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Relationships between impulsivity, anxiety, and risk-taking and neural correlates of attention in adolescents

James W. B. Elsey^{1,2}, Michael J. Crowley¹, W. Einar Mencil³, Cheryl M. Lacadie⁴, Linda C. Mayes^{1,5,*}, and Marc N. Potenza^{1,6,7,*}

¹Child Study Center, Yale University School of Medicine, United States ²University of Amsterdam, Department of Clinical Psychology, the Netherlands ³Haskins Laboratories, Yale University, United States ⁴Department of Diagnostic Radiology, Yale University School of Medicine, United States ⁵Departments of Epidemiology, Pediatrics and Psychology, Yale University School of Medicine, United States ⁶Departments of Psychiatry and Neurobiology and CASAColumbia, Yale University School of Medicine, United States ⁷Connecticut Mental Health Center, United States

Abstract

Although impulsivity, anxiety, and risk-taking may relate to attentional processes, little research has directly investigated how each may be associated with specific facets of attentional processes and their underlying neural correlates. Nineteen adolescents performed an fMRI task involving simple, selective and divided attention. Out-of-scanner-assessed impulsivity, anxiety and risk-taking scores were not correlated with each other and showed task-phase-specific patterns of association. Results are discussed in light of research and theory suggesting a relationship between these domains and attention and may serve to focus future research aiming to understand these relationships.

Keywords

Adolescence; fMRI; anxiety; attention; impulsivity; risk-taking

Attention serves to focus the brain's processing on particular stimuli, from which our conscious experience of the world is derived. In the words of James (1890): "my experience is what I agree to attend to" (p. 402). Since James (1890), researchers have distinguished between top-down attention (the volitional orientation of attention based upon an individual's goals), and bottom-up attention (in which the salience of an environmental stimulus demands attention) (Buschman & Miller, 2007). Normal cognitive functioning and mental health are linked to the ability to direct one's focus and appropriately respond to salient stimuli as they arise in the environment.

The central role of attention function in mental health is highlighted by research showing disturbances in attentional processing in multiple psychiatric disorders, including anxiety

* Authors contributed equally to the manuscript.

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disorders (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van Ijzendoorn, 2007), depression (Gotlib, Krasnoperova, Yue, & Joormann, 2004), eating disorders (Dobson & Dozois, 2004), addictive disorders (Field & Cox, 2008; Field, Munafò, & Franken, 2009), attention-deficit hyperactivity disorder (ADHD) (Biederman, 2005), and schizophrenia (Cornblatt & Keilp, 1994). In turn, key symptoms including anxiety, impulsivity, and risk taking have long been recognized as important risk factors for the development of anxiety disorders, ADHD, and substance-use disorders, respectively, in otherwise healthy individuals (Wills, Vaccaro, & McNamara, 1994; Adlaf & Smart, 1983). As adolescence is a period during which the incidence of psychiatric illness increases (Paus, Keshavan, & Giedd, 2008), particularly among those at risk, assessment of the relationship between predisposing symptoms and attentional processing in otherwise healthy adolescents may provide insight into how risk factors for psychiatric illness vary with attention function.

This study was designed to elucidate the association between several potential risk factors for psychiatric disturbance (anxiety, impulsivity, and risk-taking) and neural features of attentional processing during the adolescent developmental period. In support of this line of inquiry, we first review neuroimaging research on selective and divided attention, followed by work on impulsivity and attention. Next we discuss possible links between anxiety and attention, and between risk-taking and attention. Finally, these lines of work are used to delineate hypotheses for the present study.

Neural underpinnings of selective and divided attention

Selective attention refers to the ability to focus on particular elements of incoming sensory information. This can occur within modalities, such as when listening to a conversation partner whilst ignoring other conversations, or crossmodally, such as when focusing on a book whilst ignoring surrounding chatter. People are also often faced with situations in which dividing attention between visual and auditory stimuli is required, such as maintaining a conversation with a passenger whilst driving a car. Divided attention requires that one sustain attention across channels to pick up any important information, whereas selective attention requires the intentional blocking out of one or more information streams. These capacities play pivotal roles in the ability to select important elements from the environment for deeper processing, ignoring irrelevant information that would unnecessarily expend limited resources (Lavie, 2010).

Varying levels of attention to stimulus features differentially activates regions of sensory cortex associated with the processing of that feature. For instance, Corbetta and colleagues (Corbetta, Miezin, Dobmeyer, Shulman, & Peterson, 1991) found that selective attention to visual cues was associated with heightened activity in primary visual cortex and adjacent occipital regions, more so than when these same stimuli were presented without active attention. Extrastriate regions extending into the fusiform gyrus, parahippocampal gyrus, and temporal cortex/superior temporal gyrus (STG) were activated for attention to shape. Inferior parietal cortex activation was associated with attention to motion, whereas attention to color was associated with dorsolateral occipital activation. These regions were suggested to be those involved in the processing of the respective visual feature, with increased activation relating to top-down modulatory signals to enhance processing of that feature.

