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Maladaptive social information processing in childhood predicts young men's atypical amygdala reactivity to threat

Daniel Ewon Choe¹, Daniel S. Shaw¹, and Erika E. Forbes^{1,2}

¹Department of Psychology, University of Pittsburgh, PA, USA

²Department of Psychiatry, University of Pittsburgh, PA, USA

Abstract

Background—Maladaptive social information processing, such as hostile attributional bias and aggressive response generation, is associated with childhood maladjustment. Although social information processing problems are correlated with heightened physiological responses to social threat, few studies have examined their associations with neural threat circuitry, specifically amygdala activation to social threat.

Methods—A cohort of 310 boys participated in an ongoing longitudinal study and completed questionnaires and laboratory tasks assessing their social and cognitive characteristics between 10- and 12-years-old. At age 20, 178 of these young men underwent functional magnetic resonance imaging and a social threat task. At age 22, adult criminal arrest records and self-reports of impulsiveness were obtained.

Results—Path models indicated that maladaptive social information processing at ages 10 and 11 predicted increased left amygdala reactivity to fear faces, an ambiguous threat, at age 20 while accounting for childhood antisocial behavior, empathy, IQ, and socioeconomic status. Exploratory analyses indicated that aggressive response generation—the tendency to respond to threat with reactive aggression—predicted left amygdala reactivity to fear faces and was concurrently associated with empathy, antisocial behavior, and hostile attributional bias, whereas hostile attributional bias correlated with IQ. Although unrelated to social information processing problems, bilateral amygdala reactivity to anger faces at age 20 was unexpectedly predicted by low IQ at age 11. Amygdala activation did not mediate associations between social information

Correspondence: Daniel Ewon Choe, One Shields Ave., 1347 Hart Hall, Davis, CA 95616, USA; danchoe@ucdavis.edu.

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Supporting Information

Additional Supporting Information is provided along with the online version of this article.

Appendix S1 Hariri and colleagues' (2002) social threat task

Table S1 Correlations and descriptive statistics of fMRI behavioral data

Appendix S2/MRI Acquisition, Preprocessing, and Analysis

Appendix S3 Threat-related amygdala activity by contrast and region of interest (ROI): Main effect of task

Appendix S4 Descriptive Statistics and Correlations

Appendix S5 Maladaptive SIP and antisocial behavior (left amygdala)

Appendix S6 Threat-related whole brain activity for *Fear Faces* > *Shapes* contrast: Main effect of task

Appendix S7 Maladaptive SIP and antisocial behavior (whole brain)

Appendix S8 Threat-related whole brain activity for *Anger Faces* > *Shapes* contrast: Main effect of task

processing and number of criminal arrests, but both impulsiveness at age 22 and arrests were correlated with right amygdala reactivity to anger facial expressions at age 20.

Conclusions—Childhood social information processing and IQ predicted young men's amygdala response to threat a decade later, which suggests that childhood social-cognitive characteristics are associated with the development of neural threat processing and adult adjustment.

Keywords

Amygdala; social information processing; hostile attribution; aggression; functional magnetic resonance imaging

Introduction

Maladaptive social information processing (SIP) is believed to mediate effects of social threat on aggressive behavior and is associated with childhood maladjustment (Crick & Dodge, 1994). According to Crick and Dodge (1996), two salient SIP problems in childhood include tendencies to attribute hostile intent to others when intent is ambiguous and to generate aggressive behavioral responses to address perceived threats. During ambiguous social conflicts evoking feelings of threat, children with a *hostile attributional bias* are prone to misinterpreting others' intentions as being hostile via situational and socioemotional cues they encode (Orobio de Castro, Veerman, Koops, Bosch, & Monshouwer, 2002). Under similar conditions, socially-rejected and aggressive boys tend to show a hostile attributional bias (Dodge & Somberg, 1987) and generate, recall, or enact aggressive behavioral responses to perceived threats, increasing their risk of antisocial behavior (Criss & Shaw, 2003; Hyde, Shaw, & Moilanen, 2010). *Aggressive response generation* is moderately correlated with boys' actual reactive aggression and represents individuals' access to hostile schemas or cultivation of aggressive responses to novel threats that reinforce aggressive behavior (Calvete & Orue, 2012; Schultz & Shaw, 2003). Thus, children's hostile attributional bias and aggressive response generation contribute to social maladjustment, specifically reactive aggression and peer rejection.

