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Executive Function in Adolescence: A Commentary on Regulatory Control and Depression in Adolescents: Findings from Neuroimaging and Neuropsychological Research:

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Abstract

This commentary addresses the manner in which executive control processes and their development is impacted by major depressive episodes during adolescence. Strengths of the papers within this special section include the breadth of executive functions that were examined, incorporation of biological probes to understand neural mechanisms involved in observed impairments, the use of longitudinal paradigms to assess developmental timing, consideration and modeling of comorbid conditions, and the identification of individual difference factors that may serve as both liabilities and resilience factors. This work is timely; a close examination of negative emotions and how they change during adolescence is needed if we are to fully understand motivation-cognition interactions and how they are impaired by psychopathology.

As someone who has, for many years, researched the development of executive functions (EFs) and their neural underpinnings in children and adolescents from a clinical neuroscientific perspective, I am intrigued by the question of how such functions are impacted by depressive illnesses and how deficits in EF skills might lead to vulnerabilities to disorder, particularly during critical periods of the lifespan. This interest stems not only from an appreciation of the many facets of EF as they develop but also from an awareness of motivation-cognition interactions and how such interactions are behaviorally expressed between childhood and adulthood. This special section is timely in bringing such issues to the forefront of empirical inquiry, particularly given that publications within the expanding area of adolescent brain development have largely emphasized associations between executive dysfunction, reward-seeking, and externalizing forms of risk-taking (Gruber et al., 2012; Paulsen et al., 2014; Pharo et al., 2011). It is time to take a closer look at the impact of internalizing tendencies on the development and manifestation of EF skills in adolescence as well as how individual differences in EF skills impact the severity and course of internalizing disorders.

Deficits in executive function have been invoked to explain nearly every form of psychopathology; examples abound within the psychological, clinical, developmental, and neuroscientific literatures. At present, a Google Scholar search of “executive function and psychopathology” yields over 17,000 hits! This prominence is well-justified given that executive skills, at their highest levels, are, by many accounts (c.f., Barkley, 2001), what makes us human. The human capacity to self-organize behavior in the midst of an

unpredictable environment, to engage in what has been termed “mental time travel” (Suddendorf & Corballis, 2007), and to plan not only minutes and days but decades into the future set us apart from other mammals. We are able to adapt under novel conditions, to strategically evade sources of threat, and to leverage available resources to attain biologically necessary incentives in the context of a complex social structure, all of which rely on an intact executive system. Indeed, EFs operate in the service of nearly every situation that is motivational in nature, albeit to varying degrees of urgency. An individual's internal milieu also fluctuates, often unpredictably, through vacillations in arousal levels, hormonal secretions, and other biological states that have motivational significance (see Berridge & Arnsten, 2013; Gregory & Sadeh, 2012 for examples). Together, these changing external and internal environments must be navigated in a momentary manner in the course of daily life, and their demands aggregate to produce what we have termed an individual's *executive load* (Luciana & Collins, 2012).

My colleagues and I have suggested that successful behavioral, emotional, and self regulation in adolescence (and indeed, through the lifespan) is a function of the magnitude of that load (e.g., how much is there to deal with?) together with the integrity of brain-based mechanisms designed to manage it (one's available tools, including coping resources) (Luciana & Collins, 2012).

Successful Regulation Depends on the Magnitude of Executive Load

The demand or load magnitude may depend on subcortically-generated emotional states, stress levels, arousal, states of fatigue, and so on. This concept of “demand” is not necessarily negative as incentive-related strivings must also be managed by the executive system for goals to be adaptively attained (Luciana & Collins, 2012; Luciana et al., 2012). Adaptive control stems from the EF-based recruitment of cognitive resources, such as attention, inhibitory control, cognitive flexibility, and working memory. Dual systems models suggest that during adolescence, there is relatively immature top-down control in the context of more mature bottom-up emotional processing (Casey et al., 2008). In contrast, our group's view is that both top-down control and bottom-up emotional processing functions are changing during adolescence (Luciana et al., 2005; Urosevic et al., 2012) but with distinct trajectories (Luciana & Collins, 2012; Luciana et al., 2012) and in a manner that creates a uniquely salient executive burden during adolescence. While regulatory failures or inefficiencies can be due to deficiencies in mechanisms of control, executive *capacities* seem to be largely intact by mid-adolescence. Thus, it seems more likely that such inefficiencies can be attributed to the experience of a motivational/emotional load that is, simply put, too difficult to regulate despite possession of otherwise appropriately engaged resources. In other words, it may be that not *all* demands *can be* efficiently managed. Although it was not an explicit goal, the papers within this special section address important features of this model as well as challenges for further study.