With respect to auditory stimuli, selective attention, particularly to spoken language stimuli, was associated with increased activation in the STG and inferior frontal gyrus (IFG) (Pugh et al., 1996), sites implicated in the processing of language. Similarly, increased activation in primary and secondary auditory cortices was observed during attention to or detection of auditory stimuli, relative to passive exposure to the same sounds (Jancke, Mirzazade, & Shah, 1999).

Cross-modal studies (Johnson & Zatorre, 2006), in which competing visual and auditory stimuli are either attended or ignored, have shown that selective attention to visual stimuli heightens lateral occipital, visual association area, peristriate area 19, and superior parietal lobe (SPL) activation. Attention to auditory stimuli increased STG activation. Similarly, selective visual and auditory forms of attention have been found to be associated with increased visual and auditory cortex activation, respectively (Loose, Kaufmann, Auer, & Lange, 2003). Conversely, selective attention to one modality has also been associated with reduced activity in those regions involved in processing the competing modality (Johnson & Zatorre, 2005). As direct projections have been found between visual and auditory cortices in primates (Rockland & Ojima, 2003), cross-modal inhibition between sensory cortices may partly explain such findings (Johnson & Zatorre, 2005).

Selective and divided forms of attention activate multiple regions beyond sensory cortices. Selective attention increases activation in the globus pallidus, caudate, posterior thalamus, premotor cortex, insula, and orbitofrontal cortex (OFC) relative to divided attention (Corbetta et al., 1993). In contrast, divided attention increases anterior cingulate (ACC) and right prefrontal cortex (PFC) activation compared with selective attention. Heightened right- and bilateral PFC activation respectively, as well as ACC activation, was observed during cross-modal divided attention (Vohn et al., 2007). Interestingly, divided cross-modal attention has been associated with increased dorsolateral prefrontal cortex (DLPFC) activation, but accuracy of performance was negatively correlated with DLPFC activation and positively correlated with recruitment of sensory cortices (Johnson & Zatorre, 2006).

However, results are not always clear cut, as cross-modal divided attention in Vohn et al., (2007) was also associated with thalamic, inferior and superior parietal lobule (IPL/SPL), claustrum, insula, globus pallidus, and STG activation relative to cross-modal selective attention, with some of these regions also subserving selective attention (Corbetta et al., 1991). Hence, it is possible that the neural substrates of selective and divided attention may not be entirely dissociable and may partly rely on a general attentional network. Similar activation in DLPFC and ventrolateral prefrontal cortex (VLPFC), IPL and SPL, and ACC has been reported with both visual selective and divided attention (Nebel et al., 2005). Differences in demands on executive control could explain differential activation of these regions in some divided attention tasks (Nebel et al., 2005), with task difficulty also potentially contributing to ACC activation in other types of tasks (Buckner, Raichle, Miezin, & Petersen, 1996). When visual selective and divided attention tasks were matched for difficulty, divided attention was associated only with increased activation in visual processing regions (Nebel et al., 2005), similar to earlier studies in which increases in attentional demands increased activation in the respective sensory cortices. Also arguing against distinct neural substrates for selective and divided attention, divided attention

between color and angle did not activate regions beyond the sum of selective attention to one or the other (Hahn et al., 2008). However, these studies did not examine cross-modal attention and thus may not be directly comparable to studies including a cross-modal component.

Hence, it seems that both selective and divided attention modulate activity in sensory processing regions. In cross-modal studies, selective attention to one modality may enhance activation in the respective sensory processing regions, and may diminish activation in the sensory regions of the ignored modality. Frontoparietal activations (e.g. in PFC, ACC, IPL, SPL) may contribute to cross-modal divided attention and may relate to a frontoparietal attentional network involved in the executive control of attention (Corbetta & Shulman, 2002). However, it has also been argued that activation of these regions could simply vary with task difficulty (Nebel et al., 2005).

Impulsivity and attention

Impulsivity has been defined as a tendency to act without forethought, or to make decisions without due consideration (Bari & Robbins, 2013). Impulsivity is a risk factor for or correlate of a range of psychopathologies, including conduct disorder/antisocial behaviour, substance abuse, and ADHD (Bari & Robbins, 2013; Moeller, Barratt, Dougherty, Schmitz, & Swann, 2014; Nigg, 2001). For example, adults diagnosed with ADHD scored higher on all three subscales of the Barratt Impulsiveness Scale (Patton, Stanford, & Barratt, 1995) relative to healthy adults, indicating a higher tendency to act on the spur of the moment (motor impulsivity), failure to focus on current tasks (attentional impulsivity), and not planning appropriately or thinking carefully (non-planning impulsivity). These adults also performed more poorly on behavioral measures of impulsivity (Malloy-Diniz, Fuentes, Leite, Correa, & Bechara, 2007).

