International Journal of Psychophysiology 132 (2018) 338–352

Contents lists available at ScienceDirect

International Journal of Psychophysiology

journal homepage: www.elsevier.com/locate/ijpsycho

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

Jason S. Moser[a,*], Megan Fisher[a], Brian M. Hicks[b], Robert A. Zucker[b], C. Emily Durbin[a]

[a] Michigan State University, United States of America
[b] University of Michigan, United States of America

ARTICLE INFO

Keywords:
Feedback negativity
Reward positivity
Development
Family study
Error-related negativity

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

* Corresponding author at: Department of Psychology & Neuroscience Program, Michigan State University, East Lansing, MI 48824, United States of America.
E-mail address: jmoser@msu.edu (J.S. Moser).

https://doi.org/10.1016/j.ijpsycho.2018.08.015
Received 18 November 2017; Received in revised form 29 August 2018; Accepted 31 August 2018
Available online 02 September 2018
0167-8760/

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.

Table 1
Summary of child demographic variables.

Characteristic	MLS sample	Community sample	Overall sample
N	53	71	124
Age (years)			
Mean (SD)	8.02(2.38)	8.87(2.55)	8.51(2.50)
Median	7.73	8.83	8.17
Range	3.70–13.89	4.19–13.85	3.70–13.89
Gender			
Male	27 (51%)	28 (39%)	55 (44%)
Female	26 (49%)	43 (61%)	69 (56%)
Race			
Caucasian	46 (87%)	32 (45%)	78 (63%)
African-American	1 (2%)	2 (3%)	3 (2%)
Native American	0	1 (1%)	1 (1%)
Asian American	0	0	0
Bi-racial	0	4 (6%)	4 (3%)
Multi-racial	1 (2%)	3 (4%)	4 (3%)
Other	1 (2%)	0	1 (1%)
Missing	4 (7%)	29 (41%)	33 (27%)
Ethnicity			
Hispanic	3 (6%)	1 (1%)	4 (3%)
Non-Hispanic	46 (87%)	41 (58%)	87 (70%)
Missing	4 (7%)	29 (41%)	33 (27%)

Table 2
Summary of parent demographic variables.

Characteristic	MLS sample	Community sample	Overall sample
N	56	68	124
Age (years)			
Mean (SD)	31.76(3.85)	37.34(6.54)	34.87(6.16)
Median	32.09	37.25	34.08
Range	20.85–39.53	24.58–55.96	20.85–55.96
Gender			
Male	29 (52%)	25 (37%)	54 (44%)
Female	27 (48%)	43 (63%)	70 (56%)
Race			
Caucasian	46 (82%)	34 (50%)	80 (64%)
African-American	1 (2%)	4 (6%)	5 (4%)
Native American	0	1 (1%)	1 (1%)
Asian American	0	1 (1%)	1 (1%)
Bi-racial	0	0	0
Multi-racial	0	0	0
Other	1 (2%)	0	1 (1%)
Missing	8 (14%)	28 (42%)	36 (29%)
Ethnicity			
Hispanic	3 (5%)	2 (3%)	5 (4%)
Non-Hispanic	45 (81%)	38 (56%)	83 (67%)
Missing	8 (14%)	28 (41%)	36 (29%)

through web advertisements (i.e., Craigslist) in the greater Lansing metro area to supplement the MLS sample (hereafter referred to as the "Community Sample"). Together, our combined recruitment efforts provided an overall sample with a broad range of SUD risk. Twenty-one children and six parents were removed from ERP analyses (see details below). Thus, subsequent individual-level analyses included 124 children and 124 parents. Some values were missing (e.g., age) across child and parent measures and thus degrees of freedom vary across analyses reported below. Demographics for included children and parents are presented in Tables 1 and 2, respectively.

At the family level, a total of 83 families (37 MLS and 46 Community) with data available from at least one child and one parent were included in subsequent analyses. Information regarding the structure of families with available data, broken down by sample (MLS and Community), is presented in Table 3.

As previously mentioned, the samples were recruited to participate in a larger study examining the familial transmission of neurobehavioral liabilities associated with risk for SUDs. Although evaluating the role of SUD risk was not a primary aim of the current investigation, we, nonetheless, describe the known SUD risk to help contextualize the present findings. The MLS families were selected based on known SUD risk of the fathers of the parents in the present study – i.e., the grandfathers of the children in the present study. Of the 37 MLS families, 24 – comprising 34 children – included one parent whose father (grandfather of the children in the present sample) was identified at the time of original enrollment as having a preschooler (i.e., the parents in the present sample), cohabitating with the child's mother and having been arrested for drunk driving. Seven families – comprising 11 children – had one parent who resided with their parents in the same neighborhoods as those with fathers with drunk driving charges but neither parent reported a lifetime alcohol use disorder (AUD). Finally, one family – comprising one child – had one parent from these same neighborhoods with a father who met criteria for AUD but had not been charged with drunk driving. None of the Community sample families were selected based on SUD risk per se.

Table 3
Summary of family characteristics.

Variable	MLS sample	Community sample	Overall sample
Families with 1 child	22 (46%)	26 (54%)	48
Families with 2 children	14 (48%)	15 (52%)	29
Families with 3 children	1 (17%)	5 (83%)	6
Children with maternal ERPs only	12 (28%)	31 (72%)	43
Children with paternal ERPs only	14 (88%)	2 (12%)	16
Children with both parent ERPs	27 (42%)	37 (58%)	64
Children with missing parent ERPs	0 (0%)	1 (100%)	1

Parents and children received $75 and $50 cash, respectively, for their participation. Parents and children over the age of eight years old were also given an additional $7.50 bonus for their performance in the feedback task (described in more detail below).

2.2. Child tasks

2.2.1. Feedback task

Children completed a guessing task that has been used in other samples of children to measure the FN and RewP (Belden et al., 2016; Bress et al., 2012). On each trial, child participants were presented with an image of two doors and were told that only one door contained a prize. Prizes varied based on child age. Children ages 3–7 earned or lost points on each trial; their total earned points at the end of the task could then be redeemed for prizes. Specifically, prior to the task, children ages 3–7 chose two small prizes (e.gs., erasers, finger puppets) and one large prize (e.gs., coloring book, jump rope) that they could win if they chose the correct door enough times and earned as many points as possible. This incentive structure was chosen for younger children because they do not possess a clear conception of money and its value, but, rather, respond quite positively to prizes (as in Belden et al., 2016 & Mai et al., 2011). Children ages 8–13, on the other hand, earned or lost money on each trial. All participants were told to earn as many points or as much money as possible. Participants were asked to select a door using one of two keyboard buttons: "C" to select the left door and "N" to select the right door. The image of the doors remained on the screen either until the participant made a selection or for a maximum of 4000 ms. Following stimulus offset, a fixation cross (+) was presented at a central location for 1000 ms, and feedback was displayed on the screen for 2000 ms. Depending on the child's age, participants were told they would gain 10 points/$0.50 or lose 5 points/$0.25 on each trial. A gain was indicated by a green "↑" arrow and a loss was indicated by a red "↓" arrow. Following offset of the feedback stimulus, one of the following messages was displayed: "Please wait for experimenter" (children ages 3–7) or "Please press the spacebar to begin the next

round" (children ages 8–13). For younger children, the experimenter pressed the left mouse button to begin the next round.

The practice block consisted of 10 trials (5 gain trials, 5 loss trials) and children were asked to repeat task instructions to ensure understanding of the task. Following administration of the practice block, children completed six blocks of 10 trials each. Feedback was provided at the end of each block regarding the amount of the prize they had earned to that point. The probability of winning on any given trial was 50% regardless of the child's choice and participants were not told that feedback was random or that they should use any specific strategy.

2.2.2. Go/no go task

Children completed a developmentally-appropriate Go/No Go task (Grammer et al., 2014) used in other samples of young children to measure the ERN (Schroder et al., 2017). Children were asked to help a zookeeper capture zoo animals that had escaped from their cages by pressing the spacebar quickly and accurately when they were presented with a target animal (go stimuli). Additionally, children were presented with three orangutans (no go stimuli) that were helping the zookeeper and did not need to be placed into their cages, and were asked to withhold pressing the spacebar upon orangutan presentation. On each trial, a centrally-located fixation cross (+) was presented (750 ms), followed by the zoo animal stimulus (750 ms), and the inter-trial interval was 500 ms.

Practice blocks consisted of 12 trials (9 go trials, 3 no go trials) and were repeated until the child demonstrated an understanding of the task. Following administration of the practice block, children completed eight blocks of 40 trials (30 go trials, 10 no go trials) with the task requiring about 20 min to complete. Feedback was presented at the end of each block to obtain a sufficient number of errors for analysis. Specifically, faster performance was encouraged if accuracy exceeded 90% and more accurate performance was encouraged if accuracy fell below 65%.

2.3. Parent tasks

2.3.1. Feedback task

Parents also completed a guessing task wherein they were presented with an image of two doors and instructed to select which door they thought hid the reward using the keyboard buttons "C" to select the left door and "N" to select the right door. The image of the doors remained on the screen either until the participant made a selection or for a maximum of 4000 ms. Following stimulus offset, a fixation cross (+) was presented at a central location for 1000 ms, and feedback was displayed on the screen for 2000 ms. Participants were told they would either gain $0.50 or lose $0.25 on each trial. A gain was indicated by a green "↑" arrow and a loss was indicated by a red "↓" arrow. Following offset of the feedback stimulus, a message saying "Please press the spacebar to begin the next round" was displayed until the participant advanced to the next trial.

The practice block consisted of 10 trials (5 gain trials, 5 loss trials) to ensure understanding of the task. Following administration of the practice block, participants completed six blocks of 10 trials each. Feedback was provided at the end of each block regarding the amount of money earned in the game to that point. The probability of winning was 50% regardless of the parents' choices and parents were not told that feedback was random or that they should use any specific strategy.

2.3.2. Go/no go task

Parents completed a Go/No Go task in which they were instructed to press the spacebar when they were presented with an upright triangle (go trial) and to withhold responses to tilted triangles (no go trial). Tilted triangles (10 degrees) were presented on 20% of trials. On each trial, a centrally-located fixation cross (+) was presented (950 ms), followed by a green triangle stimulus (200 ms), and the inter-trial interval varied between 600 and 1000 ms.

The practice block consisted of 20 trials (16 go trials, 4 no go trials). Following administration of the practice block, participants completed 8 blocks of 60 trials (48 go trials, 12 no go trials) that required approximately 15 min to complete. Feedback was presented at the end of each block to obtain a sufficient number of errors for analysis. Specifically, faster performance was encouraged if accuracy exceeded 90% and more accurate performance was encouraged if accuracy was below 65%.

2.4. Procedure

Child participants gave informed assent, and their parents gave informed consent for their participation in the study. Following the assent/consent procedures, participants were fitted with the EEG cap and EEG electrodes were applied. Child and parent participants completed a number of tasks as part of the larger parent project. They completed the go/no go task and a flanker task (not reported here), presented in a counterbalanced order, prior to the feedback task. This order of tasks was chosen to avoid carry-over effects of the incentive in the feedback task on the go/no go and flanker tasks. We reasoned that there would be less of an effect of the go/no go and flanker tasks (e.g., fatigue) on the feedback task because of the rewarding nature of the task.

2.5. Psychophysiological recording and data reduction

Continuous electroencephalographic activity was recorded using the ActiveTwo system (BioSemi, Amsterdam, The Netherlands) from 64 Ag-AgCL electrodes embedded in a stretch-lycra cap according to the 10/20 system. In addition, two electrodes were placed on the left and right mastoids. Electrooculogram activity generated by eye movements and blinks was recorded at FP1 and three additional electrodes placed inferior to the left pupil and on the left and right outer canthi. During data acquisition, the Common Mode Sense active electrode and Driven Right Leg passive electrode formed the ground. All signals were digitized at 1024 Hz using BioSemi's ActiView software.

