will tend to suppress the preference for, or

activation of other alternatives: if, say, Lisa is struggling between completing her homework or playing soccer with friends, stronger activation of the homework-making alternative will reduce the activation of the soccer preference. It is biased by goals in the sense that a dominant goal to improve her grades will support the homework alternative, which will help Lisa to suppress the soccer preference.

The key idea of metacontrol theory is that the severity with which alternatives compete and how much support is provided by goals can vary, both interindividually and intraindividually. More specifically, behavioral options compete for selection and the extent or severity of their competition and the degree to which they are biased by current goals is dictated by the present metacontrol state: Strong cognitive persistence would be accompanied by strong competition ("exclusivity" of decision-making) and a strong impact of the current goal on the competition, while strong cognitive flexibility would be accompanied by a weak competition (i.e., processing would be more integrative than selective) and a weak impact of the current goal (i.e., more goal-unrelated information would be considered). Hence, a strong persistence state would make Lisa struggle between homework-making and soccer playing, and eventually result in an unequivocal decision towards one of the options, perhaps guided by the goal to reach higher grades. In contrast, a strong flexibility state might make Lisa try to combine her two tendencies, perhaps by rushing the homework-making and then joining the soccer game, or by watching the game from her window while continuing with the homework.

As we will outline in the next section, metacontrol seems to depend on CSTC circuits (Beste, Moll et al., 2018), where competition between response options is central to many computational models of basal ganglia function (Humphries et al., 2006; Plenz, 2003; Schroll and Hamker, 2013), and is affected by alterations of neural noise during cognition (Münchau et al., 2021; Pertermann, Bluschke et al., 2019).

In general, patients with OCD seem to suffer from a broad range of cognitive deficits across several domains, such as working memory, planning, decision making, attentional shifting, and verbal fluency (Benzina et al., 2016; Kashyap et al., 2013). However, some authors (Abramovitch, Abramowitz et al., 2015; Abramovitch et al., 2019; Cameron et al., 2020), pointed out that, at this point, it is not possible to carry out a meaningful meta-analysis of cognitive dysfunction in OCD, because of significant methodological and sample differences between studies. Other issues that have not been taken into account in the field and that make it even more difficult to characterize the cognitive profile of OCD is the considerable heterogeneity of this disorder in terms of (a) the nature of the experienced symptoms (i.e., contamination obsessions vs. symmetry obsessions vs. harm obsessions); and (b) the pharmacotherapy; and (c) the fact that the relationship with other disorders (i.e., tics) can vary with age (Rothenberger and Roessner, 2019). Indeed, there seems to be a difference between OCD with onset in childhood (10–12 y, often associated with tics) and OCD with onset in adulthood (18–20 y, often closer to an obsessive-compulsive behavior style) (Wanderer et al., 2012).

These issues notwithstanding, it seems fair to say that deficits in cognitive flexibility represent the hallmark of OCD, as pointed out by Gruner and Pittenger (2017). This is evident from the phenomenology (e.g., repetitive behavior with rigid rituals, diminished behavioral flexibility; Benzina et al., 2014) as well as from different paradigms assessing OCD patients. For instance, patients with OCD perform less accurately or make more perseverative errors than healthy controls in attentional set shifting paradigms, such as the Wisconsin Card Sort Task (i.e., tasks where the rules determining the correct response change over time without notice; Shin et al., 2014; Snyder et al., 2015). Along the same lines, patients with OCD perform more poorly than healthy controls in alternation tasks (i.e., where optimal performance requires selecting/shifting to a different stimulus on every trial; Shin et al., 2014; Snyder et al., 2015), which suggested to Viswanath et al. (2009) that this cognitive deficit is a valid cognitive endophenotype of OCD. Patients with OCD also show impairments in task-switching paradigms (i.e., where the shift takes place under time pressure following an explicit rule or cue) (Shin et al., 2014; Snyder et al., 2015; but see Moritz et al., 2004; Remijnse et al., 2013), especially in tasks that require perceptual categorization (Wolff et al., 2017).

