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Incentive Processing in Persistent Disruptive Behavior and Psychopathic Traits: A Functional Magnetic Resonance Imaging Study in Adolescents

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ABSTRACT

BACKGROUND: Children with early-onset disruptive behavior disorder (DBD), especially those with callous-unemotional traits, are at risk of developing persistent and severe adult antisocial behavior. One possible underlying mechanism for persistence is deficient reward and loss sensitivity, i.e., deficient incentive processing. However, little is known about the relation between deficient incentive processing and persistence of antisocial behavior into adulthood or its relation with callous-unemotional and other psychopathic traits. In this study, we investigate the relationship between the neural correlates of incentive processing and both DBD persistence and psychopathic traits.

METHODS: In a sample of 128 adolescents (mean age 17.7) with a history of criminal offending before age 12, functional magnetic resonance imaging was performed during a monetary incentive delay task designed to assess neural responses during incentive processing. Neural activation during incentive processing was then associated with DBD persistence and psychopathic traits, measured with the Youth Psychopathic Traits Inventory.

RESULTS: Compared with both healthy control subjects and youths who had desisted from DBD, persistent DBD subjects showed lower neural responses in the ventral striatum during reward outcomes and higher neural responses in the amygdala during loss outcomes. Callous-unemotional traits were related to lower neural responses in the amygdala during reward outcomes, while other psychopathic traits were not related to incentive processing.

CONCLUSIONS: In the current study, aberrant incentive processing is related to persistence of childhood antisocial behavior into late adolescence and to callous-unemotional traits. This mechanism may underlie treatment resistance in a subgroup of antisocial youth and provide a target for intervention.

Keywords: Antisocial behavior, Callous-unemotional, fMRI, Persistence, Psychopathy, Reward

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Juvenile antisocial behavior, clinically diagnosed as a disruptive behavior disorder ([DBD], i.e., oppositional defiant disorder [ODD] or conduct disorder [CD]), causes serious personal and societal harm and is associated with substantial economic costs (1). Importantly, early onset of juvenile antisocial behavior is a potent risk factor for the persistence of such behavior into adulthood (2). General population studies in children strongly suggest that the presence of psychopathic traits (i.e., callous-unemotional traits, grandiose-manipulative traits, and impulsive-irresponsible traits) and more specifically callous-unemotional traits also increase the risk of persistent antisocial behavior [for review, see (3)].[1] Notably, these traits have been added to the DSM-5 as a specifier for conduct disorder under the label of limited prosocial emotions. However, it is largely unknown whether these traits also predict persistence of antisocial behavior in specific samples such as early-onset offenders.

[1]While the authors acknowledge the concerns of some scholars that the measurement (48) and the interpretation (52) of psychopathic traits in minors cannot be seamlessly equated with the adult construct of psychopathy, these traits will be referred to as psychopathic traits throughout this article for reasons of brevity and consistency with the (*footnote continued*) research field. Notably, psychopathic traits are not a unitary construct but consist of several dimensions with distinct behavioral and neural correlates. While studies in the past have often employed two-factor solutions for psychopathic traits [i.e., affective-interpersonal versus impulsive-antisocial (53)], more recent studies use operationalizations based on three-factor solutions [i.e., affective (callous-unemotional), interpersonal (grandiose-manipulative), and impulsive (impulsive-irresponsible) (54)] or four-factor solutions [i.e., affective, interpersonal, impulsive, antisocial (55)]. In the current study, we employ the three-factor model because it seems to be most consistent with the DSM-5 perspective of limited prosocial emotions as a specifier to conduct disorder.

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Moreover, it is unknown what the underlying neurocognitive mechanisms of persistence and psychopathic traits are in such populations, information that is essential for the development of early prevention and treatment strategies.

Most animal and human behaviors, as well as adaptive changes in behavior, are evolutionarily driven by a motivation to achieve reward and avoid punishment (4). As such, one may hypothesize that developmental processes leading to maladaptive persistent antisocial behavior are associated with aberrant sensitivity to positive or negative reinforcers (i.e., incentive processing). Aberrant incentive processing, i.e., excessive or reduced sensitivity to reward, loss, or cues associated with these outcomes, has been associated with a broad range of behavioral problems (5), including pervasive patterns of antisocial behavior during childhood (6), adolescence (7), and adulthood (8). Moreover, aberrant neural responses during incentive processing have been reported in antisocial juveniles and adults (7,9–12).