The presence of impulsive features in parallel with attention deficits in ADHD suggests a possible relationship between impulsivity and attention, with attentional deficits possibly resulting from a diminished ability to inhibit irrelevant or distracting thoughts and feelings, as well as failing to inhibit behaviors that may detract from focusing on specific tasks (Nigg, 2001). However, the precise relationship between impulsivity and attention function remains incompletely understood.

A connection between ADHD and impulsivity has been studied using measures of brain structure and activation. In children and adolescents, gray-matter reductions in the right ventral striatum negatively correlated with maternally rated impulsivity (Carmona et al., 2009). Similarly, research in both children (Scheres, Milham, Knutson, & Castellanos, 2007) and adults (Strohle et al., 2008) has found that ventrostriatal hypoactivation during reward anticipation associated with impulsivity/hyperactivity symptoms in ADHD.

In a meta-analysis of 55 fMRI studies of ADHD (Cortese et al., 2012), individuals with ADHD displayed different patterns of activation in networks related to attention than control participants. For children with ADHD, hypoactivation in the frontoparietal and ventral attention networks was observed relative to controls. Adults also displayed hypoactivation of

the frontoparietal network and hyperactivation of the dorsal attention network. The frontoparietal network, which includes the IPL, DLPFC, dorsal ACC (dACC), anterior insula, and lateral cerebellum, contributes to goal-directed executive function (Rubia, 2011; Yeo et al., 2011). Similar regions in the PFC, ACC, and parietal lobes were implicated in the goal-directed control of attention in studies of selective and divided attention described above. The ventral attention network includes the temporoparietal junction (TPJ), supramarginal gyrus, and frontal operculum, and is implicated in attentional orientation to salient or behaviorally relevant stimuli. Children with ADHD have been found to show hyperactivation of regions of this network (Cortese et al., 2012). This may reflect the distractibility (i.e. attentional impulsivity) of those with ADHD (Cortese et al., 2012). The dorsal attention network, composed of the IPL and frontal eye fields (FEF), is implicated in the selection of stimuli based upon expectations and internal goals (Corbetta, Patel, & Shulman, 2008). Hyperactivation could reflect poorer efficiency of this system in adults, with increased effort required to select relevant stimuli.

Whilst the relationship between attention and impulsivity in ADHD has been well studied, less research has directly examined attentional processing in relation to impulsivity in the healthy population, particularly during developmental periods such as adolescence when impulsive behavior may be heightened (Ernst, 2014). Such studies are of particular importance, as findings in at-risk, subclinical, or healthy participants may not be the same as those in ADHD. A recent meta-analysis demonstrated that the consistent finding of ventrostriatal hypoactivation in relation to impulsivity in those with ADHD was not corroborated in those without ADHD (Plichta & Scheres, 2014). Instead, among healthy individuals, impulsivity was associated with ventrostriatal hyperactivity during reward anticipation (Plichta & Scheres, 2014). Other clinical populations, such as those with alcohol-use and gambling disorders, have also demonstrated striatal hypoactivation during anticipated reward (Balodis et al., 2012; Beck et al., 2009).

In summary, differences in the engagement of brain networks associated with attention are characteristic of ADHD and may relate to ‘attentional impulsivity’. Impulsivity is linked to differences in ventrostriatal structure and function, yet hypo- versus hyper-responsivity may depend upon ADHD status. Those with heightened impulsivity may also show dysfunction of frontoparietal attention networks.

Anxiety and attention

Anxiety may closely associate with alterations in attentional processes. In particular, individuals with anxiety disorders or high trait anxiety show a strong attentional bias to threat cues (see Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van Ijzendoorn, 2007, and Cisler & Koster, 2010, for reviews). This may result from a hyper-responsive pre-attentive threat detection system involving the amygdala (Ohman, 2005; Mathews, Mackintosh, Fulcher, & 1997). In addition, top-down executive control over attentional allocation may also be impaired in individuals with high anxiety (Bishop, 2009; Bishop, Duncan, Brett, & Lawrence, 2004).

Anxiety may also relate to inattention in non-affective contexts (Berggren & Derakshan, 2013; Bishop, 2009), as posited in the attentional control theory (ACT) model (Eysenck, Derakshan, Santos, & Calvo, 2007). Central to this model is the idea that anxiety is related to deficits in processing efficiency, such as in the inhibition of task-irrelevant distracters and shifting attention between tasks. Whilst behavioral performance may be equivalent between individuals with high and low levels of anxiety, the similar performance may relate to compensatory effort in highly anxious individuals. As task demands increase, the ability to compensate for attentional deficits may be increasingly compromised.