An important aspect of the metacontrol model we rely on is that it only characterizes the processing style of individuals as more persistent or more flexible, without considering some styles as dysfunctional per se. From a metacontrol perspective, the functionality of a particular processing style necessarily depends on the circumstances and on the task: while a strong bias towards persistence might be dysfunctional in some tasks, it may be useful in others, and the same holds for a strong bias towards flexibility (Colzato et al., 2022)). Accordingly, one would expect that individuals with a strong bias towards persistence, like presumably patients with OCD, do not only show deficits in flexibility-requiring tasks but might also show excellent performance in persistence-requiring tasks (at least if they manage to disengage from their typical ruminative "loops"). This is exactly what the literature shows: patients with OCD outperform healthy controls in tasks tapping into cognitive persistence and tasks that require the explicit maintenance of task goals and rules. For example, patients with OCD excel in tasks where the target stimulus location is explicitly determined by an underlying sequence (intentional learning), but not when they are not informed of the existence of the underlying sequence (implicit learning; Soref et al., 2008). Patients with OCD also show enhanced performance as compared to patients with ADHD in the continuous performance test, a task requiring responses to all letters of the alphabet except the letter "X" (Lucke et al., 2015), but similar performance to psychiatrically-healthy controls (Fruehauf et al., 2021; Milliery et al., 2000). In contrast, a recent study (Xie et al., 2020) showed deficits in the continuous performance test for OCD children but only with co-existent tic disorders (TD), suggesting that sustained attention deficits are intrinsic to the comorbid OCD + TD group. Patients with OCD are also better than controls in tasks that rely on persistent states, such as repetitive mental sets (Wolff et al., 2018) and automatic response selection mechanisms (Wolff et al., 2019). Hence, patients with OCD tend to outperform healthy controls in tasks that rely on cognitive persistence, either because these tasks require or benefit from a strong impact of the current goal, or because they require strong reciprocal competition between alternative cognitive representations, or both (Hommel, 2015).

In contrast to OCD, systemic reviews, meta-analyses, and empirical studies indicate that patients with ADHD typically show impairments in tasks requiring a focused/persistent cognitive state, such as sustained attention and vigilance (Barkley, 1997). Similarly, deficits in the maintenance of task-relevant information in verbal working memory have been observed (Ramos et al., 2020), which is likely to indicate low selectivity and weak mutual competition between alternative cognitive representations. Along the same lines, patients with ADHD exhibit poor performance in tasks requiring the maintenance of task goals (Gohil et al., 2017), the keeping of decision-making sets in working memory (Willcutt et al., 2005), and the discrimination of relevant and irrelevant information (interference control: Chmielewski et al., 2019; Chmielewski et al., 2018). Hence, ADHD is oftentimes associated with deficiencies regarding cognitive persistence. Even though neuropsychological deficits related to the cognitive domains mentioned above are not necessary for the diagnosis of ADHD (Willcutt et al., 2005), or specific to ADHD, and even though cognitive training in these domains has limited effects (Cortese et al., 2015), these cognitive domains can be of heuristic value to approach a mechanistic core underlying phenotypes of OCD and ADHD.

According to the metacontrol model, this implies that individuals diagnosed with ADHD might show particularly good performance in tasks that require cognitive flexibility. At first sight, this does not seem plausible, as patients with ADHD have been found to perform poorly in task-switching tasks (Rauch et al., 2012). Interestingly, however, this deficit seems to emerge only when the time interval between the different tasks within the task-switching paradigm is very short, which

suggests that the problem might not so much be the flexibility of switching to a new task per se but, rather, the inhibition of the present task-set and/or the implementation of the new set (Arabacı and Parris, 2020; Sidlauskaite et al., 2020). Indeed, the so-called preparatory switching costs do not seem to tap into cognitive flexibility but rather reflect an automatic carryover effect from the previous task (Dreisbach et al., 2002). This implies that patients with ADHD may not show deficits in cognitive flexibility if they have enough time to prepare, and may even outperform healthy controls in switching tasks that provide enough preparation time. This idea has been also supported by a meta-analysis demonstrating an altered perceptual timing abilities in ADHD (Marx et al., 2021). In line with this evidence, patients with ADHD show greater behavioural variability, as demonstrated by Bluschke et al. (2021), which is crucial for (or reflecting) a flexible cognitive state. This seems to be responsible for the observation that patients with ADHD outperform controls in divergent thinking, which requires the generation of many different ideas (Hoogman et al., 2020), however, most studies reported enhanced divergent thinking for people with high ADHD scores (subclinical) but not for people diagnosed with the disorder (clinical sample) (Hoogman et al., 2020). This greater behavioural variability might underlie also better performance of ADHD in exploratory foraging patterns (Van den Driessche et al., 2019), and in the implicit learning of an artificial grammar (Rosas et al., 2010). However, even though, adult ADHD performed as good as controls in an implicit sequence learning, they revealed reduced efficiency of the inhibition of incorrect responses (Pedersen and Ohrmann, 2018), suggesting that implicit learning is still intact in ADHD but only when the task does not depend on sustained attention. Hence, ADHD seems to be associated with excellent performance in tasks requiring cognitive flexibility.

Unfortunately, despite the frequent co-existence of OCD and ADHD, and to the best of our knowledge, only one study compared "pure" ADHD to ADHD with comorbid OCD in terms of cognitive performance, but without finding any statistically significant difference between the two groups (Arnold et al., 2005). The cognitive effect of "pure" OCD compared to OCD with co-existing ADHD has not yet been examined. Accordingly, future studies are needed to unequivocally disentangle the effect of OCD and ADHD on cognitive performance, ideally using a 2×2 factorial design with the factors OCD (yes/no) and ADHD (yes/no) addressing the inclusion of four groups (OCD only, OCD+ADHD, ADHD only, controls).

