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The interacting role of media violence exposure and aggressive–disruptive behavior in adolescent brain activation during an emotional Stroop task

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ABSTRACT

Only recently have investigations of the relationship between media violence exposure (MVE) and aggressive behavior focused on brain functioning. In this study, we examined the relationship between brain activation and history of media violence exposure in adolescents, using functional magnetic resonance imaging (fMRI). Samples of adolescents with no psychiatric diagnosis or with disruptive behavior disorder (DBD) with aggression were compared to investigate whether the association of MVE history and brain activation is moderated by aggressive behavior/personality. Twenty-two adolescents with a history of aggressive behavior and diagnosis of either conduct disorder or oppositional-defiant disorder (DBD sample) and 22 controls completed an emotional Stroop task during fMRI. Primary imaging results indicated that controls with a history of low MVE demonstrated greater activity in the right inferior frontal gyrus and rostral anterior cingulate during the violent word condition. In contrast, in adolescents with DBD, those with high MVE exhibited decreased activation in the right amygdala, compared with those with low MVE. These findings are consistent with research demonstrating the importance of fronto-limbic structures for processing emotional stimuli, and with research suggesting that media violence may affect individuals in different ways depending on the presence of aggressive traits.

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1. Introduction

The extent to which exposure to media violence increases aggressive and violent behaviors across age and gender has been the subject of decades of research and debate (Bandura et al., 1963; Anderson and Bushman, 2001; Huesmann et al., 2003; Bushman and Huesmann, 2006). Increases in the popularity, availability, and variety of violent media (e.g., television and video games) necessitate an understanding of how such exposure may affect both behavior and cognitive functioning (Kronenberger et al., 2005a). The General Aggression Model (GAM) posits that long-term exposure to violent media content alters internal states (arousal, cognition, and affect), leading to consolidation of aggressive mental schemas (Anderson and Bushman, 2001; Carnagey et al., 2007). Empirical support for the GAM emerged from research showing that viewing violence on television increases aggressive thoughts, feelings, and behaviors (Anderson and Bushman, 2001). In addition, individuals who play violent computer/console games also exhibit an increase in short-term aggressive behaviors (Uhlmann and Swanson, 2004; Bartholow et al., 2005; Carnagey and Anderson, 2005), long-term aggressive traits (Lemmens et al., 2006), and desensitization as measured by a reduced P300 event-related brain potential to violent images (Bartholow et al., 2006).

Recent advances in the quality and availability of neuroimaging techniques, particularly functional magnetic resonance imaging (fMRI), have allowed researchers to evaluate relationships between brain functioning and media violence exposure (Mathews et al., 2005; King et al., 2006; Mathiak and Weber, 2006; Murray et al., 2006; Wang et al., 2009; Hummer et al., 2010). These latter studies have investigated neural activity related to the observed behavioral and cognitive sequelae of media violence exposure (MVE) and highlight the potential link between MVE, brain functioning, and related components of behavior and cognition, such as aggression. Brain imaging during violent television shows and while playing violent video games suggests engagement of similar, but not identical, brain networks across the different types of media. For example, whereas violent-game playing modifies activity in prefrontal cortex, cingulate cortex and amygdala (King et al., 2006; Mathiak and Weber, 2006; Wang et al., 2009), viewing violent television may recruit additional

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processing circuitry, including the hippocampus, pulvinar, and inferior parietal lobe (Murray et al., 2006). In addition to activation of brain regions during exposure to media violence, research has demonstrated that changes in brain activation may continue after exposure ends (Wang et al., 2009; Hummer et al., 2010). For instance, in a recent experimental study with adolescent subjects randomly assigned to play either a violent or a non-violent video game, subjects exposed to the violent game exhibited a greater amygdala response to subsequent presentation of violent words, compared with the nonviolent game group (Wang et al., 2009). Furthermore, this activation did not demonstrate the negative functional connectivity with medial prefrontal cortex that was present in those who played the nonviolent game. These data suggest that short-term exposure to violent television and video games may affect frontal inhibitory and subcortical limbic structures, as well as connectivity between these regions.

The relationship between media violence, aggression, cognition, and brain functioning is complex and likely moderated by multiple factors. In particular, individuals with preexisting aggressive–hostile personality traits have demonstrated increased vulnerability to effects of violent media in behavioral studies (Bushman, 1995), reflecting more readily accessible scripts of beliefs, behavior patterns, and angry affect that are exacerbated by violent media exposure. GAM supports this position that exposure to environmental stimuli, in conjunction with parallel aggressive predispositions, may consolidate and strengthen disruptive behavior schemas (Cohen, 1973; Bushman and Anderson, 2002). Furthermore, media violence exposure is associated with poor executive functioning, a relationship that is stronger in adolescents with a disruptive behavior disorder (Kronenberger et al., 2005a).

