BRIEF REPORT

Neural Responses to Gains and Losses in Children of Suicide Attempters

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Suicidal behavior aggregates within families, yet the specific mechanisms of suicide-risk transmission are poorly understood. Despite some evidence that abnormal patterns of reward responsiveness might constitute one such potential mechanism, empirical evidence is lacking. The goal of this study was to examine neural responses to gains and losses in children of suicide attempters with no personal history of suicide attempt (SA) themselves. To objectively assess these neural responses, we used feedback negativity (FN), a psychophysiological marker of responsiveness to reward and loss. Participants were 66 parents and their 7–11-year-old children (22 with parental history of SA and 44 demographically and clinically matched children of parents with no SA history). Diagnostic interviews were used to gather information about psychiatric diagnoses, symptoms, and histories of suicidal thoughts and behaviors. Children also completed a guessing task, during which continuous electroencephalography (EEG) was recorded. The FN was scored as the mean amplitude, 275–375 ms, following gain or loss feedback at frontocentral sites (Fz and FCz). Children of suicide attempters exhibited significantly more negative ΔFN (i.e., FN to losses minus FN to gains) than children of parents with no SA history. We found that this difference in ΔFN was due specifically to children of parents with a history of SA exhibiting a stronger response to loss, and no group differences were observed for responses to gains. The results suggest that an increased neural response to loss might represent one of the potential pathways of the familial transmission of suicide risk.

General Scientific Summary
Despite clear evidence that suicidal behavior aggregates within families, little is known about specific mechanisms of risk. This study is the first to show that heightened neural responses to loss differentiate children of parents with a history of attempted suicide from those without. The findings may have important clinical implications by suggesting hyperresponsiveness to loss as a potential target for early interventions designed to reduce future risk for suicidal thoughts and behaviors in children.

Keywords: suicide risk, reward, EEG, feedback negativity, children

Suicide is a major public health concern and constitutes the second leading cause of death in the United States for 10–14 year olds (Centers for Disease Control and Prevention, 2016). Despite decades of research, however, there was a 45% increase in the suicide rates among females and a 16% increase in the suicide rates among males between 1999 and 2014, with those aged 10–14

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having the greatest percent increase (National Center for Health Statistics, 2016). That said, there is strong evidence that suicidal behavior aggregates within families and that the intergenerational transmission of suicide risk is at least partially independent of the familial transmission of psychiatric disorders (for a review, see Brent & Melhem, 2008). However, the specific mechanisms of risk in children of parents with suicide attempt (SA) history remain unclear. This area of inquiry is important because rates of self-harming thoughts and behaviors increase dramatically during the transition from childhood to adolescence (e.g., Kessler, Berglund, Borges, Nock, & Wang, 2005). Thus, an increased understanding of these mechanisms might contribute to prevention efforts by improving our ability to identify and target specific vulnerabilities in at-risk children.

There is evidence from several lines of research that abnormal patterns of reward responsiveness (i.e., responses to reward and loss of reward) might be one mechanism in the familial transmission of suicidal behavior. Indeed, neuroimaging and postmortem research provides evidence of structural and/or functional changes associated with suicidal behavior in a number of brain regions implicated in reward responsiveness, including ventral and dorsal striatum, anterior cingulate cortex (ACC), orbitofrontal cortex, and ventral and dorsal insula (for a review, see van Heeringen & Mann, 2014). Studies also suggest an association between self-reported anhedonia and SA (e.g., Auerbach, Millner, Stewart, & Esposito, 2015; Nock & Kazdin, 2002). Further, although not yet examined in the context of parental SA, patterns of abnormal reward responsiveness have been observed in never-disordered children of depressed parents (e.g., Gotlib et al., 2010; Kujawa, Proudfit, & Klein, 2014; Luking, Pagliaccio, Luby, & Barch, 2016; McCabe, Woffindale, Harmer, & Cowen, 2012).