Based on the evidence available so far, we suggest a metacontrol model to characterize both OCD and ADHD within the same theoretical framework. In particular, we propose that people diagnosed with OCD tend to show a processing style that is biased towards persistence, which creates a more focused state (i.e., cognitive persistence) facilitating perseverative behaviour, such as rituals and compulsions, but comes at the cost of cognitive flexibility, see Fig. 1. In contrast, we suggest that people diagnosed with ADHD tend to show a processing style that is biased towards flexibility, leading to greater behavioural variability but at the cost of cognitive persistence, see Fig. 1. Among other things, our view calls for a revision of the current research focus, which is almost exclusively directed at the impairments of OCD or ADHD. While such impairments undoubtedly exist, it seems premature, if not unfair to characterize the underlying processing style as entirely dysfunctional. It certainly is dysfunctional with respect to diagnostically relevant tasks, especially if they call for a processing style that is located far from the individual's current default on the metacontrol scale. But it may not be dysfunctional at all, and may even be beneficial for tasks that require a control style that fits better with the individual's current default. Spending more research efforts on identifying these latter kinds of tasks seems to make reasonable use of societies' human resources from the perspective of both, the society and the diagnosed individual. We thus call for a more balanced view that does not neglect the possible positive side of OCD and ADHD. From a clinical standpoint, having research programs focusing on the potential mental gains of OCD and ADHD might play a pivotal role in the classroom, given that OCD and ADHD

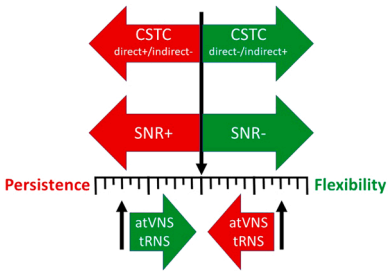

Fig. 1. Metacontrol can be depicted as a scale ranging from extreme persistence to extreme flexibility. The processing style of people is assumed to show both inter-individual and intra-individual variability, that is, individuals might differ with respect to their preferred processing style, and they can also adjust their own processing style by becoming more persistent or more flexible under appropriate circumstances. With respect to OCD and ADHD, patients diagnosed with OCD are assumed to exhibit a processing style biased towards the left part of the scale, while patients diagnosed with ADHD are assumed to show a processing style biased towards the right part of the scale. The individual location on the scale is assumed to be determined by at least two factors: the functionality of the CSTC circuit and the individual signal-to-noise ratio. For the CSTC circuit, the balance between the direct and the indirect path matters: greater dominance of the direct path and/or reduced contributions from the indirect path are assumed to bias the processing style towards persistence, while greater dominance of the indirect path and/or reduced contributions from the direct path are assumed to bias towards flexibility. With respect to the signal-to-noise ratio, a higher ratio tends to bias processing towards persistence (especially in sensorimotor processing and decision-making), while a lower ratio tends to bias processing towards flexibility (especially in sensory processing). As indicated in the lower part of the figure, systematic biases towards persistence or flexibility could be reduced by noninvasive brain stimulation techniques, such as tVNS and tRNS, which are able to modulate the SNR.

students show notorious academic underachievement and low self-esteem (Packer and Pruitt, 2010). That is, we believe that the assessment of cognitive gains and their active support might enhance educational achievement and improve self-esteem in these conditions.

Before we turn to the neural underpinnings of OCD and ADHD, and their relationship to the metacontrol model, we would like to address an interesting and important open question with respect to our current approach: the observation of co-existing disorders. If we simply place OCD on the left and ADHD on the right of a unidimensional persistence-persistence scale, how is it possible that one given individual can show evidence for both conditions? Given the lack of decisive data to resolve this question, we hesitate to commit ourselves to a particular theoretical possibility, as we believe that this question needs to be tackled empirically. Nevertheless, we do want to present a number of possibilities that might guide such empirical endeavors:

1. The probably most obvious possibility is that the diagnostic categories are not sufficiently informed by mechanistic knowledge to allow for a clear-cut categorization of patients. After all, clinical categories often emerge from clinical practice and more or less systematic observations of practitioners. The resulting categories are not necessarily optimized for perfect discriminability, so that what looks like co-existence of disorders might actually indicate a semantic overlap of the underlying categories. This can be avoided by a more theory-driven approach that is more informed by a mechanistic understanding of the underlying factors. The metacontrol framework provides such an understanding and the required theoretical framework. Accordingly, we suggest replacing the clinical categories by a more systematic framework that is based on metacontrol theory. Indeed, while a processing style can only persistent or flexible at one given point in time, we simply do not know whether one can fall into the OCD category and the ADHD category at the same time.
2. The second scenario considers the possible existence of a second, not yet identified dimension in addition to the hypothesized persistence-

flexibility continuum. This second dimension could represent the impulsive–compulsive continuum (Allen et al., 2003), on which patients with co-existing disorders differ from more "unipolar" patients. However, given that the impulsive-compulsive continuum does not refer to theoretically motivated mechanisms but summarizing descriptions with considerable semantic overlap, we doubt that this dimension—if it can be considered a dimension at all—will be a useful addition to our model. Nevertheless, there might be other dimensions that need to be added to receive a full picture.

