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Response to monetary and social feedback during development: Associations with depressive symptomatology and risk

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Abstract of the Dissertation

Response to monetary and social feedback during development: Associations with depressive symptomatology and risk

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Depression represents a major public health concern, with particularly harmful effects in children. Existing interventions show relatively low response rates and high relapse rates, and there has been a recent effort to identify biomarkers of depression that may improve interventions and guide the way for novel treatments. One potential biomarker is the neural response to reward. However, existing studies typically focus on monetary reward, which may be less relevant to children and adolescents than social feedback. The current study focused on the feedback negativity (FN), an event-related potential (ERP) typically elicited by monetary feedback, in the context of a novel, ecologically valid social feedback task. The sample was composed of 213 8- to 16-year-old girls and parents. The goals of the study were to determine the degree of similarity between ERPs elicited by monetary and social feedback, and to assess unique relationships between the social FN and pubertal development, depression, and familial risk for depression when accounting for the response to monetary feedback. Although a robust

FN response was not apparent in the ERP waveform for the social task, principal components analysis revealed an underlying component similar to the FN, which correlated strongly with the monetary FN and, unexpectedly, was larger for rejection compared to acceptance feedback. This component showed unique negative associations with latent variables representing both pubertal development and depression, and did not differ between participants at high and low familial risk for depression. The current results suggest that the social FN may reflect aspects of outcome salience in addition to reward value. The unique relationship between the social FN and depression, in the absence of an effect of maternal risk, indicates a possible role for the social FN as a state measure of decreased attentional engagement or emotional responsiveness in individuals with depression. Potential environmental influences on the relationship with pubertal development are also discussed. Although additional studies will be needed to further characterize the social FN, this component could serve as a useful measure of treatment response in future clinical studies.

Table of Contents

List of Tables

List of Figures

List of Abbreviations

CDI: Children's Depression Inventory

CFA: confirmatory factor analysis

CFI: Comparative fit index

DSM-IV-TR: Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision

EFA: exploratory factor analysis

ERP: event-related potential

fMRI: functional magnetic resonance imaging

FN: feedback negativity

GnRH: gonadotropin-releasing hormone

K-SADS-PL: Kiddie Schedule for Affective Disorders and Schizophrenia – Present and Lifetime

MDD: major depressive disorder

PBIP: Picture-Based Interview about Puberty

PCA: principal components analysis

PDS: Pubertal Development Scale

PROMIS: Patient-Reported Outcomes Measurement Information System

RDoC: Research Domain Criteria

RMSEA: root mean squared error of approximation

SCID-I: Structured Clinical Interview for the DSM-IV-TR Axis I Disorders

SEM: structural equation modeling

TLI: Tucker-Lewis index

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Introduction

Major depressive disorder (MDD) is widespread, with far-reaching – and often devastating – consequences. Lifetime prevalence of MDD has been estimated at 16% (Kessler et al., 2003), and its annual economic impact in the United States alone has been estimated at $83.1 billion (Greenberg et al., 2003). MDD is characterized by low mood and/or decreased sensitivity to rewarding experiences, as well as a number of other symptoms (American Psychiatric Association, 2000) that can be disruptive to sufferers' quality of life and ability to interact effectively with their environment. In addition to these immediate psychological impacts, MDD is associated with a range of negative outcomes in other domains of life including poor academic performance (Birmaher et al., 1996; Heiligenstein, Guenther, Hsu, & Herman, 1996), poor health habits (Katon, 2003), high morbidity and mortality associated with chronic medical conditions (Katon, 2003), interpersonal difficulties (Joiner & Timmons, 2010), substance abuse and dependence (Grant, 1995; Regier et al., 1990), unemployment (Whooley et al., 2002), and an increased risk of suicide (Harris & Barraclough, 1997).

Although depression is less prevalent before adulthood, it represents a problem in a substantial minority of children. Lifetime prevalence of MDD in children has been estimated at rates of 10-13% by age 16 (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Merikangas et al., 2010), with incidence increasing for girls compared to boys at around the age of 14 (Wade, Cairney, & Pevalin, 2002; Wichstrøm, 1999). Similar to depression in adults, depression in children is associated a wide array of concurrent problems, including poor interpersonal relationships (La Greca & Harrison, 2005) and academic performance (Mesman & Koot, 2000). Childhood depression is also associated with an economic toll on parents, who must devote resources and take off time from work to care for them (Busch & Barry, 2007).

In addition to these immediate problems, depression during childhood and adolescence increases risk for negative outcomes later in life. These outcomes include a higher likelihood of unemployment (Fergusson & Woodward, 2002), alcohol abuse and dependence (Fergusson & Woodward, 2002), early parenthood (Fergusson & Woodward, 2002), and depression and suicide in adulthood (Fergusson & Woodward, 2002; Harrington et al., 1994). Even in children who do not meet the full criteria for MDD, subthreshold depressive symptomatology can have serious implications (Pine, Cohen, Cohen, & Brook, 1999): for each additional symptom of depression in girls between ages 8-10, the risk of a major or minor depressive disorder during early adolescence increases by 50-80% (Keenan, Feng, Hipwell, & Klostermann, 2009). Moreover, early-onset depression is particularly heritable (Kupfer, Frank, Carpenter, & Neiswanger, 1989; Mendlewicz & Baron, 1981; Weissman et al., 1984) and is particularly persistent (Fombonne, Wostear, Cooper, Harrington, & Rutter, 2001) and pernicious (Gollan, Raffety, Gortner, & Dobson, 2005). Depression that begins before the age of 20 is associated with a greater number of symptoms and higher relapse than later-onset depression (Gollan et al., 2005).

Given the high prevalence of depression, its proximal and distal consequences, and the vast array of negative long-term outcomes with which it is associated, there is a clear need for effective treatments. Antidepressant medications are commonly prescribed, but these have small effect sizes in all but the most severe cases of depression (Fournier et al., 2010; Kirsch et al., 2008). Behavioral interventions have also been shown to reduce depressive symptomatology to some extent (Weisz, McCarty, & Valeri, 2006; Westen & Morrison, 2001), but even the most effective interventions are far from perfect. Remission rates are far below 100%, and even when

interventions are effective in the short term, they tend to be associated with high relapse rates (Vittengl, Clark, Dunn, & Jarrett, 2007; Westen & Morrison, 2001).

Some childhood preventive interventions for depression have also been developed. One meta-analysis of school-based social and emotional learning programs found that such programs had an effect size of .24 on symptoms of emotional distress; however, this effect size was reduced to .15 at follow-ups a mean of 52 weeks later (Durlak, Weissberg, Dymnicki, Taylor, & Schellinger, 2011). Other meta-analyses of prevention programs for depression in children and adolescents have found mean effect sizes of .15-.16, with programs targeted at higher-risk children showing greater efficacy (Horowitz & Garber, 2006; Stice, Shaw, Bohon, Marti, & Rohde, 2009). Another recent meta-analysis of preventive interventions for depression across several age groups found that the interventions reduced the incidence of depression by 22% (Cuijpers, van Straten, Smit, Mihalopoulos, & Beekman, 2008). However, the number of children who need to be treated in order to see a difference from control groups is relatively high, even when the interventions are targeted at high-risk individuals (Cuijpers et al., 2008). Taken together, these studies suggest that treatments and preventive interventions for depression are somewhat efficacious, and that they are more helpful when they are targeted, but that there remains a great deal of room for improvement.

One means by which this might be accomplished is by identifying pathophysiological processes that are associated either with the maintenance of depressive symptoms or a predisposition toward depression; these may provide clues for new avenues of intervention. Neural biomarkers – i.e., measures of core neural processes that relate to psychopathology (Luck et al., 2011) – are a particularly promising means of assessing both mechanisms and risk factors for depression. Research initiatives such as the Research Domain Criteria (RDoC) (Insel et al.,

2010; Sanislow et al., 2010) have encouraged the use of such measures, emphasizing that dysfunction in core neural systems is more likely to capture underlying processes that characterize and predict the onset and course of mental disorders than standard measures based on the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 2000). From this point of view, rather than relying on common phenotypes that may have heterogeneous etiologies or mechanisms, it might be possible to begin at the level of neural mechanisms and create more useful clinical classifiers based on these measures. Identifying reliable neural biomarkers would also shed light on the etiopathogenesis of depression, potentially paving the way for novel treatments.

A large body of evidence suggests that a possible biomarker for current and future depression is an impaired neural sensitivity to rewards. Depression has been linked to decreased interest in rewarding experiences (Kasch, Rottenberg, Arnow, & Gotlib, 2002), decreased positive emotional response to the receipt of monetary rewards (McFarland & Klein, 2009), and a decreased tendency to adjust behavior to attain maximal rewards in both adults (Henriques & Davidson, 2000; Henriques, Glowacki, & Davidson, 1994; Pizzagalli, Iosifescu, Hallett, Ratner, & Fava, 2008) and children (Forbes, Shaw, & Dahl, 2007). Depression is also associated with abnormal activity in mesocorticolimbic circuits (Nestler & Carlezon, 2006; Pizzagalli et al., 2009; Steele, Kumar, & Ebmeier, 2007) – brain areas central to reward processing. In response to a monetary reward-based decision task, for instance, 9- to 17-year-olds with MDD show reduced activity in reward-related brain areas (Forbes et al., 2006). Likewise, increased depressive symptoms in healthy 12-year-olds is associated with reduced striatal response to monetary rewards (Forbes et al., 2010), and 8- to 16-year-olds with MDD show reduced striatal activation during reward anticipation after winning money on a previous trial (Olino et al.,

2011). Reward sensitivity also appears to relate to risk for depression in adolescents: never-depressed adolescent girls with depressed mothers have decreased activity in the left putamen and the left insula during anticipation of point-based rewards (Gotlib et al., 2010). Indeed, anhedonia – a decreased ability to enjoy rewarding stimuli – is a key feature of MDD (American Psychiatric Association, 2000) and has been suggested as a possible endophenotype of depression based on its apparent heritability, stability over time, and tendency to precede other depressive symptoms (Hasler, Drevets, Manji, & Charney, 2004).

In the sections that follow, I will provide an explanation of the theoretical framework linking depression to impaired sensitivity to rewards; I will then review the existing evidence that supports this framework. Additionally, I will review the evidence for a common neural representation of monetary and social rewards, particularly during adolescence, which suggests that it may be possible to evaluate the neural response to social rewards using a similar approach to the established methods for evaluating the response to monetary rewards. I will also discuss the importance of pubertal considerations when assessing relationships between depression and reward sensitivity. Finally, I will discuss the aims of the current study in the context of this collected evidence.

Reward Sensitivity and Depression: Theoretical Framework

Impaired reward sensitivity may represent an intermediary step in a causal chain between familial risk and the development of depressive symptoms. Parental – and particularly maternal (Brennan, Hammen, Katz, & Le Brocque, 2002; Klein, Lewinsohn, Rohde, Seeley, & Olino, 2005) – history of depression is one of the strongest predictors of depression in offspring (Beardslee, Versage, & Gladstone, 1998); by age 15, the incidence of depression is twice as high in adolescents with depressed mothers as in those with never-depressed mothers (Hammen &

Brennan, 2003). The transmission of depression from parent to child has been attributed to several different factors, including parenting style and depression-related changes in neuroendocrine levels or blood flow in the intrauterine environment during pregnancy (Connell & Goodman, 2002; Field, Hossain, & Malphurs, 1999; Goodman & Gotlib, 1999). Heredity also plays a substantial role: 37% of observed variance in depression has been attributed to genetic factors (Sullivan, 2000).

Theorists have suggested that a genetic predisposition leads certain children to develop hypohedonia, a decreased responsiveness to reward that is instantiated in dopaminergic neural systems (Hamburg, 1998; Meehl, 1975). In healthy individuals, actions that are rewarded are reinforced, and the individual becomes more likely to repeat the action. Even actions that are rewarded intermittently – as many actions are in daily life – are reinforced and, in fact, are more resistant to extinction than consistently rewarded behaviors (Skinner, 1953). When there is no reward value associated with an outcome, however, there is no reinforcement and therefore no motivation to repeat the behavior. Hypohedonic children may therefore respond to intermittently rewarded experiences as if they were extinction schedules (Hamburg, 1998; Meehl, 1975). Hamburg (1998) gives the example of a child learning a new musical piece on the piano: a typical child would be reinforced by the experience of hitting occasional correct notes, whereas a hypohedonic child would have little response to such successes and would interpret the same experience as a series of failures. For this child, there would therefore be little motivation to continue playing the piano.

As hypohedonic children continue to accumulate unrewarding experiences like this over time, they are thought to develop a depressogenic cognitive style in which they attribute negative life events to internal, stable, and global causes (Hamburg, 1998) – the triad of attributions

posited by the learned helplessness (Abramson, Seligman, & Teasdale, 1978; Maier & Seligman, 1976; Seligman, 1975) and hopelessness (Abramson, Metalsky, & Alloy, 1989) theories to increase vulnerability to depression. It is thought that when people use this type of internal, stable, and global attributions, they begin to see a disconnect between actions and their outcomes. That is, an individual failure to achieve a goal is interpreted as being caused by some failure in the character of the individual; that failure is seen as being unchangeable over time, and it is also seen as being just one of many character faults. This leads to a belief that one's actions will rarely lead to one's desired outcomes, and that one is powerless to change this pattern. Such thinking is thought to lead to a sense of helplessness and a subsequent experience of depression.

Peterson and Seligman (1984) attribute the development of a depressogenic cognitive style – in some cases – to an early experience of negative life events. However, Hamburg (1998) argues that early adversity is not a sufficient condition for the development of the attributional triad because it sets an unrealistically high threshold for the severity and global nature of the life events. In support of this idea, Hamburg points out people's tendency towards resilience after tragedies, their ability to differentiate between situations that can be controlled and those that cannot, and the tendency for helplessness to decrease over time. He cites these as evidence that negative life events alone – even when traumatic – are not enough to lead most people to the conclusion that they are helpless. Taking this line of thought to its conclusion, Hamburg remarks that such experiences should lead to a depressogenic cognitive style "only in hideously exceptional cases" (Hamburg, 1998) – which is clearly not the case for the majority of individuals with depression. Instead, he posits that negative life experiences of a less severe

nature can be sufficient to trigger depressogenic thinking – but only provided that the individual finds positive experiences less reinforcing than others would.

Under this conceptualization, hypohedonia is a necessary backdrop to the depressogenic cognitive style that leads to a sense of helplessness. It decreases the reward value of reinforcers such that positive life experiences are seen as less positive, events are therefore more universally negative, and actions are rarely associated with desired outcomes. Once the depression has been established, the individual continues to experience events as unrewarding and actions as useless, and the depression is perpetuated. If this line of thinking is correct, then blunted sensitivity to reward should act both to maintain ongoing symptomatology and to increase risk in those who have not yet developed depressive symptoms.

