THE JOURNAL OF CHILD PSYCHOLOGY AND PSYCHIATRY
ACAMH THE ASSOCIATION FOR CHILD AND ADOLESCENT MENTAL HEALTH
Journal of Child Psychology and Psychiatry 55:7 (2014), pp 781–783 doi:10.1111/jcpp.12274

Commentary: Improving treatment for youth with callous-unemotional traits through the intersection of basic and applied science – reflections on Dadds et al. (2014)

Luke W. Hyde,[1] Rebecca Waller,[2] and S. Alexandra Burt[3]
[1]Department of Psychology, Center for Human Growth and Development, Survey Research Center at the Institute for Social Research, University of Michigan, Ann Arbor, MI, USA; [2]Department of Psychology, University of Michigan, Ann Arbor, MI, USA; [3]Department of Psychology, Michigan State University, East Lansing, MI, USA

There is increasing evidence that callous-unemotional (CU) traits delineate a distinctive group of youth with antisocial behavior (AB). While controversies surrounding the construct of CU traits remain, the study by Dadds' et al. (2014) in this issue is emblematic of recent research that has focused on understanding the development of CU traits among antisocial youth. We explore four pertinent issues in the study of youth AB highlighted by this study.

Translating from basic to clinical science – the importance of studying mechanisms

The current study has significant strengths, not least because it highlights the feasibility and importance of using basic etiological research to inform new treatments. Making the leap from 'bench to bedside' is a daunting task for most researchers, as the gap between basic and intervention research seems so wide. This study, particularly when viewed alongside other research from this and other investigative teams, shows us that not only is the distance between etiological and intervention research a passable one, but, that it can also be navigated if researchers focus their attention on the specific mechanisms underlying differences seen across various clinical groups and are precise and creative in their approach. In this case, prior studies conducted by Dadds, as well as coauthors of the current study, have examined genetic (Moul, Killcross, & Dadds, 2012), neural (Viding & McCrory, 2012), and behavioral (Dadds, El Masry, Wimalaweera, & Guastella, 2008; Dadds, Jambrak, Pasalich, Hawes, & Brennan, 2011) mechanisms underlying CU traits. These studies, as well as the current one, build on previous findings in the field that youth high on CU traits (1) have difficulty recognizing emotions, particularly fear, in others, (2) demonstrate blunted neural reactivity to these same emotions in others, and (3) evidence particularly severe trajectories of problem behavior and thus need more effective interventions (see Viding & McCrory, 2012).

Dadds et al. (2014) further advanced this work by targeting a specific aspect of these emotional deficits (i.e., eye gaze). The authors found that youth with CU traits attend less to the eye region of their mothers' faces compared with youth without CU traits, and this impaired eye contact was largely independent of maternal eye contact toward her child. These findings could help explain the blunted neural reactivity to facial expressions and deficits in fear recognition seen in children high on CU traits. As noted by the authors, youth with CU traits may miss out on important opportunities to learn about others by reduced eye contact. This failure to orient to important emotional stimuli may lead to a cascade across development, which interferes with the development of empathy contributing to more severe AB. These results are exciting, in that they provide a good test of the hypothesized model by employing a novel task. They also inform possible new treatment targets (i.e., eye contact and interpersonal interactions; Allen et al., 2012; Dadds, Cauchi, Wimalaweera, Hawes, & Brennan, 2012) with the express goal of making interventions more effective. In short, Dadds et al. (2014) serves as an impressive model for any of us studying basic or applied issues in youth psychiatric disorders by highlighting how thoughtful mechanistic research can inform innovative interventions.

Gene–environment correlations

Dadds et al. (2014) also found that father's fearlessness was correlated with child's eye contact deficits and that these deficits were linked to less positive maternal feelings toward the child. We would posit that these very interesting associations highlight the likely importance of gene–environment correlations (rGE) in the development of AB. In this case, the rGE could reflect both passive and/or evocative effects. Passive rGE refers to the fact that the environment that parents provide to their biological child (at least partially) reflects their own genetically influenced tendencies. In the current study, if reduced eye contact is in part a function of the tendency to be high on CU traits and fearlessness, and if CU traits have a genetic component, then biological parents

and children could share both the genes for CU traits/fearlessness and the corresponding tendency to make less eye contact with others. Additionally, evocative rGE would be present if genetically influenced characteristics of the child (i.e., CU traits) evoke responses from parents (i.e., less positive feelings), which go on to further exacerbate their genetic risk for CU traits. Interestingly, a recent meta-analysis showed that much of the heritable aspect of parenting in fact appears to be driven by child behavior via these kinds of evocative effects (Klahr & Burt, 2013). It is thus critically important for researchers examining the role of parenting in child outcomes to consider that the effects of parenting on child outcomes may reflect passive and evocative rGE, at least in part (Waller, Gardner, & Hyde, 2013).

