# Evaluating Orbital–Ventral Medial System Regulation of Personal Attention: A Critical Need for Neuropsychological Assessment and Intervention

**Article** *in* Applied Neuropsychology Child · March 2015

DOI: 10.1080/21622965.2015.1005486 · Source: PubMed



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APPLIED NEUROPSYCHOLOGY: CHILD, *0*: 1–10, 2015

ISSN: 2162-2965 print/2162-2973 online
DOI: 10.1080/21622965.2015.1005486

# Evaluating Orbital-Ventral Medial System Regulation of Personal Attention: A Critical Need for Neuropsychological Assessment and Intervention

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Attention to self and environment form the basis of effective social exchange and relationships. Although implicit in this basic social competency is the ability to be self-aware and responsive to circumstances of others, many neuropsychologists have yet to understand or measure its basic functions, let alone recognize the brain–behavior relationships that govern this area. Several years ago, interest in "emotional intelligence" rose to the forefront of popular psychology, but we are still unraveling the cortical, subcortical, and neurocellular interactions that produce this nebulous construct, and we are determining how dysfunctional frontal-subcortical and cortico-cerebellar circuitry can lead to aberrant social dynamics and ultimately psychopathology when maladaptive patterns become routinized. In this article, we explore the orbital-ventral medial circuitry thought to govern emotional attention, personal self-regulation, social concern and exchange, and affective aspects of interpersonal relationships. Our examination notes both the dearth of and need for neuropsychological research on the biological basis and measurement of executive regulation of emotional attention, behavioral regulation, and social competence. We conclude with a call for development of neuropsychological measures and methods that can foster differential diagnosis and targeted treatment strategies for children with orbital-ventral medial circuit dysfunction.

*Key words:* *emotional, orbital, self-regulation, social competence, ventral-medial*

## INTRODUCTION: A CASE STUDY OF "MULTIPLE PSYCHIATRIC COMORBIDITIES"

In a Grand Rounds presentation several years ago, a neuropsychology trainee presented to his audience the case of a child who displayed significant signs of psychopathology. The 11-year-old boy had considerable problems with attention, impulse control, organization, emotional lability, and unpredictable behavior. His emotional and behavioral issues significantly interfered with academic performance, and his peer and adult relationships were quite strained. The boy could be quite distractible, talkative, affable, and gregarious, which sometimes verged on overpowering and domineering behavior, but at other times, he could be quite subdued, lethargic, aloof, and withdrawn. The neuropsychologist trainee then presented the cognitive, neuropsychological, academic, and personality/behavioral evaluation results. As the data from the attention and executive function measures were presented, test result after test result showed mostly average or above-average performance. There were some qualitative signs that attention and executive function were problematic, and the

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personality- and behavior-rating scale results showed significant signs of attention, hyperactivity, depression, anxiety, opposition, and atypicality problems, with adaptive functioning quite low, especially in functional communication. The behavior ratings of executive function, particularly in the area of self-regulation, were significant concerns according to parent and teacher reports (but not self-report, which was within normal limits).

The neuropsychology trainee was perplexed by this child's presentation. Why were there "no" signs of neuropsychological impairment in a child with obvious executive and psychosocial concerns? Then someone in the audience offered a conclusion: This case was obviously a "clinical" and "psychiatric" case, not a neuropsychological case, as if the two were distinct and unrelated. Continuing with this position, this clinical psychology colleague suggested this case had multiple "comorbidities" that could account for the behaviors displayed, including attention-deficit hyperactivity disorder (ADHD), depression, generalized anxiety disorder, oppositional defiant disorder, and learning disability. So the broad consensus of the audience was to give multiple diagnoses, consult with psychiatry, and provide an extensive list of learning and psychosocial interventions. One audience member concluded this child must be "on the autism spectrum," and pediatric bipolar disorder was considered by several audience members, but the audience questioned the validity of the diagnosis in young children and noted that the other diagnoses had more of an "evidence base" in the literature.