SIP problems in childhood have longstanding consequences for adolescents and adults' antisocial behavior (e.g., Hyde et al., 2010). According to Dodge (2006), children's frequent and recent access to hostile representations and aggressive responses embed hostile schemas into memory, making them readily accessible in both threatening and benign situations. Accordingly, socioeconomic disadvantage is believed to contribute to maladaptive SIP by exposing children to interpersonal conflict and violence (Crick & Dodge, 1994), thereby repeatedly evoking threat responses and facilitating children's internalization of hostile schemas. Thus, children of low socioeconomic status (SES) are at risk of developing stable cognitive tendencies that contribute to their hypervigilance and reactive aggression. Such enduring patterns of SIP may have an underlying biological basis as researchers have linked socioeconomic disadvantage in childhood with increased amygdala reactivity to threatening facial expressions in young adulthood (Gianaros et al., 2008), which suggests that early harsh environments alter the development of brain function involved in threat processing.

No study we know of has examined relations between neural threat processing and maladaptive SIP despite the latter's association with heightened physiological responses to social threat (Chen & Matthews, 2001). Experimental evidence indicates that hostile men show excessive increases in blood pressure, heart rate, and cortisol reactivity to social threat (Suarez, Kuhn, Schanberg, Williams, & Zimmerman, 1998). Functional magnetic resonance imaging (fMRI) may help designate neural substrates of hostile attributional bias and aggressive response generation as underlying mechanisms of social maladjustment. In the current study, we use fMRI to examine whether maladaptive SIP in childhood is associated with young men's amygdala reactivity to threat, and whether amygdala activation mediates effects of childhood characteristics on adjustment outcomes in young adulthood.

Although other brain regions are implicated in aggressive behavior and socioemotional processing, the amygdala is considered a key component of threat processing and is the brain structure most consistently related to emotional processing in the fMRI literature (Sabatinelli et al., 2011). A recent study found that damage to the basolateral amygdala contributed to adults' hypervigilance to threat cues from fearful facial expressions; hence, the critical role of the amygdala in the threat vigilance system (Terbug et al., 2012). Accordingly, atypical amygdala activation to threat may reflect biases in threat detection that impede processing of social information. Thus, the current study focuses on the amygdala as a hypothesized region of interest, but we acknowledge that other brain areas are involved in social threat processing and present whole brain findings as supplemental appendices.

Increased amygdala reactivity to social threat is associated with reactive aggression (Coccaro, McCloskey, Fitzgerald, & Phan, 2007), which suggests a link between SIP problems and threat-related amygdala response. The amygdala has several putative functions related to emotion-behavior integration thought to modulate aggressive behavior, such as fear conditioning, memory of emotional stimuli, and recognition of emotional facial expressions (Adolphs, 2008). Facial expressions of fear and anger are salient stimuli for eliciting amygdala activity (Adams, Gordon, Baird, Ambady, & Kleck, 2003). Fear facial expressions indicate a potential and ambiguous environmental threat, whereas anger facial expressions indicate a clear and direct threat; both are associated with increased activation in the left amygdala (Adams et al., 2003; Hardee, Thompson, & Puce, 2008). Left amygdala reactivity to anger faces is positively associated with lifetime aggressive behavior (Coccaro et al., 2007). Despite more evidence showing activation in the left amygdala in response to threatening stimuli, we examined both hemispheres of the brain to consider lateralization of amygdala reactivity to social threat.

Although children with a hostile attributional bias could misinterpret ambiguity in neutral facial expressions as threatening, fear and anger facial expressions elicit more robust amygdala activity than neutral faces or other negative, non-threat-related expressions, such as sadness (Adams et al., 2003; Coccaro et al., 2007). Moreover, children and adults visually detect anger and fear faces faster than happiness and sadness (LoBue, 2009); thus, anger and fear faces may elicit exaggerated amygdala activity in individuals with SIP problems, as children and young adults are highly vulnerable to intentionality biases when making rapid decisions (Dodge, 2006; Rosset, 2008). Exaggerated amygdala reactivity to threat,

especially in ambiguous situations, may disrupt adolescents' transition into young adulthood by skewing social cognitive processing and increasing risk for reactive aggression. Disruptions while transitioning to young adulthood have serious consequences for adult mental health and psychopathology, as progress through this developmental transition mediates effects of stressors in childhood and adolescence on well-being in adulthood (Schulenberg, Sameroff, & Cicchetti, 2004). Thus, neuroimaging studies and experiments suggest that SIP problems in childhood may be related to increased amygdala reactivity to threatening facial expressions in young adulthood.