3. The third scenario considers that patients may not necessarily be "fixed" on a particular location on the persistence-flexibility dimension but exhibit considerable intra-individual variability in different situations. It could be that they do show symptoms of both poles but not at the exact same time—not unlike patients diagnosed with manic-depression. Consistent with this idea, patients with ADHD treated with stimulants have been found to show an increase of co-morbid obsessive–compulsive symptoms (Geller et al., 2002). This third possibility would suggest that patients with comorbidities show substantially more symptomatic variability over time as compared to "unipolar" patients. To clarify this issue, as pointed out by Abramovitch et al. (2015), prospective pharmacological studies are mandatory with the aim to investigate the effect of developmental trajectory of ADHD-OCD co-existence on the cognitive profile. Indeed, such studies would be crucial in clarifying factors disentangling ADHD-like symptoms in OCD from genuine co-existence between the two conditions (Abramovitch, Dar et al., 2015).

In the next section, we continue a dimensional approach and propose that the persistence-flexibility continuum is based on a shared neurobiological vulnerability continuum with OCD, on one side, and ADHD, on the other. We will argue that the specific constellations of cognitive gains and cognitive deficits in OCD and ADHD may arise from alterations of CSTC circuits and alteration of neural noise during cognition, two neurobiological underpinnings on which metacontrol seems to depend (Beste, Moll et al., 2018; Münchau et al., 2021).

4. Shared neurobiological vulnerability: abnormal functioning of CSTC circuits and alteration of neural noise

If it is true that the cognitive and behavioral characteristics of individuals diagnosed with OCD and ADHD can be accounted for by a metacontrol dimension ranging from extreme persistence to extreme flexibility, and if OCD can be characterized by a bias towards persistence and ADHD by a bias towards flexibility, we need to understand which individual neural peculiarities underlie such biases. Hence, which neural mechanisms and which impairments thereof are responsible for biasing an individual towards persistence or flexibility? We argue that the available evidence points to two neural conditions that, either separately and in parallel, or in interaction, or both can generate biases of metacontrol: the relative dominance of direct and indirect pathways in cortico-striato-thalamo-cortical (CSTC) circuits and the individual signal-to-noise ratio (SNR).

4.1. CSTC circuits and their pathways

Parallel CSTC circuits have been suggested to control different aspects of behavior by cognitive processes, such as spatial attention and set-shifting, working memory, and decision-making (Chudasama and Robbins, 2006). These circuits include connections between the cortex, the basal ganglia, the thalamus, and back to the cortex. Parallel CSTC circuits involve *direct* and *indirect pathways* projecting from the specific cortical regions to the specific sections of the striatum and thalamus with loop projections to the cortex (van den Heuvel et al., 2016). These circuits are innervated by both dopaminergic (from the pars compacta of the substantia nigra and ventral tegmental area) and glutamatergic projections (from the cortex to the striatum including direct excitatory "Go" and inhibitory indirect "NoGo" pathways that relay back to the cortex (Albin et al., 1989). Even if parallel CTSC circuits are in most parts segregated, higher-order behavior relies on between-circuit communication, likely acting via dopamine (Groenewegen et al., 2003). This between-circuit communication is crucial, given that the subprocesses promoted by the different CSTC circuits cannot be executed independently (van den Heuvel et al., 2016). It has been suggested that functional behavior relies on the flexible equilibrium between the direct pathway, which operates as a self-reinforcing positive feedback loop and promotes the initiation and maintenance of behavior, and the indirect pathway, which operates via negative feedback supporting the inhibition of behavior and behavioral variability (van den Heuvel et al., 2016).

Against this background, insufficiencies of the flexible balance between these pathways is a likely cause for maladaptive behavior, such as for example, the excessive fear of making a catastrophic mistake that in turn elicits checking behavior (Burguière et al., 2015) or emotional overeating (Moore et al., 2017). In line with this assumption, several meta-analysis and systemic reviews have shown that both OCD and ADHD patients display reduced volume of the basal ganglia (Ellison--Wright et al., 2008; Piras et al., 2015). These structural abnormalities of the basal ganglia reflect functional alterations of the CSTC circuits in both OCD and ADHD (Brem et al., 2014). Specifically, several neuroimaging studies revealed that OCD is associated with a hyper-activated direct, and a hypo-activated indirect pathway of the CSTC circuits (Brem et al., 2012; Menzies et al., 2008; van den Heuvel et al., 2016), suggesting a systematic alteration of the balance between the two pathways. Hence, *increases of the contributions from the direct pathway and/or decreases of the contributions from the indirect pathway* may be responsible for the bias towards cognitive persistence that in our view characterizes OCD. In contrast, neuroimaging studies have demonstrated that ADHD is associated with a functional alteration of the balance between the two pathways within the CSTC circuits towards the indirect pathway (Dickstein et al., 2006; Makris et al., 2009; Rubia et al., 2014; Sonuga-Barke, 2003). Hence, *increases of the contributions from the indirect pathway and/or decreases of the contributions from the direct pathway* might be responsible for the bias towards cognitive flexibility typical of ADHD. However, given that our hypothesis is so far supported by only a few studies, future meta-analytic evidence should be added to test its validity.

