Developmental Cognitive Neuroscience 8 (2014) 98–99

Contents lists available at ScienceDirect

Developmental Cognitive Neuroscience

journal homepage: http://www.elsevier.com/locate/dcn

Commentary

# Response to Helfinstein & Casey

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We thank Sarah Helfinstein and B.J. Casey for raising some important issues in their commentary to our paper 'Exciting fear in adolescence: Does pubertal development alter threat processing?' (http://dx.doi.org/10.1016/j.dcn.2014.01.004, this issue).

In response, we welcome the opportunity to express agreement with a few of their key points, to respectfully disagree on one point, and (hopefully) to clarify some issues that we regard as potential misunderstandings.

First of all, we agree that the paradoxes of adolescent behavior represent a complex and fascinating set of issues that will require better integration of several conceptual and empirical approaches. We certainly did not mean to imply that the goal of our paper was an effort to explain all (or even most) of adolescent risk taking with a simple model, but rather to address one developmental component contributing to this complex picture, focusing on a hypothesis that may provide insight into one (affective) dimension of developmental changes in association with pubertal maturation. More specifically, the goal of our paper was *not* to suggest that [in their words]: "adolescence [is] one big roller coaster ride of thrills in the face of potential danger…"; moreover, we do *not* regard the central premise of our paper as being that "teens find threatening situations exciting."

More generally, we feel strongly that there is value in moving beyond general statements about "the teen brain" (and what we regard as on over-reliance on results from cross-sectional studies that often span several of the 'teen' years) and instead focus on studies designed to understand specific developmental processes. Accordingly, our study used a longitudinal design focused on puberty (including youth ages 11 and 12 at time 1) and focused on the role of a specific pubertal hormone—not because we believe these factors are necessarily the most important aspects of risk-taking (or affective changes in adolescence in general), but rather because this approach allowed us to test specific developmental hypotheses.

We hypothesized that puberty is associated with a maturational shift toward a more complex (and more ambiguous) processing of threat cues—which may contribute to adolescent tendencies to explore and enjoy some types of risky experiences. We believe that the affective underpinnings of these changes (associated with a pubertal increase in sensation-seeking and risk-taking, and possibly an increased capacity to experience a 'mixed' state of fear/excitement) may play a role in some adolescent capacities to *learn* how to approach some frightening situations (particularly in some social contexts). However, this seems a far cry from implying that all (or most) 'teens' find all (or most) sources of threat to be thrilling.

A second important set of issues raised by Helfinstein and Casey, is the need to move beyond any simplistic one-to-one mappings of the ventral striatum and amygdala to positive and negative valence, respectively, and instead to recognize the importance of understanding the distinct computational roles of these structures in learning (Li et al., 2011). We agree wholeheartedly. There is a critical need to better understand how the ventral striatum and amygdala work together to influence adaptive action in response to risk/reward tradeoffs during adolescent development, including a focus on the role of maturational changes in prediction-error processing during adolescence (e.g., Cohen et al., 2010), and focusing on how these systems are involved in reward *learning* in adolescence.

Broadly speaking the main thrust of the commentary by Helfinstein and Casey is that the pattern of findings that we presented may be accounted for by theories other than the

DOIs of the original articles:http://dx.doi.org/10.1016/j.dcn.2014.02.007, http://dx.doi.org/10.1016/j.dcn.2014.01.004.

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http://dx.doi.org/10.1016/j.dcn.2014.02.008

one we offered. We agree. In fact we would never claim that any set of findings could be explained by only one theory. We welcome alternative models that may explain these findings and that may provide better heuristics for understanding these important paradoxes of adolescent development.

There is however, a second more specific implication at the center of Helfinstein and Casey's critique involving the particular brain regions that would best serve to test (i.e., attempt to falsify) our hypotheses. They suggest that amygdala and nucleus accumbens (NAc) are poor choices, because these two areas do not respond solely to threat and reward. Respectfully, we argue that the necessary criterion here is not "which brain region responds only to reward/threat," but rather "which brain region responds most reliably to reward/threat."

To take this a step further, to avoid their critique, we would have needed to choose a brain region that responds exclusively to reward (if such a region even exists). Finding activation in that region would allow us to say that the data are consistent with our theory. However, a failure to find activation would be relatively uninformative, unless we also knew that the reliability of reward-related activation in this region was also high. We chose to examine NAc, which is known to respond reliably to reward (Bartra et al., 2013). Finding activation in NAc, we can say only that the data are consistent with our theory. However, a failure to find activation in NAc would have been relatively informative, because the NAc response to reward is relatively reliable. We formed our hypotheses based on this line of thinking (and the very large number of studies consistent with this pattern of reliable, but not necessarily exclusive responses).

There is, however, a larger set of issue here, which we believe cross back to an area of agreement with Helfinstein and Casey. We found greater activation in *both* amygdala and NAc in response to threat faces in those youth who had the largest rise in testosterone (using a within-subject longitudinal design focusing specifically on pubertal maturation). We believe this is consistent with our interpretation of a more complex processing of threat. Yet we also agree that this raises several unanswered questions as to *how* this combination of both amygdala and ventral striatal responses to threat signals (working together to perform computational processes regarding risk *and* reward) may contribute to understanding why this same maturational interval (and perhaps this same hormonal mechanism) appears to be associated with increased sensation-seeking and some tendency to 'want' or 'like' high-intensity 'excitement', and in at least some adolescents, a tendency for thrill-seeking and risk-taking.

More generally, I think we all agree that this is a very complex scientific frontier with important clinical and public health implications, which deserves a great deal of additional study, along with healthy debates and discussions to move the field forward—conceptually and empirically. We appreciate the opportunity to try to clarify our perspective and look forward to learning more from other perspectives and approaches. Clearly, this is a relatively early period in the fields of developmental cognitive, affective and social neuroscience, but also a time of rapid expansion in ways that are beginning to provide exciting (dare we say, 'thrilling'?) advances to understanding these important issues.

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