This longitudinal study is one of the first to use *f*MRI to elucidate brain correlates of social maladjustment by examining prospective relations between SIP problems in childhood and amygdala reactivity to threat in young adult men. The current sample was at risk for developing antisocial behavior because of its gender and low-income status. We examined effects of SIP problems and risk correlates assessed between ages 10 and 12 on amygdala reactivity to fear and anger facial expressions at age 20. Risk correlates of SIP problems and amygdala function pose potential problems when relating SIP to amygdala activity, as third variables could contribute to spurious effects. Thus, we included childhood antisocial behavior, family SES, and race as covariates. We also accounted for childhood empathy to capture motivations for moral behavior that inform aggressive behavioral choices and are distinct from cognitive processes highlighted in most SIP studies. Because of its associations with hostile attributional bias and aggression, child IQ also was included as a covariate (Choe, Lane, Grabell, & Olson, 2013; Runions & Keating, 2007). We hypothesized that childhood SIP problems would predict young men's increased amygdala reactivity to fear and anger faces, while accounting for this diverse group of covariates. We then deconstructed SIP into hostile attributional bias and aggressive response generation to specify which component was associated with altered amygdala activity. Lastly, we examined whether amygdala reactivity to threat mediated effects of childhood SIP problems on number of arrests and impulsiveness in young adulthood, our indicators of adult maladjustment.

Method

Participants and Procedure

A cohort of 310 toddler boys was recruited in 1991 from Women, Infants, and Children Nutritional Supplement Program Clinics in a metropolitan area for an ongoing longitudinal study (Shaw, Hyde, & Brennan, 2012). Mean monthly family income when boys were 1.5-years-old was approximately \$1,045. Boys were assessed 14 times with their families (except at age 20) in their homes and/or the laboratory from ages 1.5- to 20-years-old. At age 20, 186 (60%) of these young men returned to the laboratory and were administered computerized experimental tasks while undergoing *f*MRI. Before neuroimaging, participants were required to take a saliva drug test to ensure they were not under the influence of drugs, and appropriate measures were taken to reduce confounds of substance use (e.g., controlling for self-reported alcohol, tobacco, and marijuana use at age 20 did not alter results). Eight participants who underwent *f*MRI were excluded for the following reasons: one for missing over 20% of amygdala coverage, three for being under the influence of drugs or psychosis,

one for not responding during tasks, and three for distorted data. The final sample included 178 young men (54% Caucasian, 38% African American, 8% other). At age 22, 164 (92%) participants completed questionnaires in the lab and adult criminal court records were ascertained through an online database. All participants provided informed consent.

Measures

Age 10 and 11 maladaptive social information processing—Interviewers presented the child with eight vignettes and accompanying pictures (Dodge & Somberg, 1987). In each vignette, the behavior of another boy leads to a negative outcome for the target child (e.g., being bumped), with the other boy's intentions left ambiguous. Following each vignette, the child was asked to imagine that he were the target child, to make an attribution of the other boy's intent (e.g., "did the other boy hurt you on purpose?"), and to indicate how he would respond (e.g., "yell at the boy"). Hostile attributions were coded as 1's if the child responded that the other boy performed the action on purpose, whereas attributions of non-hostile or benign intent were coded as 0's. Responses were summed at each age to create two scales of hostile attributional bias at ages 10 ($\alpha = .65$) and 11 ($\alpha = .64$). Hypothetical responses to situations indicating retaliatory aggression or threats were coded as 1's, whereas verbally engaging, but non-hostile or ambiguous (e.g., doing nothing) responses were coded as 0's. Responses were summed at each age to create two scales of aggressive response generation at ages 10 ($\alpha = .76$) and 11 ($\alpha = .71$). Aggressive response generation scores at ages 10 and 11 were moderately correlated ($r = .42, p < .001$), as were hostile attributional bias scores ($r = .47, p < .001$). Aggressive response generation and hostile attributional bias were modestly correlated with each other at ages 10 ($r = .18, p = .005$) and 11 ($r = .26, p < .001$). All scores were standardized and averaged into an index of maladaptive SIP ($\alpha = .78$). We also examined unique effects of mean hostile attributional bias and aggressive response generation scales on amygdala activation in an exploratory model.