Event-related potentials (ERPs) from electroencephalography (EEG) are well-suited for the assessment of neural processes related to reward responsiveness across development (Nelson & McCleery, 2008). Feedback negativity (FN) has been used as a psychophysiological quantifier of the evaluation of outcomes as either favorable or unfavorable (Hajcak, Moser, Holroyd, & Simons, 2006). The FN peaks approximately 300 ms after feedback, is maximal over frontocentral recording sites, and is thought to be driven by the value of one outcome relative to another, as opposed to the absolute value of any single outcome (Hajcak et al., 2006). A more negative deflection in FN corresponds to larger neural responsiveness to an outcome, whereas a less negative deflection in FN reflects a blunted neural response. According to the reinforcement-learning theory, FN reflects phasic activity in the midbrain dopamine system (Holroyd & Coles, 2002). Source-localization research suggests that it is generated by activity in the striatum (e.g., Foti, Weinberg, Dien, & Hajcak, 2011) and ACC (e.g., Potts, Martin, Burton, & Montague, 2006). In line with these findings, evidence from functional magnetic resonance imaging (fMRI) has demonstrated a direct link between the FN amplitude and reward-related activation in the ventral striatum (Carlson, Foti, Mujica-Parodi, Harmon-Jones, & Hajcak, 2011), which makes this ERP component ideally suited for the investigations of neural reward-responsiveness abnormalities.

To isolate neural sensitivity to gains versus losses, many studies have relied on the ΔFN (i.e., FN to losses, FN to gains; e.g., Foti & Hajcak, 2009; Foti et al., 2011; Kujawa et al., 2014), which is associated with reward-related neural activity (e.g., Carlson et al.,

2011) as well as with behavioral and self-report measures of reward sensitivity (e.g., Bress & Hajcak, 2013). To date, one study has examined neural responses to reward using the ΔFN in at-risk children. This study found that children of mothers with a history of major depressive disorder (MDD) without a comorbid anxiety disorder exhibited a blunted ΔFN, and greater maternal depression severity was associated with greater blunting of the ΔFN (Kujawa et al., 2014). Consistent with this finding, a number of fMRI studies demonstrated blunted striatal responses to the receipt of reward in never-depressed offspring of depressed parents, compared with children of nondepressed parents (for a review, see Luking et al., 2016). However, no studies have examined whether similar alterations in neural response to reward might also be observed in children of parents with a suicide attempt history. Further, it is unclear whether group differences in the ΔFN amplitude among children with a family history of SA would be driven by reduced sensitivity to gains, greater sensitivity to losses, or a combination of the two. Indeed, although few studies to date have investigated reactivity to punishment or loss of reward, their findings consistently demonstrate enhanced response to negative feedback in high-risk children and adolescents (for a review, see Luking et al., 2016).

In summary, there is robust evidence that suicidal behavior runs in families (Brent & Melhem, 2008) and that reward responsiveness abnormalities are observed in never-disordered children of depressed parents (e.g., Gotlib et al., 2010; Kujawa et al., 2014; McCabe et al., 2012). Further, studies have demonstrated structural or functional alterations associated with suicidal behavior in a number of brain regions implicated in reward responsiveness (Van Heeringen & Mann, 2014) and the relation between self-reported anhedonia and SA (e.g., Auerbach et al., 2015; Nock & Kazdin, 2002). However, although suicide is a transdiagnostic behavior, it is unclear whether children of suicide attempters would demonstrate abnormalities in neural response to reward independently of familial history of depression (or anxiety), and whether this neural response would be driven by the sensitivity to gains, losses, or a combination of the two. Therefore, the goal of this study was to address these gaps in the literature by examining neural response to gains and losses, assessed via the FN, in children of suicide attempters with no personal history of SA themselves. We hypothesized that parental suicide attempt history would be associated with a reduced response to gains and a heightened response to losses.

