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Title

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Permalink

Journal

ISSN

Authors

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Publication Date

DOI

Supplemental Material

Peer reviewed

Maladaptive Social Information-Processing in Childhood Predicts Young Men's

Atypical Amygdala Reactivity to Threat

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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-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.

Maladaptive Social Information-Processing in Childhood Predicts Young Men's

Atypical Amygdala Reactivity to Threat

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, 2009).

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., 2009). 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 hyper-vigilance 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 *f*MRI 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 *f*MRI literature (Sabatinelli et al., 2011). A recent

study found that damage to the basolateral amygdala contributed to adults'

hyper-vigilance 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 fMRI 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 (α = .65) and 11 (α

= .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 (α = .76) and 11 (α = .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 (α = .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

(α = .75) and 11 (α = .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 (α = .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 Appendix A, 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 A). 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 (log10) 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 (α = .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 x 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 B

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 C for amygdala activity by task condition

and ROI). After preliminary analysis of missing data, attrition, descriptive

statistics and bivariate relations (see Appendix D), data were transferred to

M*plus* 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 *f*MRI 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 E for scatterplot and

Appendix F 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 G for scatterplots and Appendix H 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 D, number of criminal arrests was

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

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 D, bivariate correlations revealed greater right 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 reactively 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 hypo-reactivity, 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., 2009; 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 hyper-

vigilant 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 meditational pathways from childhood vulnerabilities to young men's

brain function and behavior. Nonetheless, we have a limited understanding

of how amygdala hyper-reactivity 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 E and G 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 *f*MRI 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.

Key points

- Social information-processing problems are associated with reactive aggression and physiological hyper-reactivity 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.

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References

Adams, R. B., Gordon, H. L., Baird, A. A., Ambady, N., & Kleck, R. E. (2003).

Effects of gaze   on amygdala sensitivity to anger and fear faces. *Science*,

300, 1536.

Adolphs, R. (2008). Fear, faces, and the human amygdala. *Current Opinion in*

Neurobiology, *18*,

166–172.

Calvete, E., & Orue, I. (2012). Social information processing as a mediator

between cognitive

schemas and aggressive behavior in adolescents. *Journal of Abnormal*

Child Psychology, 40, 105–117.

Carré, J. M., Fisher, P. M., Manuck, S. B., & Hariri, A. R. (2012). Interaction

between trait

anxiety and trait anger predict amygdala reactivity to anger facial

expressions in men but   not women. *Social Cognitive and Affective*

Neuroscience, *7*, 213–221.

Chen, E., & Matthews, K. A. (2001). Cognitive appraisal biases: An approach

to understanding

the relation between socioeconomic status and cardiovascular

reactivity in children. *Annals of Behavioral Medicine*, *23*, 101–111.

Choe, D. E., Lane, J. D., Grabell, A. S., & Olson, S. L. (2013). Developmental

precursors of

young school-age children's hostile attribution bias. *Developmental*

Psychology, *49*, 2245–2256.

Coccaro, E. F., McCloskey, M. S., Fitzgerald, D. A., & Phan, K. L. (2007). Amygdala and

orbitofrontal reactivity to social threat in individuals with impulsive

aggression. *Biological Psychiatry*, *62*, 168–178.

Coccaro, E. F., Sripada, C. S., Yanowitch, R. N., & Phan, K. L. (2011). Corticolimbic function in

impulsive aggressive behavior. *Biological Psychiatry*, *69*, 1153–1159.

Crick, N. R., & Dodge, D. A. (1994). A review and reformulation of social information-

processing mechanisms in children's social adjustment. *Psychological

Bulletin*, *115*, 74–101.

Crick, N. R., & Dodge, D. A. (1996). Social information-processing mechanisms in reactive and

proactive aggression. *Child Development*, *67*, 993–1002.

Criss, M. M., & Shaw, D. S. (2003). Mother–son positive synchrony in middle childhood:

Relation to antisocial behavior. Social Development, 12, 379–400.

Orobio de Castro, B. O., Veerman, J. W., Koops, W., Bosch, J. D., & Monshouwer, H. J. (2002).

Hostile attribution of intent and aggressive behavior: A meta-analysis.

Child Development, *73*, 916–934.

Dodge, K. A. (2006). Translational science in action: Hostile attributional style

and the development of aggressive behavior problems. *Development*

and *Psychopathology, 18,* 791–814.

Dodge, K. A., & Somberg, D. R. (1987). Hostile attributional biases among

aggressive boys are   exacerbated under conditions of threats to the self.

Child Development, *58*, 213–224.

Elliot, D. S., Huizinga, D., & Ageton, S. S. (1985). *Explaining delinquency and*

drug use.

Thousand Oaks: Sage.

Forbes, E. E., Ryan, N. D., Phillips, M. L., Manuck, S. B., Worthman, C. M.,

Moyles, D. L. et al.