While this scenario does not rule out possible roles of other disorder-specific brain circuits, it does suggest that alterations in CSTC circuits represent an important shared neurobiological vulnerability of OCD and ADHD (Vloet et al., 2006). According to the direct/indirect pathway approach, disorders such as OCD and ADHD arise from an increased cortical excitability and/or inhibition produced by an altered direct to indirect pathway activity ratio. Even though this somewhat oversimplified model has been popular within psychiatry, another approach based on imbalances in the striatal striosome and matrix compartments of the dorsal striatum is gaining attention (Crittenden and Graybiel, 2011, 2016). Whereas the striosomes receive mainly input from limbic related regions of the prefrontal cortex (Eblen and Graybiel, 1995), the matrix compartment receives most of the striatal inputs from the motor, sensorimotor, and association cortex (Flaherty and Graybiel, 1995). Interestingly, it has been suggested that increased striosomal activity or decreased matrix activity can predict the degree of compulsions/repetitive behavior expressed in rats and monkey (see Graybiel, 2010, for a review). Based on normal and dopamine-depleted animals studies, Crittenden and Graybiel (2011) suggested that increased activity in the striosomes might foster compulsions/repetitive behavior whereas increased activity in the matrix might promote behavioral flexibility. This idea was confirmed by studies on humans suffering from X-linked dystonia-parkinsonism (XDP), a condition known to affect the striosome, but not the matrix compartment (Goto et al., 2005). In line with the hypothesis put forward by Crittenden and Graybiel (2011), XDP

patients reported deficits in tasks requiring behavioral persistence and cognitive stability necessary to monitor behavior and attain goals (Beste, Mückschel et al., 2018). In contrast, XDP patients were not impaired when they had to switch between inhibiting a response or not (Beste et al., 2017a) or when they were required to carry-out perceptual decision-making (Beste et al., 2017b), functions assumed to be related to the striatal matrix compartment which is intact in XDP patients. Translated to neuropsychiatry, it can thus be hypothesized that the OCD phenotype might be associated with hyperactivity in the striosome compartment and hypoactivity in the matrix compartment whereas the ADHD phenotype might be linked to an increased matrix to striosome activity ratio. Future studies should test the hypothesis using high-field MRI which is able to identify striosome and matrix compartments in the human striatum in vivo (Waugh et al., 2016).

In sum, alteration of activity in the balance between direct and indirect pathways in CSTC circuits might be responsible for shifts of metacontrol biases towards cognitive persistence, as observed in people with OCD, or towards cognitive flexibility, as observed in individuals diagnosed with ADHD.

4.2. Alteration of neural noise

Another factor that seems to be systematically related to the cognitive profiles of OCD and ADHD is neural noise and, in particular, the individual signal-to-noise ratio (SNR). To better understand the possible role of neural noise in the processing of information, it is important to consider that such noise does not always impair performance, but may, under some circumstance, facilitate information processing, because random fluctuations can be regarded as biologically relevant "background" that can increase the degree to which signals stick out in the nervous system (Guo et al., 2018; McDonnell and Ward, 2011a). This idea is highlighted by stochastic resonance approaches, according to which an optimal amount of noise can improve behavioral performance (McDonnell and Ward, 2011b).

An amplified SNR has been proposed to reflect an increment in neuronal gain control (Servan-Schreiber et al., 1990; Yousif et al., 2016; Ziegler et al., 2016). Like the volume control on television, our encephalon requires gain control to fine-tune the balance between neuronal input, which can vary substantially depending on changes in the environment, and neuronal output, which in effect occurs within a limited range of amplitudes (Priebe and Ferster, 2002). The relation between neuronal input and output can be illustrated by an S-shaped (sigmoid) function where the net-input is depicted by the horizontal axis and the net-output is depicted by the vertical axis. A steep S-shaped function indicates robust gain modulation processes which results in enhanced ability to disentangle signal and noise (i.e., enhanced SNR)—a condition that is likely to be particularly useful for a persistent (i.e., highly focused, selective) processing style but counterproductive for flexible, adaptive behavior (Findling and Wyart, 2021). In contrast, if the S-shaped function is too flat, the SNR is too low bringing too much neural noise for persistent processing, but promoting cognitive flexibility and impulsivity (Söderlund et al., 2007). If OCD would indeed be characterized by a metacontrol bias towards persistence, one would thus expect particularly high SNR in patients with OCD, and if ADHD is characterized by a bias towards flexibility, one would expect a particularly low SNR in patients with ADHD.