Evidence for Hypohedonia in Depression

In adults, depressive symptoms have been associated with measures of hypohedonia – i.e., decreased sensitivity to reward – across a number of domains including self-report, behavior, and neural activity. Adults with MDD endorse less reward responsiveness (Kasch et al., 2002) and score lower on other measures of positive affectivity (Mineka, Watson, & Clark, 1998) than non-depressed adults. Depression in adults is also associated with a decreased tendency to adjust behavior to attain maximal rewards; women with dysphoria show a more conservative behavioral response bias toward potentially rewarding stimuli than those without dysphoria (Henriques et al., 1994), and adults with MDD do not show the same reward-maximizing behavior in a verbal memory tasks as healthy adults do (Henriques & Davidson, 2000).

Moreover, depression is associated with abnormal activity in mesocorticolimbic circuits – brain areas central to reward processing (Nestler & Carlezon, 2006). In a monetary incentive delay task, adults with MDD show reduced blood-oxygen-level dependent (BOLD) activation of

the caudate and the left nucleus accumbens in response to rewarding outcomes as compared to a group of healthy controls (Pizzagalli et al., 2009); these results are specific to rewards and not to losses or neutral feedback. Similarly, in an fMRI study using a gambling task, adults with clinical depression do not show the same activation of the ventral striatum to rewarding outcomes that healthy controls do (Foti, Carlson, Sauder, & Proudfit, 2014; Steele et al., 2007).

A number of studies in children and adolescents have found a similar relationship between depression and reduced sensitivity to reward, also across multiple domains. In a sample of 11-year-old boys, those with recent depression did not alter their decision-making behavior to differentiate between high-probability rewards of small or large magnitudes; this was in contrast to their healthy peers, who were more likely to choose the larger rewards (Forbes et al., 2007). In response to a monetary reward-based decision task, 9- to 17-year-olds with MDD show reduced activity in reward-related brain areas including the caudate compared to a healthy control group (Forbes et al., 2006). Likewise, increased depressive symptoms in unselected 12-year-olds is associated with reduced striatal response to monetary rewards (Forbes et al., 2010).

Reduced reward sensitivity also appears to relate to *risk* for depression over the course of development: Olino and colleagues found that healthy 8- to 17-year-olds at high familial risk for depression had reduced striatal response during anticipation of monetary reward compared to those at low risk (Olino et al., 2014), and Gotlib and colleagues found that never-depressed adolescent girls with familial risk for depression had decreased activity in the left putamen and the left insula during anticipation of point-based rewards (Gotlib et al., 2010). Additionally, adolescents in mid- to late- puberty show an association between reduced striatal activity during reward anticipation and an increase in depressive symptoms over the following two years (Morgan, Olino, McMakin, Ryan, & Forbes, 2013).

Social Reward During Adolescence

Despite numerous findings relating depression to reduced neural response to reward, little emphasis has been placed on determining the impact of different *types* of reward; in fact, this has been highlighted as an area in need of further research (Forbes & Dahl, 2012; Forbes, 2009; Morgan et al., 2013). Studies of neural reward sensitivity have often used feedback indicating monetary loss and gain (Bress, Foti, Kotov, Klein, & Hajcak, 2013; Bress, Smith, Foti, Klein, & Hajcak, 2012; Forbes et al., 2006, 2007; Foti & Hajcak, 2009; Foti, Weinberg, Dien, & Hajcak, 2011; Gotlib et al., 2010; Hajcak, Moser, Holroyd, & Simons, 2007). Reward in other domains has been neglected – in particular, the domain of *social relationships*, which becomes increasingly important during adolescence.

During late childhood and early adolescence, individuals begin to place progressively greater emphasis on social relationships with peers, becoming less invested in the parental relationships that played a substantial role during earlier childhood (Steinberg & Morris, 2001). Although fourth-graders report that their social support is provided primarily by their parents, seventh-graders rank social support from same-sex friends equally as highly as parental support; by the time children reach tenth grade, they rank peer support more highly (Furman & Buhrmester, 1992). Adolescents also spend four times as much time talking to peers as they do talking to parents, and they rate their social relationships as making them most happy (Csikszentmihalyi, Larson, & Prescott, 1977). The growing importance of social relationships is particularly pronounced in girls: girls in third through sixth grades rate their peer friendships as more reciprocal and more positive than boys do, and their social relationships also suffer more than boys' when they become depressed early in adolescence (Rudolph, Ladd, & Dinella, 2007).

Depression has been described as a protective state that evolved to reduce social risk at times when social acceptance is perceived to be low. According to the social risk hypothesis (Allen & Badcock, 2003), transient and mild depressive symptoms are common, and are therefore likely to serve an adaptive evolutionary purpose. Social relationships and status are seen as increasing access to resources, which increases reproductive fitness. When social status is threatened, it becomes imperative to increase the ratio of one's perceived value within the social context with respect to one's perceived burden. The social risk hypothesis posits that this is accomplished through the development of a depressed mood, which promotes social-risk-reducing cognitions and behaviors, including a reduction in the perceived value of positive social feedback (Allen & Badcock, 2003).

Social factors are thought to play an especially important role in the development of depression during adolescence. Research has focused on the contribution of relational victimization during adolescence – which includes behaviors such as friendship withdrawal and social exclusion – to depressive symptoms, and relational victimization has been found to contribute to depressive symptoms even when controlling for other aspects of the social life such as social group membership and dating status (La Greca & Harrison, 2005). Consistent with these findings, adolescents with MDD also show increased neural reactivity to peer rejection feedback compared to healthy peers (Silk et al., 2013).

Indeed, one of the three etiological pathways to depression modeled by Kendler and colleagues (2002) relates to a history of interpersonal difficulties; Hamburg (1998) also suggests that, because social situations provide relatively few objective cues to guide learning, they may be particularly non-reinforcing to individuals with pre-existing hypohedonia. The increased importance placed on social relationships is more pronounced for girls than for boys (Furman &

Buhrmester, 1992); in line with this phenomenon, brain activity in response to social acceptance versus rejection increases in girls as they become older (Guyer, McClure-Tone, Shiffrin, Pine, & Nelson, 2009). The increased importance of social affiliation and acceptance has been hypothesized to contribute to the gender disparity in depression rates that begins in adolescence (Cyranowski, Frank, Young, & Shear, 2000; Wichstrøm, 1999).

A small number of existing studies provide evidence that there may also be a connection between *risk* for depression and socioemotional processing. In one longitudinal study of 13-year-olds, it was found that girls whose mothers had experienced depression during the postnatal period showed a higher degree of emotional sensitivity (Murray, Halligan, Adams, Patterson, & Goodyer, 2006). Two additional studies have found that girls with a maternal history of depression attend selectively to negatively valenced emotional faces, whereas their peers do not (Joormann, Talbot, & Gotlib, 2007; A. J. Kujawa et al., 2011). Similarly, a group of 10- to 18-year-old offspring of parents with a lifetime history of MDD showed decreased amygdala and nucleus accumbens activation to images of happy faces when compared to a control group (Monk et al., 2008). Another recent study found that adolescents whose mothers showed more negative affect during a discussion task had less activation in the left nucleus accumbens, amongst other areas, in response to acceptance feedback in a chatroom task (Tan et al., 2014). Although these studies have found a connection between familial risk for depression and differences in emotion processing, to our knowledge no studies have examined the relationship between maternal depression diagnosis and response to social reward. Thus, the neural response to social reward may represent an important, but understudied, biomarker for depressive symptoms and risk in adolescent girls.

Evidence from the ERP Literature

Studies from the ERP literature have frequently measured the neural response to monetary outcomes using the feedback negativity (FN), an apparent negative deflection in the ERP waveform that is apparent approximately 300 ms after winning or losing money (Gehring & Willoughby, 2002; Hajcak et al., 2007; Yeung & Sanfey, 2004). Consistent with studies in other modalities, one ERP study in adults found that depressive symptoms were correlated with a smaller differentiation between the FN to monetary gains and losses (i.e., the ΔFN) (Foti & Hajcak, 2009). Two other recent studies have found an association between FN amplitude and MDD diagnosis. Liu and colleagues reported that individuals with MDD showed less differentiation between the FN to monetary losses and gains than a control group, and that this effect was driven specifically by a decreased FN to gains (Liu et al., 2014). Foti and colleagues likewise reported a reduced ΔFN in participants with MDD, and they additionally found that ΔFN amplitude was associated with striatal activation in depressed individuals (Foti et al., 2014). Similar results have been found in samples of children: a smaller ΔFN was related to individual differences in depressive symptoms in an unselected sample of children between the ages of 8 and 13 (Bress et al., 2012), and this relationship between the FN and concurrent depressive symptoms remained apparent two years later (Bress, Meyer, & Proudfit, 2015).

In addition to its association with concurrent depression, the FN also predicts the onset of *subsequent* depressive episodes: we recently found that the onset of a first major depressive episode is preceded by a blunted FN in never-depressed adolescent girls (Bress et al., 2013). The size of this effect (Cohen's d = .50) was independent and similar to the effect of baseline depressive symptoms (Cohen's d = .59) – an established risk factor for subsequent depression (Pine et al., 1999). These findings have since been replicated in a larger sample of adolescents

(B. D. Nelson, Perlman, Klein, Kotov, & Hajcak, in press). Another recent study found that the FN is also reduced in the 9-year-old offspring of mothers who had a history of depression, and that the severity of the mother's depression relates to the degree of reduction of the child's FN (A. J. Kujawa, Proudfit, & Klein, 2014). The FN therefore represents a strong candidate biomarker of current symptomatology and risk for depression in children and adolescents. However, no studies have examined the response to *social* rewards with respect to maternal depression diagnosis. For the current study, I aimed to examine and compare associations between maternal risk and the FN in response to monetary and social rewards.

Relatively few studies have used ERPs to study the response to social rewards. Many existing studies use the Cyberball task (Williams & Jarvis, 2006), in which a participant plays a virtual game of catch with two other "players" – actually generated by the computer. After a few rounds, the other "players" begin to exclude the participant from the game, and the virtual ball is not thrown to the participant for the remainder of the task. ERP studies using the Cyberball task have produced mixed findings, often reporting weak or absent effects relating to the FN (Crowley, Wu, Molfese, & Mayes, 2010; Kawamoto, Nittono, & Ura, 2010; Themanson, Khatcherian, Ball, & Rosen, 2013).

However, the Cyberball task was originally designed to elicit feelings of exclusion over time, and acceptance and rejection events are not equally probable throughout the game; this makes it suboptimal for eliciting ERPs, which are strongly modulated by probability. Moreover, because there is no explicit goal to the game, some participants may not find it engaging; the "players" also have very little information about each other, and therefore an acceptance or rejection may feel somewhat arbitrary and not personally relevant to the participant. Finally,

because the task is designed to create a sense of exclusion over time, and individual ball-toss that excludes the participant may not be interpreted immediately as a rejection.

Our novel Kinder task, described in the Methods section, was specifically designed to assess neural responses to social acceptance and rejection. Acceptance and rejection were equally probably throughout the task. Moreover, the task itself was designed to be engaging, and participants were told that the other players would see their personalized profile, making acceptance and rejection by other players more personally relevant. Finally, the interface was designed carefully to appear as realistic as possible.

Monetary and Social Reward: Shared Neural Substrates

Although evidence from the ERP literature relating to social reward is minimal, some fMRI studies suggest that social and monetary reward are processed by overlapping reward networks in the brain. In adults, information about both social and monetary rewards has been associated with activity in mesolimbic regions. Adults given feedback about having a good reputation show robust activation in the striatum, an area also activated in response to monetary gains (Izuma, Saito, & Sadato, 2008); Izuma and colleagues have referred to this shared neural substrate as the "common neural currency" of reward. These results are corroborated by findings from other fMRI studies, which have found that the nucleus accumbens is activated in response to feedback from both monetary incentive delay and social incentive delay tasks (Spreckelmeyer et al., 2009) and that the ventral tegmental area and parts of the striatum are activated in response to both monetary rewards and decisions to give up money for the benefit of another person (Moll et al., 2006).

Similar to adults, adolescents show common activation in striatal areas in response to both monetary and social rewards (Guyer, Choate, Pine, & Nelson, 2012; Guyer et al., 2009).

Davey and colleagues found that a group of 15- to 24-year-olds who received accepting feedback from purported peers showed activation in a variety of brain areas, including the nucleus accumbens, as well as areas specific to social processing (Davey, Allen, Harrison, Dwyer, & Yücel, 2010). In another study, adolescents between the ages of 9 and 17 showed activation in numerous areas, including the striatum, in response to positive social feedback from desirable peers (Guyer et al., 2012). Collectively, these studies suggest that similarly to adults, adolescents show neural reactivity to social rewards that overlaps with the reactivity to monetary rewards, supporting the idea of the "common neural currency" from Izuma and colleagues.

Given the overlap in neural correlates of monetary and social reward processing, it was hypothesized that social reward would elicit an FN similar to the one elicited by monetary reward, making it possible to study both social and monetary reward using ERP methodology.

Pubertal Considerations

In assessing relationships between reward responsiveness and depression during childhood and adolescence, it is necessary to consider the influence of pubertal maturation; adolescents' behavioral sensitivity to rewards peaks during mid-adolescence as compared to childhood and to later adolescence and adulthood (Crone & Dahl, 2012). During adolescence, the balance of gonadal hormones shifts dramatically: levels of gonadotropin-releasing hormone (GnRH) increase, triggering increased follicle-stimulating hormone and luteinizing hormone, which then increase levels of either testosterone or estradiol (Spear, 2000). Testosterone has been found to increase sensitivity to rewards (Bos, Panksepp, Bluthé, & van Honk, 2012); a study of adult women, for instance, found that those who had been administered a dose of testosterone showed increased striatal response to monetary rewards during a monetary incentive delay task (Hermans et al., 2010). Moreover, levels of circulating testosterone and estradiol – correlates of

pubertal development – relate to the striatal response to monetary reward (Forbes et al., 2010; Op de Macks et al., 2011).

Changes in testosterone during puberty may also reorient the adolescent brain to social rewards (Crone & Dahl, 2012). Testosterone and estradiol are involved in the release and binding of neuropeptides related to social bonding (Peter et al., 1990; Quiñones-Jenab et al., 1997), and animals incapable of metabolizing these hormones show deficits in social recognition (Bos et al., 2012). Increases in testosterone are also associated with approach behavior in threatening social situations, including anticipation and winning of social competitions (Eisenegger, Haushofer, & Fehr, 2011). Crone and Dahl (2012) describe testosterone as a hormone that "alters the appraisal of threats and rewards – particularly when these are relevant to social status" (p. 647).

Laurence Steinberg (Steinberg, 2008) hypothesizes that the increase in risk-taking observed during puberty is due to changes in the dopaminergic network – specifically, alterations in the relative density of dopamine receptors in cortical and subcortical brain areas, which may in turn lead to an increase in the salience of rewards – and that the overlap in neural systems that encode monetary and social rewards may explain the increase in risk-taking within a social setting. A complementary view is held by Crone and Dahl (2012), who explain the changes in dopamine receptor density during puberty as related to increasing levels of testosterone during development. Some have cautioned against assuming that the relationship between pubertal hormones and neural remodeling is a directly causal one; but the two do appear to have, at minimum, a correlational relationship (Spear, 2000; Steinberg, 2008).

