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Understanding emotion processing in offspring of mothers with depression – A commentary on Burkhouse and Kujawa (2023)

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Understanding the mechanisms underlying the intergenerational transmission of depression is fundamental if we are to develop more effective preventive interventions for the offspring of parents with depression. According to the most prominent model of familial transmission of depression (Goodman & Gotlib, 1999), parental depression confers a risk for child psychopathology via a combination of biological (e.g. genetic predispositions, prenatal stress) and environmental (e.g. parenting, childhood adversity) factors. Importantly, it is proposed that rather than having a direct effect on child psychopathology, these mechanisms cause psychobiological vulnerabilities in the child, which make children more susceptible to the effects of negative life events (Goodman & Gotlib, 1999). The review by Burkhouse and Kujawa (2023) addresses one of the most prominent psychobiological vulnerability factors for youth depression: maladaptive emotion processing (Goodman & Gotlib, 1999). Emotion processing encompasses both the initial emotion 'reactivity' to an event (e.g. the emotional pain of being rejected) as well as implicit and explicit 'regulation' of this emotion (e.g. attempts not to take the rejection personally). Deficits in emotion processing have been shown to prospectively predict the onset of depression in adolescence (Gonçalves et al., 2019). Emotion processing is a plausible mechanism in the transgenerational transmission of depression because as Burkhouse and Kujawa (2023) describe, it is associated with both the biological and environmental transgenerational processes proposed by Goodman and Gotlib (1999).

A number of cross-sectional self-report, observational and behavioural studies show that impaired emotion processing in 'high-risk' (HR) children of parents with depression compared with 'low-risk' (LR) children of parents without mental illness. For example, HR children (compared with LR children) have greater difficulty identifying facial emotions (Joormann et al., 2010), tend to interpret emotionally ambiguous sentences more negatively (Sfärlea et al., 2019) and less frequently engage in adaptive emotion regulation strategies (Loechner et al., 2020). Interestingly, findings from a study of a family and group-based cognitive-behavioural (FGCB) intervention for HR children suggest that improvements in HR children's coping with stress mediated the positive effects of the intervention on depression risk (Compas et al., 2010). However, longitudinal and experimental studies are needed to establish the causal role of emotion processing in the familial transmission of depression. In addition, uncovering the intergenerational processes by which this takes place is important. A large body of evidence suggests that parenting has an important influence on emotion regulation during childhood and adolescence (see Morris et al., 2017 for a review). However, the extent to which parenting is responsible for emotion-processing deficits in HR children is relatively understudied.

Since patients with depression show deficits in emotion processing also on neurophysiological levels, the extent to which these differences act as precursors for depression in HR children is worth investigating (Foland-Ross et al., 2013). Burkhouse and Kujawa (2023) perform the first systematic review of physiological (e.g. heart rate) and neural (e.g. functional MRI) studies of emotion processing in HR children. In their comprehensive and novel review, Burkhouse and Kujawa (2023) incorporated findings from 64 studies using a wide range of physiological and neural measures. They found consistent evidence from EEG and resting state fMRI studies to show that HR children show alterations in both negative valence systems (e.g. heightened amygdala activation to threat) as well as positive valence systems (e.g. reduced left frontal EEG activation to reward). Resting state functional connectivity differences in regions associated with cognitive control and reduced reward processing were also observed in HR youth. Finally, in EEG studies, relatively consistent support was found for reduced left frontal activity, although this finding may be explained by generally reduced activity in HR youth. The latter findings were stronger for infants and young children than for adolescents, perhaps indicating that biological vulnerability decreases with age. Effect sizes in ERP studies were relatively small. Whereas the overall quality of the review itself was very good, the validity of the findings was largely

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limited by the quality of the included studies. This includes the small sample sizes in the MRI studies, the lack of control for multiple comparisons, and the use of dimensional measures of depression rather than diagnostic assessment.