What the trainee and the audience had failed to appreciate was that most—if not all—cases of developmental psychopathology have executive dysfunction, so there are few "clinical" or "psychiatric" cases that do not have executive impairment—we just do not typically measure it well using traditional neuropsychological assessment (e.g., Weyandt et al., 2014), and we often find little agreement between direct performance measures and indirect informant report of executive dysfunction (e.g., McAuley, Chen, Goos, Schachar, & Crosbie, 2010), thereby making symptom coherence and case conceptualization exceedingly difficult. Instead of a laundry list of "multiple comorbidities," cases can and should be conceptualized as reflecting a more coherent picture of underlying and interacting neural mechanisms that produce the clinical presentation we observe. Despite their importance in understanding developmental psychopathology, the problem is that we just do not have the sophisticated neuropsychological tools available, and/or perhaps sufficient clinical acumen because of this limitation, to effectively measure and understand deficits in self-regulation of affective attention, interpersonal relationships, and social judgment—functions of the orbital-ventral medial prefrontal cortical networks. Even the most skilled clinician will struggle with case conceptualization when ventral system dysfunction is present, or they will recognize how it can lead to many quite different psychopathologies, including ADHD and bipolar, conduct, autism, and anxiety disorders (e.g., Reddy, Weissman, & Hale, 2013). In an attempt to address this limitation in clinical practice, we offer an overview of the circuitry involved in self-regulation of emotional and behavioral attention and provide implications for neuropsychological training, research, and practice.

## NEURODEVELOPMENT AND FUNCTIONAL ORGANIZATION OF FRONTAL-SUBCORTICAL CIRCUITS

There are numerous cortical and subcortical structures implicated in emotional attention to self and the environment, including the cerebellar vermis, thalamic pulvinar, amygdala, hypothalamus, hippocampus, basal ganglia, nucleus accumbens, insula, right temporal-parietal association cortices, ventral cingulate, and orbital-cortical regions (Alexander, DeLong, & Strick, 1986; Koziol & Budding, 2009; Lichter & Cummings, 2001; Miller & Cummings, 2007; Rubia, 2011; Stuss & Alexander, 2000). We focus our discussion here on the top-down orbital and ventral-medial circuitry thought to govern this "emotional intelligence" or self-regulation of affective and intrapersonal attention. These areas responsible for development of social competence and personality, affective responsiveness, and self-awareness may be the most important function of the human frontal lobes (Stuss & Alexander, 2000), even though they are often poorly understood, measured, evaluated, and treated.

Although the prefrontal area occupies one third of the cerebral cortex (Fuster, 1997), delineation of the different frontal-subcortical circuit (FSC) functions has only begun relatively recently in human neuropsychology. A picture has emerged that the FSCs do not process information per se, but they regulate and govern posterior brain region processing of information. Although five to seven "prototypical" FSCs have been identified (e.g., motor, oculomotor, dorsolateral, orbital, cingulate; Lichter & Cummings, 2001; see Figure 1), there is an evolutionary trend that leads to dorsal and ventral system differences—a task-oriented metacognitive dorsal system and affective-motivational ventral medial system (Ardila, 2008). Although anatomically and functionally different, these two systems are highly related given that emotion can have a positive or negative effect on cognitive control during executive task performance (Mitchell & Phillips, 2007). For instance, we have known for some time that the dorsolateral region is important for working memory and for long-term memory encoding and retrieval (Fletcher, Shallice, & Dolan,

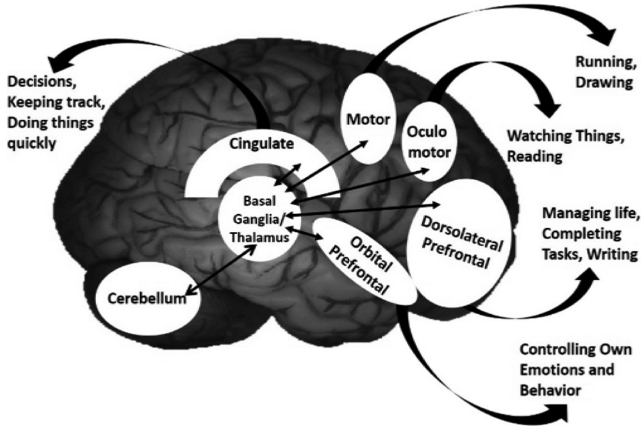


FIGURE 1 Frontal-subcortical circuits and their functions.