Rates of depression also increase sharply in girls during mid-puberty (Angold, Costello, & Worthman, 1998; P. Cohen et al., 1993), which has been attributed to a combination of vulnerability factors that occur in conjunction with an increased number of negative life events

for girls during this time (P. Cohen et al., 1993; Hyde, Mezulis, & Abramson, 2008). Pubertal maturation is a stronger predictor than age of sex differences in depression (Angold et al., 1998), and gonadal hormones have been linked to depressive symptoms, possibly due to the sensitizing effects of these hormones on neurotransmitter systems (Steiner, 2003). Recent conceptualizations have attributed some of the increase in depression during adolescence to pubertal changes in reward-related neural processes (Paus, Keshavan, & Giedd, 2008) or in emotional processing of social stimuli (E. E. Nelson, Leibenluft, McClure, & Pine, 2005). Additionally, adolescents with abnormal neural responses to social cues may be at greater risk of developing weaker interpersonal relationships than their peers, which may in turn act as a diathesis to the stressors of adolescent life and increase the risk for developing depression (E. E. Nelson et al., 2005).

Existing evidence suggests that the monetary FN may change over the course of development. In a study of the monetary FN over the life span, Hämmerer and colleagues found that the ERP response to monetary gains and losses become progressively more positive from childhood to adulthood (Hämmerer, Li, Müller, & Lindenberger, 2010); similarly, a recent study from our lab found that the ERP response to monetary gain becomes larger with advancing age during adolescence (Meyer, Bress, & Hajcak, unpublished data). This is consistent with findings from the puberty literature that point to increased behavioral reward-seeking (Steinberg, 2008) and striatal response to reward (Crone & Dahl, 2012; Van Leijenhorst et al., 2010) in adolescence. Striatal activation during a gambling task has also been found to peak during mid-adolescence, with decreased activation earlier and later in development (Van Leijenhorst et al., 2010). However, the influence of puberty has not been assessed with respect to the social FN. In order to account for variance that may be explained by differences in maturation, pubertal

development was measured within the proposed study using multiple measures, including automated interviews, questionnaires, and hormonal assays.

Research Aims and Hypotheses

Although it is clear that reward sensitivity relates to depression, and that social rewards are increasingly important as girls progress through adolescence, it remains unclear whether social reward relates differently than monetary reward to depressive symptoms or to known markers of risk. The proposed study aimed to examine a novel ERP response – the social FN – in relation to depressive symptoms and maternal depression, and to examine how this response changes across development. Because the importance of social feedback increases during puberty, and because reward sensitivity is tied to depression, it was hypothesized that the response to social reward would relate uniquely to measures of puberty, depression, and risk when accounting for the response to monetary reward. If these hypotheses were supported, this research would provide a basis for future longitudinal studies to determine whether the social FN prospectively predicts increased symptoms and subsequent diagnoses of depression. In the current study, I addressed four primary research aims:

1. **Compare the social FN and the monetary FN.** Although a growing body of evidence suggests that the response to monetary and social rewards share certain common neural underpinnings, very little research has been devoted to investigating commonalities between the two using ERP methodology. One aim of the proposed research was to determine whether an ecologically valid social reward task would produce a neural response to social reward (i.e., an FN) – and if so, to relate the social FN to the neural response to monetary reward (i.e., the monetary FN). I hypothesized that, although both social and monetary feedback would elicit an FN, they would share only a moderate degree of variance.

2. **Assess relationships between puberty and social reward sensitivity. S**tudies of social development suggest that social reward becomes more highly valued over the course of puberty. I hypothesized that the neural response to social outcomes would increase with pubertal development, and the social FN would be more strongly tied to pubertal development than the monetary FN.
3. **Assess relationships between depression and social reward sensitivity.** Depression has been linked to a reduced monetary FN (Bress et al., 2012) and a tendency to underestimate peer acceptance (Zimmer–Gembeck, Hunter, & Pronk, 2007). However, the relationship between the neural response to social reward and depressive symptoms has rarely been explored. Therefore, a third aim of the proposed research was to relate the neural response to social rewards to current depressive symptoms. Based on the existing evidence, I hypothesized that increased depressive symptoms would be associated with a blunted neural response to both social and monetary rewards. Moreover, I hypothesized that the social FN would predict additive variance in current depressive symptoms after controlling for the monetary FN. Both of these hypotheses were examined considering depressive symptoms continuously and dichotomously (i.e., comparing non-depressed participants to participants with threshold or subthreshold depression).
4. **Assess associations with risk.** A final aim of the proposed research was to examine the relationship between the neural response to social outcomes and risk for depression based on maternal history of depression. Maternal depression is a well-established risk factor (Beardslee et al., 1998; Brennan et al., 2002; Klein et al., 2005), and causal theories suggest that offspring of depressed mothers should be predisposed to low reward sensitivity even before the onset of a depressive episode (Hamburg, 1998; Meehl, 1975). Therefore, I

hypothesized that a maternal history of depression would be associated with a smaller social FN; that the relationship between the social FN and maternal depression would remain in the absence of a diagnosis in the offspring, and when controlling for subclinical symptoms in offspring; and that the relationship between the social FN and maternal depression would be stronger than the relationship between the monetary FN and maternal depression. An additional part of this aim was to begin teasing out the extent to which the effects of maternal depression are genetic or environmental. If, as hypothesized, the relationship between the social FN and depression in the child is influenced by a genetic predisposition toward hypohedonia, then this effect was expected to be stronger in children whose mothers had been depressed than in those whose mothers have not, even if the mother's depression resolved before the child was born.

Methods

Participants

Participants were 213 8- to 16-year-old girls and their parents, recruited from the community surrounding Stony Brook University as part of a larger study investigating depression and reward sensitivity during adolescence. Participants for this study were recruited using a commercial mailing list of families in the area with daughters in the specified age range. An initial letter describing the study was sent to each family, and a follow-up phone call was made a week later. Some participants were additionally recruited through online advertisements, flyers posted in doctors' offices and other community settings, and word-of-mouth from other participants. Each family that expressed interest was given a short phone screen by a trained undergraduate research assistant. If the daughter was within the target age range, was free of neurological illness and traumatic brain injury, lived with at least one biological parent, spoke English and had an English-speaking parent, and did not have metal in her body that could not be removed (e.g., orthodontic braces), she and her parent were invited to participate in the study.

Thirteen participants were excluded from analyses due to low-quality EEG data, and an additional four were excluded because they had an FN more than three standard deviations from the mean. Therefore, the final sample consisted of 196 participants and their parent. As the final sample included a number of participants accompanied by their fathers rather than their mothers (n = 23), these participants were excluded from analyses of maternal risk.

Clinical Interviews

Child psychopathology. In order to assess MDD and other diagnoses in child participants, the Kiddie Schedule for Affective Disorders and Schizophrenia – Present and Lifetime Version (K-SADS-PL; Kaufman et al., 1997) was administered to children and their

parent. The K-SADS-PL is a semi-structured interview designed to assess both past and current diagnoses in children and adolescents across a number of categories – including mood and anxiety disorders – according to *DSM-IV-TR* (American Psychiatric Association, 2000) criteria. The K-SADS-PL has shown high inter-rater reliability in assessing both current and lifetime diagnoses and good to excellent test-retest reliability (κ = .78 to .90 for mood and anxiety disorders, and .63 to .74 for ADHD and oppositional defiant disorder) over a period of two to 38 days (Kaufman et al., 1997).

Maternal psychopathology. In order to assess MDD and other depressive diagnoses in mothers, the Structured Clinical Interview for the *DSM-IV-TR* Axis I Disorders, Research Version, Non-Patient Edition (SCID-I; First, Spitzer, Gibbon, & Williams, 2002) was administered to the mother of each child participant. Like the K-SADS-PL, the SCID-I is a semi-structured interview designed to assess both past and current diagnostic categories – also including mood and anxiety disorders – according to *DSM-IV-TR* criteria; however, this interview is specifically targeted toward adults. The SCID-I has been used across many studies and is demonstrated to have moderate to high inter-rater reliability (κ = .57 to 1.0 depending on the diagnosis) (Lobbestael, Leurgans, & Arntz, 2011; Zanarini et al., 2000).

In order to maximize consistency of administration, the same interviewer administered both the K-SADS-PL and the SCID-I. Case conference meetings were held regularly to reach consensus diagnoses and resolve discrepancies between child and parent reports. Meetings included all of the clinical interviewers and Dr. Greg Hajcak, who provided feedback to the interviewers and aided in consensus diagnoses.

Clinical Questionnaires

Child depression. Children completed a self-report version of the Children's Depression Inventory (CDI:SR; Kovacs, 2003), a 28-item questionnaire assessing core symptoms of depression over the previous two weeks. Items are rated on a scale from 0 (e.g., "I am sad once in a while") to 2 (e.g., "I am sad all the time"). Mothers completed a parent version of the Children's Depression Inventory (CDI:P; Kovacs, 2003). This measure consists of 17 items similar to those included in the CDI:SR (e.g., "My child looks sad"); however, these are rated on a scale from 0 ("not at all") to 3 ("much or most of the time"). Internal reliability of the CDI is moderate; most psychometric studies report alpha coefficients of .80 or higher (Sitarenios & Kovacs, 1999).

Children also completed the short form of the Patient-Reported Outcomes Measurement Information System (PROMIS) Depression scale, self-report version (PROMIS:SR; Pilkonis et al., 2011). The PROMIS:SR is an 8-item depression scale that evaluates core symptoms of depression (e.g., "I felt unhappy") over the previous 7 days. Items are rated on a scale from 0 ("never") to 4 ("almost always"). Parents completed a parent version of the PROMIS (PROMIS:P), which consists of 6 items similar in content and structure to those in the PROMIS:SR. As for the PROMIS:SR, items are rated on a scale from 0 ("never") to 4 ("almost always").

Pubertal Assessment

Children and their parents completed self- and parent-report versions of the Pubertal Development Scale (PDS:SR and PDS:P, respectively; Petersen, Crockett, Richards, & Boxer, 1988). The PDS is a 5-item questionnaire assessing physical development in terms of growth spurt, body hair, changes in skin, breast development, and menstruation. Menstruation is rated

dichotomously as either 1 (“no”) or 4 (“yes”); all other items are scored on a scale from 1 (“not yet started”) to 4 (“seems complete”). In fifth- and sixth-grade girls, child and parent ratings on the PDS tend to correlate highly (*r*s from .71 to .80), and internal consistency of both the self-report and parent-report versions is moderate to high (Cronbach’s α of .67 to .78; Carskadon & Acebo, 1993).

A subset of children and their parents also completed the Picture-Based Interview about Puberty (PBIP:SR and PBIP:P, respectively; Dorn & Sussman, 2002). The PBIP is a two-item measure assessing physical development in terms of pubic hair growth and breast development. Items are scored from 1 to 5, and each rating is anchored by a picture of the corresponding developmental stage, along with a verbal description. The PBIP correlates highly with the PDS (*rs* of .72 to .81) and a physical examination (*r*s of .75 to .88; Shirtcliff, Dahl, & Pollak, 2009). Although the PBIP was originally designed to be administered by an interviewer, the current study employed a version of the measure in the form of an automated slideshow with a corresponding audio narration, timed to correspond to the images on the screen. Before beginning the slideshow, participants were given paper rating forms and were asked to circle their response for each of the two items as they watched the slideshow. This format allowed the child participants and their parents to complete the measure separately, in private rooms, which was intended to increase participants’ comfort given the sensitive subject matter. Moreover, this format allowed for greater consistency across administrations than the standard interview. Administration of this measure was discontinued partway through the study due to its extremely high correlation with the PDS; therefore only a subset of participants (n = 103) in the current study completed the PBIP.

In addition to these self- and parent-report measures, levels of testosterone and estradiol were assessed using salivary assays. Passive drool samples were collected in polypropylene vials, which were stored in a deep freezer until being sent out for analysis by Salimetrics. To the extent possible, samples were collected at approximately the same time of day for each participant. Although hormone data was collected throughout the study, results were not yet available for 47 subjects at the time of the current analyses; therefore the current analyses included hormone data for 166 participants.

Reward Tasks

Monetary reward task. Children's neural response to monetary reward was assessed using the Doors task, a guessing paradigm that has been used in many prior studies to elicit reward-related neural activity (Dunning & Hajcak, 2007; Foti & Hajcak, 2010; Hajcak, Moser, Holroyd, & Simons, 2006; Hajcak et al., 2007). Participants were shown an image of two doors, side-by-side, on a computer screen and were instructed to choose whichever one they thought was correct by clicking the corresponding mouse button. They were told before the task began that a correct choice would result in a reward of $0.50, and an incorrect choice would result in a monetary loss of $0.25; they were further told that they would receive their earnings from this task at the end of the study visit.

The Doors task consisted of 60 trials divided into three blocks of 20. Each trial began with an image of the two doors, which remained onscreen until the participant made a choice (Figure 1). A fixation cross then appeared for 1000 ms and was followed by feedback lasting for 2000 ms: either a green upward-facing arrow indicating a gain of $0.50, or a red downward-facing arrow indicating a loss of $0.25. After the feedback screen, a fixation mark was presented for 1500 ms; the participant was then shown the message "click for next round," which remained

onscreen until the participant clicked a mouse button. Exactly half (i.e., 30) of the trials resulted in gains and half resulted in losses; outcomes were randomized throughout the task.

Social reward task. Children's neural response to social reward was assessed using the Kinder task, a novel paradigm developed for the purposes of the current study. This task was designed to resemble a social networking game. Before coming into the lab, participants were asked to bring a picture of themselves that they would be willing to show to peers. When they arrived, they were asked to set up a profile that included their name, their photograph, and a short description of themselves; participants were told that this profile would be shown to peers in other labs, who would vote on whether or not they would like to be friends with the participant. They were additionally told that that they would have the opportunity to see and vote on their peers' profiles. After completing their profiles, participants saw a message indicating that their profiles were being uploaded for peers to view.

After a break of approximately 30-60 minutes, participants completed the main portion of the Kinder task, in which they were shown the other players' profiles, were asked whether they would want to be friends with each of the other players, and then viewed the other players' feedback about them. Participants first saw the profile they had set up earlier, after which they viewed a screen indicating that the other players' responses and profiles were being downloaded. Participants were told that they would see the profiles of 80 other players, and that they would be given 40 "yes" votes and 40 "no" votes in total.

Each trial consisted of two phases: a voting phase (Figure 2) and a feedback phase (Figure 3). During the voting phase, participants first viewed a fixation cross for 500 ms. This was followed by another player's profile, which consisted of a central image of that player with a brief description at the bottom of the screen and the player's name at the top of the screen. The

photograph was first presented alone for 2000 ms, and the player's name and description were presented together with the photograph for an additional 5000 ms. A fixation cross appeared for 500 ms, followed by an image with the text "Yes or no?" written at the top, prompting the participant to vote. This text was accompanied by an image of a green upward-facing arrow under the word "yes" and a red downward-facing arrow under the word "no"; the number of remaining "yes" and "no" votes remaining was shown under their respective arrows (Figure 2). This image remained onscreen until participants made their selection by clicking the corresponding mouse button. If there were no remaining votes of one type, and the participant attempted to select that option, the count remained at 0 and the screen did not disappear until the participant chose the other option. Once the participant had voted, a rectangle appeared around the arrow corresponding to her vote for 2000 ms, followed by a fixation cross for 500 ms.

