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Feedback-related neurophysiology in children and their parents: Developmental differences, familial transmission, and relationship to error-monitoring

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ABSTRACT

The feedback negativity (FN) and reward positivity (RewP) are event-related brain potentials (ERPs) that follow the presentation of negative and positive feedback information, respectively, and have become the focus of recent research on psychopathology because of their associations with symptom severity of and risk for depression. We advanced our understanding of these feedback-related ERPs by examining developmental differences, familial transmission, and associations with error-monitoring ERPs. Parents and their children completed parallel, developmentally-tailored guessing and go/no-go tasks while feedback- and error-related ERPs were measured. We found that the Δ FN and RewP amplitudes increased with age and were larger in males than females among the child participants. The RewP also demonstrated familial transmission between fathers and their children. Finally, the FN and RewP were associated with error-related ERPs in children and adults, albeit in different ways. The current findings demonstrate that the FN and RewP have promise as developmentally-sensitive neural markers of reward and action monitoring processes associated with risk for psychopathology.

1. Introduction

Event-related brain potentials (ERPs) that follow feedback to an action have become a focus of recent research in psychopathology because of their associations with symptoms, risk and onset of depression across development (Bress et al., 2013; Bress et al., 2015a; Bress et al., 2012; Foti et al., 2014; Foti and Hajcak, 2009; Kujawa et al., 2014; Nelson et al., 2016). Two ERPs of special interest are the feedback negativity (FN)—a negative deflection elicited by negative feedback—and the reward positivity (RewP)—a positive deflection elicited by positive feedback—recorded at frontocentral sites around 250–350 ms (for a review see Proudfit, 2015). The FN is also referred to by other names in the literature, such as the feedback-related negativity and the feedback error-related negativity, however, for the purposes of this paper we will refer to this ERP component as the FN. Given the robustness of associations between feedback-related ERPs and depression symptoms and risk, researchers have suggested that these ERPs represent valuable biomarkers of disease and have proposed them as measures of Positive Valence Systems in the National Institute of Mental Health (NIMH) Research Domain Criteria (RDoC). Although several studies have demonstrated the utility of the FN and RewP as biomarkers of reward-related processes and depression, we aimed to advance the science in three key domains: 1) developmental differences, 2) familial transmission, and 3) associations with neurophysiological markers of conceptually related processes, specifically, error monitoring.

Identifying developmental differences in the modulation of the FN and RewP is important for determining at what ages these ERPs may be valid measures of reward processes that have relevance to depression. Additionally, establishing their familial transmission will help clarify if they can serve as vulnerability markers of disease within families. Finally, examining whether they relate to other neurophysiological markers of the broader construct of action monitoring will help us situate them in a larger nomological network to build more robust, multi-measure indicators of risk and symptom severity (Moser et al., 2015).

Developmental studies of the FN and RewP have thus far revealed mixed findings. Eppinger et al. (2009) used a reinforcement learning task and found a larger Δ FN (FN – RewP) in children (ages 10–12 years) than young adults (ages 19–24 years), which was driven by enlarged responses to negative feedback (i.e., FN) in the pre-adolescent children. In contrast, Hämmerer et al. (2011) – also using a reinforcement learning task – found that children (ages 9–11 years) and older adults (ages 65–75 years) evidenced a smaller Δ FN compared to

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adolescents (ages 13–14 years) and young adults (ages 20–30 years). However, two additional studies, one using a virtual maze task (Lukie et al., 2014) and the other a doors guessing task (Bress et al., 2015b), found no age effects (ages ranging from 8 to 23 years) for the Δ FN score. Moreover, although many studies demonstrate significant modulation of the FN and RewP (i.e., that the FN is more negative than the RewP) in older children and adolescents (Ethridge et al., 2017), some do not (Groen et al., 2007).

Studies in younger children are sparser but have also produced contradictory results (Belden et al., 2016; Mai et al., 2011). Mai et al. failed to find any modulation of the FN or the RewP to feedback in a prize box guessing game in preschoolers. Belden et al., however, reported a more negative FN than RewP using a doors guessing game in healthy, but not depressed, 4–7 year olds. One caveat of the Belden et al. study is that the Δ FN in the healthy children only emerged at a parietal site (Pz) rather than the prototypic fronto-central sites (e.g., FCz).

The developmental trajectory of the FN/RewP is therefore unclear. Task and age-group differences between the above-reviewed studies may contribute to the discrepancies. Some studies employed learning tasks whereas others utilized guessing tasks. Although it seems fairly clear that older adolescents and young adults show greater negativity in the FN compared to the RewP, it is less clear what to expect in younger samples. Moreover, most previous studies included relatively small samples within each age group and only two examined the FN and RewP in preschool-aged children. To address these limitations, we examined the FN and RewP in a relatively large sample ($N > 100$) of youth covering a broad age range from preschool to adolescence. We utilized the doors guessing task for its simplicity and because it consistently generates FN and RewP amplitudes that are related to depression (Moran et al., 2017).