1998; Fletcher, Shallice, Frith, Frackowiak, & Dolan, 1998) and that orbital-mediated emotional valence has an inverted U-shaped effect on the accuracy of memory (Bechara, Damasio, & Damasio, 2000; Poletti, 2010), so clearly these FSCs work together in processing both cognitive and affective aspects of executive control of attention (Hale et al., 2009). As a result, the dorsolateral-dorsal cingulate is considered the ‘‘cool’’ circuit responsible for *external* executive control and the orbital-ventral cingulate may be considered the ‘‘hot’’ circuit responsible for *internal* self-regulation (Ardila, 2013; Arnsten & Rubia, 2012; Hale, Reddy, Weissman, Lukie, & Schneider, 2013).

Given the complexity of FSC function, it is not surprising that this area is the last to fully develop (Giedd & Rapoport, 2010; Waber et al., 2007) or to be assessed effectively (Anderson, 2002), with neurodevelopmental trajectories suggesting that considerable changes happen just before a child enters school, again in adolescence, and continuing on to early adulthood. Interestingly, of the white-matter pathways so critical to social functioning (Johansen-Berg, 2010), the uncinate fasciculus is the last to fully develop (Hasan et al., 2009). This suggests that of all brain functions, advanced social competence and interpersonal attachment may not reach their full potential in all individuals until early adulthood. Neurodevelopmental disorders can be mapped longitudinally in relation to typical brain development, suggesting differences in FSC neurodevelopmental delays or aberrant pathways can differentiate several childhood disorders such as ADHD, schizophrenia, and autism (Marsh, Gerber, & Peterson, 2008; Shaw, Gogtay, & Rapoport, 2010).

Neurodevelopmental differences in executive control of emotional attention and self-regulation are likely affected by the subcortical areas that the orbital and ventral-medial circuits regulate. A graphical display of these circuits is represented in Figure 2. As can be seen, the connections with the striatum are somewhat different in that the orbital cortex has a relationship with the caudate, whereas the cingulate is also related to putamen and nucleus accumbens function. Although both the caudate and putamen are excitatory, the caudate has been linked to sensorimotor functions, whereas the putamen has been related to appetitive drives, including motor functions, and the nucleus accumbens relationship explains its role in reinforcement and punishment (Voorn, Vanderschuren, Groenewegen, Robbins, & Pennartz, 2004). Additional features of these circuitries include the nigrostriatal (excitation, movement, behavior maintenance), mesolimbic (reward and emotional valence), and mesocortical (motivation) pathways. The mesolimbic profile, in particular, with influences on the nucleus accumbens, amygdala, hippocampus, and hypothalamus, is heavily involved in the regulation of emotional valence, whereas the mesocortical profile influences higher-level emotional regulation by impacting the cingulate or orbital cortex (Barbas, 2007; Robbins, 2010; Tomasi & Volkow,

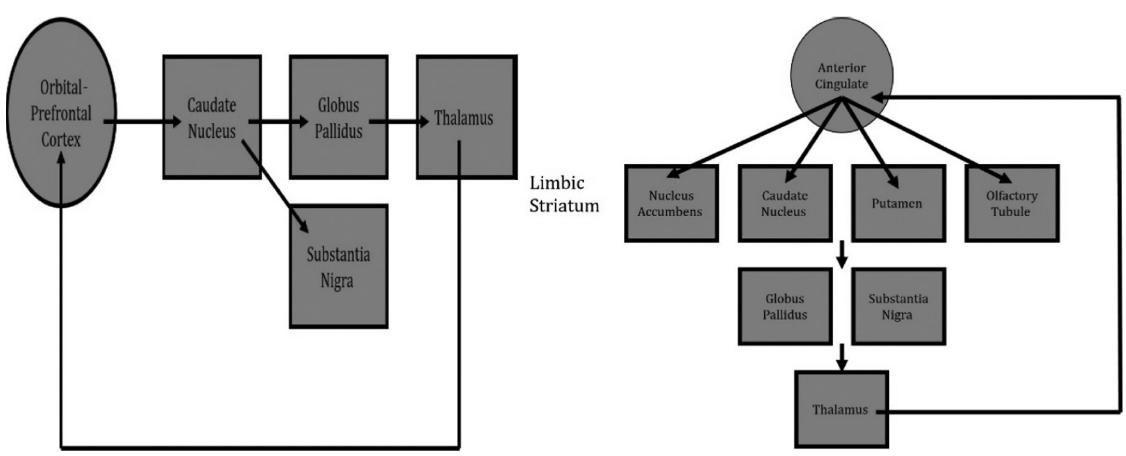


FIGURE 2 Orbital and anterior cingulate circuits (adapted from Lichter & Cummings, 2001).