During the feedback phase of the trial, the participant viewed an image of the other player again for 3000 ms, with the caption, "[other player's name] voted…", followed by a fixation cross for 1000 ms and a feedback screen for 1000 ms. The feedback consisted of either an upward-facing green arrow, indicating that the other player had voted to be friends with the participant, or a downward-facing red arrow, indicating that the other player had voted not to be friends with the participant. This was followed by a fixation mark lasting 1500 ms, and a screen with text reading "Click for next round."

Although participants were led to believe that they were playing against peers in other labs, all of the other players' profiles were computer-generated; half (i.e., 40) of the profiles were male, and half were female. Photographs of children and adolescents who appeared to be between the ages of 8 and 14 were selected from databases of non-copyrighted images, and captions were written by research assistants who were informed of the target age range for the

task; these include lines such as “I looove field hockey and volly ball” and “I have a weird cat that hides from me”. Player names were selected from a list of the most common baby names in 2003 (BabyCenter LLC, 2014), approximately 11 years before the current study was conducted. For each administration of the task, name/photograph pairings were randomized within each gender, and profile captions were randomized across all profiles. The presentation of feedback was restricted such that half of the players rejected by the participant gave acceptance feedback and half gave rejection feedback; likewise, half the players accepted by the participant gave acceptance feedback and half gave rejection feedback. Feedback was randomized within these constraints.

After the initial 75 participants, the task was shortened from 80 trials to 40; this decision was made because it substantially reduced the duration of the task, which had previously lasted approximately 30 minutes. Additionally, it was anticipated that shortening the task might reduce participant fatigue during the task and could lead to greater overall task engagement, which could also maximize significant findings. Furthermore, reducing the duration of the Kinder task allowed for additional time in which to run other tasks, which was an important consideration for the parent study.

In the shortened version of the task, participants were allowed 20 “yes” votes and 20 “no” votes. Half (i.e., 10) of the players the participant voted to accept gave the participant acceptance feedback and half gave rejection feedback; likewise, half the players she voted to reject gave acceptance feedback and half gave rejection feedback.

Procedure

Whenever possible, all study procedures were conducted within a single session, which lasted approximately 4-5 hours. Occasionally a session took place in two parts, which were

scheduled as close together as possible. When the participant and her parent arrived for the study visit, a trained research assistant or the lab coordinator explained the study and gave them an opportunity to ask questions. Written informed consent was obtained from the parent, and written assent was obtained from the child participant.

Interviews, questionnaires, saliva sample collection, and ERP tasks were conducted in the context of other tasks and measures throughout the visit. The K-SADS-PL was always administered to the parent about the daughter before it was administered to the daughter herself. The Doors and Kinder tasks were administered in the context of other ERP tasks; the order of these tasks was randomized, and instructions were given before each task.

Participants' families were paid $20 per hour for their participation. Participants also won $7.50 for the Doors task and were given the chance to win up to an additional $25 during other tasks throughout the visit.

Psychophysiological Recording and Data Reduction

EEG was recorded from a custom 34-channel BioSemi electrode cap, including electrodes FCz and Iz, arranged according to the 10/20 system. Electrooculographic (EOG) activity was recorded from electrodes positioned approximately 1 cm from the outer corners of the eyes, and 1 cm above and below the right eye. EEG signals were converted at the electrode with a gain of one and digitized at a 24-bit resolution with a sampling rate of 1024 Hz. The data were filtered using a low-pass fifth order sinc filter with a half-power cutoff of 204.8 Hz. Electrodes were measured online with respect to a common mode sense electrode forming a monopolar channel.

EEG data were analyzed offline using BrainVision Analyzer (Brain Products, Munich, Germany). Each channel was re-referenced to the mean of the mastoid signals and band-pass

filtered using cutoffs of 0.1 and 30 Hz. Eyeblink artifacts were removed from the data using the procedure described by Gratton and colleagues (Gratton, Coles, & Donchin, 1983). Additional artifacts were removed using a semi-automated artifact rejection procedure with a maximum allowed voltage step of 50 μV/ms between sample points, maximum voltage difference of 300 μV in any 200-ms interval, and a minimum voltage of .50 μV in any 100-ms interval; remaining artifacts were identified by visual inspection and removed manually.

For both the Doors and Kinder tasks, the EEG data were segmented into epochs from -200 to 600 ms relative to feedback onset, and separate means were created for desirable feedback (i.e., monetary gain or social acceptance) and undesirable feedback (i.e., monetary loss or social rejection) in each task. For each task, the FN was scored at electrode FCz, where it is typically maximal. For the Doors task, the FN was quantified as the mean amplitude between 250 and 350 ms after feedback onset; for the Kinder task, it was quantified as the mean amplitude between 220 and 320 ms after feedback onset. These time windows were selected based on visual inspection of the ERP grand average. For both tasks, the ΔFN was calculated as the FN to undesirable feedback minus the FN to desirable feedback. The data were corrected using the 200-ms interval immediately preceding feedback onset as baseline.

Data Analysis

Relationships between responses to social and monetary reward. Similarities between the social and monetary FNs were assessed by conducting Pearson product-moment correlations between the two ERPs. Both the social and monetary FNs were then decomposed into their underlying components using temporal-spatial principal components analysis (PCA). PCA analyses were run using the ERP PCA Toolkit, version 2.53 beta (Dien, 2010) with parameters chosen according to published guidelines for conducting PCAs on ERP data (Dien &

Frishkoff, 2005; Dien, Khoe, & Mangun, 2007). For both the Kinder and Doors data sets, a set of factors reflecting temporal variance was acquired using a temporal PCA with Promax rotation. Data from all timepoints of each participant's averaged ERP waveform were used as variables, with condition (desirable/undesirable feedback), electrode site, and participant constituting separate observations. Twenty-four temporal factors were extracted from the Kinder data set based on the Scree plot (Cattell, 1966), and twenty-two were extracted from the Doors data set. Factor scores were generated for each combination of condition, electrode, and participant. A spatial PCA with Infomax rotation was then conducted using electrode sites as variables, with condition, participant, and temporal factor scores constituting separate observations; based on the Scree plots, four spatial factors were extracted for rotation from the Kinder data set, and three were extracted from the Doors data set. Temporospatial factors were scored using their peak value for each condition at the electrode at which they were maximal.

Because both ERPs showed similar FN-like morphology – i.e., PCA-derived factor combinations associated with a frontocentral positivity close to 300 ms after feedback onset – the relationship between the amplitudes of the social and monetary FN-like PCA factor combinations was assessed using Pearson product-moment correlations in order to assess the extent of their shared variance.

Associations with puberty. In order to assess pubertal effects, relationships among pubertal measures (i.e., age, PDS:P, PDS:SR, PBIP:P, PBIP:SR, estradiol, and testosterone) were measured using Pearson product-moment correlations. The pubertal measures were then modeled as observed indicators in an SEM measurement model using Mplus (version 7.11) (Muthén & Muthén, 2013). The PBIP was excluded from this model due to the strong correlation between the PDS and the PBIP, the similarity in methodology between the two measures, and the high

number of missing PBIP values due to its discontinuation partway through the study. A latent dimensional factor representing the common variance across the other measures of pubertal development was estimated using confirmatory factor analysis (CFA; Bollen, 1989; Loehlin, 2004) guided by exploratory factor analysis (EFA). Extraneous covariance related to data collection methods and the reporter of the information was extracted using correlated residuals when appropriate to reduce measurement error associated with the latent factor (Brown, 2006). Observed variables with non-normal distributions were accounted for with a robust maximum likelihood estimator (MLR; Muthén & Muthén, 2013).

Comparative fit index (CFI), Tucker-Lewis index (TLI), and root mean squared error of approximation (RMSEA) were used as measures of model fit. Published guidelines have suggested that CFI and TLI greater than or equal to 0.95, and RMSEA less than or equal to .06, represent good model fit (Hu & Bentler, 1999).

Using SPSS, the latent puberty factor was entered into a series of correlations with the PCA-derived social and monetary FNs. For the FN variables that showed significant correlations with puberty, the puberty factor was then regressed simultaneously on the social and monetary FN variables in order to test for unique associations between pubertal development and the response to social feedback.

Associations with depression in the child. The measures of depressive symptoms (i.e., PROMIS:P, PROMIS:SR, CDI:P, CDI:SR, and K-SADS-PL depression diagnosis) were used as indicators for a latent dimensional factor representing common depression-related variance. Similarly to the puberty factor, this factor was estimated in Mplus using CFA informed by EFA. Correlated residuals were used to extract method-related variance as appropriate, and the binary

K-SADS-PL variable – i.e., presence or absence of depression diagnosis – was treated as categorical.

Using SPSS, the latent depression factor was entered into a series of correlations with the PCA-derived social and monetary FNs. For the FN variables that showed significant correlations with depression, the depression factor was then regressed simultaneously on the social and monetary FN variables in order to test for unique associations between depression and the response to social feedback.

To examine the relationship between dichotomously classified depression and the social FN, separate 2 (condition: acceptance/rejection) x 2 (absence/presence of a threshold or subthreshold MDD diagnosis) mixed model ANOVAs were conducted with the PCA-derived social FN factors as dependent variables.

Associations with maternal history of depression. To assess relationships between dichotomously classified maternal depression and children's social and monetary FNs, a 2 (condition: acceptance/rejection) x 2 (maternal depression: absent/present) mixed model ANOVA was conducted for each of the PCA-derived FN factors. These analyses were then repeated, excluding children who met criteria for a depressive disorder, and controlling for the child's depressive symptoms (i.e., the latent depression factor) in order to determine the extent to which the effects of maternal depression on the social FN were independent of the child's current symptomatology.

In order to begin teasing out genetic and environmental influences of maternal depression on the social and monetary FNs, the above analyses were repeated with maternal depression divided into three categories: absent, present but resolved before the child's birth, and present during the child's lifetime.

Exploratory analyses. A series of Pearson correlations was conducted to examine relationships between variables of interest and a set of unanticipated ERP components that emerged from the Kinder task.

Results

Features of the ERP Waveforms

Raw ERP. Figure 4a depicts grand average stimulus-locked ERPs from the Kinder task in response to social acceptance and rejection, as well as the difference between conditions (rejection – acceptance) at electrode FCz. Although there was a slight relative negativity in the difference wave that peaked at approximately 260 ms following feedback onset, the Kinder task did not elicit a robust FN. Confirming this observation, the responses to desirable (i.e., social acceptance; M = 10.45 μV, SD = 6.52) and undesirable (i.e., social rejection; M = 10.51 μV, SD = 7.58) conditions did not differ significantly at FCz from 220-320 ms, $t(195) = -.18$, $p = .86$.

Figure 4b depicts grand average stimulus-locked ERPs from the Doors task in response to monetary gain and loss, as well as the difference between conditions (loss – win) at electrode FCz. An FN peaking at approximately 300 ms was visible in the waveform from the Doors task, and the responses to win (M = 14.00 μV, SD = 8.73) and loss (M = 9.78 μV, SD = 7.91) differed significantly $t(193) = 9.78$, $p < .001$.

Two additional components were apparent in the raw ERP waveform produced by the Kinder task (Figure 5). The first was a positive-going component maximal at occipital sites and peaking approximately 130 ms following the onset of feedback; this component was similar in timing and topographical distribution to the P1, a component thought to be involved in early visual processing (Luck, Woodman, & Vogel, 2000). The response to rejection (M = 6.03 μV, SD = 4.56) at 100-200 ms at electrode Iz was significantly larger than the response to acceptance (M = 3.11 μV, SD = 3.91), $t(194) = 13.02$ ($p < .001$). Additionally, a slow-wave positivity was apparent from approximately 200 ms – 600 ms following feedback onset, which showed maximal differentiation between win and loss conditions at electrode site Oz. This component

was similar in timing and topographical distribution to the P3, an ERP that is sensitive to both manipulations of attention (Johnson, 1986) and to emotional salience (Hajcak, MacNamara, & Olvet, 2010; Macnamara, Foti, & Hajcak, 2009). Like the P1, the P3 was significantly larger in response to rejection feedback (M = 11.13 μV, SD = 6.06) than acceptance feedback (M = 9.14 μV, SD = 5.64), $t(195) = 6.76$ ($p < .001$).

A similar set of components was visible in the waveform elicited by the Doors task. When these were scored within the same time window and at the same electrode as in the Kinder task, the P1 differed significantly by condition such that there was a larger P1 in response to monetary gain (M = 2.86 μV, SD = 3.24) than monetary loss (M = 2.31 μV, SD = 3.33), $t(192) = 2.96$, $p < .01$. The P3 did not differ significantly between conditions ($p > .05$).

Principal components analysis. Means and standard deviations for the PCA-derived temporospatial factors are presented in Table 1. In the Kinder data set, the PCA produced two factors resembling the FN in timing and topographical distribution (Figure 6): TF3SF1 (hereafter referred to as Kinder-1), a positivity maximal at Cz, 223.83 ms after stimulus onset that accounted for 4.57% of variance in the data, and TF8SF1 (hereafter referred to as Kinder-2), a positivity maximal at FCz, 326.37 ms after stimulus onset that accounted for 2.11% of variance. Despite their similarity to the FN, these positivities were both larger for the undesirable (i.e., rejection) condition than for the desirable (i.e., acceptance) condition (Kinder-1: $t(195) = 4.80$ ($p < .001$), Kinder-2: $t(195) = 4.40$ ($p < .001$)).

In the Doors data set, the temporospatial PCA also produced two factor combinations resembling the FN in timing and topographical distribution (Figure 6): TF3SF1 (hereafter referred to as Doors-1), a positivity maximal at Cz, 234.57 ms after stimulus onset that accounted for 3.88% of variance in the data, and TF6SF1 (hereafter referred to as Doors-2), a positivity

maximal at Cz, 298.05 ms after stimulus onset that accounted for 2.84% of variance. Consistent with previous PCA studies using the Doors task (Carlson, Foti, Mujica-Parodi, Harmon-Jones, & Hajcak, 2011; Foti et al., 2011; Weinberg, Riesel, & Proudfit, 2014), this positivity was larger for the desirable (i.e., monetary gain) condition than for the undesirable (i.e., monetary loss) condition for both factors (Doors-1: $t(194) = 5.08$ ($p < .001$), Doors-2: $t(194) = 7.90$ ($p < .001$)).