In addition to providing a comprehensive summary of existing literature, Burkhouse and Kujawa (2023) make a number of excellent suggestions for future research. One important aspect is the need for genetically sensitive designs (e.g. adoption studies, epigenetic studies) to understand the relative contribution of genetic, prenatal, parenting and life events to emotion-processing deficits in HR youth. This will be hugely informative for the development of improved preventive interventions. Another important aspect of emotion processing in HR youth which needs to be better investigated is the specificity (e.g. parental depression vs. adverse childhood experiences) and sensitivity (e.g. internalising vs. externalising symptoms) of biomarkers of emotion processing. Similarly, examining the potentially moderating role of child-related (e.g. sex, age, and puberty) and parent-related (e.g. depression severity and timing and sources of support/treatment) factors on emotion processing will be important for determining the conditions under which emotion processing is most likely to be dysregulated in HR youth. The authors also advocate the use of large, shared datasets and multimethod approaches (e.g. combining EEG, fMRI and behavioural measures) to answer these more complicated questions. Finally, the authors emphasise the need to use more robust methods for investigating emotion processing such as (a) preregistering studies, (b) increasing sample sizes to prevent overestimates of true effect sizes and (c) using measures with psychometrically sound properties. From my perspective, it would also be important that researchers consider child psychopathology as a potentially confounding variable in analyses of emotion-processing vulnerabilities. As Burkhouse and Kujawa (2023) note, a number of biopsychological findings were only apparent in HR youth who had elevated symptoms of psychopathology. Since it is possible that these neural markers are the product of child psychopathology rather than representing a vulnerability factor of it, this should be considered in statistical modelling of data.

Despite the novel contributions the review makes to theoretical models of transgenerational transmission and future research in the field, the clinical implications of the findings might be more cautiously interpreted. Physiological and neural studies of emotion processing have seen increasing popularity, perhaps due in part to paradigm shifts such as the Research Domain Criteria (RDoC), which assumes that mental illnesses are fundamentally 'brain' disorders for which the biological mechanisms underlying them are yet to be fully understood (Insel & Cuthbert, 2015). However, there are several limitations to this reductionist approach to the study of emotion processing in depression. Although often described as a means to identify the 'substrates' or 'underlying mechanisms' of psychological phenomena, the extent to which neural mechanisms are causally related to emotion processing, rather than representing a correlate or consequence, is questionable (Eronen, 2021). Longitudinal and experimental designs are necessary for establishing the causality of psychobiological markers of emotion processing in the risk of depression in HR youth, yet just a handful of longitudinal studies exist (and experimentally manipulating neural markers of emotion processing is methodologically challenging). A second limitation is that even if researchers were able to identify the underlying neural correlates of emotion regulation (or the heritability of depression within families), it does not necessarily follow that this knowledge would make a significant contribution to improved interventions to prevent transgenerational transmission (Eronen, 2021) for at least two reasons. First, 'distal' measures of emotion processing (e.g. BOLD responses) tend to carry less predictive power than more 'proximal' measures (e.g. self-report) or risk-factors (e.g. parental depression) in determining the onset of depression. Second, many implications of neural studies can also be obtained using direct and indirect behavioural methods. The authors argue that one implication of their findings is that children with positive valence deficits may benefit from positive affect interventions, whereas children with reduced functional connectivity may benefit from cognitive control training. Whilst there is certainly a case for the development of personalised preventive interventions for HR youth, behavioural measures are also able to identify HR children with positive valence and cognitive control deficits, such that the unique contribution of biological studies of emotion processing may be modest. Currently, knowing the underlying biology is unlikely to impact prevention efforts, and it may be a while before knowledge of the underlying biology will provide value-added data for intervention, to the same degree that knowledge of specific DNA mutations informs personalised therapies for lung cancer, for example.

In summary, improved theoretical models of the intergenerational transmission of depression are necessary if improved preventive interventions are to be developed. Behavioural, observational, and self-report studies suggest that emotion processing represents a plausible means by which the risk of depression is conferred from parent to child, although it remains unknown whether emotion-processing deficits are transferred via biological versus environmental pathways. Burkhouse and Kujawa (2023) should be commended for their comprehensive review of physiological and neural studies of emotion processing in HR youth. The review provides an excellent summary of research findings to date and concrete suggestions for future research studies in

this field. It nevertheless also highlights the relative infancy of research in this field. Particularly given the cross-sectional nature of the majority of studies included, caution should be taken in drawing clinical implications from the findings. I look forward to what future experimental and longitudinal studies of emotion processing bring and an improved understanding of how to better prevent psychopathology in children of parents with depression.

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