2012)—a contrast we discuss in terms of "cool" cognitive versus "hot" emotional attentional and behavioral control.

## MOVING BEYOND THE CORTICO-CENTRIC "COOL" DORSAL AND "HOT" VENTRAL EXECUTIVE SYSTEMS

Despite the allure of thinking that "cool" dorsal and "hot" orbital-ventral medial systems govern *external* (environment-focused) and *internal* (self-focused) executive control, respectively, we need to recognize that these circuits are not orthogonal and influence each other in more complex ways than this oversimplified dichotomy suggests. For instance, *cognitive* impulse control would be governed by the dorsal system, but *emotional* impulse control would be a component of the ventral medial system. Thus, how does a practitioner determine whether a child's impulsivity is more cognitive or emotional in nature, especially when internal-state and external task or environment demands might differentially influence these systems? For one child, impulsive behavior could be the result of external distraction (e.g., ADHD), whereas for another child, it could help release internal tension and pressure (e.g., obsessive-compulsive disorder [OCD]). This is just another clear example of why informant reports do not lead us to clear-cut diagnostic pictures or effective treatment regimens for affected children and why a neurobiological approach governed by careful neuropsychological evaluation is needed to understand and serve children with emotional and/or behavior disorders.

Attentional constructs typically thought of as "cognitive," such as selective, sustained, and divided attention, may be influenced by the orbital-ventral medial regions more than one might initially think, especially when one considers circuit subcortical connectivity. Through striatal connections, these regions would be associated with all three aspects of attention, because this could affect planning and modulation of movement for goal-directed behavior (Kringelbach, 2005). Their limbic connectivity seems to be of prime importance for *selective* attention for integration of information into behavioral output (Balleine & O'Doherty, 2010). Connectivity with the thalamic region suggests a role in inhibitory and excitatory regulation of the autonomic and sensorimotor function (Cavada, Company, Tejedor, Cruz-Rizzolo, & Reinoso-Suarez, 2000) and is thus, again, related to *selective* and *sustained* attention, with impairment perhaps leading to *emotional* impulsivity or perseveration. This connectivity combined with cerebellar (particularly vermis) functions provides both for automaticity and amplification of behavioral repertoires, which if dysfunctional, could amplify or depress volitional activity or provide automatic maladaptive responses, both significant factors in the manifestation of psychopathology (Koziol, Budding, & Hale, 2013). In essence, this complex frontal-striatal-thalamic-cerebellar circuitry, in combination with other cortical areas, governs volition over time to determine what an individual does, the circumstances that suggest when to do it, and once activated, how it will be carried out (Bostan, Dum, & Strick, 2013; Koziol et al., 2014).

Unlike the largely externally focused dorsal system, the orbital and medial-ventral regions appear to play a balancing act to govern internal self-focused states (e.g., medial) in relation to external environmental circumstance (e.g., lateral), making its function more complex than the dorsal system. Perhaps that is why most of our neuropsychological attention has been focused on the "easy"-to-understand dorsal system and external executive control. Whereas the medial region is more associated with making stimulus–reward associations with reinforced behaviors, the lateral region is more associated with evaluation of stimulus–outcome associations (Walton, Behrens, Buckley, Rudebeck, & Rushworth, 2010). Thus, while the medial region likes or dislikes circumstances, the lateral region is more cognitive in nature, as it encodes expectations about outcomes and social reprisal (Campbell-Meiklejohn et al., 2012; Tanferna, López-Jiménez, Blas, Hiraldo, & Sergio, 2012). Interestingly, the lateral region appears to be more responsive to punishment or negative outcomes (perhaps more externally focused), whereas the medial region appears to be more responsive to reward (perhaps more internally focused; e.g., Elliott, Agnew, & Deakin, 2009).