(2010). Healthy adolescents' neural response to reward: Associations

with puberty, positive affect, and depressive symptoms. *Journal of the*

American Academy of Child & Adolescent Psychiatry, *49*, 162–172.

Frick, P. J., & Nigg, J. T. (2012). Current issues in the diagnosis of attention

deficit hyperactivity

disorder, oppositional defiant disorder, and conduct disorder. *Annual*

Review of Clinical   *Psychology*, 8, 77–107.

Gianaros, P. J., Horenstein, J. A., Hariri, A. R., Sheu, L. K., Manuck, S. B.,

Matthews, K. A., &

Cohen, S. (2008). Potential neural embedding of parental social

standing. *Scan*, *3*, 91–96.

Hardee, J. E., Thompson, J. C., & Puce, A. (2008). The left amygdala knows

fear: Laterality in

the amygdala response to fearful eyes. *Scan*, *3*, 47–54.

Hariri, A. R., Mattay, V. S., Tessitore, A., Kolachana, B., Fera, F., Goldman, D., et al. (2002).

Serotonin transporter genetic variation and the response of the human amygdala. *Science*, *19*, 400–403.

Hollingshead, A. B. (1975). Four-factor index of social status. Unpublished manuscript, Yale

University, New Haven, CT.

Hyde, L. W., Shaw, D. S., & Moilanen, K. L. (2010). Developmental precursors of moral   disengagement and the role of moral disengagement in the development of antisocial   behavior. *Journal of Abnormal Child Psychology*, *38,* 197–209.

Lahey, B. B., Applegate, B., Chronis, A. M., Jones, H. A., Williams, S. H., Loney, J., &

Waldman, I. D. (2008). Psychometric characteristics of a measure of emotional dispositions developed to test a developmental propensity model of conduct disorder. *Journal of Clinical Child and Adolescent Psychology, 37*, 794–807.

LoBue, V. (2009). More than just another face in the crowd: Superior detection of threatening   facial expressions in children and adults. *Developmental Science*, *12*, 305–313.

Marsh, A. A., & Blair, R. J. R. (2008). Deficits in facial affect recognition among antisocial

populations: A meta-analysis. *Neuroscience and Biobehavioral*

Reviews, *32*, 454–465.

Marsh, A. A., Finger, E. C., Mitchell, D. G., Reid, M. E., Sims, C., Kosson, D. S.,
et al. (2008).

Reduced amygdala response to fearful expressions in children and

adolescents with callous-unemotional traits and disruptive behavior

disorders. *American Journal of Psychiatry*, *165*, 712–720.

Patton, J. H., Stanford, M. S., & Barratt, E. S. (1995). Factor structure of the
Barratt

impulsiveness scale. *Journal of Clinical Psychology*, *51*, 768–774.

Rosset, E. (2008). It's no accident: Our bias for intentional explanations.
Cognition, *108*, 771–

780.

Runions, K. C., & Keating, D. P. (2007). Young children's social information
processing:

Family antecedents and behavioral correlates. *Developmental*

Psychology, *43*, 838–849.

Sabatinelli, D., Fortune, E. E., Li, Q., Siddiqui, A., Krafft, C., Oliver, W. T.,…
Jeffries, J.

(2011). Emotional perception: Meta-analyses of face and natural scene
processing.

NeuroImage, *54*, 2524–2533.

Schulenberg, J. E., Sameroff, A. J., & Cicchetti, D. (2004). The transition to

adulthood as a   critical juncture in the course of psychopathology and

mental health. *Development and   Psychopathology*, *16*, 799–806.

Shaw, D. S., Hyde, L. W., & Brennan, L. M. (2012). Early predictors of boys'

antisocial

       trajectories. *Developmental Psychopathology*, *24*, 971–988.

Schultz, D., & Shaw, D. S. (2003). Boys' maladaptive social information

processing, family   emotional climate, and pathways to early conduct

problems. *Social Development*, 12, 440–460.

Suarez, E. C., Kuhn, C. M., Schanberg, S. M., Williams, R. B., & Zimmerman,

E. A. (1998).

       Neuroendocrine, cardiovascular, and emotional responses of hostile

       men: The role of interpersonal challenge. *Psychosomatic Medicine*, *60*,

       78–88.

Terbug, D., Morgan, B. E., Montoya, E. R., Hooge, I. T., Thornton, H. B., Hariri,

A. R.,…van

       Honk, J. (2012). Hypervigilance for fear after basolateral amygdala

       damage in humans. *Translational Psychiatry*, *2*, e115.