Offline, EEG processing and analysis were performed using BrainVision Analyzer 2 (BrainProducts, Gilching, Germany). Scalp electrodes were re-referenced to the numeric mean of the mastoids and bandpass filtered with cutoffs of 0.1 and 30 Hz (12 db/oct roll-off). Ocular artifacts were corrected using the method developed by Gratton et al. (1983) A computer-based algorithm detected physiologic artifacts such that trials in which the following criteria were met were rejected: a voltage step exceeding 50 μV between contiguous sampling points, a voltage difference of > 200 μV within a trial, or a maximum voltage difference < 0.5 μV within a trial. To ensure the algorithm's effectiveness in detecting artifacts in children – who tend to have more artifactual data because of excessive movement – a trained technician manually checked a subset of child EEG data from the feedback and go/no go tasks. This procedure confirmed the effectiveness of the algorithm.

2.5.1. Child ERPs

Twenty-one children were removed from data analysis: 13 due to poor data quality on the feedback task, 2 because of too few trials for calculation of average waveforms, four due to task discontinuation, one due to a computer error that resulted in the loss of the data file and one who did not understand the task. Thus, subsequent analyses included 124 children (69 female) ages 3.70 to 13.89 years ($M = 8.51$, $SD = 2.50$). An additional seven children were removed from analyses involving error-related ERPs: four due to poor data quality on the Zoo task, one because of too few trials for calculation of average waveforms, one due to task discontinuation, and one due to a computer error that resulted in the loss of the data file.

Stimulus-locked ERPs were averaged separately for each type of feedback (reward or loss) and baselined relative to a − 200 ms to 0 ms pre-feedback stimulus time window. Following visual inspection of the

grand averaged waveforms, we defined the FN and RewP as the average amplitude in the 250 ms to 350 ms post-feedback stimulus time window. Response-locked ERPs were averaged separately for each response (correct or error) and baselined relative to a −400 ms to −200 ms pre-response time window. Following visual inspection of the grand averaged waveforms, the ERN and CRN were then scored as the average amplitude in the 0 ms to 100 ms post-response time window. Reward- and error- related ERPs were evaluated across five midline electrode locations (Fz, FCz, Cz, CPz, Pz).

2.5.2. Parent ERPs

Six parents were removed from data analysis: four due to poor data quality on the feedback task, one because of too few trials for calculation of average waveforms, and one due to a computer error that resulted in the loss of the data file. Thus, a total of 124 parents (70 female) ages 20.85 to 55.96 years old (M = 34.87, SD = 6.16)[3] were available for all subsequent analyses. An additional 10 parents were removed from analyses involving error-related ERPs: five due to poor data quality on the Go/No Go task and five because of too few trials for calculation of average waveforms.

Stimulus-locked ERPs were averaged separately for each type of feedback (reward or loss) and baselined relative to a −200 ms to 0 ms pre-feedback stimulus time window. Following visual inspection of the grand averaged waveforms, we defined the FN and RewP as the average amplitude in the 210 ms to 310 ms post-feedback stimulus time window. Response-locked ERPs were averaged separately for each response (correct or error) and baselined relative to a −400 ms to −200 ms pre-response time window. Following visual inspection of the grand averaged waveforms, the ERN and CRN were then scored as the average amplitude in the 0 ms to 100 ms post-response time window. Reward- and error-related ERPs were evaluated across five midline electrode locations (Fz, FCz, Cz, CPz, Pz).

2.6. Child and parent analyses

Repeated measures analyses of covariance (rANCOVAs) models included within-subjects factors Site (F, FCz, Cz, CPz, Pz) and Feedback (reward vs. loss)/Response (correct vs. error) and between-subjects factors Gender (Male vs. Female) and Sample Type (MLS vs. Community) with Age entered as a covariate. Gender was included in all models to account for documented gender differences in feedback-related neurophysiology and to answer the call from the NIH to treat Gender/Sex as a biological variable. Sample Type was included in analyses to account for potential differences between our two recruitment methods. Finally, Age was included in child analyses to test the developmental questions of interest in the current study.

2.7. Family transmission analyses

To account for the non-independence of data among family members (parents and children nested within families), we evaluated within-family associations between parent and child psychophysiology phenotypes using multi-level modeling. Child EEG variables were nested within families (including siblings within families), along with maternal and paternal variables. One hundred and seven children (from 70 families) were included in analyses of associations between mother and child feedback-related ERPs and 91 children (from 51 families) in analyses of associations between father and child feedback-related ERPs. Ninety-eight children (from 64 families) were included in analyses of associations between mother and child error-related ERPs and 75 children (from 50 families) in analyses of associations between father and child error-related ERPs. Data were analyzed using HLM 7.0, using full maximum likelihood estimation, with child variables as the dependent variable and maternal and paternal variables as the predictors. These models yield an estimate of the association (similar to coefficients in regression analyses) between individual differences in parent and child EEG phenotypes, accounting for nonindependence of data from siblings and from parents and offspring.

3. Results

3.1. Child results

3.1.1. FN and RewP

Child feedback-related grand average ERP waveforms are presented in Fig. 1. Descriptive statistics for child FN, RewP and the Δ FN at representative electrode sites FCz and Cz are presented in Table 4.

Most pertinent to the current investigation, the 5 Site × 2 Feedback × 2 Gender × 2 Sample Type × Age rANCOVA revealed a main effect of Feedback (F (1, 119) = 5.58, p = .02, η_p^2 = 0.05) and significant Feedback × Age (F (1, 119) = 7.86, p = .006, η_p^2 = 0.06) and Feedback × Gender (F (1, 119) = 4.16, p = .04, η_p^2 = 0.03) interactions. There were no other significant effects involving Feedback (Fs < 1.56, ps > .21).[4]

To probe the Feedback × Age interaction, we conducted follow-up correlational analyses collapsing across Gender, Sample Type, and Site. As age increased, the Δ FN increased (r (124) = −0.24, p = .006; see Fig. 2). This relationship was mostly driven by rewards, as increases in age were related to increases in the RewP (r (124) = 0.19, p = .03) but not FN (r (124) = −0.02, p = .87).[5]

To further understand the Feedback × Gender interaction, we tested the effect of Feedback in each gender using paired samples t-tests after collapsing across Age, Sample Type, and Site. For females, there was no effect of Feedback (t (68) = 0.70, p = .49, d = 0.08; M losses = 6.87, SD losses = 6.79; M rewards = 6.36, SD rewards = 7.42). For males, however, the effect of Feedback was significant and in the expected direction (t (54) = −2.22, p = .03, d = −0.30; M losses = 7.79, SD losses = 6.99; M rewards = 9.62, SD rewards = 6.84). Figs. 3 and 4 present feedback-related grand average waveforms for females and males, respectively. We also probed the Feedback × Gender interaction by testing the effect of Gender at each level of Feedback (reward vs. loss) using independent samples t-tests after collapsing across Age, Sample Type, and Site. For losses, the effect of Gender was not significant (t (122) = 0.74, p = .46, d = 0.13). For Rewards, however, the effect of Gender was significant (t (122) = 2.52, p = .01, d = 0.46) such that males evidenced larger RewPs than females. Finally, the Δ FN difference was larger in males (M = −1.82, SD = 6.10) than females (M = 0.52, SD = 6.13; t (122) = −2.12, p = .04, d = −0.38). Thus, across age, males showed significant modulation of the RewP/FN, whereas females did not, with the primary difference between genders being the larger response to rewards for males than females.

There were also significant Site × Age (F (4, 116) = 13.83, p < .001, η_p^2 = 0.10) and Site × Gender (F (4, 116) = 5.80, p = .006, η_p^2 = 0.05) interactions. Although not predicted effects, the Site × Gender interaction indicated greater overall positivity for males than females at centro-parietal, but not fronto-central, sites (F_{lin} (1, 119) = 6.95, p = .009, η_p^2 = 0.06) and the Site × Age interaction indicated greater overall positivity for older than younger children at centro-parietal, but not fronto-central, sites (F_{lin} (1, 119) = 15.29, p < .001, η_p^2 = 0.11). No other significant effects emerged (Fs < 2.90,

[3] Age data were missing from four parents and thus age data reported above are based on 120 parents.

[4] Although Feedback did not interact with the Site variable, effects reported collapsed across sites are consistent with those observed at representative electrode sites FCz and Cz.

[5] Accounting for non-independence of scores because of inclusion of siblings did not change any results.

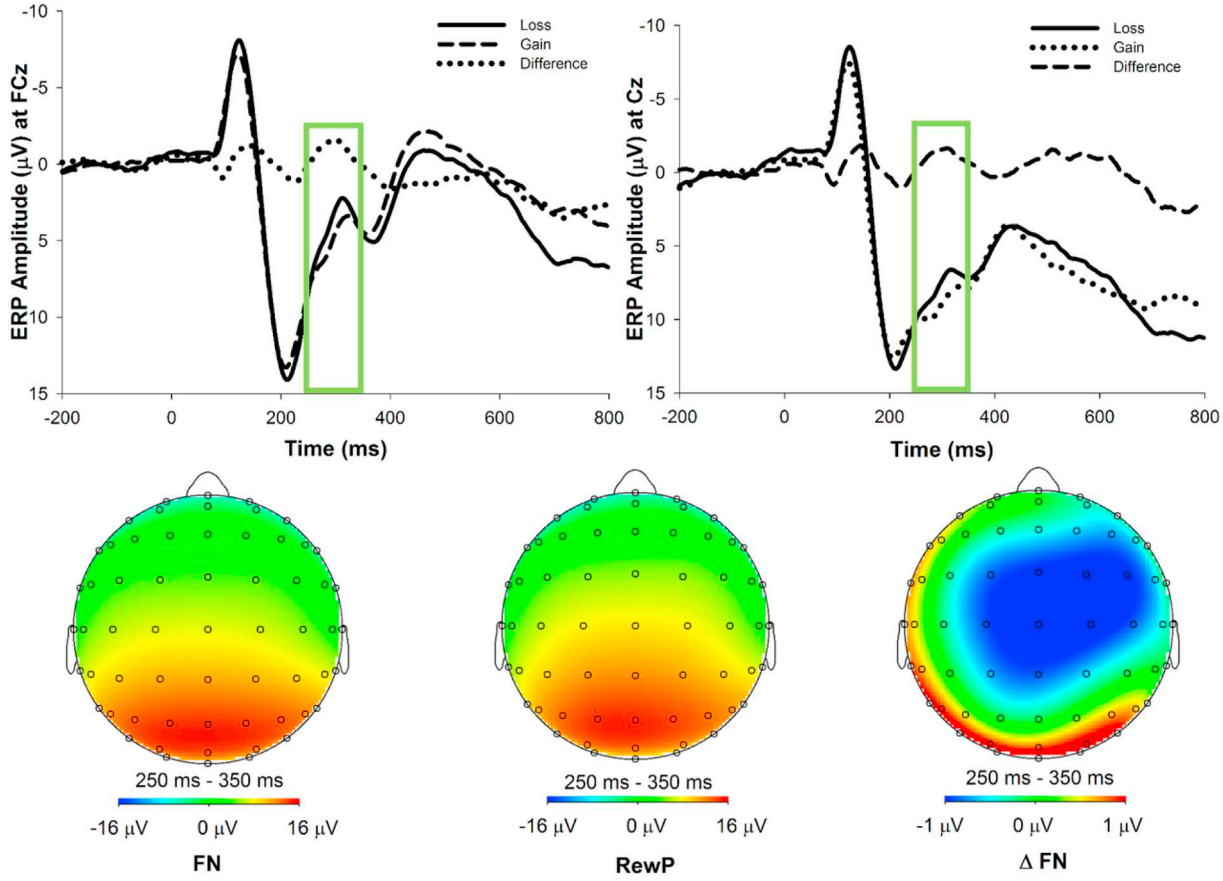

Fig. 1. Top left: Child feedback-locked, grand-average waveforms depicting loss, gain, and the loss-gain difference at electrode FCz. Time 0 is the stimulus onset. Top right: Child feedback-locked, grand-average waveforms depicting loss, gain, and the loss-gain difference at electrode Cz. Time 0 is the stimulus onset. Bottom: Child scalp topographies depicting voltages for FN, RewP, and the Δ FN, respectively, quantified as the average activity between 250 ms and 350 ms following stimulus onset.