This lateral-medial divide can be further understood by examining interconnections with the amygdala, hypothalamus, and hippocampus (Barbas, 2007; Zald et al., 2014). First, the medial regions are essential in self-regulation of response selection based on anticipated emotional valence of positive or negative outcomes (e.g., orbital-amygdala; Baxter, Parker, Lindner, Izquierdo, & Murray, 2000; Dolan, 2007). This could be considered an aspect of what is often termed *selective attention* as an individual chooses to focus on a particular set of behaviors over another in anticipation of outcomes based on prior experience. Second, the medial region is involved in self-regulation of motivational states for autonomic drive regulation and maintenance of behavior to meet individual needs (e.g., orbital-hypothalamus; Lichter & Cummings, 2001; Price, 2007). This function would be important for maintaining goal-directed behavior over time to meet one's needs, so it could be considered important in governing *sustained attention*. Finally, the lateral regions appear to be important in providing emotional tone during memory encoding and retrieval (e.g., orbital-hippocampus; Churchwell & Kesner, 2011; Hoover & Vertes, 2011). With evidence suggesting memory function is impaired during excessive or minimal emotional states as discussed earlier, this function could influence how individuals acquire and use memory in adaptive ways for goal-directed behavior (Young & Shapiro, 2011).

Thus, the lateral and medial divisions indirectly influence selective, sustained, and divided attention in a competitive fashion, with the lateral system more related to cognitive and ''cool'' dorsolateral circuitry than the medial areas. In our treatise, perhaps the lateral part of the orbital cortex is ''cooler'' than the ''hotter'' ventral-medial circuitry and serves a critical bridge function between cognitive and affective executive control of attention. This position is in part supported by meta-analytic findings of lateral and ventral orbital regions, with the lateral regions influencing cognitive skills like language and memory, while the medial region is more associated with autonomic and limbic systems that govern internal states (Zald et al., 2014).

To add a final layer of complexity in our cursory examination of the complex circuitry, we briefly examine neurotransmission and its influence on circuit function. Neurotransmitters often have multiple effects that unfortunately are not straightforward or simple, hence the need for extensive psychopharmacology research efforts. Exceedingly complex relationships among neurotransmitters and their influence on each other is often the case in these studies, with dopamine, norepinephrine, serotonin (primarily excitatory), and gamma-aminobutyric acid (GABA; primarily inhibitory) neurotransmitters interacting to differentially influence cortical and subcortical function. Considered within the context of the FSCs, the excitatory caudate and putamen and the direct and indirect pathways of the inhibitory globus pallidus influence FSC function in subtle ways that are not easily ascertained during neuropsychological assessment. Considered at the neurotransmitter level, there are different types of neurotransmitter receptor sites (e.g., D1, D2, D3, D4, D5 dopamine receptors) that can be excitatory or inhibitory and reciprocal tonic and phasic neurotransmitter influences (e.g., steady-state levels vs. short-term bursts and dips), so relationships on neuropsychological test performance or observable behavior are seldom clear or straightforward. For instance, considerable evidence suggests insufficient striatal dopamine availability as the cause of ADHD, and this leads to FSC hypoactivity and the symptoms displayed (e.g., Arnsten & Rubia, 2012). This is also supported by the plethora of research supporting stimulant treatment (dopamine agonists) of ADHD. However, if it were just insufficient dopamine, then treating ADHD with L-dopa (dopamine precursor) would make sense, but L-dopa is ineffectual in treatment of the disorder. In addition, dopamine can have both excitatory (D1 receptors) *and* inhibitory (D2 receptors) effects, suggesting decomposition of such brain–behavior relationships is exceedingly complex at the neurocellular and neurochemical level. However, this is beyond the scope of this article.