In addition to the FN-like factors described above, the Kinder PCA also produced factors similar in timing and topographical distribution to the P3 and P1 observed in the raw ERP (Figure 7). TF1SF2, a relatively slow-wave positivity that appeared similar to the P3, was maximal at electrode site O2, 376.17 ms after stimulus onset. This positivity accounted for 5.38% of variance in the data and was larger for rejection ($M = 11.00$ μV, $SD = 5.85$) than acceptance ($M = 10.14$ μV, $SD = 5.61$) feedback, $t(195) = 3.07$, $p < .01$. Another factor, TF6SF2, was similar to the P1 and peaked at O2, 127.15 ms after stimulus onset. This factor accounted for 1.50% of variance in the data and was also larger for rejection ($M = 7.59$ μV, $SD = 7.12$) than acceptance ($M = 2.53$ μV, $SD = 5.78$) feedback, $t(195) = 11.08$, $p < .001$.

Relationships Between Responses to Social and Monetary Reward

Pearson product-moment correlations between the social and monetary FN ERPs are shown in Table 2. The FN to acceptance and the FN to wins were strongly correlated, as were the FN to rejection and the FN to losses (Cohen, 1992).

Pearson product-moment correlations between the PCA-derived temporospatial factors for each task are presented in Table 3. Factors correlated across tasks, with particularly strong associations between Kinder-1 to acceptance and Doors-1 to win ($r = .63$, $p < .001$), and between Kinder-1 to rejection and Doors-1 to loss ($r = .65$, $p < .001$).

Assessment of Pubertal Development

Pearson correlations between pubertal measures are presented in Table 4. Associations between pubertal measures were generally high (*r* values ranging from .30 to .90), with particularly strong associations between the PDS and PBIP (*r* values from .80 to .90). As noted above, when creating the latent variable, PBIP:SR and PBIP:P were excluded from the final CFA model due to their similarity to the PDS and their discontinuation partway through the current study. The final model therefore included estradiol, testosterone, age, PDS:P, and PDS:SR. A correlated residual between estradiol and testosterone was included in the model based on modification indices and residual fit. The resulting model produced an excellent fit with the data (CFI/TLI = 1.00, RMSEA = 0.00). Loadings for the latent puberty variable were as follows (all *p* values < .001): age = 0.82, estradiol = 0.37, testosterone = 0.50, PDS:SR = 0.92, PDS:P = 0.96.

Associations With Puberty

Bivariate correlations. Correlations between the latent puberty variable and the PCA-derived FN components for the Doors and Kinder tasks are presented in Table 3. Puberty was negatively associated with Kinder-1 to acceptance, such that there was a smaller positivity to social acceptance feedback with more advanced puberty. Puberty was also negatively associated with Doors-1 to loss, such that there was a smaller positivity to monetary loss feedback with more advanced puberty.

Unique associations with response to social acceptance. To determine whether the response to social acceptance *uniquely* predicted pubertal development when accounting for the response to monetary gain, Kinder-1 to acceptance was entered as a predictor of puberty in a linear regression controlling for Doors-1 to monetary gain. The overall model was significant, $F(2,191) = 3.35$, $p < .05$, $R^2 = .03$. The response to acceptance predicted a significant portion of

variance in puberty when accounting for the response to monetary gain, β = -.21, t(191) = -2.30, p < .05; in the same model, the contribution of the response to monetary gain was not significant, β = .05, t(191) = .50, p = .62. In a separate analysis, Kinder-1 to acceptance was entered as a predictor of puberty in a linear regression controlling for Doors-2 to monetary gain. Again, the overall model was significant, F(2,191) = 3.71, p < .05, R^2 = .04, and the response to social acceptance contributed significant variance in puberty, β = -.20, t(191) = -2.69, p < .01, whereas the response to monetary gain did not, β = .07, t(191) = .97, p = .33.

Assessment of Depression

Pearson correlations between depression measures are presented in Table 5. Associations between depression questionnaire measures were moderate to high (r values ranging from .36 to .75) (J. Cohen, 1992). KSADS diagnosis showed low to moderate associations with self-reported depression, such that a diagnosis of MDD was associated with higher self-reported depression scores. Correlations between KSADS diagnosis and parent-reported depression were not significant.

A latent depression variable was created using methods described previously. Guided by an EFA, a single factor representing depression was parameterized using CFA; a correlated residual between parent-reported CDI and PROMIS was included in the model based on modification indices. The resulting model produced an excellent fit with the data (CFI/TLI = 1.00, RMSEA = 0.00) (Hu & Bentler, 1999); it was therefore determined to be unnecessary to include correlated residuals to extract method variance associated with self-reported depression measures. Loadings for the depression factor were as follows (all p values < .001): CDI:SR = 0.85, CDI:P = 0.51, PROMIS:SR = 0.89, PROMIS:P = 0.41, KSADS diagnosis = 0.70. PROMIS:P and CDI:P were correlated at .68.

Associations with Depression

Bivariate correlations. The latent depression and puberty variables showed a weak to moderate correlation ($r = .20$, $p < .01$), such that depression increased with more advanced puberty. Correlations between the latent depression variable and the PCA-derived FN components for the Doors and Kinder tasks are presented in Table 3. Depression was negatively associated with Kinder-1 to acceptance and rejection, such that there was a smaller positivity to social acceptance and rejection feedback with greater depression, and with Kinder-2 to rejection, such that there was a smaller positivity to rejection feedback with greater depression. Depression was negatively associated with Doors-1 to both monetary gain and loss, such that there was a smaller positivity to monetary gain and loss feedback with greater depression. Scatter plots depicting the relationships between depression and Kinder-1 to acceptance and rejection are presented in Figure 8.

Unique associations with social acceptance. To determine whether the response to social acceptance *uniquely* predicted depression when accounting for the response to monetary gain, Kinder-1 to acceptance was entered as a predictor of depression in a linear regression controlling for Doors-1 to monetary gain and Doors-2 to monetary gain. The overall model was significant, $F(3,189) = 3.87$, $p = .01$, $R^2 = .06$, and the response to social acceptance predicted unique variance in depression, $t(189) = -2.06$, $p < .05$, $\beta = -.19$, whereas the responses to monetary gain did not, Doors-1: $t(189) = -.87$, $p = .39$, $\beta = -.09$; Doors-2: $t(189) = .42$, $p = .67$, $\beta = .03$.

Unique associations with social rejection. To determine whether the response to social rejection uniquely predicted depression when accounting for the response to monetary loss, Kinder-1 to rejection was entered as a predictor of depression in a linear regression controlling

for Doors-1 to monetary loss and Doors-2 to monetary loss. The overall model was significant, $F(3,189) = 4.46$, $p < .01$, $R^2 = .07$, and the response to social rejection accounted for unique variance in depression, $t(189) = -2.64$, $p < .01$, $\beta = -.24$, whereas the responses to monetary loss did not, Doors-1: $t(189) = -.30$, $p = .76$, $\beta = -.03$; Doors-2: $t(189) = 1.06$, $p = .29$, $\beta = .08$.

A similar set of regressions was run to assess for unique associations between depression and Kinder-2 to social rejection. Kinder-2 to rejection was entered as a predictor of depression in a linear regression controlling for Doors-1 to monetary loss and Doors-2 to monetary loss; the overall model was also significant, $F(3,189) = 4.29$, $p < .01$, $R^2 = .06$, and the response to social rejection predicted unique variance in depression, $t(189) = -2.54$, $p < .05$, $\beta = -.19$, whereas the responses to monetary loss did not, Doors-1: $t(189) = -1.85$, $p = .07$, $\beta = -.14$; Doors-2: $t(189) = 1.38$, $p = .17$, $\beta = .10$.

In addition to defining depression as a continuous variable, depression was also classified dichotomously (i.e., as the presence or absence of a threshold or subthreshold MDD diagnosis). In separate 2 (condition) x 2 (MDD diagnosis) mixed-model ANOVAs with Kinder-1 and Kinder-2 as dependent variables, there was a main effect of condition such that the response to rejection was larger than the response to acceptance (Kinder-1: $F(1,194) = 12.96$, $p < .001$; Kinder-2: $F(1,194) = 6.04$, $p < .05$). There was no main effect of depression diagnosis, and no significant interaction, for either analysis (p values > .05).

Associations With Maternal History of Depression

A 2 (condition: acceptance/rejection) x 2 (maternal risk: low/high) mixed-model ANOVA was conducted on Kinder-1. There was a main effect of condition such that there was a larger positivity to rejection ($M = 12.04$ μV, $SD = 8.33$) than acceptance ($M = 10.08$ μV, $SD = 8.27$), $F(1,189) = 17.07$, $p < .001$; there was no main effect of maternal depression and no

interaction effect (*p* values > .05). The same pattern of results emerged when children with a threshold or subthreshold depression diagnosis were excluded and the child's depression was added as a covariate: there was a main effect of condition such that there was a larger positivity to rejection (*M* = 12.10 μV, *SD* = 8.41) than acceptance (*M* = 10.14 μV, *SD* = 8.23), *F*(1,168) = 15.57, *p* < .001, and there was no main effect of maternal depression and no interaction effect (*p* values > .05). No effects of maternal depression emerged when puberty was included as a covariate; when subjects with a maternal history of anxiety disorders were excluded; when maternal depressive symptoms on the Beck Depression Inventory were added as a covariate; or when subjects were categorized trichotomously into those whose mothers had never been depressed, those whose mothers' depression had resolved before their birth, and those whose mothers had been depressed during their lifetimes (*p* values > .05).

Likewise, in a separate ANOVA with Kinder-2 as a dependent variable, there was a main effect of condition such that there was a larger positivity to rejection (*M* = 3.26 μV, *SD* = 5.90) than acceptance (*M* = 1.49 μV, *SD* = 5.08), F(1,189) = 11.29, *p* < .01, but there was no main effect of maternal depression and no interaction between maternal depression and condition (*p* values >.05). The same pattern of results emerged when children with a threshold or subthreshold depression diagnosis were excluded and the child's depression was added as a covariate: there was a main effect of condition such that there was a larger positivity to rejection (*M* = 3.33 μV, *SD* = 5.87) than acceptance (*M* = 1.46 μV, *SD* = 5.17), *F*(1,168) = 12.31, *p* < .01, and there was no main effect of maternal depression and no interaction effect (*p* values > .05). No effects of maternal depression emerged when puberty was included as a covariate; when subjects with a maternal history of anxiety disorders were excluded; when maternal depressive symptoms on the Beck Depression Inventory were added as a covariate; or when subjects were

categorized trichotomously into those whose mothers had never been depressed, those whose mothers' depression had resolved before their birth, and those whose mothers had been depressed during their lifetimes (*p* values > .05).

Like the responses to social feedback, the responses to monetary feedback also showed a main effect of condition, but no main effect of maternal depression and no interaction (*p* values > .05). For Doors-1, there was a larger positivity to monetary gain (*M* = 13.68 μV, *SD* = 7.32) than loss (*M* = 12.21 μV, *SD* = 7.78), $F(1, 165) = 12.13$, $p = .001$. Similarly, for Doors-2, there was a larger positivity to monetary gain (*M* = 6.21 μV, *SD* = 6.78) than loss (*M* = 2.61 μV, *SD* = 5.83), $F(1,165) = 24.47$, $p < .001$. This pattern of results did not change when children with threshold or subthreshold depression were excluded and depression severity was added as a covariate; when puberty was included as a covariate; when subjects with a maternal history of anxiety disorders were excluded; when maternal depressive symptoms on the Beck Depression Inventory were added as a covariate; or when maternal depression was categorized trichotomously.

Exploratory P1 and P3 Analyses

A number of exploratory Pearson correlations were conducted to assess associations with the PCA-derived P1 and P3 factors from the Kinder task (Table 6). The latent puberty score showed moderate negative correlations with P1 to acceptance, P1 to rejection, and ΔP1; that is, more advanced puberty was associated with a smaller response to acceptance and rejection as well as a smaller differentiation between the two. Puberty also showed a moderate negative association with the P3 to rejection and acceptance, such that there was a smaller P3 to both rejection and acceptance with more advanced puberty. Additionally, depression showed a small negative correlation with P1 to rejection and ΔP1, such that greater depression severity was

associated with a smaller response to rejection and less differentiation between acceptance and rejection.

When the P1 and P3 were entered into separate 2 (condition: accept/reject) x 2 (maternal depression: absent/present) ANOVAs, the P1 showed a main effect of condition such that the response to rejection was larger than the response to acceptance, $F(1,166) = 54.87$, $p < .001$, accept $M = 2.91$ μV, $SD = 5.76$, reject $M = 7.72$ μV, $SD = 7.33$; there was no significant main effect of maternal depression and no interaction effect. There were no significant effects for the P3 (p values > .05).

Discussion

The current study was designed to evaluate the neural response to social rewards in 8- to 16-year-old girls using the Kinder task, a social feedback task modeled after popular social networking applications. An initial aim was to examine the ERP waveforms elicited by feedback indicating social rejection and acceptance; it was hypothesized that the task would elicit an FN, similar to the ERP generated by feedback indicating monetary gain and loss. Although the raw waveform did not show the expected FN in response to social feedback, a PCA revealed two FN-like components – not apparent in the raw waveform – one of which correlated strongly with a similar component generated by monetary feedback. Consistent with hypotheses, the PCA-derived social FN related uniquely to both pubertal development and depression when controlling for the monetary FN. However, the observed associations with puberty and depression were not consistently in the expected direction. Moreover, the FN did not relate to maternal history of depression. The Kinder task additionally generated components resembling a P1 and a P3, which showed significant differentiation between task conditions.

Neural Responses to Social Feedback

Contrary to hypotheses, the Kinder task did not elicit a robust FN in the ERP waveform. A typical FN was apparent in the Doors ERP waveform, in contrast, suggesting that the absence of an FN from the Kinder task in the same subjects was not simply due to a more generalized lack of response to rewarding stimuli. The absence of a robust, directly observable social FN can be explained in two possible ways: either the Kinder task simply did not elicit an FN, or the FN that it elicited was obscured by overlapping components.

In support of the first possibility, the FN is diminished under circumstances in which the participant perceives a lack of control over outcomes, such as when the participant passively

views outcome stimuli without making an active choice to influence them (Holroyd, Krigolson, Baker, Lee, & Gibson, 2009; Sambrook & Goslin, 2015). Although the Kinder task allowed participants to create their own profiles and to vote on the other "player" before receiving that player's feedback, it is possible that this was not sufficient to engender a significant sense of control. Having been told that the other player had already voted on them before the participant voted – and that the participant would simply be *viewing* the other player's pre-determined feedback after voting – the participants may not have felt that feedback could be impacted by their behavior. If participants *did* perceive a sense of control over the outcome – e.g., believing that the other player would have voted differently if the participant's profile had been different – the length of time between the participant's action and its associated outcome may have been substantially greater than is typical for FN paradigms. This is an important distinction because prior research has found that the FN is elicited only when actions and their outcomes are closely linked in time (Weinberg, Luhmann, Bress, & Hajcak, 2012). Indeed, learning of associations over a longer feedback delay appears to be linked to hippocampal, rather than striatal, processes (Foerde & Shohamy, 2011) – which would not be expected to generate an FN.