Table 4
Summary of Child ERP Variables.

Variable	MLS sample		Community sample		Overall sample	
	M	SD	M	SD	M	SD
FCz FN (μV)	5.87	7.70	3.22	7.75	4.35	7.81
FCz RewP (μV)	6.65	8.76	3.88	7.58	5.06	8.18
FCz Δ FN (μV)	−0.78	7.39	−0.66	6.70	−0.71	6.97
Cz FN (μV)	8.87	8.26	7.18	8.18	7.90	8.22
Cz RewP (μV)	10.53	9.27	7.67	8.65	8.89	8.99
Cz Δ FN (μV)	−1.67	7.07	−0.49	7.50	−0.99	7.32
FCz ERN (μV)	−8.48	6.84	−9.53	7.62	−9.09	7.29
FCz CRN (μV)	0.46	4.26	0.57	3.97	0.52	4.08
FCz Δ ERN (μV)	−8.94	7.27	−10.10	6.24	−9.61	6.69
Cz ERN (μV)	−7.06	6.87	−6.44	7.78	−6.70	7.38
Cz CRN (μV)	2.73	4.59	3.35	4.35	3.09	4.45
Cz Δ ERN (μV)	−9.79	7.49	−9.79	6.83	−9.79	7.09

ps > .09).

Because we utilized two different incentive structures for younger (3–7 years old) versus older (8 years and older) children – so as to be developmentally sensitive to their respective cognitive functioning – we also examined feedback-related neurophysiology as a function of age group (younger vs. older) in a 5 Site × 2 Feedback × 2 Gender × 2 Sample Type × 2 Age Group rANOVA (see Figs. 5 & 6 for feedback-related grand average waveforms for younger and older children, respectively). This analysis did, indeed, reveal a significant Feedback × Age Group interaction (F (1, 116) = 6.61, p = .01, η_p^2 = 0.05). Follow-up paired samples t-tests probing the effect of Feedback in each

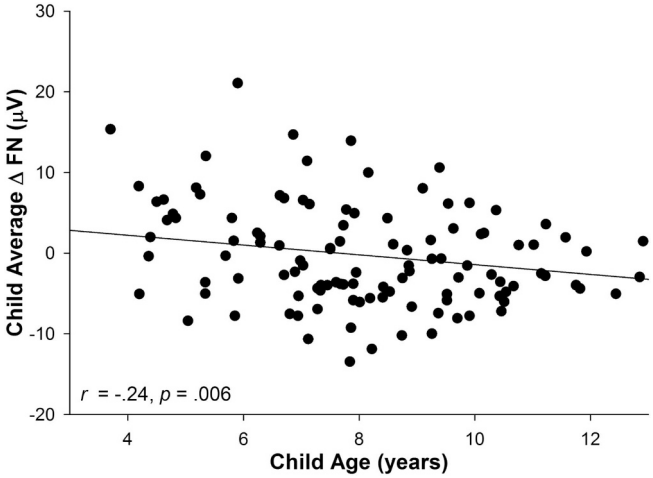

Fig. 2. Relationship between age and Δ FN in children. Increases in age were related to a larger Δ FN. FN is a negative-going ERP component and thus more negative values reflect a larger Δ FN.

Age Group collapsing across Site, Gender, and Sample Type revealed a significant effect of Feedback in the older children (t (63) = −2.93, p = .005, d = −0.37; M losses = 6.48, SD losses = 6.71; M rewards = 8.33, SD rewards = 6.76) but not in the younger children (t (59) = 0.99, p = .33, d = 0.13; M losses = 8.13, SD losses = 6.98; M rewards = 7.24, SD rewards = 7.90). Although the two Age Groups did not differ with respect to responses to rewards or losses independently

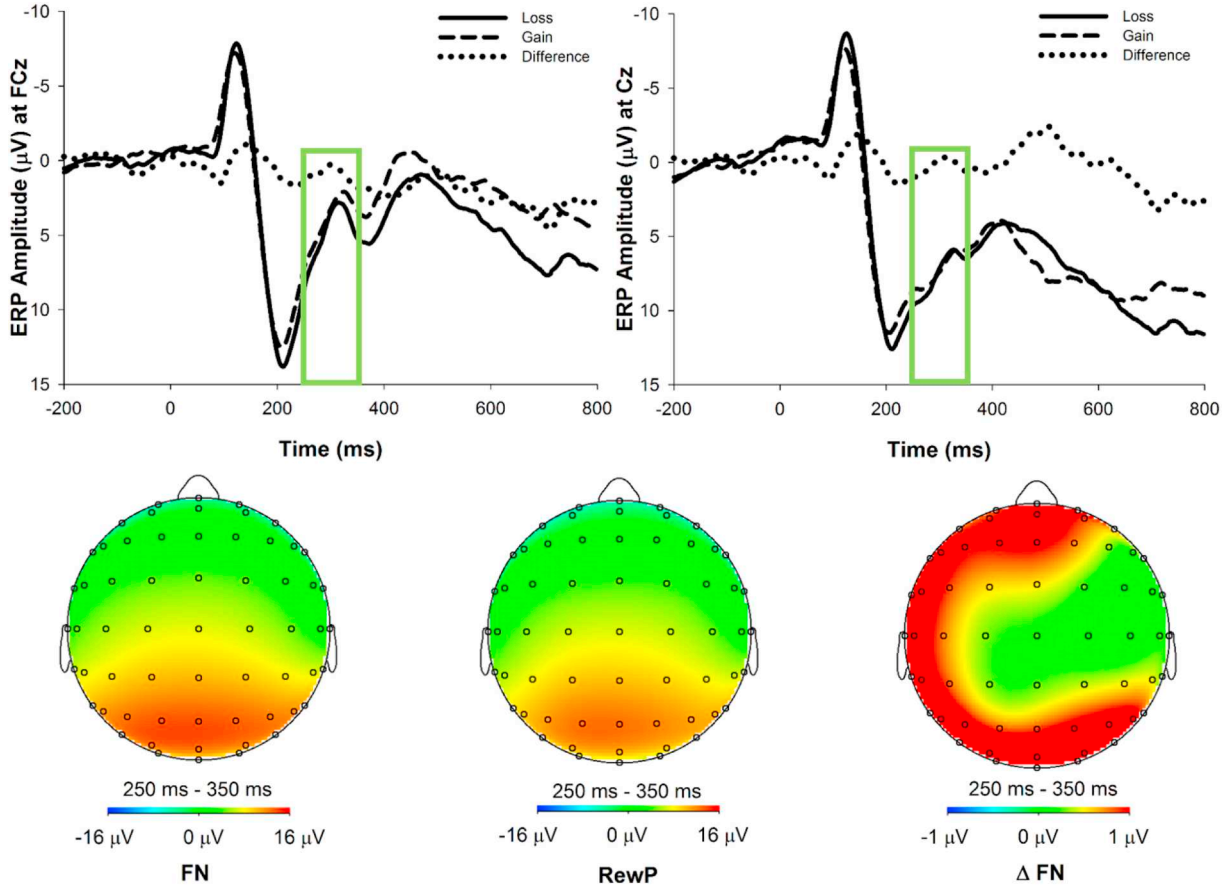

Fig. 3. Top left: Female feedback-locked, grand average waveforms depicting loss, gain, and the loss-gain difference at electrode FCz. Time 0 is the stimulus onset. Top right: Female feedback-locked, grand average waveforms depicting loss, gain, and the loss-gain difference at electrode Cz. Time 0 is the stimulus onset. Bottom: Female scalp topographies depicting voltages for FN, RewP, and the Δ FN, respectively, quantified as the average activity between 250 ms and 350 ms following stimulus onset.

(*ts* < |1.35|, *ps* > .18), the older group did show a larger Δ FN than the younger group (t (122) = −2.48, p = .02, d = −0.45; *M* older = −1.85, *SD* older = 5.04; *M* younger = 0.89, *SD* younger = 7.01). Interestingly, a follow-up 5 Site × 2 Feedback × 2 Gender × 2 Sample Type × Age rANCOVA in the younger group (N = 60) revealed a significant Feedback × Age interaction (F (1, 55) = 6.40, p = .01, η_p^2 = 0.10) such that, even in the younger group, increases in age related to larger Δ FN (r (60) = −0.31, p = .02) and RewPs (r (60) = 0.37, p = .003). This additional analysis suggests that the interaction between age and feedback observed in the full sample is not completely accounted for by the different incentive structures for younger versus older children, but rather, more possibly, by an age/developmental process.

3.1.2. ERN and CRN

Child error-related grand average ERP waveforms are presented in Fig. 7. Descriptive statistics for child ERN, CRN and the Δ ERN at representative electrode sites FCz and Cz are presented in Table 4.

As expected, the 5 Site × 2 Response × 2 Gender × 2 Sample Type × Age rANCOVA showed a main effect of Response (F (1, 112) = 13.98, p < .001, η_p^2 = 0.11), with more negative values for errors than corrects. Importantly, there was also a significant Site × Response interaction (F (4, 448) = 3.61, p = .04, η_p^2 = 0.03) and a significant three-way interaction between Site, Response and Age (F (4, 448) = 7.45, p = .001, η_p^2 = 0.06). The Site × Response interaction demonstrated a typical quadratic effect in follow-up within-subjects contrasts ($F_{quad,lin}$ (1, 112) = 11.94, p = .001, η_p^2 = 0.10) such that the difference between ERN and CRN was largest at fronto-central sites, and, in particular, FCz. The Site × Response × Age interaction further showed a robust linear effect in follow-up within-subjects contrasts ($F_{lin,lin}$ (1, 112) = 10.02, p = .002, η_p^2 = 0.08) such that children's ERN and Δ ERN tended to be larger at fronto-central sites with increasing age. There were no other significant effects involving Response (*Fs* < 2.97, *ps* > .08, η_p^2 < 0.03).

There was, however, a significant interaction between Site and Age (F (4, 448) = 12.54, p < .001, η_p^2 = 0.10). Unexpectedly, there were also significant interactions between Site and Sample Type (F (4, 448) = 5.50, p = .008, η_p^2 = 0.05) and between Site, Sample Type and Gender (F (4, 448) = 3.26, p = .05, η_p^2 = 0.03). Because these effects did not involve the critical Response variable, and are thus beyond the scope of the current paper, we did not evaluate these effects further.

3.1.3. Associations among measures of feedback-related and error-related neurophysiology

Correlation analyses were also conducted to evaluate associations between feedback- and error-related components. Given that both feedback- and error-related components and modulations/differences tended to be largest at FCz and Cz – as in prior work – we focused our analyses on relationships between components at these two sites.