Wechsler, D. (1991). *Manual for the Wechsler Intelligence Scale for Children –*

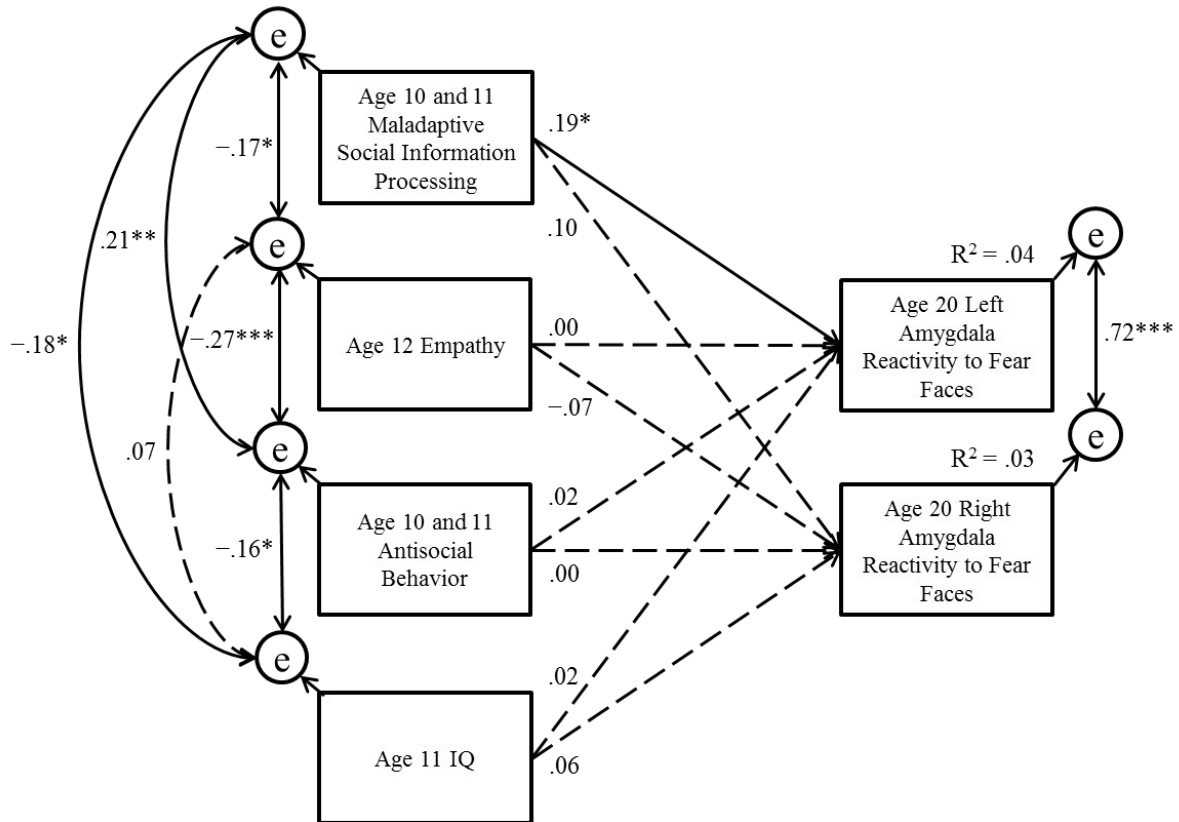*Third Edition.*   San Antonio, TX: The Psychological Corporation.