Age 10 and 11 antisocial behavior—The child reported his antisocial behavior on an adapted Self-Report of Delinquency Questionnaire (Elliot, Huizinga, & Ageton, 1985) using a 3-point scale (0 = never, 1 = once/twice, 2 = more often). Scores of all 26 items were summed into scales at ages 10 ($\alpha = .75$) and 11 ($\alpha = .91$), which were highly correlated with each other ($r = .65, p < .001$) and averaged into a composite.

Age 10 and 11 demographic information—Primary caregivers reported on caregivers' highest levels of education, occupational statuses, and child's race (1 = Caucasian, 2 = African American, 3 = other). We approximated SES by aggregating caregivers' education and occupation following Hollingshead (1975). SES scores at ages 10 and 11 were highly correlated ($r = .71, p < .001$) and averaged with a mean of 31.02 ($SD = 9.31$), placing most parents in the range of clerical and sales workers, and skilled craftsmen.

Age 11 general cognitive ability—A popular, two-scale version of the Wechsler Scales of School-Age Intelligence was administered to the child (Wechsler, 1991). Scaled scores for Block Design ($M = 9.22, SD = 4.11$) and Vocabulary ($M = 9.06, SD = 3.47$) subtests were moderately correlated ($r = .46, p < .001$). We derived a prorated full-scale IQ score (M

= 94.94, $SD = 18.69$) by converting raw scores to scaled scores and contrasting them to well-established norms.

Age 12 empathy—The child rated his empathetic behaviors on the Child and Adolescent Temperament Scale (Lahey et al., 2008) using a 4-point scale (1 = not at all; 4 = very much/very often). Scores of 10 items were summed into an empathy scale at age 12 ($\alpha = .83$).

Age 20 amygdala response to threat—During fMRI, the participant completed a widely used challenge paradigm (Hariri et al., 2002), which we will refer to as a social threat task. As described in online supplementary Appendix S1, the task interleaves four blocks of an emotional faces-matching condition (A) with five blocks of a shapes-matching sensorimotor control condition (B). Accuracy and reaction times were monitored and examined in relation to study variables and for performance differences by task condition (see Appendix S1 and supplementary Table S1). Behavioral data did not differ by emotion block and accounting for these data did not alter our main results.

Adult criminal court records of arrests—We obtained official criminal court records of arrests in Pennsylvania, which we considered a salient indicator of social maladjustment for our low-income sample of young men. Among our final sample, 46 participants (25.8%) had documented arrests. Number of arrests ($M = .72$, $SD = 1.58$) had a positively skewed distribution (skewness = 3.13, SE of skewness = 12.69; kurtosis = 12.69, SE of kurtosis = 12.69). We applied a logarithmic transformation ($\log_{10}$) to this variable after adding a constant of 1 to all values. Distributional values improved after the transformation (skewness = 1.65, SE of skewness = .18; kurtosis = 1.56, SE of kurtosis = .36).

Age 22 impulsiveness—Participants completed the revised Barratt Impulsiveness Scale, a widely-used questionnaire assessing the behavioral construct of impulsiveness (Patton, Stanford, & Barratt, 1995), which we considered an indicator of maladjustment due to its role in reactive aggression (Dodge, 2006). Participants responded to items on a 4-point scale (1 = rarely/never; 4 = almost always/always). Total impulsiveness was computed by summing responses to all 30 items ($\alpha = .80$).

fMRI Acquisition, Preprocessing, and Analysis

Participants underwent fMRI with a Siemens 3T Tim Trio scanner (Siemens Medical Solutions, Erlangen, Germany). Blood oxygenation level-dependent (BOLD) functional images were acquired with a gradient echo planar imaging sequence covering 34 axial slices (3.0 mm thick), aligned with the AC-PC plane, and encompassing the entire cerebrum and most of the cerebellum (repetition time/echo time = 2000/25 ms, field of view = 20 cm, matrix = 64×64). Whole-brain image preprocessing and analysis was completed with SPM8 and our standard procedure: segmented structural images; realigned, unwarped, co-registered, normalized, and smoothed functional images; eliminated participants with poor coverage, low response rates, or excessive movement (Forbes et al., 2010). Preprocessed data were analyzed using first-level random effects models within each subject to estimate task-specific amygdala BOLD activation. Second-level tests computed predetermined condition effects using one-sample t -tests ($p < .05$, extent = 10 voxels, corrected using

family-wise error) for two contrasts of interest: (a) *Fear Faces* > *Shapes*, (b) *Anger Faces* > *Shapes*. Appendix S2 presents a detailed account of fMRI acquisition, preprocessing, and analysis.