Since the current study was initially proposed, several ERP studies of social feedback have been published; the results of these studies are inconsistent with regard to their elicitation of FN. Although some have reported an FN (A. Kujawa, Arfer, Klein, & Proudfit, 2014), others have not (Van der Veen, Van der Molen, Sahibdin, & Franken, 2013) or have reported an FN-like deflection in the waveform that did not differ between desirable and undesirable social feedback conditions (Dekkers, van der Molen, Moor, van der Veen, & van der Molen, 2014; Van der Molen et al., 2014). One study (Sun & Yu, 2014) found an FN that differed by condition; however, the authors used a time window of 300-400 ms following feedback onset, which is later

than is typical for measuring the FN. A visual inspection of their waveforms suggests that there was little differentiation between conditions in a slightly earlier window surrounding the negative deflection, and that their measured difference may instead have reflected a difference in the time-range of the P3. Similarly, not all fMRI studies have found evidence of striatal activation in response to social reward (Davey, Allen, Harrison, & Yücel, 2011; Silk et al., 2013).

If the FN constitutes a reward learning signal, then the absence of an FN in the waveform in the Kinder task might reflect learning-related differences between Kinder and other tasks, such as Island Getaway (A. Kujawa et al., 2014), that have been found to generate an FN. In contrast with the Island Getaway task, in which participants interact with several of the other players repeatedly, the Kinder task did not provide participants with an opportunity to learn from their actions and about other players over multiple rounds. Rather, each other player's profile and feedback was viewed only once in Kinder, and therefore the feedback did not provide participants with information they would be able to use for subsequent trials.

Another possibility is that the *apparent* absence of an FN in the Kinder waveform is the result of overlap between the FN and other ERP components. A voltage peak – or the absence thereof – in an ERP waveform is not inherently meaningful (Luck, 2005). The morphology of the raw waveform reflects the summation of its underlying components, and it is therefore possible that the apparent absence of a social FN in the Kinder task was the result of an underlying FN-like component in combination with other components that differed from those produced by the Doors task. For this reason, temporospatial PCA methodology was employed with the intention of distilling the underlying components that made up the raw waveform. Of the existing ERP studies of social feedback (Crowley et al., 2010; Dekkers et al., 2014; A. Kujawa et al., 2014;

Sun & Yu, 2014; Van der Molen et al., 2014; Van der Veen et al., 2013), only one has employed this approach (Crowley et al., 2010). The authors of that study, which employed a Cyberball task, did not report a positivity with a time range and distribution typical of the FN; however, differences between the Kinder and Cyberball tasks (see Introduction) make these findings difficult to compare with the current study.

Indeed, despite the absence of a clear FN in the Kinder ERP waveform, the PCA in the current study revealed two positive-going factors within the time range and topographical distribution of a typical FN. These were similar in timing to the two factors generated by the PCA conducted on the Doors data – one at approximately 230 ms after stimulus onset, and another approximately 310 ms after stimulus onset. Both factors in both tasks had similar morphology and were frontocentrally maximal (either at Cz or FCz). For the Doors task, both of these components were similar to those found in prior PCA studies of the Doors task in adult samples (Carlson et al., 2011; Foti et al., 2011; Liu et al., 2014). Notably, the earlier FN-like social factor correlated strongly with the earlier FN-like monetary factor, suggesting that both components may reflect similar neural processes.

In contrast to the two FN-like peaks generated by each PCA in the current study, prior PCA studies with adults have reported only one (Carlson et al., 2011; Foti et al., 2011; Liu et al., 2014). The two peaks reported here may be an artifact of the PCA methodology. Because PCAs identify components based on common timing, single components that vary in latency can be treated as if they were multiple components (Luck, 2005). This phenomenon can lead to the appearance of multiple separate peaks when in fact the data would be better accounted for by a single component. Prior studies have found that younger children have longer FN latencies than

adolescents (Lukie, Montazer-Hojat, & Holroyd, 2014), and this variation in latency could have led to an artificial split in the PCA-derived FN.

As in previous studies (Carlson et al., 2011; Foti et al., 2011; Liu et al., 2014), the response to monetary gain in the Doors task was more positive than the response to monetary loss. For the Kinder task, in contrast, the effects were the opposite: the response to the undesirable condition (i.e., social rejection) was more positive than the response to the desirable condition (i.e., social acceptance). Therefore, despite similarity to the monetary FN, it appears that the FN-like factors generated by the Kinder task may not reflect reward valence. Although this finding is unexpected, it is consistent with results reported by Masten and colleagues (Masten et al., 2009), who found that social rejection, compared to acceptance, was associated with greater activation of the ventral striatum (in addition to other brain areas) in a sample of 12- to 13-year-olds. Masten et al. (2009) suggested that this activation, which had not been observed in similar studies with adults, might reflect a compensatory role for the ventral striatum in affect regulation amongst individuals for whom the prefrontal cortex – the area more typically associated with such processes – is still developing.

The current findings are also consistent with a recent proposal that, rather than reflecting reward learning *per se*, the FN may reflect a salience prediction error. In support of this conceptualization, Talmi and colleagues reported that physical punishment was characterized by a "reverse" FN – i.e., an apparent negativity that was larger for desirable feedback (a safety cue) than undesirable feedback (a cue indicating upcoming electric shock to the hand) but otherwise similar in morphology to a typical FN elicited by monetary feedback (Talmi, Atkinson, & El-Deredy, 2013). Based on this result, Talmi et al. concluded that the FN reflects the processing of stimulus salience prediction errors rather than reward prediction errors. (However, see Heydari

and Holroyd (2016) for conflicting results.) Dekkers and colleagues, too, observed an FN-like response in a social feedback task that differed based on unexpectedness of the outcome, but not valence; similarly to Talmi et al., Dekkers and colleagues argue that the FN may be an index of prediction error rather than reward value in the context of social feedback tasks (Dekkers et al., 2014).

A number of imaging studies have found evidence of striatal activity in response to undesirable outcomes, lending further support to this conceptualization. One study used a Pavlovian conditioning task involving physical pain in the form of electric shock, and found a striatal response that suggested a role for the striatum in aversive – rather than just appetitive – learning (Seymour et al., 2004). Since that time, additional studies of physical pain have produced similar results (Jensen et al., 2007; Seymour et al., 2005). More recently, a study by Metereau and Dreher (2013) found evidence of a network consisting of the striatum, anterior insula, and anterior cingulate cortex that responded to both a desirable stimulus (a taste of pleasant juice paired with a picture of a glass of juice) and an undesirable stimulus (a taste of salty water paired with a picture of a glass of brown water). The authors of that study note that the presence of an undesirable stimulus, such as a painful electric shock or an unpleasant taste, tends to be associated with an increase in striatal response, whereas the absence of a desirable stimulus, such as a monetary loss, tends to be associated with a decrease in striatal response (Delgado, Nystrom, Fissell, Noll, & Fiez, 2000; Yacubian et al., 2006).

Given that both the FN and the striatum can respond to undesirable outcomes under certain circumstances, the monetary FN and the "reverse" social FN observed in the current study could reflect a common underlying neural process. As such, the social FN may reflect the extent to which acceptance and rejection feedback are *important* to girls in the targeted age

range, rather than the extent to which they are *rewarding*. Given that rejection by peers tends to be associated with more negative long-term outcomes for children than either popularity, typical social status, or peer neglect across a variety of metrics (Ollendick, Weist, Borden, & Greene, 1992), it would be evolutionarily advantageous for feedback indicating rejection to be particularly salient – and girls may view the receipt of such feedback as an aversive experience rather than as simply the absence of a pleasant experience. Future studies might assess this possibility by employing additional measures to assess the perceived salience and aversiveness of the rejection feedback.

It is also possible that, despite their resemblance, the monetary and social FNs are driven by separate neural generators. In this case, the monetary FN might reflect a reward prediction error generated in the striatum, whereas the social FN might reflect other attentional or learning processes related to the receipt of social feedback. Although the functional significance of the PCA-derived social FN cannot be determined with complete certainty based on the current data, the fact that this component related uniquely to both pubertal development and depression is notable.

Puberty and the Response to Social Feedback

In the current study, unexpectedly, more advanced puberty was associated with a smaller FN in response to social acceptance. Given that the importance of social feedback tends to increase over the course of adolescence (Csikszentmihalyi et al., 1977; Furman & Buhrmester, 1992; Steinberg & Morris, 2001), and that pubertal hormones are associated with a neural reorientation toward social rewards (Crone & Dahl, 2012), it was expected that more advanced puberty would be associated with a *larger* response to acceptance feedback. The observed negative association between puberty and the PCA-derived social FN might reflect the influence

of a third variable that was not originally considered – for instance, a decreased belief in the task with age. That is, it is possible that older participants were less convinced that the other "players" in the Kinder task were real, decreasing the meaningfulness of acceptance feedback in these individuals. Indeed, although participants' belief in the task was not tracked systematically, a few participants spontaneously reported during the debriefing that they had not believed that they were truly playing against peers.

Another possible explanation relates to children's exposure to social networking websites. Older teens tend to use the internet more often, and are more likely to use social networking websites, than younger teens and children (Lenhart, Purcell, Smith, & Zickuhr, 2010; Roberts & Foehr, 2008); as of 2009, 82% of 14- to 17-year-olds reported using a social networking site while only 55% of 12- to 13-year-olds did (Lenhart et al., 2010). Therefore, it is likely that older participants in the current study had had substantially more experience with social networking websites than younger participants and may have been more familiar with the experience of receiving "likes". It follows that younger participants, then, might have found social feedback more rewarding or salient due to their relatively low degree of exposure to online social feedback.

Interestingly, the effect of pubertal development was specific to acceptance and not rejection feedback. This observation may relate to the fact that on the more popular social networking websites, until recently, the only feedback available was positive: if an individual created a post, that person's social contacts could click a button to indicate that they "liked" the post but could not indicate that they "disliked" it. Therefore, the specificity of the pubertal effect to social acceptance in the current study could relate to the fact that older children are more accustomed to the receipt of desirable social feedback than younger children, and therefore find

it less salient – but they have not had similar exposure to undesirable social feedback in a social networking context.

The response to *monetary* reward, in contrast, did not relate to puberty. Moreover, the response to social acceptance uniquely related to puberty when controlling for the response to monetary gain. This finding suggests that the relationship between puberty and the response to social acceptance cannot be explained by a change in reward sensitivity across puberty more generally. Following on the point above, this unique relationship could reflect the relatively high exposure that girls in the targeted age range have to online social feedback as opposed to monetary feedback. Whereas girls frequently receive social feedback via online social networking sites, they most likely rarely – if ever – receive feedback related to having won or lost money.

Depression and the Response to Social Feedback

In addition to its relationship with puberty, the response to social feedback also related to depression such that children with higher depression scores showed a smaller response to both rejection and acceptance feedback. This finding was partially consistent with initial hypotheses. Although it had been hypothesized that more depressed girls would show a smaller response to social feedback, this effect was predicted to be specific to social acceptance. If, as suggested above, the social FN reflects the salience of social feedback rather than its reward value *per se*, then it might be expected that that the response to social rejection would be *larger* in individuals with higher depression scores, given previous findings of increased relational victimization and heightened rejection sensitivity amongst individuals with depression (La Greca & Harrison, 2005; Silk et al., 2013). However, the opposite effect was observed in the current study.

The relationship between depression and decreased response to both social acceptance and rejection in the Kinder task may reflect lower task engagement amongst the more depressed individuals. Depression, by its definition, is frequently associated with decreased interest in activities (American Psychiatric Association, 2000) and has been associated with low attentional capacity and impaired automatic cognitive processing (R. M. Cohen, Weingartner, Smallberg, Pickar, & Murphy, 1982; Den Hartog, Derix, Van Bemmel, Kremer, & Jolles, 2003). It is possible, therefore, that the more depressed participants in the current study had more difficulty attending to the task or processing feedback, and that this resulted in a smaller response to social feedback in these participants.

The current results could also be consistent with the emotion context insensitivity (ECI) hypothesis (Rottenberg, Gross, & Gotlib, 2005), which posits that depression is associated with decreased emotional reactivity to both positive and negative emotional stimuli. A meta-analysis of emotional reactivity studies by Bylsma and colleagues (Bylsma, Morris, & Rottenberg, 2008) found consistent support for this hypothesis across studies employing a range of self-report, behavioral, and physiological measures. However, the design of the Kinder task did not allow for a direct examination of this possibility; in order to assess its fit with the ECI model, the task would need to include a neutral feedback condition for comparison. Without such a condition, the possibility remains that the blunted response in the acceptance and rejection conditions represents a more generalized decrease in reactivity across stimuli. In future iterations of the Kinder task, it would be informative to add a third feedback condition in which other players indicated that they felt neutral toward the participant or were not sure if they would want to be friends.

It is worth noting that for the later of the two PCA-derived social FNs, the relationship with depression was specific to the response to rejection; that is, only the response to rejection and not acceptance became smaller with higher depression severity. If, as suggested above, the two PCA factors represent the same component for different age subgroups, then this component might represent the FN for the younger participants. It is possible, then, that the association between depression and desirable social feedback appears slightly later in development than the association with negative social feedback.

Similar to the relationship observed for the responses to social feedback, the responses to monetary reward and loss also showed negative relationships with depression. When responses to monetary and social feedback were entered as simultaneous predictors of depression in a regression, the response to social feedback remained a significant predictor, whereas the response to monetary feedback did not. This finding suggests that the monetary and social responses tapped into a shared process, but that the social response accounted for an additional portion of the variance in depression scores that was not accounted for by this common process. It could be that the Kinder task was associated with a higher cognitive load and that this aspect of the task explained differences in depression beyond the common response to feedback salience or reward value across tasks; perhaps the monetary feedback required relatively little interpretation. Alternatively, it could be that social acceptance and rejection were associated with greater personal relevance to the girls in the current sample, and that the response to this feedback therefore related to depression in a way that extended beyond the reward value of the feedback. The Kinder task would need to be compared to less taxing social tasks and to more taxing non-social reward tasks in order to tease apart these possibilities.

The Social FN as a State Marker

Unexpectedly, the neural response to social acceptance and rejection did not relate to maternal risk for depression – though nor did the response to monetary feedback. It is therefore unclear whether the absence of this association reflects a lack of association with the response to social feedback in particular, or with the response to desirable and undesirable feedback in the current sample more generally. It is possible that some characteristic of the current group, such as the age range of the participants, obscured the relationship between risk and the FN that has been observed in other studies (Bress et al., 2013; A. J. Kujawa et al., 2014; B. D. Nelson, Perlman, Klein, Kotov, & Hajcak, in press). One study (Bress et al., 2013) found an association between a blunted monetary FN and new onset of a depressive episode, but participants were in their late teen years at baseline. On the other end of the age spectrum, Kujawa and colleagues (2014) found an association between a blunted monetary FN and a maternal history of depression in 9-year-old children. Notably, the ages of the participants in these samples were at the extreme ends of the sample in the current study, and it is possible that the age range from 8 to 16 represents a time in the life span when the effects of risk on reward sensitivity are different. It would be informative to study relationships between maternal risk for depression and the neural response to social feedback in a different age group. Interestingly, the effects reported by Kujawa et al. were specific to children who had a maternal history of depression *without* comorbid anxiety. However, when mothers with anxiety were excluded from the current analyses, the effect of maternal depression remained non-significant.