Method

Participants

Participants in this study were 66 parent–child pairs (57 mothers, nine fathers) recruited from the community. Using a 1:2 matching ratio, we had 22 dyads with a history of parent SA and 44 dyads with no history of SA in the parent. No children had any lifetime history of SA. The two groups were equated on (a) approximate child and parent age, (b) child and parent sex, (c) child and parent race, (d) household income, (e) child and parent MDD history, (f) child and parent history of any anxiety disorder, (g) child mean depressive and anxious symptoms, and (h) child history of suicidal ideation. All children were between the ages of 7 and 11 years and, per parent report, had no learning or devel-

opmental disorders that would preclude completing the study protocol. Only one biological child per family was included in the study. The average age of the children was 9.84 years ($SD = 1.42$) and 40.9% were girls. In terms of race, 66.7% of the children were Caucasian, 18.2% were African American, 13.6 were multiracial, and 1.5% were Asian/Pacific Islander. In terms of ethnicity, 9.1% of the children were Hispanic. The average age of the parents was 36.03 years ($SD = 6.02$). Of the parents, 86.4% were the children's mothers and the remaining 13.6% were the children's fathers. In terms of parent race, 77.3% were Caucasian, 18.2% were African American, 3.0% were multiracial, and 1.55% were Asian/Pacific Islander. In terms of ethnicity, 3.0% of the participating parents were Hispanic. The demographic and clinical characteristics of SA and no-SA groups are presented in Table 1.

Measures

Diagnoses and symptoms. The *Structured Clinical Interview for DSM–IV Axis I Disorders* (*SCID-I/P*; First, Spitzer, Gibbon, & Williams, 1995) and the Schedule for Affective Disorders and Schizophrenia for School-Age Children–Present and Lifetime Version (K-SADS-PL; Kaufman et al., 1997) were used to assess for current and past *DSM–IV* (APA *Diagnostic and Statistical Manual of Mental Disorders,* 4th ed., text rev., 2000) MDD and anxiety disorders in parents and their children, respectively. A total of 38 parents (33 mothers, five fathers) and five children (two girls, three boys) met criteria for a lifetime history of MDD. A total of 29 parents (26 mothers, three fathers) and five children (two girls, three boys) met criteria for a lifetime history of an anxiety disorder. The rates of psychopathology in this study are higher than what has been observed in epidemiological studies (e.g., Kessler et al., 2005), which we believe was due, at least in part, to the low SES nature of the sample (cf. Galea et al., 2007; Kessler et al., 2003; Lorant et al., 2003). Specifically, 64% of the parents did not

graduate from college and the median family income was $20,000–$25,000. In addition, as described below, the language of our advertising may have disproportionately attracted families with a history of depression. To assess interrater reliability, a subset of 20 *SCID* and K-SADS interviews from this project were coded by a second interviewer and κ coefficients for diagnoses of MDD and anxiety disorders in parents and children were good (all κ ≥ .86). Further, children's symptoms of depression and anxiety were assessed using the Children's Depression Inventory (CDI; Kovacs, 1981) and the Multidimensional Anxiety Scale for Children (MASC; March, Parker, Sullivan, Stallings, & Conners, 1997). Both scales exhibited good internal consistency (CDI, α = .81; MASC, α = .88).

Suicide history. As part of the assessment, interviewers assessed for the presence of a lifetime SA in parents asking the following question: "A suicide attempt is defined as intentionally hurting yourself with at least some wish to die *at that time*. How many times have you attempted suicide in your life?" As part of the K-SADS-PL (Kaufman et al., 1997) assessment, the interviewers assessed for the presence of suicidal ideation (SI) in children by asking the questions "Sometimes children who get upset or feel bad, wish they were dead or feel they'd be better off dead. Have you ever had these types of thoughts?" and "Sometimes children who get upset or feel bad think about dying or even killing themselves. Do you have these thoughts?" As noted above, the two groups were matched on children's history of SI.