High demand or Poor EF? The importance of context-dependent effects

Discerning when demand is inordinately high versus when resources are inadequate represents a challenge for empirical studies. So-called “hot” EF tasks (Gladwin & Figner,

2014; Metcalfe & Mischel, 1999) can be utilized to measure the integrity of EF when motivational demands are manipulated but potentially conflate the two. Yet, such tasks are ecologically valid. Outside of the laboratory, one's internal and external worlds are always exerting some level of demand. Accordingly, there may be relatively few real-world contexts in which EF is truly coldly cognitive.

As discussed within several papers in this issue, factor analytic studies (e.g., Miyake, 2000) suggest three major components within the overarching structure of EF: working memory/ updating, inhibition, and flexibility. When decontextualized, measures of these EF processes are extremely valuable because they directly assess basal deficits in control mechanisms. If one cannot exert appropriate control when motivational salience is low, then it stands to reason that such impairments would be even more pronounced when salience is high and the executive load increases.

Based on findings reported within this special section (i.e., Colich et al.), the question is raised as to how to validly measure activity within hot and cold systems in psychopathology. This dissociation may be especially difficult to empirically achieve in the context of affective disturbance given the perturbed state of the individual's internal milieu. Accordingly, it is critical to examine different measures of EF in depressed individuals to determine the full scope of impairment and the contexts within which such impairment manifests itself. A notable strength of the papers within this issue concerns the breadth of coverage of various EF domains; Morgan et al. and Colich et al. focus on putatively hot measures of EF through assessments of reward processing as well as inhibitory control in the context of emotional priming. Evans et al., Han et al., Vijayakumar et al., and Sommerfeldt et al. focus on laboratory measures of working memory, conflict monitoring, and attention. Together, the papers provide an opportunity to consider the conditions under which specific aspects of EF might be derailed in the context of adolescent depression.

During adolescence, executive loads are hypothesized to be higher than at other times in the lifespan due to surges in motivational strivings (Luciana & Collins, 2012). My lab has demonstrated that as information processing demands increase, the age of maturation to master a given task increases as well (Luciana et al., 2005) in support of the idea that it is one's capacity for "load-based multi-tasking" that matures during adolescence. Appreciation of the likely existence of a capacity limitation (see Callicott et al., 1999; Marois & Ivanoff, 2005) and particularly the impact of a limited capacity during development has profound implications for our understanding of how psychopathology is experienced and how it impacts high level regulatory processes.

The experience of psychopathology may compound the observed developmental peak in demand and may contribute to a scenario in which one's executive load is particularly great in magnitude. The papers within this issue are compelling in their support of this dynamic in the context of adolescent depression, which I will illustrate through a discussion of how depression impacts one's executive load, whether executive dysfunction is a cause or a consequence of affective disturbance, and implications for treatment.

