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Can positive parental reinforcement counter genetic risk for callous-unemotional traits?

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Conduct Problems (CP) are a common reason for a childhood referral to mental health and educational services and represent a substantial public health cost (1) Callous-unemotional (CU) behaviors, indicative of lack of empathy and remorse, characterize children who are at risk of developing persistent CP (2). Previous research indicates that CU behaviors are moderately to highly heritable (3) and that CP that co-occur with high levels of CU behaviors may be more heritable than CP that do not co-occur with these behaviors (4). In other words, children with CP and CU behaviors may be genetically vulnerable to developing persistent antisocial behavior. However, for complex traits genetic vulnerability does not equal destiny for developing a particular outcome, *there are no genes that directly code for CU behavior.* Genes code for proteins that influence characteristics such as neurocognitive vulnerabilities that may in turn increase risk for developing CU behaviors and CP. Although an individual's genome likely

limits a 'range for phenotypic expression' (so called 'reaction norm'), it does not pre-specify how an individual will turn out. The specific developmental trajectory of any individual is determined by a complex interplay between genetic propensities and other factors that constrain how those genetic propensities are expressed at different levels of analysis, and throughout different developmental stages. Genetic variants that are associated with CU behaviors (once such are reliably ascertained) are likely to confer advantages, as well as disadvantages, depending on the environmental context. The challenge for researchers and practitioners alike is to uncover the mechanisms via which individuals with different genetic and environmental vulnerabilities arrive at maladaptive or better-adjusted outcome. Parenting may represent one of these mechanisms.

Harsh and negative parenting has been associated with higher levels of CU behaviors, while a warm parental style has been associated with lower levels of CU behaviors in children (5). But it is not self-evident that such parenting correlates of CU behaviors reflect purely environmental causal influences of parenting on behavioral development. Parents with genetic risk factors for antisocial behavior are likely to display parenting behaviors in line with these risks (e.g. harsh parenting) and also pass these genetic risk factors, which are likely to influence CP and CU behaviors, to their offspring. This means that part of the association between less than optimal parenting strategies and CP/CU behaviors may represent a genetic confound (passive gene-environment correlation), which has been demonstrated for antisocial behavior (6). We also know that children with CP and CU behaviors are extremely challenging to

parent. They typically show diminished empathy for others, display less remorse, manipulate others, and do not seem to want to please adults or readily show affection to others. It is therefore likely that they evoke different parenting reactions from less challenging children and recent research suggests that this is the case (7)(evocative gene-environment correlation).

To date only two genetically informative longitudinal studies have investigated parenting and development of CU behaviors (8, 9). Results from the first of these studies, capitalizing on a monozygotic-twin differences design, suggest that the association between harsh and negative parenting and higher levels of CU behaviors in children may, at least in part, reflect genetic vulnerability within families (8). This could either reflect a shared genetic vulnerability for poor parenting and CU behaviors, or an effect of CU behaviors in evoking negative/harsh parenting.

Complementing and extending this work, a highly informative and exciting adoption study by Hyde and colleagues, published in this issue, demonstrates both the impact of biological risk for CP and CU behaviors, as well as a clear indication that protective environmental factors are able to moderate the expression of that risk (9). A total of 561 adopted children and their adoptive and biological families from the Early Growth and Development Study were assessed longitudinally. Adopted children were assessed on CU, oppositional and attention-deficit behaviors at age 27 months. Severe antisocial behavior was assessed in biological mothers as an index of biological risk, which may reflect totally or in part genetic risk. Adoptive mothers' positive reinforcement was

assessed when the child was 18 months old. Main findings from longitudinal structural equation modelling showed that: (i) biological mother's severe antisocial behavior predicted CU behaviors in their adopted away child (β=0.16, $p < 0.01$) but not attention deficit and oppositional behaviors; (ii) positive reinforcement by the adoptive mother exerted a protective influence on CU and oppositional behaviors (β=-0.19, $p < 0.01$ and β=-0.15, $p < 0.01$); (iii) biological mother's severe antisocial behavior did not predict CU behaviors if the adoptive mother engaged in high degree of positive reinforcement towards the child (β=0.01, $p > 0.90$). These findings are extremely encouraging, as the biological risk for early CU behaviors appeared to be completely buffered by adoptive mother's positive reinforcement.

Despite the excitement that these findings should naturally generate, key challenges regarding their potential for translation should be addressed. First, the long-term protective benefits of positive reinforcement on the development of CU in vulnerable children needs confirmation. Recent studies have highlighted the importance of genetic effects on long-term developmental trajectories of CP, whereas environmental influences tend to be short term (10). Therefore, the observed protective benefit of environmental influences, including positive reinforcement in toddlerhood, may not be maintained throughout childhood and other developmentally specific genetic and environmental factors may take over. These include genetic factors pertaining to the maturation of those brain areas involved in planning, impulse control and complex social interactions, as well as developmentally specific environmental risk factors such as peer relationships and neighbourhood factors.

Furthermore, Hyde and colleagues rightly point out that it is important to bear in mind that parents in adoptive families are typically very motivated to undertake the challenges of parenting and are also often well-resourced. By contrast, in biological families, parents of children with CU behaviors are likely to have a host of genetic and contextual risk factors, which can pose challenges for promoting interventions that seek to increase positive reinforcement behaviors toward the child – particularly if that child is challenging. Therefore, the efficacy of such interventions in biological families, as well as the size and the duration of any beneficial impact on CU and CP still need to be established.

In sum, Hyde et al. (9), have made an important contribution to our understanding of how biological and environmental risk interact in shaping the early development of CU behaviors. Follow-ups in the Early Growth and Development Study and other genetically informative studies will hopefully shed further light on the long-term significance of these findings and bring us closer to a causal understanding of risk and protective pathways to CU and CP behaviors across different development periods.

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