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. χ^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.
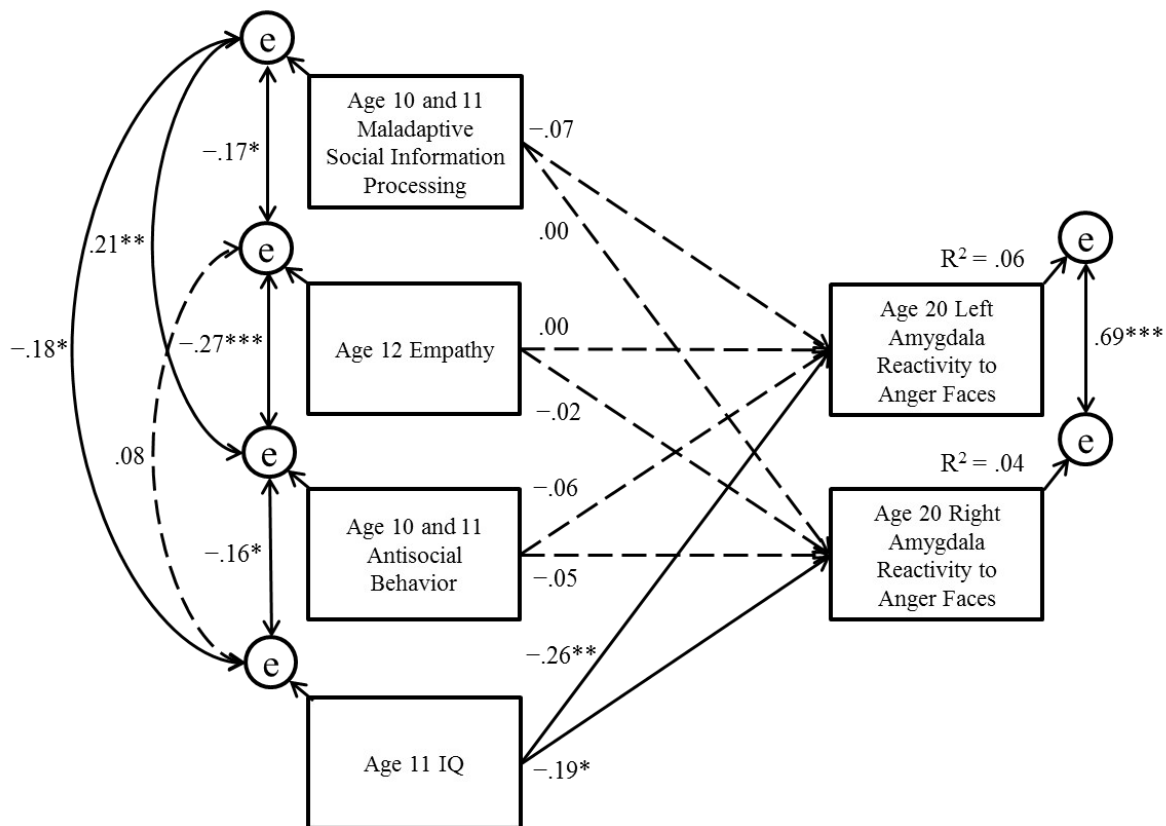
Figure 2. General cognitive ability predicts decreased bilateral amygdala reactivity to anger faces. Model controls for child race and family socioeconomic status at ages 10 and 11. All estimates are standardized and nonsignificant effects are dashed lines. e = residual error.

$\chi^2(2, N = 178) = 2.96$, $p = .227$. CFI = .99. RMSEA = 0.05, 90% CI [.00, .17]. SRMR = 0.02.

p < .05. **p* < .01. ***p* < .001.

Appendix A

Hariri and colleagues' (2002) social threat task interweaves four blocks of an emotional faces-matching condition (A) with three blocks of a sensorimotor control shapes-matching condition (B). Order of emotion types was counterbalanced using four possible orders across participants. Each emotion and control block consisted of six trials with stimuli presented sequentially for 4 seconds each. Fixation crosses (+) appeared for 2, 4, or 6 seconds between each block of trials. Each emotion block lasted 48 seconds and each control block lasted 36 seconds. (A) Subjects viewed a trio of faces expressing the same emotion (i.e., fear, anger, surprise, and neutral affect) and selected one of two faces at the bottom of the screen identical to the target face at the top of the screen. (B) Subjects viewed a trio of simple geometric shapes and selected one of two shapes (bottom) identical to the target shape (top). For the current study, two contrasts designed to elicit robust amygdala reactivity were of interest: (1) *Fear Faces > Shapes*, (2)

Anger Faces > Shapes.

We analyzed descriptive statistics and correlations for accuracy and mean reaction times (RTs) for all condition blocks included in contrasts of interest. As shown below in Table A1, accuracy and mean RTs for the faces-matching and shapes-matching conditions were significantly correlated with each other, except for accuracy on the shapes-matching condition, which was unrelated to mean RTs across all task blocks.

Table A1.   *Correlations and descriptive statistics of fMRI behavioral data*.

Variable	1	2	3	4	5	6
1. Fear Accuracy	–					
2. Anger Accuracy	.65***	–				
3. Shapes Accuracy	.63***	.69***	–			
4. Fear RT	–.29***	–.30***	–.12	–		
5. Anger RT	–.29***	–.33***	–.14	.68***	–	
6. Shapes RT	–.31***	–.34***	–.15	.74***	.72***	–
M	5.76	5.81	5.70	1241.02	1272.85	1123.65
SD	.67	.55	.50	318.06	329.10	233.63

Notes: Accuracy scores reflect number of correct reactions out of six trials. RT = Reaction Time in msec. ***$p < .001$.

Mean RT during the shapes-matching condition was negatively correlated with correct responses for the fear ($r = –.31$, $p < .001$) and anger faces-matching blocks ($r = –.34$, $p < .001$), and positively correlated with their mean RTs (Fear: $r = .74$, $p < .001$. Anger: $r = .72$, $p < .001$). Mean RT

and accuracy on the shapes-matching condition were marginally correlated

(r = –.15, p = .050). Young men who responded faster during the shapes-

matching condition responded faster and were less accurate during the

emotional faces-matching condition.