If the neural response to social feedback does not relate to maternal risk for depression, then this response may be more accurately represented as a state marker of depressive symptoms than a reflection of trait depression or risk. That is, an individual at risk for depression may show

a typical response to social acceptance and rejection until the point at which she becomes depressed; at that point, she may begin to show decreased response to both acceptance and rejection. If this is the case, then the response to social feedback might be used as an indicator of current functioning – and potentially as a means by which to examine differences in mechanisms between various therapies for depression. It would be informative to test the extent to which the social FN differed before and after psychotherapy for depression. It is possible, for instance, that interpersonal therapy might result in an increase in responsiveness to social feedback whereas standard cognitive behavioral therapy might not.

Additional Components

In addition to the FN, an unexpected set of ERPs was observed for the Kinder task: namely, components that appeared similar to the P1 and the P3, which were also apparent in the results of the PCA. These components were notable because they varied by condition, such that both were larger in response to rejection than acceptance. The P1, an early index of visual processing generated in extrastriate areas of ventral visual pathway (Luck et al., 2000), is thought to reflect early processing of visual information; it has also consistently been observed to have a larger amplitude in response to unpleasant as compared to pleasant images (Olofsson, Nordin, Sequeira, & Polich, 2008). The larger P1 in the rejection condition may indicate a modulatory mechanism by which the prior knowledge of the significance of the social feedback stimuli cues the participant to pay greater attention to these stimuli. The P1 can also be influenced by the physical characteristics of stimuli, such as color (see, e.g., Coch, Skendzel, Grossi, & Neville, 2005). However, in the Doors task – which used feedback stimuli similar to those used in the Kinder task – the P1 was larger for the desirable (i.e. monetary gain) condition.

It is therefore unlikely that the differentiation between conditions in the Kinder task reflects a simple difference in the processing of the physical properties of the stimuli.

Like the P1, the P3 showed differentiation between conditions such that there was a larger response to rejection than acceptance feedback. The P3 has been linked to the motivational salience of stimuli and is potentiated both by task-relevance of a stimulus and by its emotional content (Hajcak et al., 2010). The observed differentiation between conditions for the P3 in the Kinder task suggests that social rejection feedback was more emotionally salient to participants than social acceptance. This finding is broadly consistent with studies suggesting a particularly important role for social rejection, in comparison with social acceptance or peer neglect, amongst children (Ollendick et al., 1992). Evidence from other ERP studies of social feedback have had mixed results with regards to the P3. One study of adults, for instance, reported a larger P3 in response to social acceptance than rejection (Van der Veen et al., 2013). In contrast, a study of 8- to 12-year-olds (Crowley et al., 2010) found a P3 that was larger for rejection than "not my turn" events – i.e., trials on which one of the other players threw to each other instead of to the participant in the context of a fair-play condition – in the Cyberball task. It is possible that the larger P3 response to rejection vs. acceptance is specific to a younger age range; additional studies using the same task across a wider age range would be needed in order to clarify this possibility.

Like the FN, the P1 and P3 both related negatively to pubertal development across the acceptance and rejection conditions. Similarly to the FN, these effects may have been driven by either a decreased belief in the task amongst older participants or a greater degree of prior exposure to social feedback via social networking applications, which may have decreased the perceived importance of the feedback. In turn, this perception may have decreased the need for

earlier visual attention, as indexed by the P1, as well as the perception of feedback salience, as indexed by the P3. The P1 in response to rejection – but not acceptance – also showed a negative correlation with depression. This finding is surprising, as depression would typically be expected either to increase early attention to rejection or to decrease attention to both rejection and acceptance. Future studies will be needed to determine whether these findings are replicated in other samples.

Limitations and Future Directions

The current study had some limitations that warrant further study. First, the sample included only girls. This choice was made due to the higher incidence of depression in girls within this age range, as well as girls' tendency to place more value than boys on social relationships. It remains to be seen whether similar effects would be observed in a mixed-gender sample. Prior studies have found gender differences in the neural response to reward (Grose-Fifer, Migliaccio, & Zottoli, 2014; A. J. Kujawa et al., 2015), and preliminary evidence suggests that some of the association between risk for depression and the neural response to rewards may be stronger in boys (Bress, Infantolino, Jackson, Gibb, & Hajcak, 2015). It is therefore possible that by excluding boys from the current study, a meaningful association was overlooked. It would be informative for future studies to test gender effects in the relationships between puberty, depression, risk, and the neural response to social rewards.

The Kinder task itself had both strengths and limitations. Although the task was designed to be as realistic as possible, the current results suggest that some participants – particularly those who were older and likely savvier with social networking websites than the experimenters – may have been able to detect that the profiles of the other "players" did not actually belong to real peers. Participants' belief in the task was not assessed formally, which would have bolstered this

interpretation. In order to further strengthen its ecological validity, the Kinder task might be improved by using profiles actually generated by children in the target age range rather than by the experimenters; further, future participants could be asked explicitly about their belief in the task as a manipulation check. Moreover, in the current task, player information was randomized across gender of the player; the task might further be improved by including more gendered personal information and restricting it to female or male players as appropriate.

The task was also designed in such a way that the participants' votes were restricted, with half designated for acceptance and half designated for rejection votes. This restriction was imposed to ensure an even balance of trials across feedback conditions. Participants were shown a running tally of their remaining votes in order to allow them the leeway to allocate their votes as desired. However, the restriction of votes may have inadvertently forced participants into making choices not fully in line with their judgments of the other players – which, in turn, may have reduced the salience of corresponding feedback and weakened the associated neural response. For similar tasks in which participants were asked to predict whether peers would accept or reject them (e.g., Dekkers et al., 2014; Van der Molen et al., 2014), participants tended to predict acceptance more than rejection. If participants' own judgments of their peers follow the same pattern, then participants with unrestricted votes might have voted to accept their own peers more often.

Conclusion

Taken together, several ERP components observed in the current data suggest that girls have a particularly strong neural response to social rejection as compared to acceptance in late childhood and early adolescence. The differentiation between conditions began early in the time window following stimulus presentation, indicating a modulatory role for social feedback in

early attentional processing, and was also observable in the time range of the P3, a measure of motivational salience. The PCA methodology revealed an additional component resembling the FN; this component was also larger for rejection compared to acceptance feedback, consistent with conceptualizations of the FN and of striatal activation as reflecting learning related to salient aversive outcomes rather than rewarding outcomes alone. The social FN showed a unique relationship with pubertal development, an effect that may reflect changes in exposure to social media over the course of adolescence, as well as with depression. The association with depression could reflect either generalized task disengagement or more specific emotional disengagement amongst individuals with more intense depression, a question that will need to be clarified in future studies. Although the functional significance of this neural response remains an open question, its unique relationship with depressive symptomatology, in conjunction with the absence of a relationship with maternal risk, suggests a possible role for this component as a state marker in the assessment of future treatment interventions.

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Table 1. Means and standard deviations of the raw ERPs and PCA factor combinations for the Kinder and Doors tasks.

Task	Component	Mean (μV)	Standard Deviation
Kinder	FN – accept	10.44	6.52
	FN – reject	10.51	7.57
	ΔFN	.07	5.12
	Kinder-1 – accept	10.17	8.22
	Kinder-1 – reject	12.12	8.36
	Δ Kinder-1	1.95	5.69
	Kinder-2 – accept	1.54	5.19
	Kinder-2 – reject	3.29	5.88
	Δ Kinder-2	1.75	5.57
Doors	FN – win	13.90	8.74
	FN – loss	9.78	7.91
	ΔFN	-4.22	6.04
	TF3SF1 – win	14.11	7.40
	TF3SF1 – loss	12.28	7.74
	ΔTF3SF1	-1.84	5.05
	TF6SF1 – win	6.65	6.88
	TF6SF1 – loss	2.55	5.93
	ΔTF6SF1	-4.10	7.24

Table 2. Pearson correlations between raw ERPs for the Kinder and Doors tasks.

		Kinder			**Doors**		
		FN – accept	**FN – reject**	**ΔFN**	**FN – win**	**FN – loss**	**ΔFN**
Kinder	**FN – accept**	-					
	FN – reject	.75***	-				
	ΔFN	-.17*	.53***	-			
Doors	**FN – win**	.58***	.55***	.07	-		
	FN – loss	.60***	.62***	.15*	.74***	-	
	ΔFN	-.04	.04	.11	-.48***	.24**	-

*$p < .05$, **$p < .01$, ***$p < .001$

Table 3. Pearson correlations between PCA-derived factor combinations for the Kinder and Doors tasks, as well as their relationships with the latent puberty and depression variables.

		Kinder-1			**Kinder-2**			**Doors-1**			**Doors-2**		
		Accept	**Reject**	**Δ**	**Accept**	**Reject**	**Δ**	**Win**	**Loss**	**Δ**	**Win**	**Loss**	**Δ**
Kinder-1	**Accept**	-											
	Reject	.76***	-										
	Δ	-.32***	.37***	-									
Kinder-2	**Accept**	.33***	.20**	-.19**	-								
	Reject	.35***	.41***	.10	.50***	-							
	Δ	.05	.25**	.28***	-.41***	.59***	-						
Doors-1	**Win**	.63***	.58***	-.05	.23**	.27***	.06	-					
	Loss	.69***	.65***	-.05	.24**	.29***	.08	.78***	-				
	Δ	-.14*	-.14	.00	-.02	-.05	-.03	.27***	-.39***	-			
Doors-2	**Win**	.20**	.21**	.01	.38***	.28***	-.07	.46***	.33***	.17*	-		
	Loss	.20**	.21**	.02	.32***	.24**	-.05	.15*	.28***	-.21**	.37***	-	
	Δ	.03	.02	-.01	.10	.07	-.03	.31***	.08	.33***	.65***	-.47***	-
Puberty		-.18*	-.11	.10	.04	.08	.05	-.09	-.16*	.12	.03	-.11	.12
Depression		-.23**	-.25**	-.03	-.12	-.21**	-.11	-.19*	-.17*	-.02	-.04	.02	-.06

*$p < .05$, **$p < .01$, ***$p < .001$

Table 4. Pearson correlations between pubertal measures.

	Age	**PDS:SR**	**PDS:P**	**PBIP:SR**	**PBIP:P**	**Estradiol**	**Testosterone**
Age	-						
PDS:SR	.72***	-					
PDS:P	.76***	.87***	-				
PBIP:SR	.72***	.80***	.80***	-			
PBIP:P	.75***	.80***	.90***	.87***	-		
Estradiol	.30***	.29***	.32***	.31***	.35**	-	
Testosterone	.47***	.40***	.44***	.49***	.51***	.55***	-

*$p < .05$, **$p < .01$, ***$p < .001$

Table 5. Pearson correlations between depression measures.

	CDI:SR	**CDI:P**	**PROMIS:SR**	**PROMIS:P**	**KSADS**
CDI:SR	-				
CDI:P	.47***	-			
PROMIS:SR	.75***	.46***	-		
PROMIS:P	.37***	.75***	.36***	-	
KSADS	.29***	.14	.22**	.13	-

*$p < .05$, **$p < .01$, ***$p < .001$

Table 6. Exploratory Pearson correlations for the PCA-derived P1 and P3 factors.

	P1-accept	**P1-reject**	**ΔP1**	**P3-accept**	**P3-reject**	**ΔP3**
Puberty	-.17*	-.32***	-.20**	-.23**	-.25**	-.03
Depression	-.02	-.14*	-.14*	-.09	-.09	-.01

*$p < .05$, **$p < .01$, ***$p < .001$

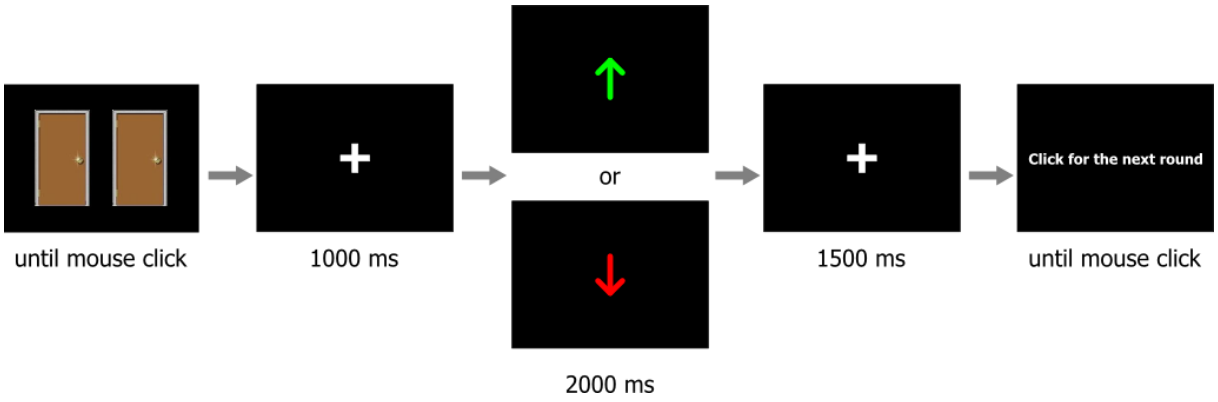

Figure 1. Diagram of the Doors task.

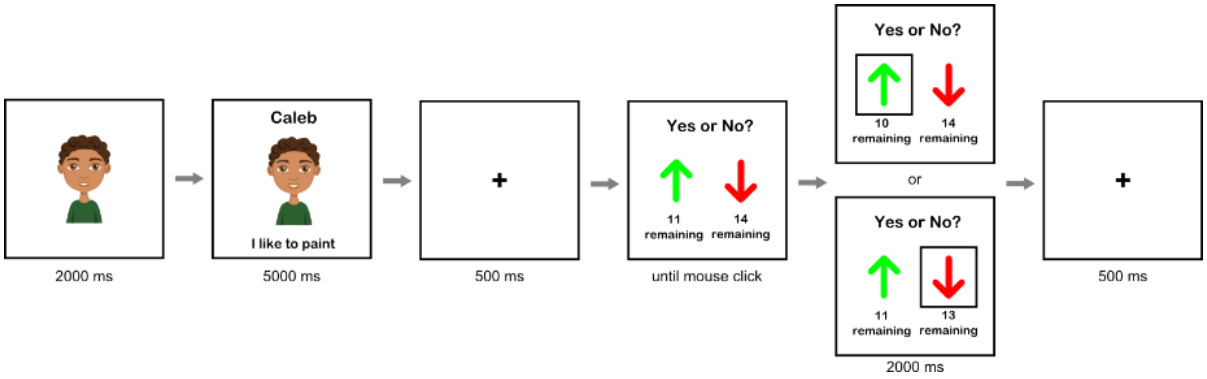

Figure 2. Diagram of the voting phase of the Kinder task.