Data Analytic Plan

Mean estimates of peak activation within significant brain clusters were extracted from SPM8 separately for the left and right amygdala for analysis in SPSS 19 (see Appendix S3 for amygdala activity by task condition and ROI). After preliminary analysis of missing data, attrition, descriptive statistics and bivariate relations (see Appendix S4), data were transferred to *Mplus* 5.21 for estimation in path models linking childhood variables with amygdala function in young adulthood. We estimated models in which separate clusters of left and right amygdala activation at age 20 were regressed on maladaptive SIP and risk correlates assessed between ages 10 and 12. Within-time correlations estimated shared variance between amygdala activation patterns in young adulthood, risk factors in childhood, and between race and SES. In an exploratory model, we deconstructed SIP into hostile attributional bias and aggressive response generation to specify which component accounted for variance in amygdala reactivity. Lastly, we estimated a series of mediation models testing pathways from significant childhood predictors to their corresponding amygdala activity in young adulthood to later behavioral outcomes, examining whether amygdala activity mediated associations from childhood to young adulthood. Race and family SES at ages 10 and 11 were covariates in all models.

Model results include chi-square (χ^2), comparative fit index (CFI), root mean square error of approximation (RMSEA) and its 90% confidence interval (CI), and standardized root mean square residual (SRMR). SRMR values < .10 are considered favorable. RMSEA values .05 indicate close approximate fit, while values .09 reflect reasonable error of approximation. CFI values > .90 reflect reasonably good fit. Only standardized beta estimates are reported for effects.

Missing Data and Attrition Analyses

Participants who underwent fMRI at age 20 did not differ from participants in the attrition group ($n = 124$) on SES, family income, race, or parent marital status at recruitment at age 1.5. The attrition group reported higher levels of aggressive response generation at age 10 ($M = .95$, $SD = 1.43$) than active participants ($M = .44$, $SD = 1.17$), $t(61) = 2.03$, $p = .047$, 95% CI [.01, 1.01]. No other pattern of systematic missing data was observed. Among the final sample, 20% were missing data on age 12 empathy, 19% were missing data on age 11 IQ, 14% were missing data on age 10 and 11 SIP, 11% were missing data on age 10 and 11 SES and antisocial behavior, and 7% were missing data on age 22 impulsiveness. Little's missing completely at random (MCAR) test with expectation maximization was not significant, $\chi^2(113) = 103.99$, $p = .716$, indicating that missing data were MCAR and did not violate assumptions underlying the use of full information maximum likelihood estimation.

Results

As hypothesized, maladaptive SIP at ages 10 and 11 predicted increased left amygdala reactivity to fear facial expressions (vs. shapes) in young adulthood ($R^2 = .04$; see Figure 1). Maladaptive SIP and antisocial behavior were positively related to one another, and both were negatively associated with empathy and IQ (see Appendix S5 for scatterplot and Appendix S6 for whole-brain findings). Although not shown, higher family SES predicted higher IQ scores ($\beta = .34, p < .001$), while racial-ethnic minority status predicted higher antisocial behavior ($\beta = .23, p = .001$) and lower IQ scores ($\beta = -.16, p = .042$).

Contrary to our hypothesis, maladaptive SIP in childhood was unrelated to amygdala reactivity to anger facial expressions (vs. shapes) in young adulthood (see Figure 2). Unexpectedly, high IQ scores at age 11 predicted decreased reactivity to anger facial expressions within the left ($R^2 = .06$) and right amygdala ($R^2 = .04$; see Appendix S7 for scatterplots and Appendix S8 for whole-brain findings). Childhood variables were related to one another similarly as in the previous model.