Depression and Executive Load

The emotional and vegetative symptoms of depression create demands upon regulatory systems through low or otherwise reactive negative moods, diminished hedonic responses, fatigue, lack of sleep, poor appetite and associated effects on nutritional status, as well as impulses to self-harm. If motivation is low, one may need to self-organize behavior in a more conscious and effortful top-down manner than is otherwise the case. The somatic state of depression is an obvious barrier to overcome if goals are to be accomplished. This principle is elegantly illustrated in the paper by Colich et al., who show that inhibitory control suffers in depressed adolescents when they are primed by a negative emotional stimulus. The mood-congruent stimulus presumably amplifies an underlying state of sadness which must then be ignored, disengaged, or otherwise dampened for efficient levels of inhibitory control to be demonstrated. It could, in fact, be argued that the affective state of sadness is inordinately salient within the ongoing stream of working memory. Efforts to dampen a negative mood state use resources that detract from the capacity of the executive system to exert full control. The finding that depressed adolescents exhibited significantly less dorsolateral prefrontal activation when they were inhibiting a response following the presentation of a sad face than following the presentation of a happy face suggests that the appropriate frontally-based resources could not be leveraged. The similar decrease in activation observed in the occipital lobe suggests that perhaps early attentional systems were also disrupted by the contextual priming. One particularly elegant aspect of this paradigm is that it incorporates lab-based emotional cues to elicit the same elevations in executive load that presumably characterize depressed individuals' day-to-day lives. The impact of those elevations on behavior and on brain activity within circuits and regions important for EF, such as the dorsolateral prefrontal cortex (DLPFC), can then be directly observed. Future work might delineate the time course of these DLPFC-occipital interactions and their impacts on inhibitory control to determine whether it is top-down control or bottom-up attention regulation that is first impacted. *Importantly, this study demonstrates that impaired inhibition in depressed adolescents is context-dependent and not a general feature of the illness.*

In addition to impacts of mood state on one's executive load, deficits in processing speed and motor slowing might derail working memory systems (as alluded to in the overview provided by Evans et al.), leading to day-to-day perceptions of the world as increasingly stressful, if not overwhelming. As a result, the context within which a healthy person has to recruit EFs to exert control is fundamentally different than what is experienced by someone in the midst of a depressive episode. *Accordingly, multiple domains of EF are likely to show inefficiencies in the context of depressive illness, as nearly all studies within this special section illustrate. The modulation of working memory and associated updating functions seems to be particularly impacted.*

Are Executive Function Deficits a Cause or a Consequence of Depression?

The demonstration of context-dependent impairments in EF (Colich et al.) is important to the understanding of cause-effect associations. It might be the case that a relatively intact EF system is overwhelmed when perceived stress is high, when negative emotions are engaged,

or when processing speed slows down due to illness. On the other hand, internal and external demands may be more salient and depressive illness more likely precisely because affected individuals have a premorbid EF deficit. If communication between subcortical areas such as the striatum and nucleus accumbens that are involved in reward processing and regions of the frontal lobe that mediate valuation and decision-making processes is impaired due to aberrant patterns of functional connectivity (as suggested by Morgan et al.), then perhaps executive load subjectively increases because of a biologically-grounded communication failure. This type of hypothesis cannot be addressed in the absence of prospective longitudinal work. A major strength of the papers within this special section is that nearly all (Han et al., Morgan et al.; Evans et al.; Vijayakumar et al.) utilize longitudinal designs. The Morgan et al. paper is particularly impressive in this regard given that trajectories of illness were examined longitudinally across seven time points. Such studies are important, because while it has been reliably observed that EF deficits characterize those with depression (Snyder, 2013), it is not clear whether cognitive disturbances predate symptom onset.

Elucidation of this timeline is critically important for intervention efforts and for understanding resilience factors, as illustrated by Evans et al. Evans et al. demonstrate that poor working memory significantly predicts increases in depressive symptoms across a four-month retest interval, even after controlling for baseline depressive symptoms. Conversely, better working memory predicted the extent to which individuals engaged in primary control coping strategies. These strategies, in turn, are hypothesized to have changed the nature of the stressor (or the individual's emotional reactions to it) as well as secondary control coping strategies that regulated attention and cognitions about the stressor. Thus, the recruitment of such strategies apparently had the effect of *diminishing the demand on executive resources*, leading to more positive outcomes. Greater use of each of these coping approaches ultimately predicted lower levels of depressive symptoms. These findings suggest that individuals with more poorly developed executive skills (e.g., working memory) may be less able to launch appropriate coping strategies when stressful circumstances are encountered. And in those who are already depressed, better “trait-like” levels of executive function may represent a resilience factor that predicts more positive responses to coping-based psychological treatments. Vijayakumar et al. take this argument a step further in demonstrating that reactive control (the transient corrective processes that are implemented once conflict has occurred, similar to coping strategies) declines as early adolescents move from a psychopathology-free state into episodes of major depressive disorder.