To normalize their behavior, youth high on CU traits need to benefit more from interventions

Given the treatment implications of this work, a third point to consider is the relative effectiveness of treatments for youth who are high versus low on CU traits. Many empirical articles focusing on CU traits often start by noting the need for improved treatments because current treatments are *less effective* for youth with high CU traits. We agree that better treatments are needed for these youth as they represent those at most risk of becoming severe and chronic offenders. Critically, however, prior work has largely neglected the confounding issue of AB severity (see Waller et al., 2013). Consider that if youth with CU traits, who typically start with the highest levels of AB, change their behavior in equal amounts to other youth (i.e., no moderation), then they still end treatment as the most antisocial youth in the group. Put another way, available studies actually suggest that youth high on CU traits *benefit equally* from current behavioral treatments for AB (Waller et al., 2013), but their recovery to "normative" levels of AB is hampered by their poorer premorbid functioning. We thus believe that the commonly highlighted notion that treatments are less effective and/or the implication that CU traits are somehow not malleable through treatment should be reconsidered, both because they are not accurate, but also because they may do a disservice to these youth, their families, and treatment providers. Instead, we should focus our attentions on the 'moderation' of treatment by CU traits *in the opposite direction*: we need treatments that promote more change in those with CU traits to normalize their behavior. This revised conceptualization could help promote the development of targeted and personalized treatments that are more effective in the presence of high levels of CU traits, which could further enable us to move these youth from clinical to normative levels of AB.

The interpretation of 'child effects'

A final issue that this study raises is the direction of effects in the development of AB and CU traits. The current study provides evidence that there may be substantial 'child effects' in the development of these behaviors, consistent with models of substantial child effects in the development of broader youth AB. This point may be very important in treatment settings and public opinion in terms of avoiding 'blaming' the parents for CU youth behaviors. On the other hand, although not an issue raised by Dadds et al., we need to be careful that these and other results are not interpreted by a wider audience in a way that 'blames' the child. AB and CU traits are complex and multifaceted phenomena that are influenced by factors across many levels, from macrolevel (e.g., poverty, government policies) to microlevel variables (e.g., amygdala reactivity), with each contributing small and interactive variance. Additionally, although etiological research can help inform specific targets for intervention, finding a mechanism at one level does not necessarily imply that the intervention needs to also occur at that level. For example, Patterson's classic coercion model specifies the important role of *child* difficult temperament in the development of coercive cycles, but interventions are still effective in focusing on *parent* behavior in response to a more difficult child. Finally, as the current study is cross-sectional and covers a range of ages, longitudinal research is needed to examine how these effects may differ or cascade over time, as this will further inform our understanding of the extent to which these are true child effects.

Looking forward

The current article by Dadds and colleagues, particularly when read alongside other research by these authors and the broader CU traits field, demonstrates how careful basic research is being used to uncover specific mechanisms within a more homogenous subgroup of antisocial youth. In turn, these mechanisms are beginning to inform more personalized interventions for CU youth. While we have highlighted ongoing issues to be considered by the field, including the presence of often unmeasured rGE, controversy surrounding the effectiveness of treatments for youth with CU traits, and the interpretation of potential child effects, the current article represents a wonderful example of the type of research program that will ultimately help youth and families through translation from the lab to the clinic.

Acknowledgement

This commentary article was invited by the JCPP editors and has been subject to internal review. The

authors have declared that they have no competing or potential conflicts of interest in relation to this article.

Correspondence

S. Alexandra Burt, Department of Psychology, Michigan State University, 105A Psychology Building East Lansing, MI 48824, USA; Email: burts@msu.edu

References

[*References cited in Dadds et al. (2014)]

Allen, J.L., Slack, K., McGregor, K., Woolgar, M., Viding, E., Scott, S., & Dadds, M.R. (2012). *Personalising treatment for antisocial children with callous-unemotional (CU) traits*. Paper presented at the British Psychological Society Divisions of Clinical Psychology Annual Conference, London, UK.

Dadds, M.R., Allen, J.L., McGregor, K., Woolgar, M., Viding, E., & Scott, S. (2014). Callous-unemotional traits in children and mechanisms of impaired eye contact during expressions of love: A treatment target? *Journal of Child Psychology and Psychiatry*, *55*, 771–780.

*Dadds, M.R., Cauchi, A.J., Wimalaweera, S., Hawes, D.J., & Brennan, J. (2012). Outcomes, moderators, and mediators of empathic-emotion recognition training for complex conduct problems in childhood. *Psychiatry Research*, *199*, 201–207.

*Dadds, M.R., El Masry, Y., Wimalaweera, S., & Guastella, A.J. (2008). Reduced eye gaze explains "fear blindness" in childhood psychopathic traits. *Journal of the American Academy of Child and Adolescent Psychiatry*, *47*, 455–463.

*Dadds, M.R., Jambrak, J., Pasalich, D., Hawes, D.J., & Brennan, J. (2011). Impaired attention to the eyes of attachment figures and the developmental origins of psychopathy. *Journal of Child Psychology and Psychiatry*, *52*, 238–245.

Klahr, A.M., & Burt, S.A. (2013). Elucidating the etiology of individual differences in parenting: A meta-analysis of behavioral genetic research. *Psychological Bulletin* Advance online publication. doi:10.1037/a0034205

*Moul, C., Killcross, S., & Dadds, M.R. (2012). A model of differential Amygdala activation in psychopathy. *Psychological Review*, *119*, 789–806.

Viding, E., & McCrory, E.J. (2012). Genetic and neurocognitive contributions to the development of psychopathy. *Development and Psychopathology*, *24*, 969–983.

*Waller, R., Gardner, F., & Hyde, L.W. (2013). What are the associations between parenting, callous-unemotional traits, and antisocial behavior in youth? A systematic review of evidence. *Clinical Psychology Review*, *33*, 593–608.

Accepted for publication: 3 December 2013