Reward task. The reward task was a simple guessing task previously used in other studies of reward processing (e.g., Foti et al., 2011; Kujawa et al., 2014; Nelson et al., 2016). The task consisted of 50 trials, presented in two blocks of 25 trials. Participants were shown an image of two doors at the beginning of each trial and instructed to guess which door had a monetary prize behind it by pressing either the left or right button on a game

Table 1

Descriptive Statistics for Children and Parents in Each of the Two Groups

Measure	Parent SA ($n = 22$)	No parent SA ($n = 44$)	$r_{effect\ size}$
Demographics			
Child age	9.52 (1.21)	9.99 (1.50)	−.16
Child sex (% female)	40.9%	40.9%	.00
Child race (% Caucasian)	72.7%	63.6%	.09
Parent age	36.23 (5.67)	35.93 (6.25)	.02
Parent sex (% female)	86.4%	86.4%	.00
Parent race (% Caucasian)	90.9%	70.5%	.23
Household income (median)	25,001–30,000	20,001–25,000	.16
Diagnoses			
Child lifetime MDD	2 (9.1%)	3 (6.8%)	.04
Child lifetime anxiety dx	2 (9.1%)	3 (6.8%)	.04
Parent lifetime MDD[1]	15 (68.2%)	23 (52.3%)	.15
Parent lifetime anxiety dx[2]	12 (54.5%)	17 (38.6%)	.15
Child symptoms and SI			
CDI	5.82 (4.10)	6.17 (5.41)	−.03
MASC	48.31 (16.59)	47.63 (16.12)	.02
SI history	9 (40.9%)	16 (36.4%)	.04

Note. MDD = major depressive disorder; dx = diagnosis; CDI = Children's Depression Inventory; MASC = Multidimensional Anxiety Scale for Children; SI = suicidal ideation.
[1] Of the parents with a history of MDD, three in the SA group were fathers and two of those in the no-SA group were fathers. [2] Of the parents with a history of anxiety disorders, two in the SA group were fathers and one of those in the no-SA group was a father.

controller. They were informed on each trial that they could either win $0.50, as indicated by a green up arrow, or lose $0.25, as indicated by a red down arrow. Feedback about having chosen correctly or incorrectly was presented for 2,000 ms, which was followed by the message "Click for the next round." This message remained on the screen until the participant responded and the next trial began. Across the task, 25 gain and 25 loss trials were presented in a random order.

EEG data acquisition and processing. During the task, continuous EEG was recorded using a custom cap and the BioSemi ActiveTwo system (Amsterdam, the Netherlands). The EEG was digitized at 24-bit resolution with a sampling rate of 512 Hz. Recordings were taken from 34 scalp electrodes based on the 10/20 system. The electro-oculogram was recorded from four facial electrodes. Offline analysis was performed using the Matlab extension EEGLAB (Delorme & Makeig, 2004) and the EEGLAB plug-in ERPLAB (Lopez-Calderon & Luck, 2014). All data were re-referenced to the average of the left and right mastoid electrodes and band-pass filtered with cutoffs of 0.1 Hz and 30 Hz. EEG data were processed using both artifact rejection and correction. Large

and stereotypical ocular components were identified and removed using independent component analysis (ICA) scalp maps (Jung et al., 2001). Epochs with large artifacts (greater than 100 μV) were excluded from analysis. EEG was segmented for each trial, beginning 200 ms before onset of the feedback stimulus and ending 600 ms after onset of the feedback stimulus. The average number of trials rejected was 4.82 ($SD = 4.40$). ERPs were separately averaged across gain and loss trials. The average number of usable trials was 22.50 ($SD = 2.47$) in the gain and 22.58 ($SD = 2.36$) in the loss condition. The FN was scored as the mean amplitude 275–375 ms following feedback at frontocentral electrode sites Fz and FCz. We examined the mean amplitude on gain and loss trials separately, as well as the ΔFN, calculated as the difference in mean amplitude to loss trials minus gain trials. See Figure 1 for ERP plots.