We then examined whether participants' performance differed by task

block in a series of paired sample t-tests. When comparing mean RTs, we

found that participants responded faster during the shapes-matching

condition (M = 1123.65, SD = 233.63) than during both the fear (M =

1241.02, SD = 318.06), t(171) = –7.24, and anger faces-matching blocks (M

= 1272.85, SD = 329.10), t(171) = –8.62, ps < .001. Mean RTs for the fear

and anger faces-matching blocks did not differ from each other, t(171) =

1.61, p = .110. When comparing accuracy, we found that participants

provided fewer correct responses when matching shapes (M = 5.70, SD

= .50) than when matching anger facial expressions (M = 5.81, SD = .55),

t(171) = –3.54, p = .001. Young men's accuracy for matching fear facial

expressions (M = 5.76, SD = .67) did not differ from their accuracy for

matching anger faces, t(171) = 1.32, p = .190, and shapes, t(171) = –1.51, p

= .133. Young men were faster at matching shapes than emotional faces,

but they made more mistakes when matching shapes than when matching

anger facial expressions.

We then examined correlations between behavioral data and study

variables of interest to evaluate whether variability in performance would

confound our tests of the association between maladaptive social

information-processing (SIP) and amygdala reactivity to threat. Mean RT during the anger faces-matching block was negatively correlated with family SES at ages 10 and 11 (r = –.21, p = .008), IQ at age 11 (r = –.26, p = .002), and empathy at age 12 (r = –.18, p = .039). Mean RTs during the fear faces-matching block (r = –.28, p = .001) and shapes-matching condition (r = –.29, p = .001) were also negatively correlated with IQ at age 11. Young men who responded faster throughout the task had higher IQs at age 11 than those who were slower.

Accuracy for the shapes-matching condition was positively correlated with right amygdala reactivity to fear faces (r = .20, p = .009) and negatively correlated with number of criminal arrests in young adulthood (r = –.24, p = .002). Young men who were more accurate matching shapes at age 20 were less likely to have an adult criminal record in Pennsylvania (PA) and showed greater right amygdala reactivity to fear faces. Accuracy for the fear faces-matching block was positively correlated with family SES at ages 10 and 11 (r = .20, p = .013) and negatively correlated with criminal arrests in young adulthood (r = –.24, p = .002). Young men who were more accurate matching fear faces at age 20 were less likely to have an adult criminal record in PA and had higher family SES in childhood than young men who were less accurate matching fear faces. Accuracy scores for the anger faces-matching block was positively correlated with right amygdala reactivity to fear facial expressions (r = .17, p = .027) and IQ at age 11 (r = .19, p = .021). Young men who were more accurate matching anger faces reported

greater empathy in childhood and showed greater amygdala reactivity to fear faces at age 20.

 In sum, behavioral data showed that participants who responded faster across task conditions were more accurate in their overall performance and that participants responded slower but more accurately when matching emotional faces, particularly anger, than shapes. Although performance for the social threat task was correlated with variables in both childhood and young adulthood, including amygdala activation, the behavioral data were unrelated to maladaptive SIP and controlling for these data did not alter results appreciably in our final models. Our contrasts of interest, which subtract activation during shapes-matching from activation during emotional faces-matching, have been widely-used and well-established as eliciting robust amygdala reactivity to emotional facial expressions (Carré, Fisher, Manuck, & Hariri, 2012). Conceivably, as with any subtraction used in fMRI data analysis, our contrasts could indicate that young men show amygdala hypo-activation when matching shapes but typical amygdala activation when matching faces. As we lacked a more generic baseline condition to contrast with emotional faces- *and* shapes-matching conditions, we were unable to test for amygdala hypo-activation within experimental blocks. Yet, the shapes-matching sensorimotor control condition has the advantage of controlling for configuration and visual processing, allowing us to isolate responses to socioemotional stimuli in the faces-matching blocks.

Appendix B

_f_MRI Acquisition, Preprocessing, and Analysis

Participants underwent _f_MRI with a Siemens 3T Tim Trio scanner

(Siemens Medical Solutions, Erlangen, Germany). An automated shim

procedure minimized magnetic field inhomogeneities. Blood oxygenation

level-dependent (BOLD) functional images were acquired with a gradient

echo planar imaging (EPI) 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 x 64). Scanning parameters were selected to

optimize BOLD signal quality while maintaining a sufficient number of slices

to acquire whole-brain data. Before collecting functional data for each

participant, a reference EPI scan was visually inspected for artifacts (e.g.,

ghosting) and good signal across the entire acquisition volume. Data from all

participants were cleared of problems.

Whole-brain image analysis was completed with SPM8 and our

standard procedure for preprocessing and analysis (see Forbes et al., 2010).

Images for each participant were grey matter segmented and visually

inspected to confirm alignment. Each functional image was realigned to the

mean volume in the time series, unwarped to correct for head motion, co-

registered to a high resolution MPRAGE structural scan, spatially normalized

into Montreal Neurological Institute template space, and smoothed with a 6

mm full-width at half-maximum Gaussian filter to minimize noise and

residual differences in gyral anatomy. Movement parameters for translation and rotation were estimated for each participant and those with mean absolute values of 3 mm or 2 degrees in any direction were deemed as possessing excessive movement and removed from analysis (no one met this cut-off). Voxel-wise signal intensities were ratio normalized to the whole-brain global mean.