What emerges in this discussion is a neuropsychological picture of competing ''interests'' in the FSCs at the cortical, subcortical, and neurocellular levels. What these multiple influences on cognition and behavior suggest is that a simple ''more is better'' or ''less is better'' perspective does not help us understand how executive control works; instead, it appears that optimal executive control is actually a *balance* of excitatory and inhibitory influences (Hale et al., 2009). It is because of this balance that a child develops meaningful understanding of status of self in relationship to the status of the environment (i.e., ''what is going on inside of me'' vs. ''what is going on around me''), and it is this competition that must be balanced for optimal executive function to occur in the real world in real time.

## CIRCUIT BALANCE THEORY: IMPLICATIONS FOR NEUROPSYCHOLOGICAL ASSESSMENT AND INTERVENTION

What emerges from this review of internal and external executive control is that a balance is needed for optimal executive control within the natural environment, as elucidated in what Hale and colleagues refer to as circuit balance theory (Hale et al., 2009, 2013). To connect with the real world in real time in a successful way, a child must adaptively respond to the external environment while maintaining awareness of his internal state. Circuit balance theory posits that deviation or imbalance—either of the internal or external executive systems—leads to maladaptive emotional or behavioral responses and to significant psychopathology (Koziol et al., 2013). Thus, optimal executive function is *average* executive function, with underactivation or overactivation of circuitry leading to a maladaptive response to environmental demands and/or internal states, which would lead to problems with selective, sustained, and divided attention, as suggested in Figure 3.

Note that while circuit underactivation could lead to ADHD symptoms due to *external* distraction, overactivation could lead to OCD symptoms and *internal* distraction, with *both* conditions leading to significant attention problems. In this perspective, perhaps the child with ADHD would compensate for FSC hypoactivity by seeking stimulation (e.g., Romer, 2010), and this explains why dopamine agonists (stimulants) have a beneficial effect by reducing hypoactivity (e.g., Levy, Wimalaweera, Moul, Brennan, & Dadds,

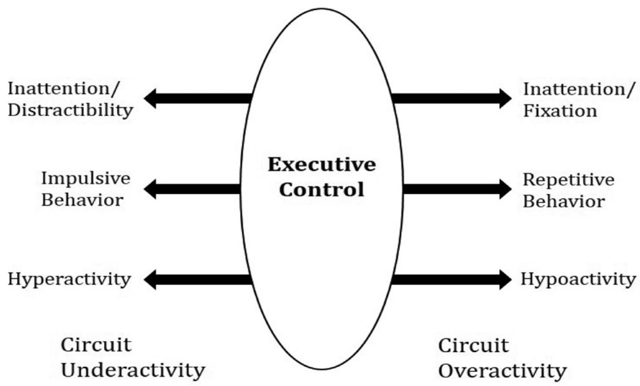


FIGURE 3 Circuit balance theory and associated symptoms.

2013). On the other hand, the FSC hyperactivity experience for a child with OCD symptoms might respond to excessive environmental stimulation by withdrawing and becoming internally focused to reduce the likelihood of becoming overwhelmed (e.g., Semrud-Clikeman, 2007), and this explains why dopamine antagonists might address this problem (e.g., Keuneman, Pokos, Weerasundera, & Castle, 2005). Thus, both would respond to environmental demands in a maladaptive way (albeit adaptive for their internal states) to compensate for their FSC imbalance. Perhaps the former child might be diagnosed with ADHD-Combined type and the latter as ADHD-Inattentive type, given both would have "attention problems" on informant reports.

Circuit balance theory also explains why comorbid depression and anxiety are so common and that the drug of choice for both depression and OCD are the selective serotonin reuptake inhibitor, which enhance serotonergic function. Depression is the result of hypoactive dorsal "cool" executive circuit dysfunction, while anxiety and OCD result from hyperactive ventral "hot" executive circuit dysfunction (Baxter et al., 2000; Fales et al., 2009; Grimm et al., 2008; Koenigs & Grafman, 2009; Milad & Rauch, 2007). Cingulate dysfunction leads to apathy and malaise in a clinical condition called abulia, characterized by lethargy, anhedonia, apathy, and malaise, so this is similar to symptoms of depression. The position in circuit balance theory is that if one or more circuits are hypoactive or hyperactive, this *imbalance* leads to compensatory and adaptive functioning of the other circuits. This has dramatic implications for both neuropsychological assessment and intervention. For instance, if a child has considerable orbital anxiety, their dorsolateral function will be depressed and the efficacy of their problem-solving skills on both neuropsychological tasks and real-life situations will be impaired. For treatment, decreasing anxiety through interventions such as systematic desensitization, thought stopping, or medication may be additionally improved by increasing dorsolateral engagement with the environment through activities that require social engagement and problem solving. The clinical treatment goal then is to not only *decrease* symptoms of one circuit, but to also *increase* the functions of the other circuit.