We then estimated a path model (not shown) similar to the previous except maladaptive SIP was separated into hostile attributional bias and aggressive response generation. The model predicting amygdala reactivity to fear facial expressions achieved an acceptable fit: $\chi^2(2, N = 178) = 4.59, p = .101$. CFI = .99. RMSEA = .09, 90% CI [.00, .19]. SRMR = .01. Aggressive response generation predicted increased reactivity to fear faces within the left amygdala ($\beta = .19, p = .031, R^2 = .05$), but only marginally within the right amygdala ($\beta = .15, p = .072, R^2 = .04$). Left and right amygdala activity were positively related ($r = .72, p < .001$). Hostile attributional bias was positively related to aggressive response generation ($r = .29, p < .001$) and negatively related to IQ ($r = -.16, p = .041$), whereas aggressive response generation was positively related to antisocial behavior ($r = .28, p < .001$) and negatively related to empathy ($r = -.21, p = .008$). Correlations and effects of family SES and child race were similar to those in previous models.

Lastly, we examined maladaptive SIP at ages 10 and 11 as the predictor and left amygdala reactivity to fear faces at age 20 as the potential mediator in two separate mediation models: one model included number of criminal arrests in young adulthood as the outcome, whereas the other model included age 22 impulsiveness. Our models did not support a mediating role of amygdala activity for childhood SIP and young adult outcomes.¹ Left amygdala reactivity to fear faces was uncorrelated with criminal arrests ($r = .10, p = .203$) and impulsiveness at age 22 ($r = .06, p = .437$). However, as shown in Appendix S4, number of criminal arrests was positively correlated with age 10 and 11 maladaptive SIP ($r = .17, p = .033$) and antisocial behavior ($r = .20, p = .014$), supporting the predictive validity of these childhood variables.

For our mediation models with IQ, left and right amygdala reactivity to anger faces were unrelated to criminal arrests and impulsiveness in young adulthood, and mediation was not supported. However, as shown in Appendix S4, bivariate correlations revealed greater right

¹Please contact the first author for detailed results of mediation models.

amygdala reactivity to anger faces was associated with fewer criminal arrests ($r = -.20, p = .008$) and higher impulsiveness ($r = .17, p = .031$) at age 22, although left amygdala reactivity to anger faces was unrelated to either ($r = -.14, p = .057$ and $r = .11, p = .159$, respectively).

Discussion

Among multiple childhood risk factors, we found SIP problems uniquely predicted men's elevated amygdala activity when viewing fear facial expressions in young adulthood. Supporting our hypothesis and previous evidence that hostile men show exaggerated cardiovascular and neuroendocrine reactivity to threat (Suarez et al., 1998), an aggregate score of hostile attributional bias and aggressive response generation at ages 10 and 11 predicted increased left amygdala reactivity to fear faces at age 20. Young men's IQ scores at age 11 predicted their reduced bilateral amygdala reactivity to anger faces at age 20. We found no evidence of amygdala activation mediating associations between childhood variables and indicators of social maladjustment in young adulthood, although impulsiveness at age 22 and number of criminal arrests were correlated with right amygdala reactivity to anger faces. We also validated our measure of maladaptive SIP as capturing a stable construct relevant to the development of social maladjustment by demonstrating its positive association with criminal arrests.

To our knowledge, this is the first study to link maladaptive SIP with increased amygdala reactivity to social threat. Specifically, our finding that SIP was related to left amygdala activity indicates an association between maladaptive threat detection and aggressive responding in childhood and amygdala hyper-sensitivity to fear facial expressions in young adulthood. Although SIP problems predicted amygdala reactivity to fear expressions (and not anger), this is not surprising given the ambiguity in fear facial expressions (Adams et al., 2003). Fear has been found to be more difficult for young people to recognize than other emotional expressions (Marsh & Blair, 2008), and SIP problems often manifest during interpersonal conflicts in which others' intentions are ambiguous (Dodge & Somberg, 1987). Without clear threat-relevant cues to rely on, young men with SIP problems may misconstrue fear facial expressions as signs of direct threat and interpersonal hostility.

Aggressive response generation was more closely associated with risk for maladjustment and threat-related amygdala activity than a hostile attributional bias, suggesting that these components of SIP have distinct neural substrates. Our exploratory model supported previous research with this sample indicating that a tendency toward responding to social conflict with aggression was associated with antisocial behavior and low empathy in childhood (Criss & Shaw, 2003; Hyde et al., 2010; Schultz & Shaw, 2003). Aggressive response generation has been linked to reactive aggression (Calvete & Orue, 2012), suggesting that impulsive aggression is due more to the accessibility of aggressive behavioral responses than over-attributions of hostile intent. Although a meta-analysis reported associations between hostile attributional bias and aggressive behavior (Orobio de Castro et al., 2002), this may have been due to studies combining hostile attributions and aggressive response generation to increase effect sizes (Dodge, 2006).