The idea of an alteration of gain control in OCD has been indirectly investigated via sensorimotor gating, the phenomenon of filtering or "gating" relevant sensory information (i.e., the signal) from irrelevant motor information (i.e., noise). A well-established index of sensorimotor gating is the pre-pulse inhibition (PPI) of startle, where the startle response is inhibited by the presentation of a weak stimulus (pre-pulse) before an intense startling stimulus (pulse), in an inter stimulus interval between 30 and 500 ms (Graham, 1975). When the startle reflex pathway (including the cortico-striato-pallido-pontine circuits) is disrupted, sensorimotor gating becomes sluggish and a strong response reaction takes place (Kohl et al., 2013). Studies investigating PPI in OCD (de Leeuw et al., 2010; Hoenig et al., 2005; Swerdlow et al., 1993) showed inconsistent results, presumably because of different methodologies being used (Kohl et al., 2013). However, it has been shown that unmedicated patients with OCD, especially with a history of a tic disorder, displayed reduced PPI (Ahmari et al., 2012). Additional evidence comes from a transcranial magnetic stimulation (TMS) study investigating short latency afferent inhibition (SAI) which is considered an index of sensorimotor integration (Turco et al., 2018). In line with the idea that patients with OCD suffer from deficient sensorimotor integration, patients with OCD showed reduced SAI (Russo et al., 2014).

The hypothesis of low SNR in ADHD has been investigated using both indirect and direct measurements. Regarding indirect indexes, patients with ADHD have been reported to display intact sensorimotor gating (as evidenced by normal PPI) but to show impaired sensory gating (as evidenced by alterations in the suppression of P50 auditory event-related potentials (ERPs)) (Holstein et al., 2013), suggesting that ADHD is characterized by alterations in "sensory noise". Concerning direct measurements of SNR, the first EEG study to investigate neural noise in ADHD used "1/f noise" as an index for the so-called "pink noise" or scale-free neural activity (Pertermann, Bluschke et al., 2019). Via the assessment of these parameters that reflect noise in neurophysiological signals, the hypothesis of low SNR in ADHD can be verified directly. Compared to "white noise" and "brown/red noise", pink noise does not reflect meaningless unstructured noise (He, 2014), but contains spatio-temporal organization relevant for information processing and brain functioning (He, 2014). In a nutshell, the idea is that neural noise is reflected by the distribution of neural activation (i.e., the power spectral density [PSD]) across the entire (EEG) frequency spectrum (Dave et al., 2018). 1/f noise can be depicted by a slope, a negative linear relationship resulting from the calculation of the logarithm of PSD across the frequency spectrum (Dave et al., 2018; He, 2014). Hence, a flatter slope reflects more neural noise (Dave et al., 2018; He, 2014). Consistent with the hypothesis of low SNR in ADHD, unmedicated patients with ADHD displayed a flatter slope compared to healthy controls (Pertermann, Bluschke et al., 2019). In contrast, after receiving methylphenidate, a dopaminergic and noradrenergic agonist, the slope of patients with ADHD became steeper—showing that the medication reduced neural noise (Pertermann, Bluschke et al., 2019). However, it is important to consider that there are many forms and definitions of noise. In fact, 1/f noise does not reflect nuisance activity (He, 2014) in the sense of background noise (Servan-Schreiber et al., 1990), which is why more neutral terms have been introduced (Donoghue et al., 2020). The question how exactly noise should be operationalized is therefore central for the metacontrol framework we apply here. Nevertheless, taken altogether, there is increasing evidence that SNRs are systematically related to OCD and ADHD: while particularly high SNRs have been observed in OCD, particularly low SNRs were obtained in ADHD. We argue that this renders individual SNR an important indicator of both metacontrol and the processing styles associated with OCD and ADHD. At this point, it is still unclear whether particularly high or low SNR levels are the cause of metacontrol biases towards persistence or flexibility, or merely indicators (functional markers) that signal particular biases. In any case, however, there is increasing evidence that SNR is systematically associated with particular metacontrol biases in OCD and ADHD (Münchau et al., 2021). In particular, OCD seems to be associated with a particularly high SNR, suggesting that this is a cause or indicator of cognitive persistence. In contrast, ADHD seems to be associated with a particularly low SNR, suggesting that this is a cause or indicator of cognitive flexibility, see Fig. 1.