Cause-effect associations cannot be discerned given that symptoms were not measured by Vijayakumar et al. during their longitudinal follow-up interval, but these findings stress the importance of reactive control as a proxy for coping mechanisms that may lessen the emotional impact of a situation before it disables other regulatory mechanisms. Since reactive control appears to develop early, younger adolescents who experience episodes of depression may be most likely to experience decrements in executive function at later ages. Since the recruitment of reactive control has been linked to the anterior cingulate cortex (Krug & Carter, 2012; De Pisapia & Braver, 2006), a region that is increasingly recognized as critical for affective regulation, one's capacity for adaptive self-regulation may be compromised in the long-term by major depressive episodes that occur at an early age due to neuroplastic changes. Similar to models of neuroplasticity that have been proposed for

substance abuse and its effects on the developing brain (Luciana et al., 2013), this patterning reinforces the importance of developmental timing and the potential for events that occur earlier in development to have far-reaching impacts into the future. Attempts to link symptom presentation, laboratory-based EF measurement, and neurobiology, as illustrated by Colich et al., Morgan et al., and Vijayakumar et al. not only permit relevant brain-based networks to be identified but also have the potential to yield novel insights regarding where anomalies will emerge first in a detectable fashion—within the brain or with respect to overt behavior.

Consideration of comorbid conditions and confounding variables

An additional strength of papers within this section concerns attempts to measure the influences of comorbid conditions and confounding variables. Han et al. demonstrate, for instance, that the presence of EF deficits, as measured by the Wisconsin Card Sort, in the context of a depressive episode predicts later anxiety. This finding is important in identifying a potential cascade of effects that may emerge over time, leading to more complex forms of illness. Sommerfeldt et al. provide intriguing evidence that patterns of EF deficit may vary in those who express impulsive tendencies in a more externalizing (e.g., substance abuse) versus internalizing (e.g., suicidal behavior) manner. Given the field's current focus on dimensional modeling of psychopathology (Cuthbert, 2005), these patterns are important to ascertain in the context of longitudinal research so that specificity of effects can be determined. There may be subtypes of depressive illness that are differentially associated with specific EF impairments.

Conclusions

To conclude, this special section represents a welcome addition to the literature given its emphasis on longitudinal modeling of executive function in relation to the occurrence of major depressive disorder in adolescence. We are challenged by the reported findings to consider whether EF deficits represent a problem with the executive system itself or whether apparent deficits can be attributed to an excessive demand on regulatory resources from other sources. Both change with adolescent development. In typical development, executive systems should be gradually improving in their ability to meet contextual demands. On the other hand, motivational strivings and responses are apparently heightened during adolescence, a pattern that has been most strongly reported with respect to incentive-based motivation (Luciana et al., 2012). This special section reminds us, in keeping with triadic models of adolescent development (Ernst, 2014) that negative emotions and how they change during “healthy” adolescence should also be scrutinized.

Despite these developmental trends, individual differences are also important predictors of outcome. While psychopathology researchers may have a tendency to focus on negatives, there are many reasons for encouragement within the findings reported here. A better developed EF system is a resilience factor, clearly associated with the recruitment of more adaptive coping strategies. The leveraging of individuals' cognitive strengths may represent an important component of personalized medicine to be more thoughtfully considered in the selection of treatment strategies.

Given these age-and individual difference-related patterns, the question is reasonably raised as to what should be the primary target of intervention. Should the goal be to use training techniques to increase EF skills independent of context? Or should therapeutic interventions be aimed at reducing the background level of executive load so that demands can be better aligned with available EF resources? Given the strong suggestion from papers in this section that EF deficits in depressed adolescents are context-dependent, strategies to reduce real and perceived stress, as suggested by Sommerfeldt et al., may be particularly efficacious in reducing the magnitude of one's executive load, resulting in improved levels of EF.

Current technologies now permit the neurobiological and behavioral correlates of successful interventions to be rigorously studied, representing an important next step in the understanding, treatment, and prevention of adolescent depression. The authors of the papers represented within this special section are likely to be leaders in this endeavor.

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