Across the full child sample, the Δ FN and Δ ERN were significantly positively correlated at the FCz site (r (117) = 0.24, p = .009; see Fig. 8). This relationship indicates that a larger Δ FN was related to a larger Δ ERN. Thus, greater loss-related activity was related to greater error-related activity. Interestingly, the relationship between the difference scores seemed to be driven mostly by the association between losses and errors at FCz (r (117) = 0.29, p = .002) and not rewards and

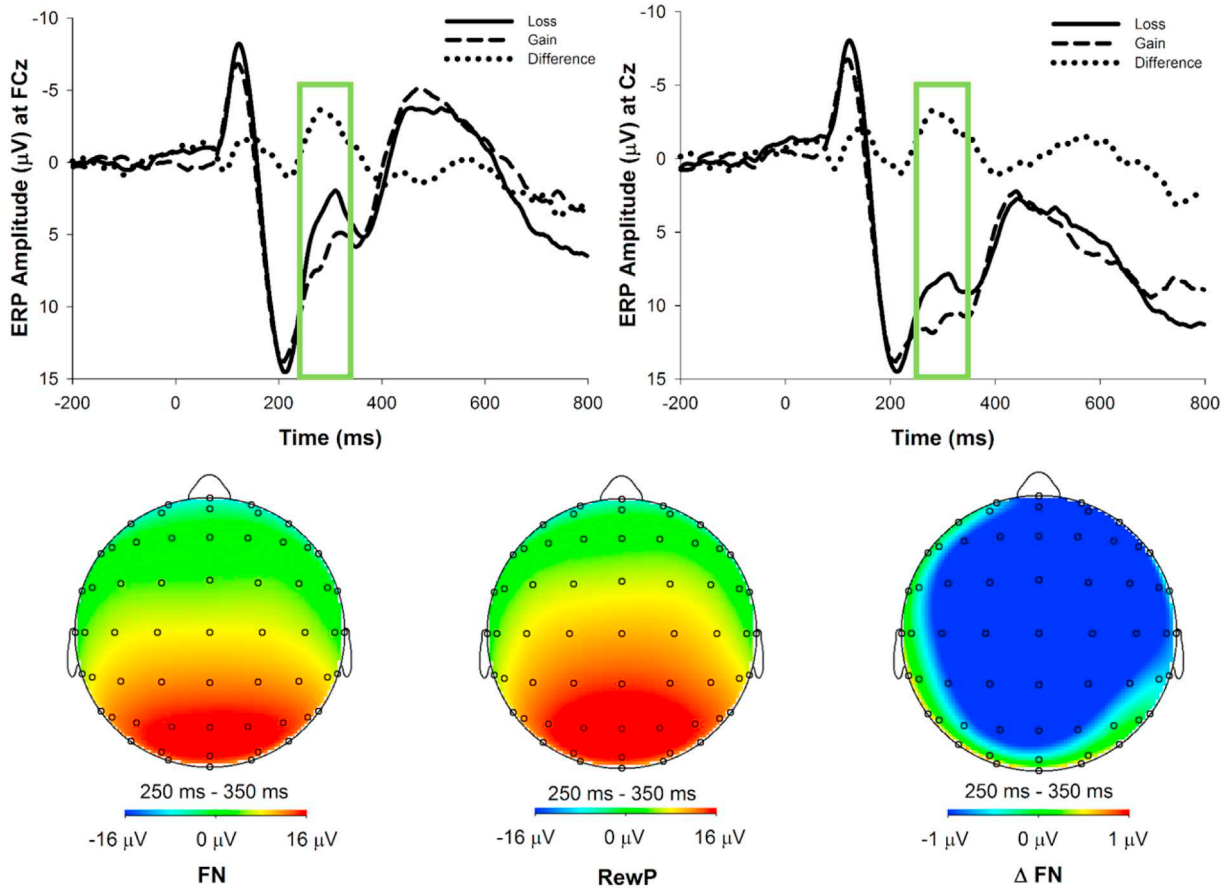

Fig. 4. Top left: Male feedback-locked, grand average waveforms depicting loss, gain, and the loss-gain difference at electrode FCz. Time 0 is the stimulus onset. Top right: Male feedback-locked, grand average waveforms depicting loss, gain, and the loss-gain difference at electrode Cz. Time 0 is the stimulus onset. Bottom: Male scalp topographies depicting voltages for FN, RewP, and the Δ FN, respectively, quantified as the average activity between 250 ms and 350 ms following stimulus onset.

corrects (r (117) = 0.04, p = .66). That is, larger FN was related to larger ERN but RewP and CRN were unrelated. The relationship between the two difference scores was smaller and non-significant at the Cz site, but still in the same direction (r (117) = 0.16, p = .08). There were, however, no significant associations between losses and errors or rewards and corrects at Cz (rs < 0.10, ps > .34).

Based on the findings above indicating age and gender effects on the RewP/FN, we also tested moderation of these relationships by Age and Gender using the PROCESS MACRO for SPSS (v3.0; Hayes, 2017). Age did not moderate the relationship between the Δ FN and Δ ERN scores at FCz or Cz (coefficients < 0.03, ps > .56). Age also did not moderate the relationship between losses and errors at FCz or Cz (coefficients < |0.07|, ps > .09). Nor did age moderate the relationship between rewards and corrects at FCz or Cz (coefficients < −0.03, ps > .75).

Gender did, however, moderate the relationship between the Δ FN and Δ ERN scores (coefficient = 0.41 (SE = 0.17), p = .02) at FCz such that males (coefficient = 0.42 (SE = 0.11), p < .001), but not females (coefficient = 0.01 (SE = 0.12), p = .92), demonstrated a strong positive relationship between the two (see Fig. 9). The same moderation by Gender held true at Cz (coefficient = 0.49 (SE = 0.18), p = .008) such that males (coefficient = 0.44 (SE = 0.14), p = .002), but not females (coefficient = −0.05 (SE = 0.12), p = .67), demonstrated a strong positive relationship between the two difference scores. Gender did not quite moderate the relationship between losses and errors at FCz (coefficient = 0.32 (SE = 0.19), p = .09). Gender clearly did not moderate the relationship between losses and errors at Cz (coefficient = 0.23 (SE = 0.21), p = .30) or between rewards and corrects at FCz or Cz (coefficients < 0.18, ps > .62).

Overall, results indicate an association between feedback- and error-related ERPs in children and adolescents. Moreover, this relationship seems to be driven primarily by an association between losses and errors that manifests most clearly in males.

3.2. Parent Results

3.2.1. FN and RewP

Parent feedback-related grand average ERPs are presented in Fig. 10. Descriptive statistics for parent FN, RewP and the Δ FN at representative electrode sites FCz and Cz are presented in Table 5.

Most pertinent to the current investigation, the 5 Site × 2 Feedback × 2 Gender × 2 Sample Type rANOVA confirmed the main effect of Feedback (F (1, 120) = 36.58, p < .001, η_p^2 = 0.23) with more positive values for the RewP than the FN. There was also a significant interaction between Feedback and Site (F (4, 480) = 4.02, p = .02, η_p^2 = 0.03). Follow-up within-subjects trend analysis indicated a significant quadratic effect ($F_{quad,lin}$ (1, 120) = 11.43, p = .001, η_p^2 = 0.09) such that the FN and RewP were most different from each other at fronto-central sites, namely FCz and Cz. There was also a main effect of Site (F (4, 480) = 36.58, p < .001, η_p^2 = 0.23), with greater overall positivity at parietal sites.

There was an unexpected interaction between Gender and Sample type (F (1, 120) = 8.21, p = .005, η_p^2 = 0.06) as well as an unexpected four-way interaction between Feedback, Site, Gender and Sample Type (F (4, 480) = 3.80, p = .03, η_p^2 = 0.03). Because the Gender × Sample Type interaction did not involve the critical Feedback variable and the

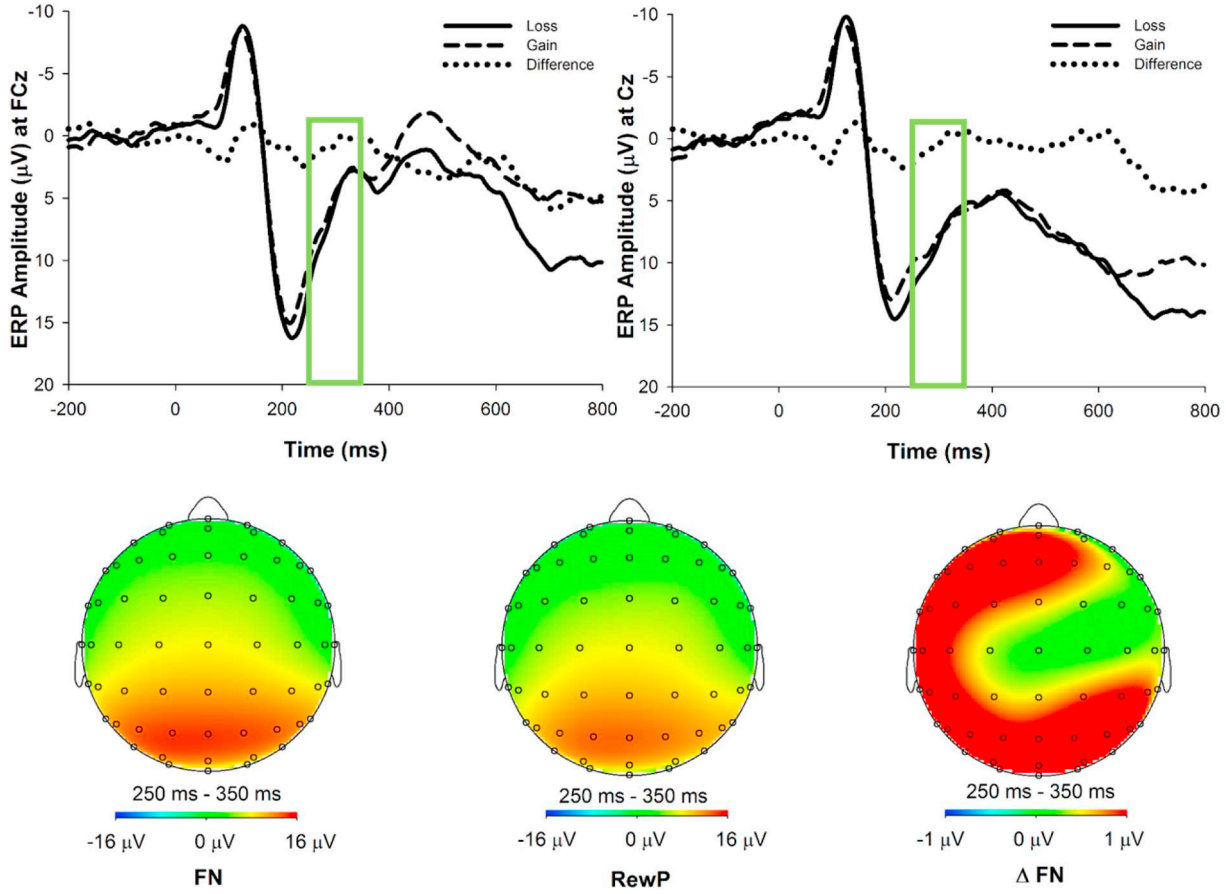

Fig. 5. Top left: Young child (ages 3–7) feedback-locked, grand average waveforms depicting loss, gain, and the loss-gain difference at electrode FCz. Time 0 is the stimulus onset. Top right: Young child (ages 3–7) feedback-locked, grand average waveforms depicting loss, gain, and the loss-gain difference at electrode Cz. Time 0 is the stimulus onset. Bottom: Young child (ages 3–7) scalp topographies depicting voltages for FN, RewP, and the Δ FN, respectively, quantified as the average activity between 250 ms and 350 ms following stimulus onset.

four-way interaction is difficult to interpret, we did not examine these interactions further. No other significant effects emerged (*Fs* < 1.73, *ps* > .18, η_p^2 < 0.02).