Not surprisingly, orbital dysfunction has been associated with emotional lability, indifference to social situations or tactlessness, poor social judgment, emotional impulsivity, and irresponsible behavior, with bipolar and conduct disorders also related to orbital-ventral medial dysfunction. The question remains as to how orbital-ventral medial dysfunction can lead to both internalizing anxiety-OCD and externalizing conduct disorders. Again, circuit balance theory can be extended to consider these possibilities, with circuit underactivity found in conduct disorder (e.g., Rubia, 2011) and circuit overactivity related to anxiety and obsessive-compulsive symptoms (e.g., Stern et al., 2012). There may be a lateralizing effect as well here and in other conditions, with the left FSC dysfunction or underactivation leading to depressive symptoms (Grimm et al., 2009) and ADHD symptoms related to right FSC dysfunction or underactivation (Arnsten & Rubia, 2012; Bush, 2011), which would be consistent with hemispheric studies of emotion and behavior (Beraha et al., 2012; Holtgraves & Felton, 2011). It would also explain, for instance, why ADHD tends to be a right dorsolateral hypoactivity problem, why depression appears to be a left dorsolateral hypoactivity problem, and how the orbital-ventral medial circuits are involved in the "comorbidities" associated with them (hyperactive orbital functioning in OCD and hypoactive orbital functioning in conduct disorder). Clearly, what is emerging is a movement away from a global executive dysfunction approach to understanding child psychopathology to one in which different circuits are related to different disorders, and from these efforts, we can move away from behavioral diagnostic criteria toward criteria in which neurobiological and genetic markers can be ascertained (Insel, 2013).

How does circuit balance theory and neuropsychological assessment relate to administration and interpretation of neuropsychological tests? The traditional neuropsychological approach to cognitive aspects of attention does not consider how orbital-ventral medial circuitry might influence performance on our executive measures. That is because orbital-ventral medial circuitry would only *indirectly* influence performance on traditional "cognitive" neuropsychological executive measures. As it reflects the cortical representation of limbic system functions, it considers the best time, place, and strategy to invoke a behavioral response (Bonelli & Cummings, 2007). In other words, these orbital-ventral medial areas do not necessarily carry out the cognitive functions necessary to perform traditional executive tasks, but instead, they have subtle (in typical populations) and potentially significant (in clinical populations) effects on our cognitive neuropsychological data designed to largely tap dorsal system function. Traditionally, the main function of the orbital-ventral medial circuits is to modulate affective and social behavior and integrate memory and emotional valence, both of which clearly influence cognitive decision making by considering expected outcomes of action (Bechara et al, 2000; Murray, O'Doherty, & Schoenbaum, 2007) for adaptive (Baxter et al., 2000) and even maladaptive response selection (Wise, Murray, & Gerfen, 1996). The greater the discrepancy between the predicted and the actual outcome, the more that is learned about the environment and/or the choice made (Schoenbaum, Roesch, Stalnaker, & Takahashi, 2009), so clearly, cool cognitive executive control of the decision-making process is influenced by hot affective orbital-ventral medial functions.

Even for dorsal system function, for which many measures have been developed, the problem is that we expect our tools to measure static *trait*-determined executive functions, and we do not take into account an important consideration of *state* in our measurement (Koziol et al., 2013). A child's neuropsychological state will vary from one assessment to the next, one day to the next, and one situation to the next, depending on biochemical, neuroanatomical, neurofunctional, task demands, and environmental determinants at any given time. In fact, the *consistent* clinical finding for children with executive dysfunction is *inconsistent* state-determined behavior and performance (Stuss, Murphy, Binns, & Alexander, 2003), suggesting that the quest for stable trait variance in our executive tools has led us down the garden path when it comes to neuropsychological assessment of child psychopathology and has therefore led to inconsistent results across disorders and measures (Weyandt et al., 2014).