Preprocessed data were analyzed using first-level random effects models accounting for scan-to-scan variability and second-level random effects models accounting for participant-to-participant variability to estimate task-specific regional BOLD activation. For each participant and scan, predetermined condition effects at each voxel were calculated in SPM8 using one-sample t-tests ($p < .05$), producing a statistical image for contrasts of interest. Two contrasts were the foci of analyses: (a) *Fear Faces > Shapes* and, (b) *Anger Faces > Shapes*. Participants with less than 80% amygdala coverage in first-level analyses were excluded from further analysis.

Region-of-interest (ROI) analyses estimated group-level main effects of task within an amygdala mask defined as a 723-voxel sphere using an anatomical region in WFU PickAtlas 3.0.3 (Wake Forest University, School of Medicinehttp://fmri.wfubmc.edu). Based on our a priori hypothesis of differential amygdala responses to emotionally-salient facial expressions and our conservative statistical models, we used a statistical threshold of $p <$ 0.05 with a family-wise error (FWE) rate correction for multiple comparisons.

Whole-brain analyses using FWE were conducted to confirm the role of the

amygdala in the models tested.

Appendix C

Threat-related amygdala activity by contrast and region of interest (ROI):

Main effect of task

Contrast and ROI	Talairach Coordinates *x y z*	MNI Coordinates *x y z*	*t*	Cluster Size	Cluster *p*FWE
Fear Faces >					
Shapes					
Left	–17.48, –3.84, –	–18, –4, –18	8.86	113	< .00
Amygdala[a]	12.6	22, –4, –16	7.57	89	1
Right	19.52, –4.21,				< .00
Amygdala	–10.2				1
Anger Faces >					
Shapes					
Left	–23.05, –3.98,	–24, –4, –16	7.55	85	< .00
Amygdala	–10.9				1
Right	19.52, –4.21, –	22, –4, –16	7.07	89	< .00
Amygdala	10.2				1

Note.   Coordinates correspond to peak voxel. [a]Maximum peak voxel located

at Brodmann Area 28. *p*FWE = Family-wise error corrected significance level

for cluster.

Appendix D

Descriptive Statistics and Correlations (N = 178)

Variable and Age in Years	1	2	3	4	5	6	7	8	9	10	11	12
1. Race[a]	--											
2. Family SES 10/11	-.02	--										
3. Maladaptive SIP 10/11	.11	-.11	--									
4. Antisocial Behavior 10/11	.24**	-.02	.23**	--								
5. IQ 11	-.15	.34**	-.22*	-.20*	--							
6. Empathy 12	.06	.13	-.18*	-.26*	.11	--						
7. Left Amygdala (Fear) 20	-.02	-.04	.19*	.05	-.02	-.05	--					
8. Right Amygdala (Fear) 20	.07	.11	.08	.02	.10	-.06	.71**	--				
9. Left Amygdala (Anger) 20	-.07	-.07	-.03	-.03	-.24*	-.01	.07	-.03	--			
10. Right Amygdala (Anger) 20	-.10	.08	.01	-.01	-.18*	-.01	.01	-.01	.69**	--		
11. Number of Criminal Arrests	.27**	.04	.17*	.20*	-.09	.09	.10	.08	-.14	-.20*	--	
12. Impulsiveness 22	-.20*	.06	.02	-.08	.04	.00	.06	.11	.11	.17*	-.03	--
M	–	31.25	-.03	1.70	96.26	2.89	.33	.34	.36	.36	.14	60.88
SD	–	8.91	.67	1.86	17.43	.54	.56	.61	.65	.70	.25	9.90

Note. ᵃCaucasian (1), African American (2), other (3). SES = socioeconomic status. SIP = social information-processing. Bold values statistically significant.

p < .05. **p* < .01. ***p* < .001.