3.2.2. ERN and CRN

Parent error-related grand average ERPs are presented in Fig. 11. Descriptive statistics for parent ERN, CRN and the Δ ERN at representative electrode sites FCz and Cz are presented in Table 5.

The 5 Site × 2 Response × 2 Gender × 2 Sample Type rANOVA confirmed the main effect of Response (F (1, 109) = 42.42, p < .001, η_p^2 = 0.28), with larger negativity for errors compared to corrects. The expected interaction between Response and Site was also significant (F (4, 436) = 29.75, p < .001, η_p^2 = 0.21). Follow-up within-subjects trend analysis indicated significant linear (p = .04, η_p^2 = 0.04), quadratic (p < .001, η_p^2 = 0.51) and order 4 contrasts (p = .02, η_p^2 = 0.05), the largest of which was quadratic. The quadratic effect indicated, as expected, that errors and corrects were most different from each other at fronto-central sites, namely FCz and Cz. There was also a main effect of Site (F (4, 436) = 87.51, p < .001, η_p^2 = 0.45), with greater overall negativity at frontal sites.

Unexpectedly, there was also a significant Gender × Sample Type interaction (F (1, 109) = 6.16, p = .02, η_p^2 = 0.05) that was further qualified by a significant Response × Gender × Sample Type interaction (F (1, 109) = 7.20, p = .008, η_p^2 = 0.06). These effects were not expected and beyond the scope of the current investigation and are thus not examined further. No other significant effects emerged (*Fs* < 3.22, *ps* > .06, η_p^2 < 0.03).

3.2.3. Associations among measures of feedback-related and error-related neurophysiology

Correlation analyses were also conducted to evaluate relationships between feedback- and error- related components. Given that both feedback- and error-related components and modulations/differences tended to be largest at FCz and Cz – as in prior work – we focused our analyses on relationships between components at these two sites.

There was a marginal negative association between the Δ FN and Δ ERN scores at FCz (r (113) = −0.18, p = .05). This relationship indicates that a larger Δ FN was related to a smaller Δ ERN – the reverse of what was found in children and adolescents. However, examining the relations by feedback- and response- type separately revealed the expected positive relationship between FN and ERN (r (113) = 0.34, p < .001), but an unexpected positive relationship between RewP and ERN as well (r (113) = 0.38, p < .001; see Fig. 12). Although the RewP was not related to the CRN (r (113) = 0.13, p = .16), the FN was positively related to the CRN (r (113) = 0.22, p = .02).

At Cz, the Δ FN and Δ ERN scores were not related (r (113) = −0.09, p = .37). However, again, the FN and ERN were positively correlated (r (113) = 0.32, p < .001) as were the RewP and ERN (r (113) = 0.31, p = .001). Neither the FN nor the RewP were related to the CRN (*rs* < 0.18, *ps* > .06) at Cz. Using the PROCESS MACRO to test for Gender moderation of the above relationships revealed no significant effects (coefficients < 0.21, *ps* > .14).

Overall, results indicate that there is support for an association between feedback- and error-related ERPs in adults, however, the relationship is different from that in children and adolescents. For adults, correlational analyses indicate that increases in loss- and decreases in

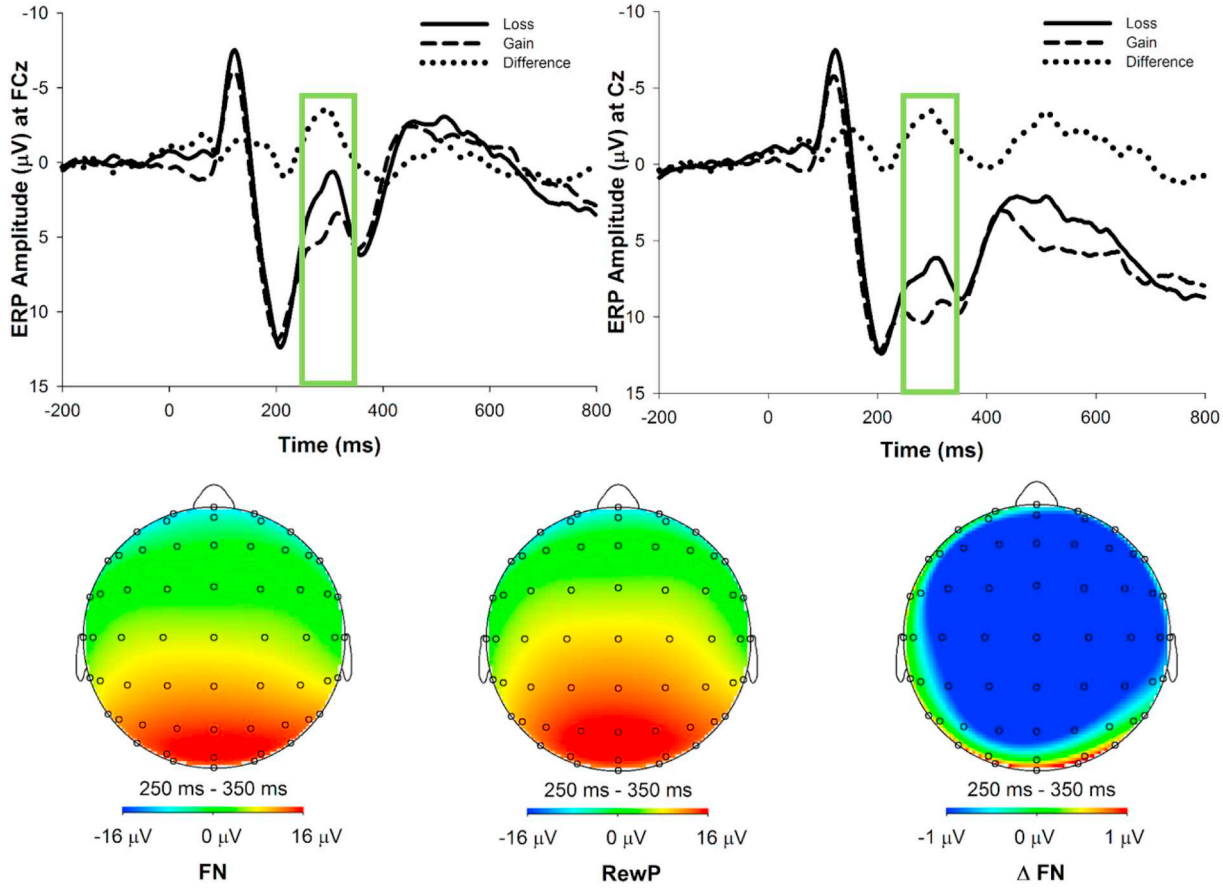

Fig. 6. Top left: Older child (ages 8–13) feedback-locked, grand average waveforms depicting loss, gain, and the loss-gain difference at electrode FCz. Time 0 is the stimulus onset. Top right: Older child (ages 8–13) feedback-locked, grand average waveforms depicting loss, gain, and the loss-gain difference at electrode Cz. Time 0 is the stimulus onset. Bottom: Older child (ages 8–13) scalp topographies depicting voltages for FN, RewP, and the Δ FN, respectively, quantified as the average activity between 250 ms and 350 ms following stimulus onset.

reward- related activity are related to increased error-related activity.

3.3. Familial transmission analyses

As with other correlations reported above, we focused familial transmission analyses on the FCz and Cz sites where feedback- and error- related ERPs tended to be largest. There was some evidence of familial transmission of feedback-related ERPs. The most straightforward relationship was observed between father and child RewPs at FCz (coefficient = 0.40 (SE = 0.18), $p = .03$) such that larger RewPs in fathers were related to larger RewPs in their children. The only other relationship that was significant was between mother and child Δ FN at Cz such that larger Δ FN in mothers was related to smaller Δ FN in their children (coefficient = −0.49 (SE = 0.16), $p = .003$). All other relationships between parent and child FNs, RewPs, and Δ FNs at FCz and Cz were nonsignificant ($ps > .14$).

There was also some evidence that error-related ERPs were familial. The strongest relationship was observed between mother and child ERNs at Cz (coefficient = 0.24 (SE = 0.12), $p = .05$) such that a larger ERN in mothers was related to a larger ERN in their children. Although attenuated and only marginally significant, this relationship between mother and child ERNs emerged at FCz as well (coefficient = 0.17 (SE = 0.10), $p = .11$). Mother and child CRNs also tended to be positively related at Cz (coefficient = 0.18 (SE = 0.11), $p = .09$). All other relationships between parent and child ERNs, CRNs, and Δ ERNs at FCz and Cz were nonsignificant ($ps > .30$).

4. Discussion

We advanced the research on feedback-related neurophysiology by probing developmental differences, familial transmission, and associations with error-related neurophysiology in children and their parents. With regard to development, the current findings suggest that the Δ FN increases with increasing age from preschool to adolescence. This relationship appears to be primarily driven by increases in the RewP across development. Parents also demonstrated larger mean-level amplitudes for Δ FN and RewP than their children (see Figs. 4 and 5), consistent with increases in these components across development. Familial transmission analyses further indicated that father and child RewPs were related and mother and child ERNs/CRNs were related. Results also indicated that feedback- and error- related neurophysiology were associated with each other in both children and parents but in different ways. Specifically, the relationship in children suggested a fairly clear positive relationship between loss- and error-related activity – i.e., between the FN and ERN. In adults, however, both loss- and reward- related activity were associated with error-related activity. Finally, analyses of gender differences confirmed a larger Δ FN in males than females in children that was primarily driven by larger RewPs in males. There were no clear gender differences in parents. We discuss each of these findings in turn in the context of existing theory and research.

First, results demonstrated that increasing age was related to a larger Δ FN, which was driven by increases in the RewP from 3 to 13 years of age. Modulation of the FN/RewP was also clearly present in parents and the Δ FN and RewP were numerically larger in parents than