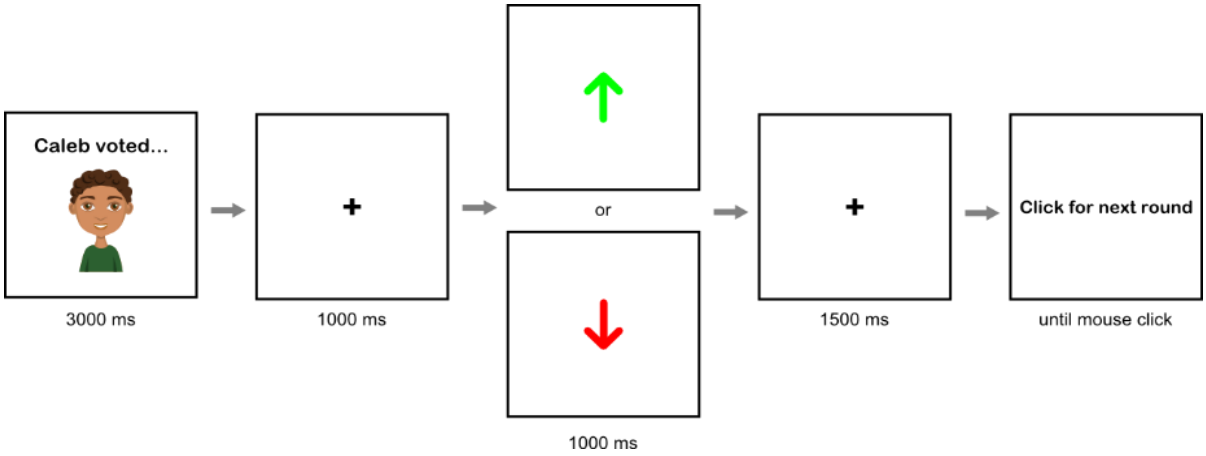

Figure 3. Diagram of the feedback phase of the Kinder task.

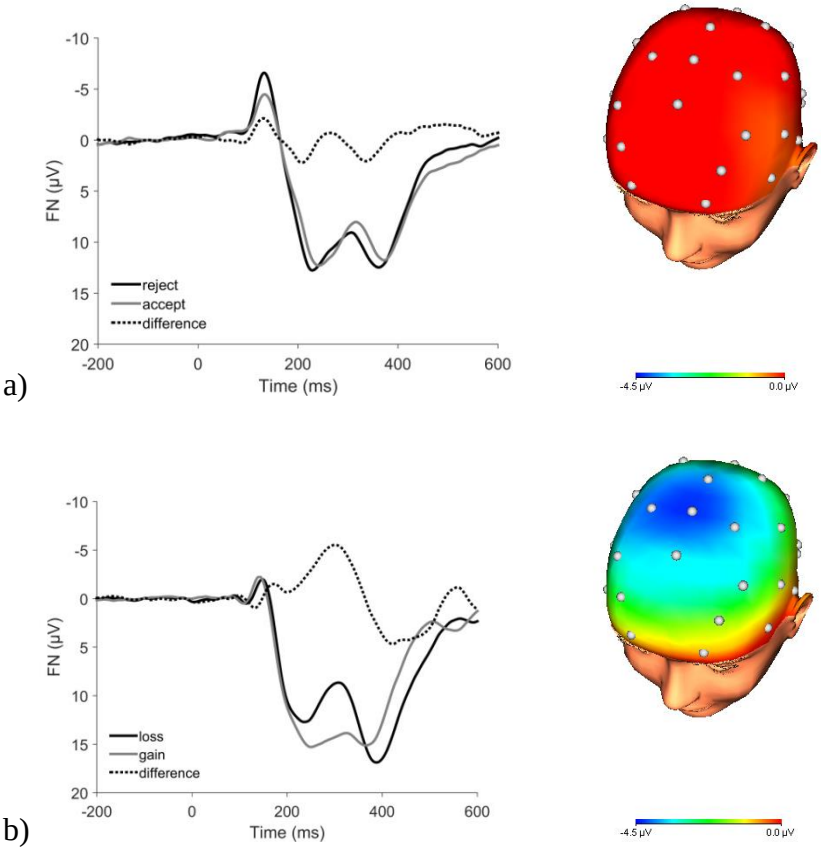

Figure 4. (a) ERP waveform in the time range of the FN at electrode FCz of the response to rejection and acceptance feedback in the Kinder task (left) and topographical distribution of the rejection-acceptance difference wave (right). (b) ERP waveform in the time range of the FN at electrode FCz of the response to loss and win feedback in the Doors task (left) and topographical distribution of the loss-win difference wave (right).

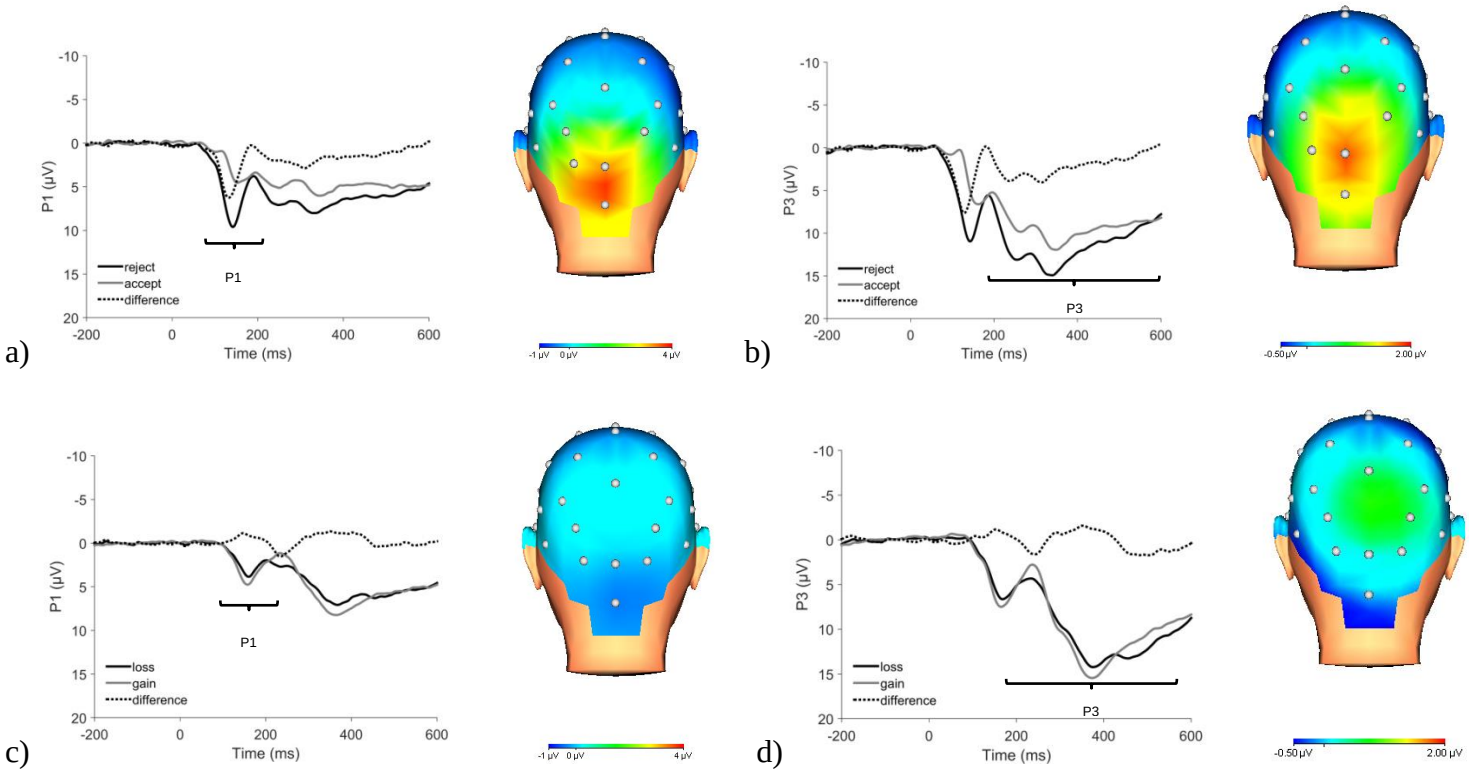

Figure 5. (a) ERP waveform in the time range of the P1 at electrode Iz of the response to rejection and acceptance feedback in the Kinder task (left) and topographical distribution of the rejection-acceptance difference wave (right). (b) ERP waveform in the time range of the P3 at electrode Oz of the response to rejection and acceptance feedback in the Kinder task (left) and topographical distribution of the rejection-acceptance difference wave (right). (c) ERP waveform in the time range of the P1 at electrode Iz of the response to monetary gain and loss feedback in the Doors task (left) and topographical distribution of the loss-gain difference wave (right). (d) ERP waveform in the time range of the P3 at electrode Oz of the response to monetary loss and gain feedback in the Doors task (left) and topographical distribution of the loss-gain difference wave (right).

Note: Amplitude scales differ between P1 and P3 head maps.

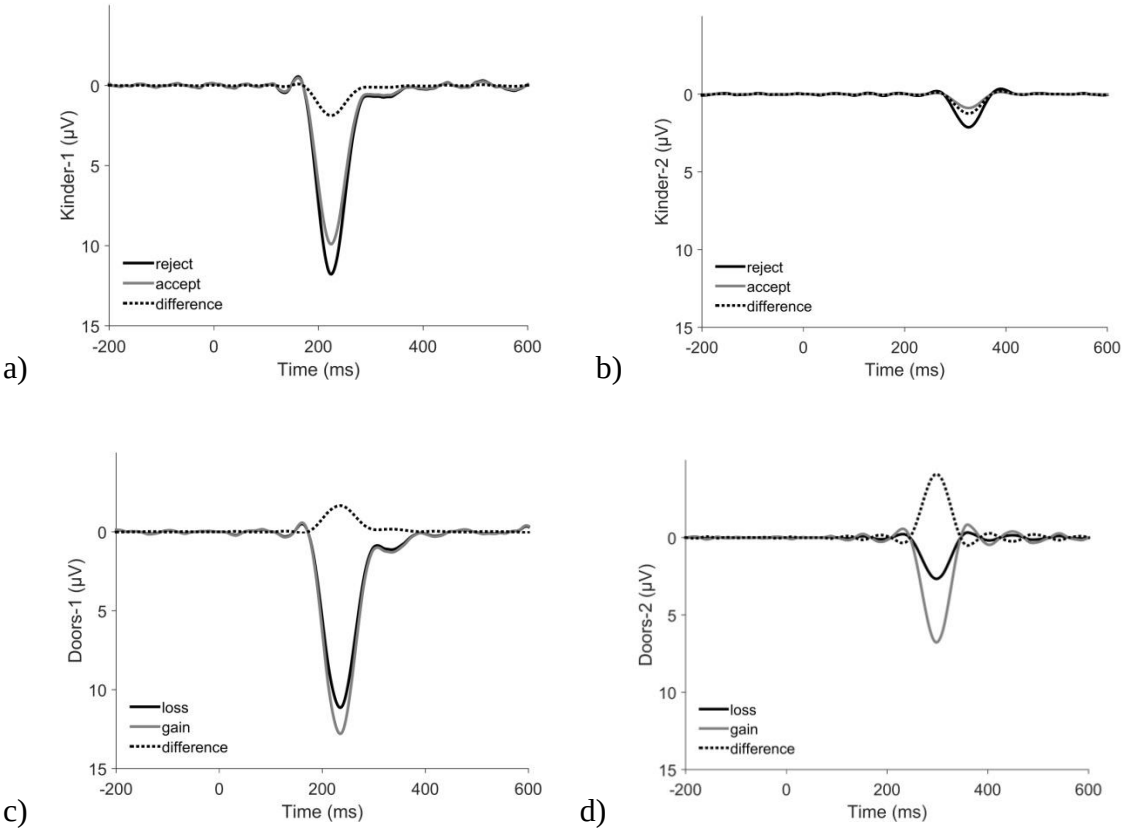

Figure 6. PCA-derived factor combinations from the Kinder (top) and Doors (bottom) tasks. (a) Kinder TF3SF1, (b) Kinder TF8SF1, (c) Doors TF3SF1, (d) Doors TF6SF1.

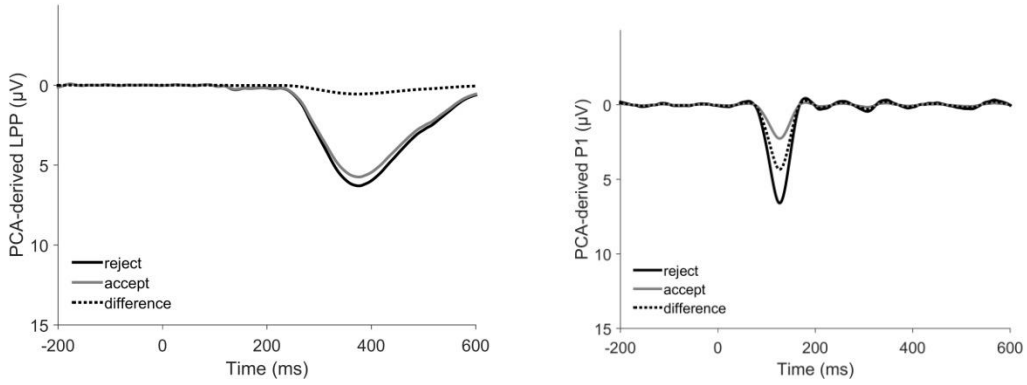

Figure 7. Additional PCA-derived factor combinations from the Kinder task: TF1SF2, which resembles the P3 (left), and TF6SF2, which resembles the P1 (right)

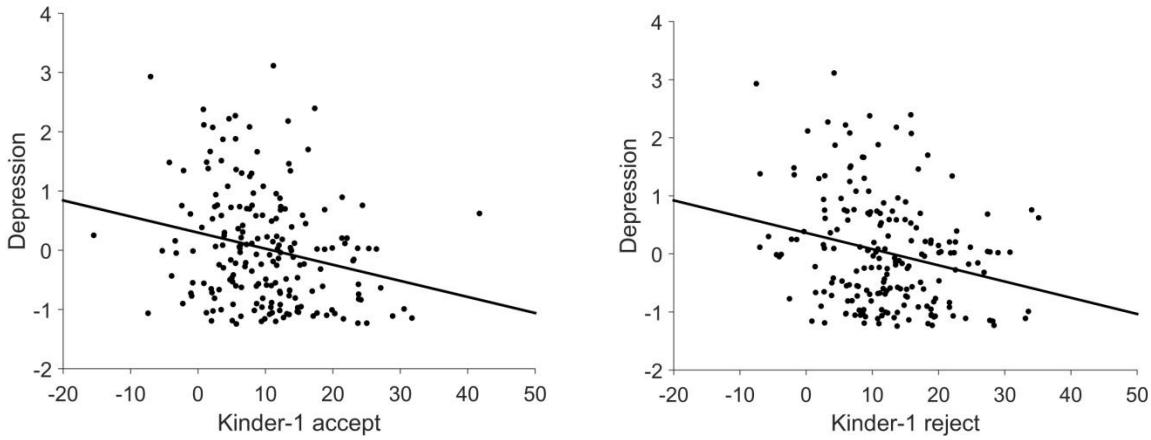

Figure 8. Scatter plots depicting the relationship between the latent depression variable and the response to social acceptance (left) and rejection (right).