Appendix E
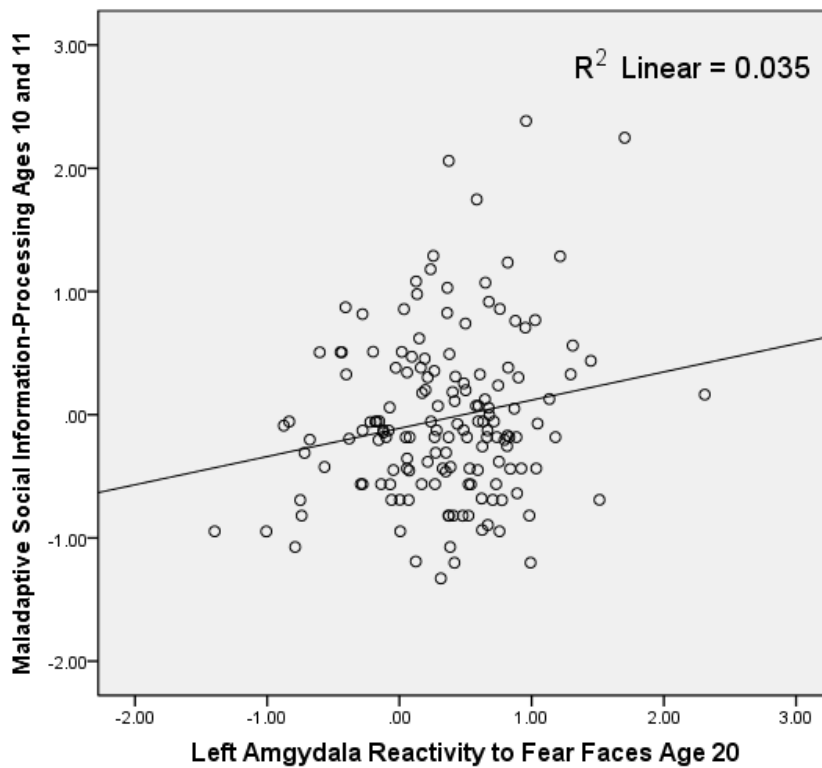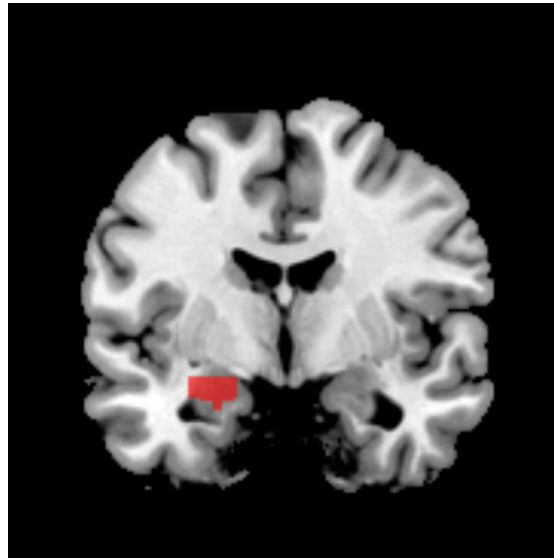
Maladaptive social information-processing (SIP) in childhood is associated

with increased left amygdala reactivity to fear faces in young adulthood.

Mean activation pattern in the left amygdala cluster (113 voxels) was

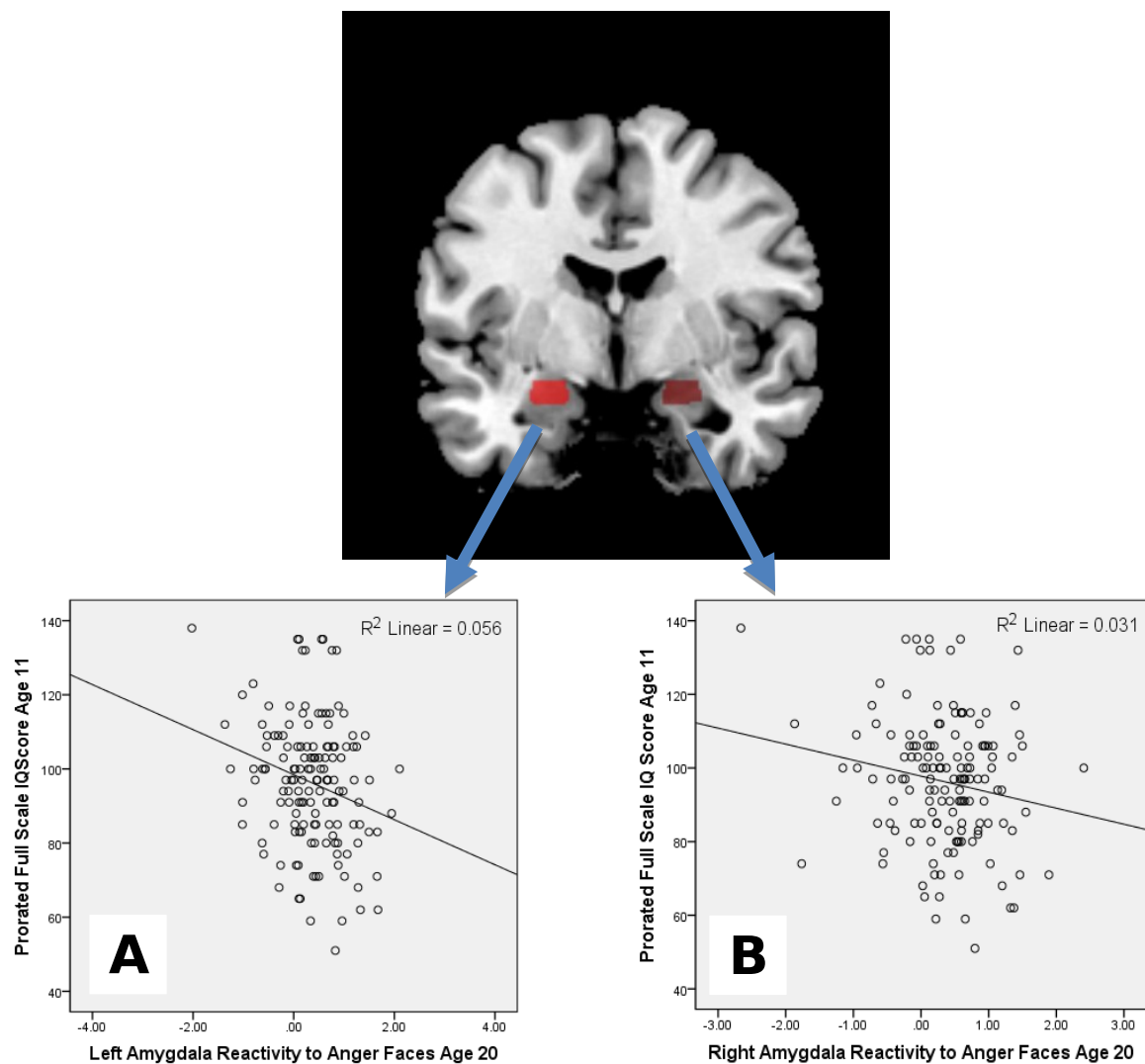extracted from SPM8 and plotted with maladaptive SIP as a scatterplot in