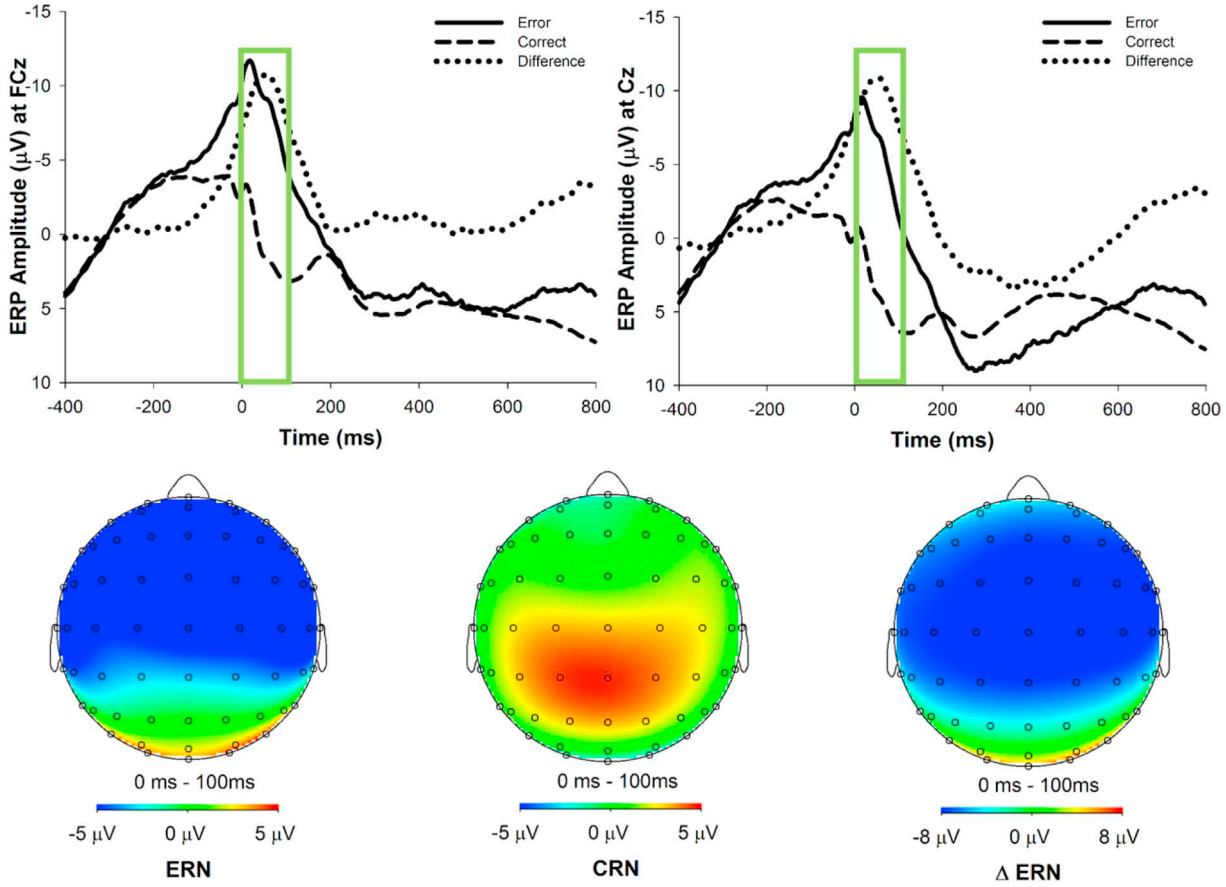

Fig. 7. Top left: Child response-locked, grand-average waveforms depicting error, correct, and error-correct difference at electrode FCz with a baseline correction of −400 ms to −200 ms pre-response time. Time 0 is the response onset. Top right: Child response-locked, grand average waveforms depicting error, correct, and error-correct difference at electrode Cz. Time 0 is the response onset. Bottom: Child scalp topographies depicting voltages for ERN, CRN, and the Δ ERN, respectively, quantified as the average activity between 0 ms and 100 ms following response onset.

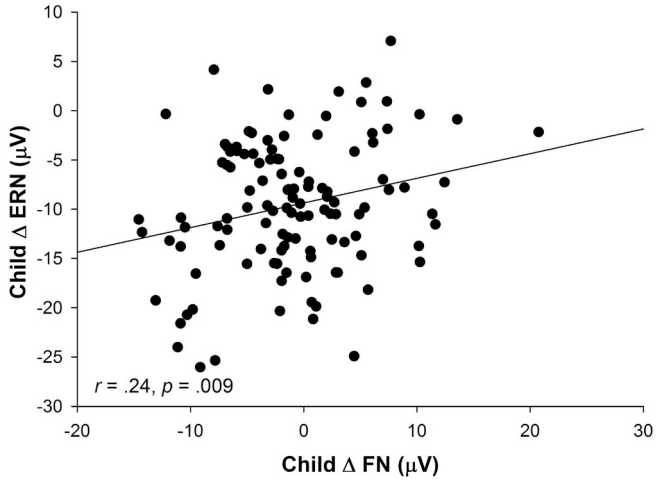

Fig. 8. Relationship between Δ FN and Δ ERN in children. Increases in loss-related activity were related to increases in error-related activity. FN and ERN are negative-going ERP components and thus more negative values reflect a larger Δ FN and Δ ERN.

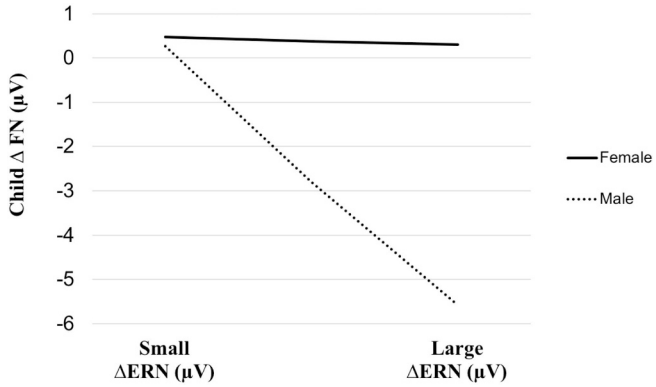

Fig. 9. Gender moderation of the relationship between Δ ERN and Δ FN. Females show no relationship between Δ ERN and Δ FN. However, males show a strong positive relationship between the two such that increases in Δ ERN were related to increases in Δ FN. FN is a negative-going ERP component and thus more negative values reflect a larger Δ FN.

children. Previous research in children has demonstrated inconsistencies regarding the differentiation between FN and RewP (Eppinger et al., 2009; Hämmerer et al., 2011; Lukie et al., 2014) and thus we were agnostic as to this effect in the current sample. Our RewP findings, however, are consistent with theory and research suggesting a protracted development of dopaminergic reward-related processes that become more mature as adolescence proceeds (Casey, 2015; Rubia, 2013; Wahlstrom et al., 2010).

What separates the current investigation from prior developmental work in this area is that we included a large sample ($N = 124$) of youth spanning preschoolers to adolescents. Indeed, our age range – i.e., 10 years – was double that of previous studies in youth (e.g., Bress et al., 2015a, 2015b). In particular, we were able to characterize the development of the FN and RewP starting in early childhood – most previous

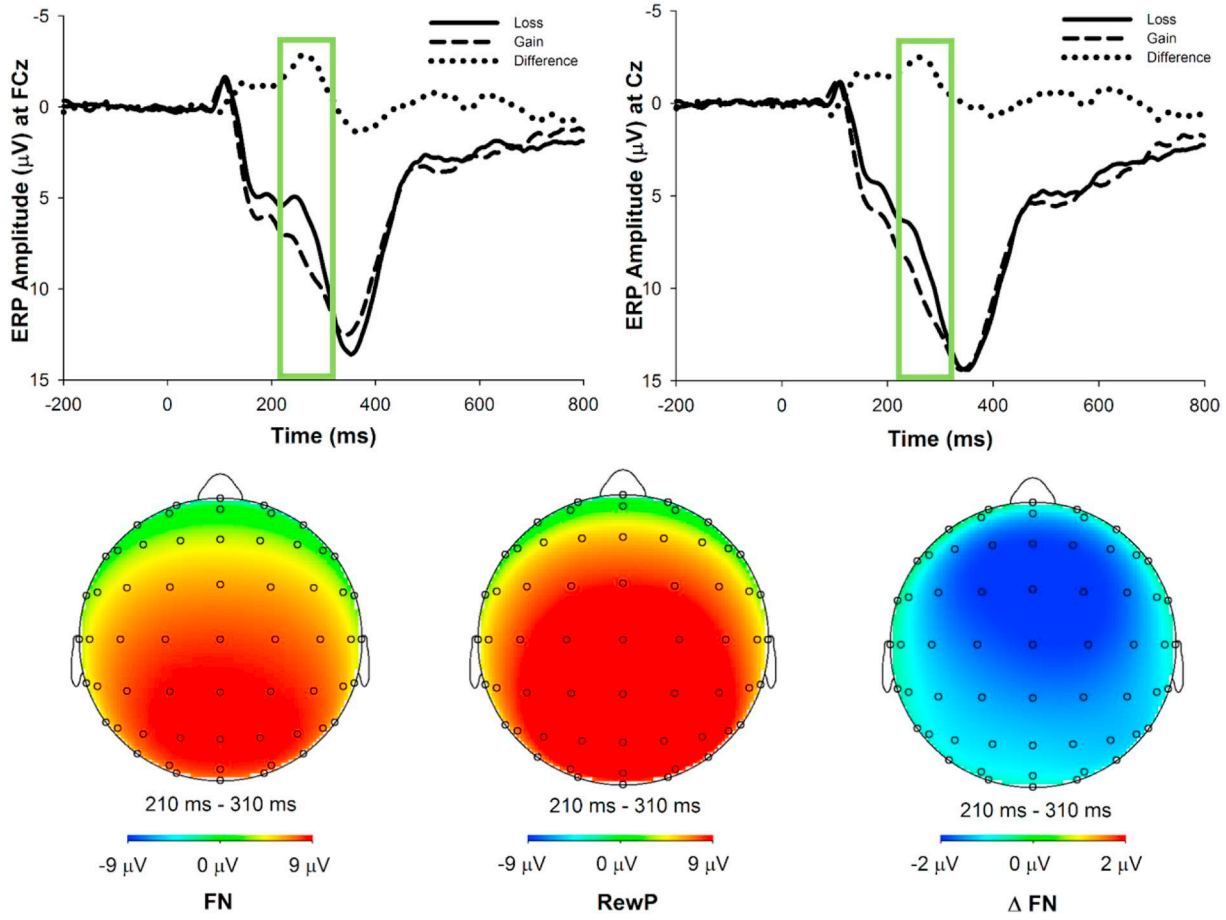

Fig. 10. Top left: Parent feedback-locked, grand average waveforms depicting loss, gain, and the loss-gain difference at electrode FCz. Time 0 is the stimulus onset. Top right: Parent feedback-locked, grand average waveforms depicting loss, gain, and the loss-gain difference at electrode Cz. Time 0 is the stimulus onset. Bottom: Parent scalp topographies depicting voltages for FN, RewP, and the Δ FN, respectively, quantified as the average activity between 210 ms and 310 ms following stimulus onset.

Table 5
Summary of parent ERP variables.

Variable	MLS sample		Community sample		Overall sample	
	M	SD	M	SD	M	SD
FCz FN (μV)	6.28	4.67	6.21	4.65	6.24	4.64
FCz RewP (μV)	7.91	5.49	8.70	4.27	8.34	4.85
FCz Δ FN (μV)	−1.63	3.26	−2.49	2.79	−2.10	3.03
Cz FN (μV)	7.68	4.92	8.44	4.70	8.10	4.79
Cz RewP (μV)	9.21	5.95	10.62	4.68	9.99	5.32
Cz Δ FN (μV)	−1.53	3.73	−2.19	2.90	−1.89	3.30
FCz ERN (μV)	−1.69	6.93	−1.13	5.78	−1.40	6.33
FCz CRN (μV)	2.50	3.75	1.76	4.42	2.11	4.11
FCz Δ ERN (μV)	−4.19	6.55	−2.90	5.35	−3.52	5.96
Cz ERN (μV)	−0.79	7.26	−0.01	5.39	−0.38	6.34
Cz CRN (μV)	3.99	3.49	3.49	4.38	3.73	3.97
Cz Δ ERN (μV)	−4.78	6.49	−3.50	4.31	−4.11	5.47

work examined the FN and RewP in older children and adolescents ages 8 years and older. Only two studies had examined the FN and RewP in young children between the ages of 4 and 7, with one showing differentiation between FN and RewP (Belden et al., 2016) and another failing to find differentiation of FN and RewP (Mai et al., 2011). Two caveats of the Belden et al. study should be noted. First, the only differentiation of the FN and RewP was observed in the control group, which had been screened for various mental and physical health problems. Second, the FN-RewP differentiation was only observed at Pz and not the prototypic fronto-central locations. These caveats therefore restrict the generalizability of these findings to other groups of young children.