While neuropsychological studies have provided a neurocognitive framework implicating both hyposensitivity to loss and hypersensitivity to reward in antisocial juveniles (13), the neuroimaging literature is not entirely consistent with this framework [for a review, see (14)], suggesting that other neural mechanisms may also be involved. In addition, this inconsistency may result from sample differences and neurobiological heterogeneity within the population of antisocial juveniles. These latter issues can be addressed by taking into account longitudinal (15) and cross-sectional (16) markers of heterogeneity, i.e., by characterizing antisocial youth in terms of distinct developmental profiles (i.e., persisters versus desisters) or phenotypical differences (i.e., psychopathic traits), respectively.

Regarding the latter, psychopathic traits are characterized by a continuous distribution (17), continuous criterion validity (18), and neurobiological specificity (19). Moreover, they have been associated with neuropsychological measures of reward dominance (20). Most functional magnetic resonance imaging (fMRI) studies in children with high levels of psychopathic traits, however, focused on the processing of fear and emotional pictures. Although these studies provide clear evidence for reduced amygdala responsivity in such paradigms [e.g., (19,21–23)], they do not allow conclusions about incentive processing. The only studies on the relation between psychopathic traits and neural responses during incentive processing have focused on ventral striatum (VS) responsiveness in healthy adults and reported atypical responses during reward processing (24–26) in relation to impulsive-antisocial traits. It is unknown, however, if these findings generalize to antisocial youths and if such effects can also be observed in other key incentive processing regions (27,28), such as the amygdala and medial prefrontal cortex (mPFC), which have been implicated in decision-making deficits observed in antisocial and psychopathic development (29–31).

The current study has three main objectives: 1) to investigate if early-onset antisocial youths with persistent DBD differ from those who desist from DBD and from healthy control subjects with respect to neural responses in the ventral striatum, amygdala, and mPFC during incentive processing; 2) to ascertain that associations between these neural responses and persistence indeed pertain to stable patterns of dysfunction, rather than to current DBD severity, by assessing their association with current DBD symptoms; and 3) to investigate the association between these neural responses and psychopathic traits. We performed an fMRI study in a large group of childhood arrestees (i.e., first offense before age 12) followed up until late adolescence using a well-established incentive processing paradigm, i.e., the monetary incentive delay (MID) paradigm (32). Given the inconsistency of previous fMRI studies on incentive processing in antisocial juveniles but consistent theoretical accounts on the relevance of the VS, mPFC, and amygdala for dysfunctional decision making in antisocial development (30,31), we hypothesized that in all these regions, reduced neural responses during reward feedback (7,10,11,33) and higher responses during loss feedback (7,33,34) would uniquely characterize the DBD persister subgroup, as compared with desisters and control subjects, but would be less strongly associated with current DBD severity. We also hypothesized that impulsive-irresponsible traits would be related to lower neural responses during reward anticipation (24) and reward feedback (26), whereas callous-unemotional traits would be related to higher neural responses during reward feedback (26).

METHODS AND MATERIALS

Participants

Participants were recruited from a Dutch cohort of 364 adolescents who had become known to local police services before the age of criminal responsibility (12 years) for a range of acts that would be prosecutable above the age of 12 (e.g., petty theft, arson, vandalism, trespassing, burglary, assault, sexual abuse, and robbery), excluding status offences.[2] This longitudinal study had three previous data collection waves (35): mean age at study entrance was 10.9 (SD 1.4) years and 13.1 (SD 1.5) years at wave 3.

For the current neuroimaging study (wave 4; mean age 17.7 [SD 1.6] years), a subsample (total $n = 150$) representing the entire severity range was recruited, including those at low, medium, and high risk for antisocial development (see Supplement 1 for recruitment strategy) and partly overlapping with our previously published fear conditioning study (36). For the current report, 22 out of the original 150 participants were excluded from analyses because of invalid (i.e., with movement artifacts or poor coverage) or missing MRI data ($n = 10$), drug use in last 24 hours before scanning ($n = 3$), or task performance rates deviating more than 3 SD from the mean ($n = 9$). The excluded group did not differ from the study sample ($n = 128$) with respect to current and previous aggression or psychopathic traits scores, DBD diagnosis, age, IQ, gender, ethnicity, or socioeconomic status (all $p > .1$).

To answer our first research question, i.e., how do DBD-persisters (DBD-p) differ from DBD-desisters (DBD-d) and healthy control subjects (HC), these subgroups were defined as follows (see flow chart in Figure S1 in Supplement 1): 1) DBD-p ($n = 22$): participants meeting full criteria of DBD on the National Institute of Mental Health Diagnostic Interview Schedule for Children version IV in any of the previous waves

[2]Status offenses are acts that are punishable only in minor populations, e.g., running away from home and truancy.