In addition to tracking the development of FN and RewP, we examined their familial transmission as this has important implications for basic and translational science. Indeed, establishing familiality of biomarkers aids in the identification of risk markers of illness. To date, we are aware of only one study to look at the familiality of feedback-related ERPs. Weinberg et al. (2015) found that the RewP – and to a lesser extent the FN – were correlated within adult sibling pairs. To build on this work, we employed a family design in which we included parents and their children to establish a more robust indicator of familiality. Further, examining these associations in children and adolescents provides the added benefit of testing whether the FN and RewP might serve as *early* risk markers prior to the onset of psychopathology. If we can first establish that the amplitudes of the FN and RewP are transmitted within families, this sets up future studies to examine whether these familial components are involved in familial transmission of related psychopathology (e.g., depression).

Finally, the current study addressed whether the FN and RewP relate to other neurophysiological markers of the broader construct of action monitoring – i.e., detecting and adjusting actions in the service of optimizing goal-directed behavior – in children and adults. Specifically, we examined the associations between the FN and RewP and the error- and correct- related negativity (ERN and CRN, respectively). The ERN and CRN are fronto-centrally maximal negativities that occur within the first 100 milliseconds following erroneous and correct responses, respectively, in speeded response tasks (Simons, 2010). Although the FN is elicited by external feedback and the ERN is elicited by an internally-generated response, foundational reinforcement learning theories propose that the FN and ERN reflect activity of the same neural system dedicated to action monitoring (Holroyd and Coles, 2002). Such theories suggest that both components originate from the anterior cingulate cortex (ACC) following the impact of a phasic decrease in midbrain dopamine that tags actions as worse than expected (e.gs., suboptimal choices or response errors). However, a number of subsequent investigations found that the two were dissociable. For instance, studies show that the FN and ERN have separable source contributions (Potts et al., 2011). The RewP might be especially different from the ERN in source contribution, as studies show it has a primary source in striatum (Carlson et al., 2011). Individual differences studies are also mixed, as some argue that FN and ERN share common variance with psychopathology (Cavanagh and Shackman, 2015) whereas others show dissociable relationships in adults (Horan et al., 2012) and children (Bress et al., 2015a, 2015b). Amidst this confusion, studies generally do not report the direct associations between feedback- and error- related components and very few examine these relationships in children. We therefore aimed to address these limitations in the current study by directly examining the relationships between FN and RewP and ERN and CRN in both adults and children.

In sum, we aimed to advance the science on feedback-related neurophysiology by probing developmental changes, familial transmission and associations with other conceptually related neurophysiological measures. Toward this end, we measured the FN, the RewP, and error-related neurophysiology measures (ERN, CRN) in parents and their children using parallel, developmentally-tailored tasks. We also examined gender[1] differences in the FN and RewP, as prior work in children (Kujawa et al., 2014), adolescents (Crowley et al., 2009) and adults (Yi et al., 2012) has reported larger ΔFN in males than females. Moreover, the National Institutes of Health have recently called for studies to include analyses of gender differences as such differences have clear relevance across the health spectrum (Clayton and Collins, 2014). Indeed, given gender differences in neural mechanisms of reward and decision making and related psychopathology – including depression and substance use (for a review see Hammerslag and Gulley, 2015) – studying gender differences in the FN and RewP is clearly warranted. Studies showing a relationship between blunted FN/RewP and depression in females, in particular (e.g., Nelson et al., 2016), also point to the important role of gender. Based on the mixed findings, we made no specific predictions about developmental change in FN and RewP nor about their associations with the ERN and CRN. However, we felt more confident predicting that the FN/RewP would demonstrate familial transmission and be larger in males than females.

2. Method

2.1. Participants

Because our aims were to characterize developmental differences in and familial transmission of feedback-related neurophysiology, as well as associations between feedback- and error-related neurophysiology, data were analyzed at both the individual – child and parent – and family level.

At the individual level, participants included 145 children (78 female) ages 3.30 to 13.89 ($M = 8.30$, $SD = 2.64$) years old and their biological parents ($N = 130$, 76 females, $M_{age} = 34.81$ years, $SD_{age} = 6.08$ years)[2] primarily drawn from southcentral Michigan to participate in a larger study examining the familial transmission of neurobehavioral liabilities associated with risk for substance use disorders (SUDs). Families were screened for living in the greater Lansing community and were required to have at least one biological child between the ages of 3 and 13 years old. One hundred twenty-five (65 children and 60 parents) of the 275 total participants were recruited through the Michigan Longitudinal Study (MLS), a multi-decade study examining the intergenerational transmission of risk for SUDs and related psychopathology (Zucker et al., 1996; Zucker et al., 2000). An additional 150 participants (80 children and 70 parents) were recruited

[1] Because we did not, and most studies do not, confirm biological sex, and because of the difficulty in disentangling sex from gender in humans we use the term gender throughout the remainder of the paper.

[2] Age data were missing from 5 parents and thus the age data reported above are based on 125 parents.

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