Interestingly, follow-up analyses between younger (3–7) and older (8–13) children in the present study demonstrated clear differentiation between the FN and RewP in older but not younger children, consistent with the Mai et al. study. This lack of differentiation between losses and rewards in young children may reflect the relatively immature reward and learning mechanisms involved in self-control mentioned above. Although, the current ERN findings suggest that, at least, certain aspects of the self-control network are online in young children. It may be, then, that the current findings indicate relatively less mature feedback-related processes in children that are specific to guessing tasks. One reason this lack of differentiation may be specific to guessing tasks is what C. B. Holroyd et al. (2009) previously noted; that task engagement and learnable response contingencies are critical to the modulation of FN and RewP. In young children, the guessing task might not be engaging enough to reliably modulate the neural systems underlying these components. Along these lines, it is possible that because younger children (3–7) gained or lost points rather than money, the incentive structure was not salient enough to elicit significant modulation of the FN and RewP. Our analysis showing modulation of the FN and RewP in the older children (8–13) who received money but not in the younger children (3–7) who received points is consistent with this idea. We chose this different incentive structure based on past studies of the FN and RewP in preschoolers (Belden et al., 2016; Mai et al., 2011), which argued that points for prizes are more salient for younger children who have less of a concrete notion of the value of money.

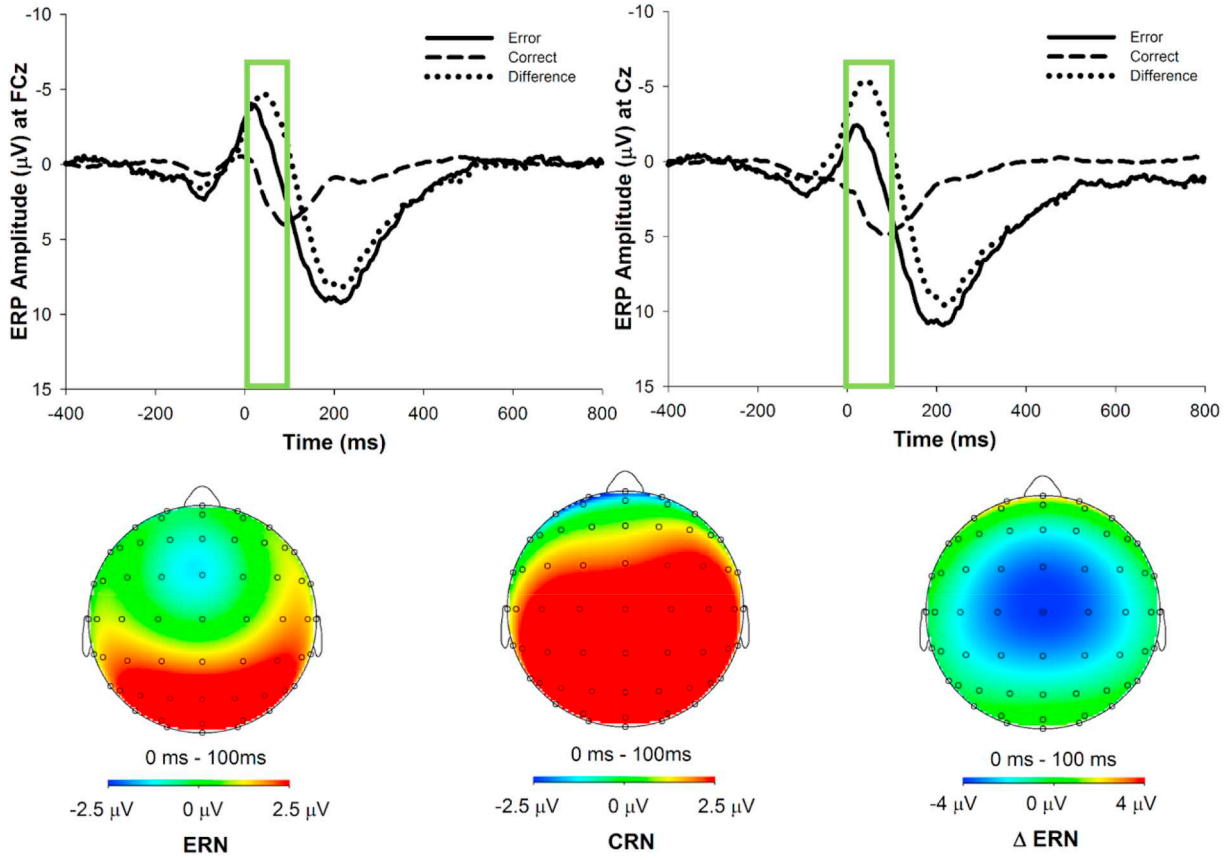

Fig. 11. Top left: Parent response-locked, grand-average waveforms depicting error, correct, and the error-correct difference at electrode FCz with a baseline correction of −400 ms to −200 ms pre-response time. Time 0 is the response onset. Top right: Parent response-locked, grand-average waveforms depicting error, correct, and the error-correct difference at electrode Cz. Time 0 is the response onset. Bottom: Parent scalp topographies depicting voltages for ERN, CRN, and the Δ ERN, respectively, quantified as the average activity between 0 ms and 100 ms following response onset.

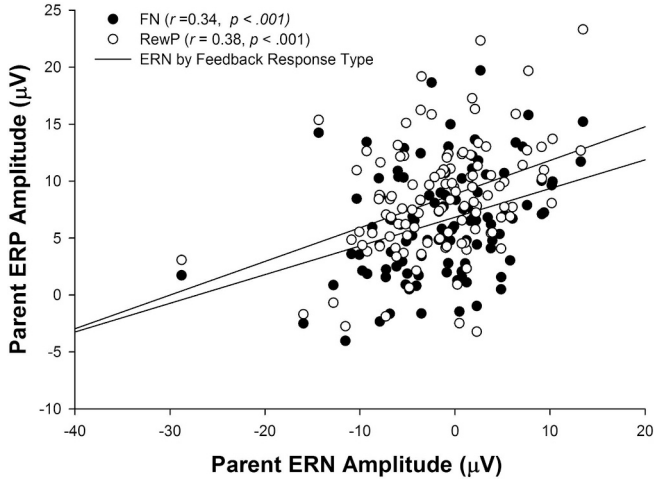

Fig. 12. Relationship between FN and RewP and ERN in parents. Increases in loss- and decreases in reward- related activity were related to increases in error-related activity. FN and ERN are negative-going ERP components and thus more negative values reflect a larger FN and ERN. Removal of seeming outlier in left half of figure does not change relationships.

Importantly, we did find significant age effects in the younger age group that mirrored those observed in the overall sample – such that increases in age were related to increases in Δ FN and RewP – suggestive of the presence of a developmental process even at a young age. Thus, incentive structure does not seem to be solely responsible for age effects reported in the current study.

The reason we used the guessing task here is because it produces feedback-related ERPs that are more closely linked with depression than are reinforcement learning tasks (Moran et al., 2017). Indeed, one study in preschoolers successfully used the guessing task to demonstrate reduced reward processing in depressed children (Belden et al., 2016). This raises another possibility why we did not find differentiation between FN and RewP in younger children in the current sample, namely, because some of the children were at risk for psychopathology given their family history of substance use. We plan to investigate this possibility directly in a future study that takes psychopathology into account. Sorting out these different possibilities will be important for future studies aiming to utilize the FN and RewP as markers of psychopathology symptom severity and risk in children.

The significant gender differences in feedback processing observed in the current study might also help explain the developmental differences in FN and RewP. Consistent with previous work across development (Crowley et al., 2009; Kujawa et al., 2014; Yi et al., 2012), we found that males demonstrated a significant modulation of the FN and RewP whereas females did not. Moreover, the difference between genders was most pronounced for the RewP such that males demonstrated a larger RewP than females. That this effect held across ages suggests a stable gender difference beginning in early development. It also suggests that incentive structure alone cannot account for modulations of the FN and RewP. The larger Δ FN and RewP in males than females are consistent with reported gender differences in mesocorticolimbic system activation (e.g., Hoeft et al., 2008). These gender differences in the FN/RewP may further help explain hyperresponsivity to reward and risk for substance use in males and hyporesponsivity to reward and risk for depression in females (Hammerslag and Gulley,

2015; Heitzeg et al., 2018). What is novel in the current study is that we demonstrated these gender differences in a large sample of children spanning preschoolers to young adolescents.

Consistent with expectations, the current study also provided some evidence that feedback-related ERPs are familial. The most straightforward effect was between father and child RewPs. This finding supports the potential use of this component as a familial risk marker. It also extends the only other existing study on the familiality of the FN and RewP (Weinberg et al., 2015) by showing that the RewP is not only related between siblings but also between fathers and their children. Interestingly, both the current study and Weinberg et al. found stronger familial relationships for the RewP than FN. Our study also extends the Weinberg et al. finding by examining familial relationships between parents and their child and early adolescent offspring. That the RewP was related between fathers and their children has important implications for the intergeneration transmission of reward-related psychopathology. Specifically, sensitivity to reward may be passed down by fathers and thus the amplitude of the RewP in fathers may provide an *early* indicator of risk for reward-related psychopathology in their children that could be identified well before the onset of illness in the child that would likely occur later in adolescence and young adulthood (Hammerslag and Gulley, 2015; Heitzeg et al., 2018). What was less clear was a significant negative association detected between mother and child Δ FNs – i.e., larger Δ FNs in mothers were related to smaller Δ FNs in children.

The current results also suggested that the ERN and CRN are familial. Specifically, mother and child ERNs and CRNs were related to each other. To our knowledge, this is the only family study of the ERN/CRN. Our findings are consistent, however, with a small twin study that demonstrated the heritability of the ERN and CRN (Anokhin et al., 2008). Given that the ERN tends to be more related to anxiety problems (Cavanagh and Shackman, 2015; Moser et al., 2013), ERN amplitudes in mothers may provide markers of risk for anxiety in their children.

We will directly consider the role of feedback- and error-related ERPs in the intergenerational transmission of psychopathology in future studies. Future large-scale behavioral genetic work could also help sort out whether genetic or environmental factors are at play in these familial associations.

Finally, the present findings indicated that feedback- and error-related ERPs were associated in children and adults, albeit in somewhat different ways. In general terms, these results are consistent with models suggesting these ERPs reflect activity of a common action monitoring system (Cavanagh and Shackman, 2015; Clay B Holroyd and Coles, 2002). They also suggest that these components could be used in multivariate studies to index individual differences in action monitoring to build toward multi-method measures in line with the RDoC framework across development (Patrick and Hajcak, 2016; Patrick et al., 2013). Across children and their parents (i.e., adults), a larger FN was related to a larger ERN. Thus, adults and children share a developmentally preserved relationship between neurophysiological markers of negative outcomes (Cavanagh and Shackman, 2015; Clay B Holroyd and Coles, 2002). Children and adults differed, however, with respect to the way in which the RewP and ERN were related. There was no direct relationship between RewP and ERN in children but there was a positive association in adults such that a larger RewP was related to a smaller ERN and vice versa. In the context of neurodevelopmental models of self-control, these findings may reflect the greater integration of cortical structures with subcortical rewards centers (e.g., striatum) in adults than children and adolescents (Casey, 2015; Rubia, 2013). That gender moderated the relationship between Δ FN and Δ ERN scores in children, but not in adults, further points to developmental differences in the functional integration of self-control circuitry that may have relevance to gender differences in the emergence of reward-related psychopathology (Hoeft et al., 2008).

In sum, the current findings advance the science on the FN and RewP by demonstrating their developmental differences, familial transmission, and relations to error-monitoring. Together, the current findings demonstrate that reward-related processes increase from childhood to adolescence and adulthood. They furthermore confirm the familial transmission of the RewP, which is important for its potential use as a risk marker within families. Feedback- and error- related ERPs were generally related to each other, although the relationship was somewhat different between children and adults. Finally, gender differences in children suggested greater reward-related activity and relations between reward- and error-related activity in males than females. The FN and RewP therefore have strong potential for indexing developmentally-sensitive neural markers of reward and action monitoring processes of significant relevance to psychopathology.