Clinically, individuals with disruptive behavior disorders (DBD; defined in the Diagnostic and Statistical Manual for Mental Disorders, Fourth Edition [DSM-IV] as oppositional defiant disorder or conduct disorder; American Psychiatric Association, 2000) show a pattern of rule-breaking behavior, anger, and/or oppositionality toward others that overlaps with the construct of aggressive–hostile personality traits. Consistent with emotional and behavioral traits of this population, abnormalities in frontal and temporal cortices have been reported in individuals with a history of aggressive behavior and/or DBD (Li et al., 2005; Marsh et al., 2008; Yang and Raine, 2009). Of particular note, individuals with DBD or related disorders have a lower amygdala response to emotionally arousing stimuli than healthy controls (Kiehl et al., 2001; Sterzer et al., 2005; Marsh et al., 2008), indicating desensitization or neural dysfunction. Such functional abnormalities are consistent with aberrant information processing within the emotional regulatory network. Moreover, studies show substantial overlap in brain regions sensitive to aggressive behavior/DBDs and activity in adolescents exposed to high levels of violent media (Mathews et al., 2005).

Evidence of shared neural correlates between aggression/DBD and media violence processing suggests that individuals with DBD could show abnormal effects of media violence on brain activation. However, little is known about the relationship between history of media violence exposure and brain activation in individuals with DBD, particularly involving brain circuits related to emotional response and regulation. Most prior research in this area has focused either on concurrent effects of media violence and brain activation (e.g., during viewing or playing of violent media; Mathiak and Weber, 2006; Murray et al., 2006) or short-term effects (30–45 min) on later brain activity (Wang et al., 2009; Hummer et al., 2010). Mathews et al. (2005) investigated the relationship between history of MVE and brain functioning during executive functioning. They found differences between individuals with high and low MVE in activation of prefrontal cortical areas. However, they did not use emotionally provocative tasks designed to recruit circuits related to emotional processing and control.

To address this research gap, the primary aim of the current study was to examine the association between history of media violence exposure and activation of brain regions related to emotional processing, in adolescents with a history of clinically significant aggressive–disruptive behaviors and adolescents with no behavior problems. Unlike prior neuroimaging studies regarding MVE, this research used an emotionally provocative paradigm consisting of aggression-related words, with the intent of focusing on neural circuitry related to emotional response and regulation. Based on separate bodies of imaging research in the areas of MVE and aggressive–disruptive behavior (Speltz et al., 1999; Li et al., 2005; Mathews et al., 2005; Mathiak and Weber, 2006; Murray et al., 2006; Plessen et al., 2006; Marsh et al., 2008; Wang et al., 2009), we hypothesized that adolescents with a history of aggressive–disruptive behavior would show abnormalities in activation of frontal and limbic regions compared with adolescents with no diagnosis.

In particular, we hypothesized that youth with high MVE, and especially those also with DBD, would have lower amygdala response to violent stimuli, as desensitization has been related to both factors (Sterzer et al., 2005; Bartholow et al., 2006). This limbic activity was expected to be inversely related to frontal and anterior cingulate activity, particularly in individuals with better emotional control (i.e., controls and low MVE), due to top-down effects of these regions (Etkin et al., 2006). In addition, MVE was hypothesized to be associated with poor behavioral performance, a relationship which is strongest in individuals with DBD (Kronenberger et al., 2005a). Furthermore, we expected behavioral reactions to violent stimuli to be delayed unless participants were desensitized by MVE.

2. Materials and methods

2.1. Subjects

Participants were recruited via informational flyers at pediatric settings, a psychiatry clinic, community organizations and schools. Participants were between ages 13 and 17 (inclusive), with an estimated IQ (as measured by the Kaufman Brief Intelligence Test; Kaufman and Kaufman, 1990) of at least 70. Adolescents with a current diagnosis of major depressive disorder or lifetime diagnosis of bipolar disorder or schizophrenia were excluded. Two matched diagnostic groups, of 22 participants each, were formed from adolescents who qualified for the study and completed the experiment. Groups were matched on age (controls 14.86 ± 0.34; DBD 14.64 ± 0.28), gender (male/female: controls 13/9; DBD 13/9), past violent media exposure (see later for description; controls -0.5 ± 3.3; DBD -0.8 ± 2.5) and estimated IQ (controls 99.90 ± 1.65; DBD 96.59 ± 1.55) (all $P > 0.05$).

Participants in the DBD with aggressive features subsample met the DSM-IV criteria for oppositional-defiant disorder or conduct disorder based on the Behavioral Disorders module of the Schedule for Affective Disorders and Schizophrenia for School-Aged Children, Present and Lifetime Version (K-SADS) (Chambers et al., 1985) semi-structured diagnostic interview with the primary caretaker. Additionally, participants in the DBD group had at least one of the seven K-SADS conduct disorder symptoms involving aggression to people or animals. Six participants with DBD were being treated with medication (2 methylphenidate, 2 mixed salts amphetamines, 1 venlafaxine, and 1 bupropion). However, participants were instructed (based on evaluation of symptoms and medication status by a study physician) to not take psychostimulant medication for 24 h prior to study visits, in order to minimize the effects of those medications on fMRI results.

Participants in the healthy control subsample had no psychiatric diagnosis (based on results of the K-SADS and the Adolescent Symptom Inventory — 4 [completed by parents]) (Gadow and

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