Aggressive response generation may be related to amygdala hyper-sensitivity to ambiguous threat cues in particular, given that the amygdala is thought to play a key role in guiding behavioral choices under uncertain conditions (Adolphs, 2008). While researchers have linked fear processing to increased amygdala activity in aggressive youth (Coccaro, Sripada, Yanowitch, & Phan, 2011), others have reported that impaired fear-recognition is related to amygdala hyporeactivity, antisocial personality disorder, and psychopathy (Marsh & Blair, 2008). Low empathy characterizes callous-unemotional traits (Frick & Nigg, 2012), which have been linked with reduced amygdala reactivity to fear faces in boys with conduct disorder (Marsh et al. 2008). While childhood empathy was unrelated to young men's amygdala activity, it was concurrently associated with antisocial behavior and SIP problems. Because we utilized a community sample rather than a criminal or antisocial population, we included childhood empathy and antisocial behavior as predictors of amygdala activation instead of violent behavior or psychopathy in adulthood. These childhood variables were unrelated to amygdala reactivity, as young men's most robust risk factors were SIP problems and low IQ.

When we considered other factors relevant to SIP, general cognitive ability emerged as the sole predictor of young men's amygdala reactivity to anger facial expressions. Increased amygdala reactivity to anger faces has been linked with aggressive behavior (Coccaro et al., 2007), suggesting boys' low IQ scores may be associated with altered threat-related amygdala function and reactive aggression. It is possible that poor verbal and visuospatial abilities impede problem-solving involved in non-aggressive responding, such as the identification of benign intent. Previous research and the current study indicate that boys' low IQ is associated with greater antisocial behavior and hostile attributional bias, but not aggressive response generation (Choe et al., 2013; Hyde et al., 2010; Runions & Keating, 2007). Moreover, meta-analytic findings indicate that low general cognitive ability and SIP problems, while interrelated, contribute distinctively to child maladjustment (Orobio de Castro et al., 2002). Overall, these findings suggest that low general cognitive ability is associated with erroneous threat detection, which in turn increases the likelihood of aggression and antisocial behavior.

Low child IQ was the only variable related to family SES and may have been a proxy for socioeconomic disadvantage, which also has been linked to increased amygdala reactivity to anger facial expressions (Gianaros et al., 2008). Early disadvantage may alter the development of neural stress responses in threat processing through general cognitive abilities involved in facial recognition, such as perceptual processing and working memory (Marsh & Blair, 2008). Hostile attributional bias has been shown to mediate effects of socioeconomic disadvantage on young people's increased cardiovascular reactivity to social threat (Chen & Matthews, 2001). Both low IQ and hostile attributional bias have been linked to socioeconomic disadvantage and high physiological reactivity to threat, suggesting that these interrelated factors may operate in tandem to influence the brain's threat processing. Thus, boys' low IQ and hostile attributional bias may reflect the effects of disadvantage on their stress regulatory systems, evident in this study as increased amygdala reactivity to clear threat cues in anger facial expressions.

Although amygdala activity did not emerge as a mediator of childhood variables on arrests or impulsiveness in young adulthood, young men's right amygdala reactivity to anger facial expressions was positively correlated with impulsiveness and negatively correlated with number of arrests. This inverse association with arrests is inconsistent with research linking heightened left amygdala reactivity to anger faces with greater lifetime aggression in adults (Coccaro et al., 2007). Young men in our low-income sample may have been arrested for drug and property offenses that are unrelated to SIP rather than for violent crimes, which may explain the negative correlation between amygdala activity and total arrests. Theoretically, we would expect SIP problems to directly contribute to problems of reactive aggression, which we did not measure at age 20 or 22. However, young men's impulsiveness, which amplifies risk for reactive aggression (Dodge, 2006), was positively associated with right amygdala reactivity to anger faces, aligning with Coccaro and colleagues' work (2007, 2011) implicating the amygdala's role in the initiation and expression of aggressive impulses and threat detection.