Procedure

Potential participants were recruited from the community through a variety of means (e.g., Facebook and TV ads) focused on "how stress
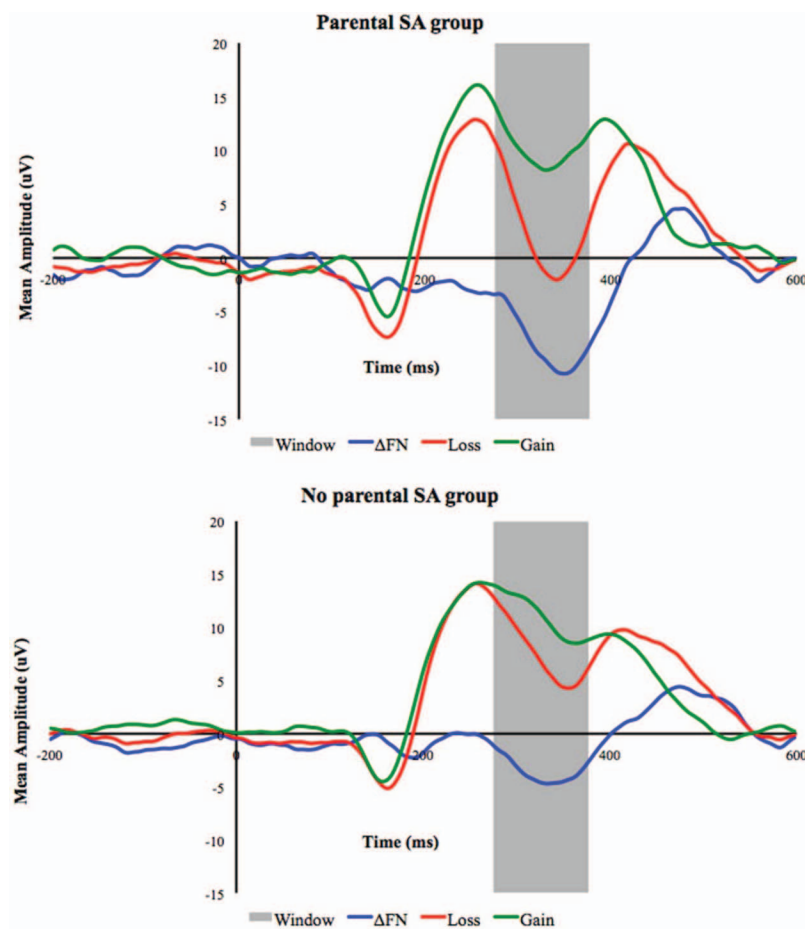

Figure 1. Stimulus-locked event-related potentials to feedback indicating monetary loss (red, middle) and gain (green, top), as well as the difference waveform for loss minus gain trials (blue, bottom) for children of parents with (upper panel) and without (lower panel) a history of SA. The gray region (window) shows the measurement window for FN (275–375 ms). Waveforms are averaged across Fz and FCz locations. See the online article for the color version of this figure.

and moods run in families" and "why some children are happier than others." Parents responding to the advertisements were initially screened over the phone to determine potential eligibility. Upon arrival at the laboratory, parents were asked to provide informed consent and children were asked to provide assent to be in the study. Next, the child completed the reward task. During this time, the *SCID-I/P* (First et al., 1995) and then the K-SADS-PL (Kaufman et al., 1997) were administered to the parent by two separate trained interviewers. Following this, the same interviewer who had administered the K-SADS-PL to the parent also administered it to the child. The institutional review board approved all procedures. Families were compensated a total of $90 for their participation in the study. Children also received a bonus of $5 for completing the reward task.