It is important to consider that different disorders have been hypothesized to suffer from alterations of neural noise at different stages of information processing, such as sensory processing (i.e., sensory noise), sensorimotor processing (i.e., sensorimotor noise), motor processing (i.e., motor noise) or decision-making processing (i.e., decision-making noise). For example, tic disorders seem to be characterized by altered

motor noise (Beste and Münchau, 2018; Münchau et al., 2021) affecting motor processing (i.e. the process by which muscular movements are combined in the production of simple or complex motor acts). Instead, OCD has been hypothesized to be associated with alterations in decision-making noise affecting decision-making processes (i.e., the process of selecting a response among several alternative options) and sensorimotor noise impacting sensorimotor processes (i.e., the process by which sensory information or input is coupled or integrated to a related motor response in the CNS) (Ahmari et al., 2012; Russo et al., 2014). In contrast, ADHD has been claimed to be associated with alterations in sensory noise (i.e., the ability of CNS to collect, process and regulate responses to sensory information) (Söderlund et al., 2007).

5. Novel treatments to modulate the SNR: tRNS and atVNS

We have argued that the relationship between direct and indirect pathways in CSTC circuits and the individual SNR are important factors in accounting for particular metacontrol biases towards persistence and flexibility in OCD and ADHD, respectively. Especially the second factor, the SNR level, points to an interesting new therapeutic option. If SNR indeed reflects a particularly strong bias towards persistence or flexibility, techniques that are able to change this bias might be useful to treat these cognitive/neural mechanisms underlying OCD and ADHD. Indeed, in addition to current first-line pharmacological treatments targeting the dopaminergic and serotoninergic systems (Cortese, 2020; Golmirzaei et al., 2016; Rothenberger and Roessner, 2019), neural noise may also be modulated directly or by other neurotransmitter systems that are of relevance in OCD and ADHD. In this regard, the noradrenaline (NE) and GABAergic systems are of particular interest (Bandelow et al., 2017; Del Campo et al., 2011; Puts et al., 2020). We propose that a way to verify our dimensional hypothesis of OCD and ADHD, based on a shared neurobiological vulnerability, is applying novel noninvasive brain-stimulation based treatment approaches affecting the SNR directly or via targeting the NE and GABAergic systems. Brain stimulation treatments have seen an increase in popularity to modulate OCD and ADHD symptoms (Rapinesi et al., 2019; Wong and Zaman, 2019). Even though further randomized clinical trials with larger sample sizes are required, several meta-analysis and empirical studies suggest that popular neurotherapy of OCD is TMS (Rapinesi et al., 2019; Trevizol et al., 2016) and of ADHD is neurofeedback (Bluschke et al., 2016, 2018, 2020; Riesco-Matías et al., 2021; Van Doren et al., 2019). However, it is important to keep in mind that, as pointed out by a consensus paper (Faraone et al., 2021), neurofeedback has "no effect on inattention symptoms, but a small-to-medium reduction in hyperactivity-impulsivity symptoms (Van Doren et al., 2019)." Relevant for our purpose, novel methods to modulate the SNR are transcranial random noise stimulation (tRNS), as proposed by Pavan et al. (2019), and auricular transcutaneous vagus nerve stimulation (atVNS), as recently reviewed by Colzato and Beste (2020). Noninvasive brain stimulation might be especially interesting for severe cases of OCD and ADHD, i.e., when pharmacological treatment becomes insufficient due to augmentation, the development of tolerance or contraindication for catecholaminergic and serotonergic treatment. Given that emotional dysregulation is an important aspect in ADHD and that pharmacological interventions for ADHD are not overly effective in treating this aspect compared to ADHD core symptoms (De Crescenzo et al., 2017), atVNS might be an interesting option in this regard. Indeed, atVNS has been found to enhance cognitive emotion regulation (De Smet et al., 2021), likely via its modulation of the anterior cingulate and lateral PFC, areas known to be dysregulated in ADHD patients (Qiu et al., 2011). Anyhow, it should be mentioned that especially in the case of non-pharmacological interventions, the ideal evaluation of treatment efficacy should always include blinding by design and blinding by reporter in order to avoid any potential bias on study outcomes (Coghill et al., 2021).

5.1. tRNS

tRNS is a type of transcranial electrical stimulation (tES) that can directly affect the SNR (Pavan et al., 2019). Typically, low intensity currents (1–2 mA) are applied via electrodes located on the cranium (Paulus, 2011). In contrast to TMS, that produces action potentials, tES modulates spontaneous firing rates of cortical neurons and elicits variations in cortical excitability. These changes can last for up to one hour after the cessation of the stimulation (Nitsche and Paulus, 2000; Paulus et al., 2016). In tRNS, a low-intensity alternating current is administered in a way that the intensity and the frequency of the current fluctuate in a randomized fashion (Paulus et al., 2016). The effects gained by tRNS can be elucidated within the context of stochastic resonance (Gammaitoni et al., 1998). Namely, a signal that would usually be too low to be detected by a sensor can be augmented by adding white noise to it. White noise contains a wide spectrum of frequencies and, because of that, the frequencies in the white noise complementing the original signal's frequencies elicit resonance. That is, the original signal, but not the residual white noise, is amplified increasing the SNR and getting the original signal easier to be detected. Therefore, we propose tRNS as a suitable tool to normalize the SNR and to restore the balance between cognitive persistence and cognitive flexibility via stimulating the cortical areas linked to the respective functions affected in OCD and ADHD. On the one hand, the stimulation of the orbitofrontal cortex (OFC), which is linked to cognitive flexibility (Klanker et al., 2013), might compensate for deficits in switching between mental sets in OCD. On the other hand, the stimulation of the dorsolateral prefrontal cortex (PFC), which is linked to cognitive persistence/stability (Fallon et al., 2013) might compensate for deficits in focusing on one goal and on relevant information in ADHD.