High levels of SIP problems and antisocial behavior at ages 10 and 11 were correlated with a greater number of criminal arrests in young adulthood. To our knowledge, this is the first evidence linking SIP problems in childhood to arrest records in a community sample. Boys who were hypervigilant or antisocial were more likely to be arrested by their early 20's. As alluded to above, we may have found evidence of mediation had we focused on violent offenses; however, reducing arrest records to only violent crimes would have yielded an outcome with insufficient variability to explain in analyses. Perhaps with a larger sample of antisocial men, we might have found mediational pathways from childhood vulnerabilities to young men's brain function and behavior. Nonetheless, we have a limited understanding of how amygdala hyperreactivity to anger and fear faces affected the behavior of young men in our sample. Our findings are correlational and require replication with samples drawn from other populations and experimental analogues to elucidate causal relations between social-cognitive vulnerabilities, amygdala reactivity to threat, and adjustment outcomes.

Our findings suggest school-age boys' SIP and IQ are associated with affective threat processing within the amygdala almost a decade later; however, we did not assess SIP and amygdala activity at the same age or amygdala function longitudinally. Although we are uncertain of whether young men in our sample showed SIP problems at age 20, intentionality and attentional biases to threat-relevant cues are salient problems for young adults (Dodge, 2006; LoBue, 2009; Rosset, 2008). Identifying emotional facial expressions is a multifaceted task requiring visual scanning, perceptual processing, effortful attention, working memory, and semantic processing, suggesting that SIP problems may be related to activity in other brain regions involved in socioemotional processing, such as the ventromedial prefrontal cortex (PFC), thalamus, superior colliculus, and fusiform gyrus (Marsh & Blair, 2008). Whole brain analyses reported in Appendices S5 and S7 showed right dorsolateral PFC reactivity to anger faces was positively associated with maladaptive SIP, while left occipital cortex reactivity to fear faces was negatively associated with age 11 IQ. As maturation rates of brain areas involved in threat processing likely differ from childhood to adulthood, longitudinal fMRI studies with a range of behavioral and cognitive measures may be best suited to delineate neural substrates of SIP. Moreover, exploring the

amygdala's functional connections with other brain regions may reveal networks of activation that mediate relations between SIP and aggressive behavior.

Although the transition to young adulthood is studied less often than other developmental phases, it is critical to adult mental health and psychopathology as it mediates experiences in childhood and adolescence on well-being in adulthood (Schulenberg et al., 2004). The onset of serious conduct problems by 11-years-old doubles children's risk of developing antisocial personality disorder by adulthood (Frick & Nigg, 2012); thus, examining maladaptive SIP and other risk factors at this phase of childhood in relation to neural threat circuitry in young adulthood may elucidate biomarkers of chronic antisocial behavior. In one of the first studies to explore neural correlates of maladaptive SIP, we found that SIP problems and low IQ scores in childhood predicted young men's altered amygdala circuitry 9 to 10 years later, and on occasion, these factors were related to social maladjustment in adulthood. Our findings indicate an enduring association between early social-cognitive characteristics and the development of neural threat circuitry and adult adjustment.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Key points

- Social information processing problems are associated with reactive aggression and physiological hyperreactivity to social threat in children and young adults.
- Increased amygdala reactivity to salient threat-cues from anger and fear facial expressions is associated with reactive aggression and antisocial behavior.
- Results indicate that childhood social information processing problems are associated with young men's increased amygdala reactivity to fear facial expressions, an ambiguous threat, whereas low child IQ is associated with increased amygdala reactivity to anger facial expressions, a clear threat.
- Although the amygdala did not mediate associations between childhood vulnerabilities and young men's maladjustment, amygdala reactivity to anger facial expressions was correlated with young men's criminal arrests and impulsiveness.

Figure 1.

Maladaptive social information processing predicts increased left amygdala reactivity to fear faces. Model controls for child race and family socioeconomic status at ages 10 and 11. All estimates are standardized and nonsignificant effects are dashed lines. e = residual error. $\chi^2(2, N = 178) = 4.62, p = .100$. CFI = .99. RMSEA = 0.09, 90% CI [.00, .19]. SRMR = 0.01. * $p < .05$. ** $p < .01$. *** $p < .001$.

Figure 2.

General cognitive ability predicts decreased bilateral amygdala reactivity to anger faces. Model controls for child race and family socioeconomic status at ages 10 and 11. All estimates are standardized and nonsignificant effects are dashed lines. e = residual error. $\chi^2(2, N = 178) = 2.96, p = .227$. CFI = .99. RMSEA = 0.05, 90% CI [.00, .17]. SRMR = 0.02. * $p < .05$. ** $p < .01$. *** $p < .001$.