Results

Focusing first on the ΔFN, we conducted a one-way analysis of variance (ANOVA) with the parental SA group serving as the independent variable and the ΔFN magnitude serving as the dependent variable. We found a significant group difference in ΔFN, $F(1, 64) = 5.85$, $p = .02$, $\eta_p^2 = .08$, with children of parents with a history of SA exhibiting significantly more negative ΔFN than children of parents with no SA history (see Figure 1). Because significant differences in ΔFN can be driven by differences in response to gains, losses, or both, we next examined responses to gains and losses separately. Specifically, we conducted a 2 (group: parent SA, no parent SA) × 2 (condition: gain, loss) repeated-measures ANOVA with children's FN amplitude serving as the dependent variable. Although the main effect of parent SA group was not significant, $F(1, 64) = 1.57$, $p = .22$, $\eta_p^2 = .02$, there was a significant main effect of condition, $F(1, 64) = 27.64$ $p < .01$, $\eta_p^2 = .30$, and a significant Group × Condition interaction, $F(1, 64) = 5.85$, $p = .02$, $\eta_p^2 = .08$. Follow-up tests revealed significant group differences in response to loss, $F(1, 64) = 5.44$, $p = .02$, $\eta_p^2 = .08$, but not gains, $F(1, 64) = 0.02$, $p = .89$, $\eta_p^2 < .001$. Specifically, the children of parents with a history of SA exhibited significantly less positive neural response to losses than children of parents with no SA history. These findings indicate that the observed group difference in ΔFN was due specifically to children of parents with a history of SA, compared with children with no parental history of SA, exhibiting stronger responses to loss, not a reduced response to gain.[1]

Although, as noted above, the two groups were matched on key demographic and clinical variables, it is possible that the group difference in FN to loss was driven by subtle differences between the groups. Therefore, we conducted a series of analyses to examine the robustness of the findings, statistically controlling for the potential influence of these other variables. The group difference in response to loss was maintained if we excluded children with a lifetime history of SI, $F(1, 39) = 5.20$, $p = .03$, $\eta_p^2 = .12$, or MDD or anxiety disorders, $F(1, 55) = 6.79$, $p = .01$, $\eta_p^2 = .11$, and when we statistically controlled for the influence of their current symptoms of depression, $F(1, 63) = 5.44$, $p = .02$, $\eta_p^2 = .08$, or anxiety, $F(1, 63) = 5.41$, $p = .02$, $\eta_p^2 = .08$, suggesting that the results were not simply due to the presence of psychopathology in the children. The findings were also maintained if we statistically controlled for the influence of parents' lifetime history of MDD, $F(1, 63) = 4.13$, $p = .046$, $\eta_p^2 = .06$, or anxiety disorders, $F(1, 63) = 4.95$, $p = .03$, $\eta_p^2 = .07$, children's history of abuse, $F(1, 63) = 5.62$, $p = .02$,

$\eta_p^2 = .08$, or children's report of parental warmth, $F(1, 63) = 5.39$, $p = .02$, $\eta_p^2 = .08$. In addition, the findings were maintained if fathers ($n = 9$) were excluded from the analyses and we only focused on mother–child dyads, $F(1, 55) = 4.49$, $p = .04$, $\eta_p^2 = .08$. Finally, the findings were maintained when the dyads with parental SA during child's life ($n = 4$) were excluded from the analyses and we only focused on SAs occurring before the child's birth, $F(1, 60) = 5.95$, $p = .03$, $\eta_p^2 = .08$, suggesting that direct exposure to the SA is not required to see the effect on neural response to loss.

Discussion

The goal of this study was to examine neural response to gains and losses, assessed using EEG and FN, in children of parents with and without a history of SA who had no personal history of SA themselves. We found that children with a parent history of SA exhibited significantly more negative ΔFN than children of parents with no SA history. Notably, when examining neural responses to gains and losses separately, the findings were specific to children of suicide attempters exhibiting a stronger response to loss than children with no parental history of SA. In contrast, the only study to date to examine the FN measure using this task in children of depressed parents found that children of depressed mothers without a comorbid anxiety disorder exhibited a blunted (i.e., less negative) ΔFN compared with children of never depressed mothers (Kujawa et al., 2014). In addition, a recent study found that blunted ΔFN prospectively predicted first-onset depressive disorder and increases in depressive symptoms in adolescent girls (Nelson et al., 2016). Although the precise reasons for the discrepancy between the current findings and these others are not clear, we should note that the intergenerational transmission of suicide risk is at least partially independent of the familial transmission of psychiatric disorders, including depression (Brent & Melhem, 2008). Therefore, the specificity of the current results to neural responses to loss may suggest a potential distinctive pathway for the familial transmission of suicide risk. Specifically, whereas blunted ΔFN might constitute a marker of the familial risk for depression, a larger (more negative) ΔFN driven by a heightened neural reactivity to losses might be more closely related to the familial risk for suicidal behavior. It should be noted, however, that the present study was the first to focus on neural responses to gains and losses, assessed via the FN, as one of the potential mechanisms of the familial transmission of suicidal behavior. Therefore, conclusions must remain tentative pending replication.