Our examination here suggests much of this state variance may be largely due to orbital-ventral medial functions and emotional response to the tasks, examiner, and other situational characteristics. Because orbital-ventral medial state characteristics are situation-dependent and can evolve in a dynamic way over time, one of the biggest limitations of our neuropsychological measures is the inability to consider evolving patterns of performance over time, and this is why idiographic (as opposed to nomothetic) interpretation is so critical for neuropsychologists conducting evaluations of children with FSC dysfunction (Hale et al., 2013). Although continuous performance tests do look at changes in response patterns over time (see Riccio, Reynolds, & Lowe, 2001), few other measures objectively assess performance over time, so the astute clinician needs to be aware of how emotional states influence test behavior and performance throughout the evaluation. It is always important to remember all children like tasks they find enjoyable and at which they are successful, whereas difficult tasks may be less pleasant and reduce selective, sustained, and divided attention as a result—an effect that would certainly be exaggerated in children with psychopathology.

Unfortunately, despite the social importance of the orbital-ventral medial circuitry, most commercially available executive measures developed to date largely measure dorsal cognitive executive processes such as planning, organization, strategizing, monitoring, evaluation, flexibility, and shifting set (Weyandt et al., 2014). These are cool cognitive (e.g., dorsolateral-dorsal cingulate) executive function measures and not hot affective (e.g., orbital-ventral cingulate-limbic) executive function measures (e.g., Zelazo & Carlson, 2012). However, tasks that tap orbital-ventral medial function do exist, such as theory-of-mind tasks (e.g., Abu-Akel & Shamay-Tsoory, 2011; Amodio & Frith, 2006; Hynes, Baird, & Grafton, 2006) and gambling-reward incentive-type tasks (e.g., Turnbull, Bowman, Shanker, & Davies, 2014; Waters-Wood, Xiao, Denburg, Hernandez, & Bechara, 2012).

Theory-of-mind tasks are certainly advantageous in our understanding of emotional attention and self-regulation of behavior, because while social perception (e.g., processing of facial affect) is more a posterior (largely right temporal parietal function), the orbital-ventral medial areas appear to be critical in perspective taking or experiencing what others are feeling. This is because to have true theory of mind, one not only has to perceive what others are feeling, but actually feel those feelings to have true empathy. This is important to consider in comprehensive neuropsychological evaluation. One can accurately perceive emotion, but not care, which would be evident for some individuals with antisocial or conduct disorder. However, one could have too much theory of mind and too much empathy. At first glance, one might ask how having too much empathy might be a problem. The idea here, according to circuit balance theory, is that the hyperactive orbital circuit could lead to excessive empathy, which in turn would lead to poor boundaries and essentially an individual who becomes an emotional chameleon, adopting the emotional state of whomever they are with at any one time. Unfortunately, most theory-of-mind tests are reported in experimental studies or were developed for use in adult populations, so there is a considerable need to develop neuropsychological measures of self-regulation of affective attention, empathy, and behavioral control in children.

If considered within a neuropsychological assessment framework that assesses different FSC functions, with results over time, and situations from both a nomothetic and idiographic interpretive approach, we can gain a better picture of how both external and internal executive functions influence emotions and behavior in producing adaptive responses to the environment. When utilized in combination with multi-informant objective rating scales, clinicians and researchers alike can begin to tackle the important problems associated with FSC functions and dysfunction. As Insel (2013) notes, identifying the genetic determinants and neurobiological markers associated with behavioral symptoms will revolutionize psychological and psychiatric practice. Efforts to understand and differentiate relationships among the FSC and cortico-cerebellar influences on cognition, emotion, and behavior are critical to advance our understanding of how they influence adaptive/maladaptive behavior. In this way, we can begin to decipher the neurobiological, structural, and functional determinants of what was labeled "emotional intelligence" many years ago in psychology. With the clarity gained from such empirical efforts, we will begin to develop measures and strategies for more effective differential diagnosis of childhood neurodevelopmental and neuropsychiatric disorders, and we can then use this information to guide targeted intervention strategies that will lead to more efficacious treatment outcomes for affected children.

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