SPSS 19.

Appendix F

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

Contrast and Brain Region	Talairach Coordinates	MNI Coordinates	*t*	Cluster Size	Cluster *p*FWE
Right Occipital Lobe (BA 19, 37)	26.4, –88.49, 5.35	30, –92, 10	14.06	757	< .001
Left Amygdala (BA 28)	–17.48, –3.84, –12.6	–18, –4, –18	8.86	132	< .001
Right Amygdala (BA 34)	19.52, –4.21, –10.2	22, –4, –16	7.57	111	< .001
Left Occipital Cortex[a] (BA 18)	–25.5, –90.61, 9.68	–26, –94, 16	6.72	41	.001
Left STG (BA 22)	–54.6, –11.8, –6.77	–58, –12, –10	5.61	18	.005
Left Perirhinal Cortex[b] (BA 35)	–15.8, –28.94, –5.94	–16, –30, –8	5.38	18	.005
Right DLPFC (BA 46)	48.87, 30.29, 20.59	54, 36, 14	5.23	13	.007

Note. Coordinates correspond to peak voxel. Clusters with fewer than 10 voxels are not reported. BA = Brodmann Area. DLPFC = Dorsolateral Prefrontal Cortex. STG = Superior Temporal Gyrus. *p*FWE = Family-wise error corrected significance level for cluster. [a]Left Occipital Cortex reactivity to fear faces is negatively correlated with age 11 IQ ($r = -.18$, $p = .032$).

[b]Left Perirhinal Cortex reactivity to fear faces is negatively correlated with age 12 empathy ($r = -.22$, $p = .008$).

Appendix G

General cognitive ability (IQ) in childhood is associated with decreased bilateral amygdala reactivity to anger faces in young adulthood. Mean activation patterns in the (A) left amygdala cluster (85 voxels) and (B) right amygdala cluster (89 voxels) were extracted from SPM8 and plotted with prorated full scale IQ scores as scatterplots in SPSS 19.

Appendix H

Threat-related whole brain activity for *Anger Faces > Shapes* contrast: Main

effect of task

Contrast and Brain Region	Talairach Coordinates	MNI Coordinates	t	Cluster Size	Cluster pFWE
Right Occipital Lobe (BA 19)	26.4, –88.49, 5.35	30, –92, 10	11.84	400	< .001
Left Amygdala (BA 34)	–23.05, –3.98, –10.9	–24, –4, –16	7.55	86	< .001
Right Amygdala	19.52, –4.21, –10.2	22, –4, –16	7.07	96	< .001
Right DLPFC[a] (BA 46, 9)	48.87, 30.29, 20.59	54, 36, 14	6.20	111	< .001

Note.   Coordinates correspond to peak voxel. Clusters with fewer than 10

voxels are not reported. BA = Brodmann Area. DLPFC = Dorsolateral

Prefrontal Cortex. pFWE = Family-wise error corrected significance level for

cluster. [a]Right DLPFC reactivity to anger faces is positively correlated with

maladaptive social information-processing (r = .17, p = .038).