The current study had a number of strengths and constitutes an important addition to the literature on potential mechanisms of the intergenerational transmission of suicidal behavior. Specifically, it is the first study to use an objective psychophysiological marker

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[1] Looked at another way, we conducted a logistic regression with parent SA history (yes/no) as the outcome variable and the FN to gains and losses both entered as predictor variables. This allowed us to examine the unique relation of FN to losses versus gains with parent SA, statistically controlling for their overlap. In this analysis, the FN to losses, Wald = 7.29, $p < .01$, OR = .88, but not gains, Wald = 3.12, $p = .08$, OR = 1.08, was significantly associated with parent SA history. We should also note that the FN to losses × FN to gains interaction was not significant, Wald = 1.66, $p = .20$, OR = 1.00. These results provide further evidence that the relation is specific to children's responses to losses.

with the goal of evaluating neural responses to gains versus losses as a potential mechanism of the familial transmission of suicidal behavior. Additional strengths included the focus on children, the use of a demographically and clinically matched sample, and the tests of robustness to rule out a number of other likely explanations for FN differences in the children. Despite these strengths, there were also some limitations that provide directions for future research. First, the study was cross-sectional and future research is needed to determine the utility of the FN in predicting suicide risk over time. In addition, our sample included primarily mother-child pairs and a limited number of father-child pairs, which prevented us from examining potential differences in the intergenerational transmission of suicide risk from mothers versus fathers. We should note, however, that our findings were maintained if father–child pairs were excluded from the analyses, suggesting that FN may be a useful marker of risk in children of mothers with a history of SA. Nonetheless, future research should examine the potential moderating role of parent sex on the FN magnitude in children at familial risk for suicidal behavior to determine whether the effects may be stronger for a history of SA in mothers versus fathers (vs. both). Future researchers should also seek to determine whether the effects are stronger for sons versus daughters of suicide attempters. Relatedly, our study focused on the impact of only one parent and no information was available about a history of SA or other variables in the other parent. Future research is needed to examine the influence of a suicide history of one versus both parents. Further, although consistent with previous studies in children of depressed parents (e.g., Kujawa et al., 2014), the magnitude of the parental SA effect on children's FN was relatively small (8% of variance explained). This suggests a need for future research to elucidate other neural processes that might be implicated in the familial transmission of suicide risk as well as factors that may moderate the link between family history of SA and children's neural response to loss and reward. In addition, although the size of our SA group was typical for an ERP study, it was still relatively small and thus it will be important for future studies to replicate our findings in larger samples. Finally, our sample was relatively low income (median family income $20,000–$25,000) with rates of parental MDD and child SI that were higher than rates typically observed in community samples. Therefore, additional research is needed to determine whether the results generalize to other populations.

In sum, the present study suggests that an increased neural response to loss might represent one of the potential pathways for the familial transmission of suicide risk. Although replications and longitudinal examinations are needed, the study contributes to the literature by suggesting a novel objective psychophysiological marker in children at familial risk for suicidal behavior. It is important to note, to the extent these findings are replicated in other samples, hyperresponsiveness to loss might represent a potentially important target for early suicide prevention in at-risk children.

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Correction to Tsypes et al. (2016)

In the article "Neural Responses to Gains and Losses in Children of Suicide Attempters" by Aliona Tsypes, Max Owens, Greg Hajcak, and Brandon E. Gibb (*Journal of Abnormal Psychology*, 2016, Advance online publication. November 3, 2016. http://dx.doi.org/10.1037/abn0000237), Figure 1 had incorrect axis labels. There was also an error in the abstract, which did not state that ΔFN was calculated as FN to losses minus FN to gains. All versions of this article have been corrected.

http://dx.doi.org/10.1037/abn0000248