To conclude, tRNS has the potential to fine-tune the SNR to enhance cognitive flexibility in OCD and enhance cognitive persistence in ADHD via stochastic resonance, a phenomenon known to adjust the SNR (Chapeau-Blondeau, 1997). Given that tES has been proven to modulate neural plasticity for minutes or hours succeeding the stimulation (Nitsche and Paulus, 2000), we propose tRNS as a promising candidate to normalize the balance between cognitive persistence and cognitive flexibility in OCD and ADHD.

5.2. atVNS

Apart from tDCS, which directly modulates the SNR via affecting the neuron membrane potential, one way to modulate gain control processes in relation to the NE and the GABAergic systems is via atVNS (L. Colzato and Beste, 2020). Even though atVNS was initially intended for purely clinical purposes, it can also be employed as a promising neuromodulation tool to enhance cognitive functions (Van Leusden et al., 2015). In contrast to neuroimaging methods merely delivering correlational data, empirically administering atVNS causal inference regarding the modulated neurotransmitters and the cognitive functions mediated by them. AtVNS is administered via a specific earplug- lookalike electrode to the outer ear, providing low intensity currents to the auricular branch of the vagus nerve also called Arnold's nerve. Functional magnetic resonance imaging studies proved that active, but not sham atVNS activates brainstem regions comprising the nucleus of the solitary tract (GABAergic center) and the locus coeruleus (NE center) (Dietrich et al., 2008; Frangos et al., 2015; Kraus et al., 2013; Yakunina et al., 2017, 2018). Interestingly, atVNS mediated modulation of the NE system plausibly enhances the SNR that has been demonstrated to be directly related to the activity of the locus coeruleus-NE system (Pertermann, Mückschel et al., 2019). As already pointed out in section "Shared neurobiological vulnerability: alteration of neural noise", the SNR mirrors neural gain control mechanisms (Servan-Schreiber et al., 1990) and is affected by NE (Kroener et al., 2009). Hence, it has been proposed that atVNS strengthens gain control processes in the PFC with the consequence that cognitive processes depending on gain modulation will be

enhanced by atVNS (Colzato and Beste, 2020). Indeed, as we recently reviewed (Colzato and Beste, 2020), several studies have shown the potential of tVNS to alter cognitive functions related to NE and GABA, such as cognitive persistence and cognitive flexibility.

In sum, atVNS has the potential to fine-tune the SNR via NE and GABA release, two neurotransmitter systems that are of relevance in OCD and ADHD (Bandelow et al., 2017; Del Campo et al., 2011; Puts et al., 2020). Accordingly, we propose atVNS as a novel tool to optimize the balance between cognitive persistence and cognitive flexibility in OCD and ADHD.

6. Conclusion

The current article provides a novel dimensional approach questioning the view of OCD and ADHD as fully separable conditions. We suggest the metacontrol model (Hommel, 2015; Hommel and Colzato, 2017) as a novel cognitive diagnostic framework to explain aspects of the specific cognitive profile linked to OCD and ADHD. We propose that OCD and ADHD are characterized by processing styles that are biased towards the extreme poles of a continuous metacontrol dimension ranging from persistence to flexibility. We suggest that these biases are associated with abnormal functioning of CSTC circuits and alterations of the individual SNR. We propose that high SNR/gain control in OCD reflects a bias towards cognitive persistence, which facilitates performance that requires the focusing on one goal and/or the neglect of irrelevant information, but impairs performance in tasks that rely on cognitive flexibility. In contrast, low SNR/gain control in ADHD reflects a bias towards cognitive flexibility, which facilitates tasks that require or benefit from the consideration or integration of various kinds of information, including non-obvious or novel stimuli, but impairs performance in tasks that rely on cognitive persistence. Future studies should test our hypothesis using treatments known to directly target SNR, such as tRNS, or indirectly via the modulation of GABA and NE, such as atVNS. Accordingly, we encourage the use of well-defined designs to examine whether tRNS and atVNS are valuable treatments in OCD and ADHD to optimize the delicate balance between cognitive persistence and cognitive flexibility.

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