Acknowledgements

This work was supported by National Institute of Drug Abuse grant 5R01DA039112 awarded to CED, BMH, RAZ, & JSM.

References

Anokhin, A.P., Golosheykin, S., Heath, A.C., 2008. Heritability of frontal brain function related to action monitoring. Psychophysiology 45 (4), 524–534.
Belden, A.C., Irvin, K., Hajcak, G., Kappenman, E.S., Kelly, D., Karlow, S., ... Barch, D.M., 2016. Neural correlates of reward processing in depressed and healthy preschool-age children. J. Am. Acad. Child Adolesc. Psychiatry 55 (12), 1081–1089. https://doi.org/10.1016/j.jaac.2016.09.503.
Bress, J.N., Smith, E., Foti, D., Klein, D.N., Hajcak, G., 2012. Neural response to reward and depressive symptoms in late childhood to early adolescence. Biol. Psychol. 89 (1), 156–162. https://doi.org/10.1016/j.biopsycho.2011.10.004.
Bress, J.N., Foti, D., Kotov, R., Klein, D.N., Hajcak, G., 2013. Blunted neural response to rewards prospectively predicts depression in adolescent girls. Psychophysiology 50 (1), 74–81. https://doi.org/10.1111/j.1469-8986.2012.01485.x.
Bress, J.N., Meyer, A., Hajcak, G., 2015a. Differentiating anxiety and depression in children and adolescents: evidence from event-related brain potentials. J. Clin. Child Adolesc. Psychol. 44 (2), 238–249. https://doi.org/10.1080/15374416.2013.814544.
Bress, J.N., Meyer, A., Proudfit, G., 2015b. The stability of the feedback negativity and its relationship with depression during childhood and adolescence. Dev. Psychopathol. 27 (4), 1285–1294. https://doi.org/10.1017/S0954579414001400.
Carlson, J.M., Foti, D., Mujica-Parodi, L.R., Harmon-Jones, E., Hajcak, G., 2011. Ventral striatal and medial prefrontal BOLD activation is correlated with reward-related electrocortical activity: a combined ERP and fMRI study. NeuroImage 57 (4), 1608–1616. https://doi.org/10.1016/j.neuroimage.2011.05.037.
Casey, B.J., 2015. Beyond simple models of self-control to circuit-based accounts of adolescent behavior. Annu. Rev. Psychol. 66 (1), 295–319.
Cavanagh, J.F., Shackman, A.J., 2015. Frontal midline theta reflects anxiety and cognitive control: meta-analytic evidence. J. Physiol. Paris 109 (1–3), 3–15.
Clayton, J.A., Collins, F.S., 2014. Policy: NIH to balance sex in cell and animal studies. Nature 509, 282–283.
Crowley, M., Wu, J., Crutcher, C., Bailey, C., Lejuez, C., Mayes, L., 2009. Risk-taking and the feedback negativity response to loss among at-risk adolescents. Dev. Neurosci. 31, 137–148.
Eppinger, B., Mock, B., Kray, J., 2009. Developmental differences in learning and error processing: evidence from ERPs. Psychophysiology 46 (5), 1043–1053. https://doi.org/10.1111/j.1469-8986.2009.00838.x.
Ethridge, P., Kujawa, A., Dirks, M.A., Arfer, K.B., Kessel, E.M., Klein, D.N., Weinberg, A., 2017. Neural responses to social and monetary reward in early adolescence and emerging adulthood. Psychophysiology 54 (12), 1786–1799. https://doi.org/10.1111/psyp.12957.
Foti, D., Hajcak, G., 2009. Depression and reduced sensitivity to non-rewards versus rewards: evidence from event-related potentials. Biol. Psychol. 81 (1), 1–8. https://doi.org/10.1016/j.biopsycho.2008.12.004.
Foti, D., Carlson, J.M., Sauder, C.L., Proudfit, G.H., 2014. Reward dysfunction in major depression: multimodal neuroimaging evidence for refining the melancholic phenotype. NeuroImage 101, 50–58. https://doi.org/10.1016/j.neuroimage.2014.06.058.
Grammer, J.K., Carrasco, M., Gehring, W.J., Morrison, F.J., 2014. Age-related changes in error processing in young children: a school-based investigation. Dev. Cogn. Neurosci. 9, 93–105. https://doi.org/10.1016/j.dcn.2014.02.001.
Gratton, G., Coles, M.G.H., Donchin, E., 1983. A new method for the off-line removal of ocular artifact. Electroencephalogr. Clin. Neurophysiol. 55, 468–484. https://doi.org/10.1016/0013-4694(83)90135-9.
Groen, Y., Wijers, A.A., Mulder, L.J.M., Minderaa, R.B., Althaus, M., 2007. Physiological correlates of learning by performance feedback in children: a study of EEG event related potentials and evoked heart rate. Biol. Psychol. 76, 174–187.
Hämmerer, D., Li, S.C., Müller, V., Lindenberger, U., 2011. Life span differences in electrophysiological correlates of monitoring gains and losses during probabilistic reinforcement learning. J. Cogn. Neurosci. 23 (3), 579–592. https://doi.org/10.1162/jocn.2010.21475.
Hammerslag, L.R., Gulley, J.M., 2015. Sex differences in behavior and neural development and their role in adolescent vulnerability to substance use. Behav. Brain Res.

298, 15–26.
Hayes, A.F., 2017. Introduction to mediation, moderation and conditional process analysis (2nd Ed): a regression-based approach. Guilford Press.
Heitzeg, M.M., Hardee, J.E., Beltz, A.M., 2018. Sex differences in the developmental neuroscience of adolescent substance use risk. Curr. Opin. Behav. Sci. 23, 21–26.
Hoeft, F., Watson, C.L., Kesler, S.R., Bettinger, K.E., Reiss, A.L., 2008. Gender differences in the mesocorticolimbic system during computer game-play. J. Psychiatry Res. 42, 253–258.
Holroyd, C.B., Coles, M.G.H., 2002. The neural basis of human error processing: reinforcement learning, dopamine, and the error-related negativity. Psychol. Rev. 109 (4), 679–709.
Holroyd, C.B., Krigolson, O.E., Baker, R., Lee, S., Gibson, J., 2009. When is an error not a prediction error? An electrophysiological investigation. Cogn. Affect. Behav. Neurosci. 9 (1), 59–70. https://doi.org/10.3758/CABN.9.1.59.
Horan, W.P., Foti, D., Hajcak, G., Wynn, J.K., Green, M.F., 2012. Impaired neural response to internal but not external feedback in schizophrenia. Psychol. Med. 42 (8), 1637–1647. https://doi.org/10.1017/S0033291711002819.
Kujawa, A., Proudfit, G.H., Klein, D.N., 2014. Neural reactivity to rewards and losses in offspring of mothers and fathers with histories of depressive and anxiety disorders. J. Abnorm. Psychol. 123 (2), 287–297. https://doi.org/10.1037/a0036285.
Lukie, C.N., Montazer-Hojat, S., Holroyd, C.B., 2014. Developmental changes in the reward positivity: an electrophysiological trajectory of reward processing. Dev. Cogn. Neurosci. 9, 191–199. https://doi.org/10.1016/j.dcn.2014.04.003.
Mai, X., Tardif, T., Doan, S., Liu, C., Gehring, W., Luo, Y., 2011. Brain activity elicited by positive and negative feedback in preschool-aged children. PLoS One 6 (4). https://doi.org/10.1371/journal.pone.0018774.
Moran, T.P., Schroder, H.S., Kneip, C., Moser, J.S., 2017. Meta-analysis and psychophysiology: a tutorial using depression and action-monitoring event-related potentials. Int. J. Psychophysiol. 111, 17–32. https://doi.org/10.1016/j.ijpsycho.2016.07.001.
Moser, J.S., Moran, T.P., Schroder, H.S., Donnellan, M.B., Yeung, N., 2013. On the relationship between anxiety and error monitoring: a meta-analysis and conceptual framework. Front. Hum. Neurosci. 7, 466.
Moser, J.S., Durbin, C.E., Patrick, C.J., Schmidt, N.B., 2015. Combining neural and behavioral indicators in the assessment of internalizing psychopathology in children and adolescents. J. Clin. Child Adolesc. Psychol. 44 (2), 329–340.
Nelson, B.D., Perlman, G., Klein, D.N., Kotov, R., Hajcak, G., 2016. Blunted neural response to rewards as a prospective predictor of the development of depression in adolescent girls. Am. J. Psychiatr. 173 (12), 1223–1230. https://doi.org/10.1176/appi.ajp.2016.15121524.
Patrick, C.J., Hajcak, G., 2016. RDoC: translating promise into progress. Psychophysiology 53 (3), 415–424. https://doi.org/10.1111/psyp.12612.
Patrick, C.J., Venables, N.C., Yancey, J.R., Hicks, B.M., Nelson, L.D., Kramer, M.D., 2013. A construct-network approach to bridging diagnostic and physiological domains: application to assessment of externalizing psychopathology. J. Abnorm. Psychol. 122 (3), 902–916. https://doi.org/10.1037/a0032807.
Potts, G.F., Martin, L.E., Kamp, S., Donchin, E., 2011. Neural response to action and reward prediction errors: comparing the error-related negativity to behavioral errors and the feedback-related negativity to reward prediction violations. Psychophysiology 48, 218–228.
Proudfit, G.H., 2015. The reward positivity: from basic research on reward to a biomarker for depression. Psychophysiology 52 (4), 449–459. https://doi.org/10.1111/psyp.12370.
Rubia, K., 2013. Functional brain imaging across development. Eur. Child Adolesc. Psychiatry 22 (12), 719–731. https://doi.org/10.1007/s00787-012-0291-8.
Schroder, H.S., Fisher, M.E., Lin, Y., Lo, S.L., Danovitch, J.H., Moser, J.S., 2017. Neural evidence for enhanced attention to mistakes among school-aged children with a growth mindset. Dev. Cogn. Neurosci. 24, 42–50. https://doi.org/10.1016/j.dcn.2017.01.004.
Simons, R.F., 2010. The way of our errors: theme and variations. Psychophysiology 47, 1–14.
Wahlstrom, D., Collins, P., White, T., Luciana, M., 2010. Developmental changes in dopamine neurotransmission in adolescence: behavioral implications and issues in assessment. Brain Cogn. 72 (1), 146–159. https://doi.org/10.1016/j.bandc.2009.10.013.
Weinberg, A., Liu, H.T., Hajcak, G., Shankman, S.A., 2015. Blunted neural response to rewards as a vulnerability factor for depression: results from a family study. J. Abnorm. Psychol. 124 (4), 878–889. https://doi.org/10.1037/abn0000081.
Yi, F., Chen, H., Wang, X., Shi, H., Yi, J., Zhu, X., Yao, S., 2012. Amplitude and latency of feedback-related negativity: aging and sex differences. Neuroreport 23 (16), 963–969. https://doi.org/10.1097/WNR.0b013e328359d1c4.
Zucker, R.A., Ellis, D.A., Fitzgerald, H.E., Bingham, C.R., Sanford, K.P., 1996. Other evidence for at least two alcoholisms: life course variation in antisociality and heterogeneity of alcoholic outcome. Dev. Psychopathol. 8, 831–848. https://doi.org/10.1017/S0954579400007458.
Zucker, R.A., Fitzgerald, H.E., Refior, S.K., Puttler, L.I., Pallas, D.M., Ellis, D.A., 2000. The clinical and social ecology of childhood for children and alcoholics: description of a study and implications for a differential social policy. In: Fitzgerald, H.E., Lester, B.M., Zuckerman, B.S. (Eds.), Children of Addiction: Research, Health, and Policy Issues. Routledge Falmer, New York, pp. 109–141.