Consistent with ACT predictions, individuals with high state anxiety performed more poorly than those with low anxiety and were especially impaired with more complex tasks (Derakshan, Smyth, & Eysenck, 2009). Individuals scoring high on trait anxiety also displayed greater attentional capture by task-irrelevant color distracters, to the detriment of performance (Moser, Becker, & Moran, 2012). High (versus low) anxious individuals were also more affected by distracters, but only under conditions of high attentional load (Sadeh & Bredmeier, 2011). Likewise, individuals with social anxiety, but not control subjects, were negatively affected by task irrelevant distracters under high attentional load (Moriya & Tanno, 2010). These results are consistent with attentional deficits linked with anxiety, which may be particularly manifest under conditions where there are heightened demands on attentional resources.

At the neural level, the possibility that anxiety leads to differential processing of neutral as well as affective cues has been supported. Comparing individuals with high and low anxiety in a target detection task in which letter distractors competed for attention (Bishop, 2009), individuals with high anxiety showed reduced DLPFC activity. This finding appeared consistent with the idea of reduced capacity to recruit top-down attentional control mechanisms in anxiety. However, other tasks have found opposite results. In a Stroop task - in which attention must selectively focus on one stimulus dimension and ignore others - increased PFC activation was observed (Basten, Stelzel, & Fiebach, 2011). Similarly, a heightened N2 component during inhibition was observed in a go-nogo task requiring sustained attention (Righi, Mecacci, & Viggiano, 2009). This was interpreted as reflecting increased, rather than reduced, recruitment of top-down control.

Potentially conflicting findings might be explained in light of ACT (Berggren & Derakshan, 2013; Eysenck et al., 2007). It is possible that when tasks are relatively simple and place low demands on participants, such as in Bishop (2009), those with high anxiety may not engage in compensatory strategies. However, as tasks increase in difficulty, greater resources may need to be marshalled in high-anxiety participants to compensate for possible deficits, leading to increased recruitment of PFC structures for top-down control. Consistent with this notion, individuals with high anxiety showed lower DLPFC activity at rest than those with low anxiety, but greater DLPFC activation during actual task performance (Fales et al., 2008).

More recent findings suggest that the processing of neutral tasks might be different in highly anxious participants in regions beyond the PFC. Individuals with high anxiety were superior in visual search tasks for neutral and affective cues (Berggren, Blonievsky, & Derakshan,

2014). This performance was linked to heightened event-related potential (ERP) responses in early visual components of the waveform. Such potentiated visual processing may be the consequence of feedback connections between the amygdala and the visual cortex (Amaral, Behnia, & Kelly, 2003).

In summary, anxiety appears to affect attentional processing in neutral contexts as well as those associated with threat. The idea of reduced processing efficiency in high anxiety (Eysenck et al., 2007) has been linked to the differential engagement of the DLPFC – a region implicated in the top-down control of attention – in those with high relative to low anxious tendencies (Berggren & Derakshan, 2013). It is also possible that earlier processing regions, such as visual cortices, may display processing differences in individuals with high anxiety (Berggren et al., 2014).

Risk-Taking and Attention

The final adolescent factor considered in this study is risk-taking. In the literature of adolescent risk-taking, risky decision-making and risky behaviors are considered to be those in which individuals place themselves in positions that may cause harm (Steinberg, 2007). Examples include –driving under the influence, unprotected sex, and drug and alcohol use. Adolescents engage in such risk-taking behavior to a greater extent than do adults or children (Steinberg, 2008). While risk-taking may be closely aligned with impulsivity (e.g. acting without thinking of harmful consequences) and risk-taking is heightened in those with ADHD (Drechsler, Rizzo, & Steinhausen, 2008; Williams & Taylor, 2005), studies investigating a potential link between risk-taking and attentional processes are rare.

Theoretically, attentional processes may be related to risk-taking. For example, heightened attention to or awareness of one's bodily feelings may affect decision-making (Bechara, Damasio, Tranel, Damasio, 1997; Bechara, Damasio, & Damasio, 2000). Shifting one's attention to consider possible gains or losses may also influence risk-taking, as occurs with framing effects (Kuhlberger, 1998; Tversky & Kahneman, 1981). Attentional biases towards addiction-related cues in addictive disorders (Field & Cox, 2008) might also interact with risk-taking propensity to increase likelihoods of engagement or relapse. Few previous studies have examined whether or not those who display higher risk-taking engage attentional processes differently than those who do not display such propensities.

The Balloon Analogue Risk Task (BART) (Lejuez et al., 2002) is a behavioral measure of risk-taking that correlates with real-world adolescent risk-taking (Lejuez, Aklin, Zvolensky, & Pedulla, 2003). Participants performing this task must decide whether to seek further gains by choosing to inflate a (computerized) balloon, at the risk of the balloon bursting and receiving nothing, or to cease inflation and obtain rewards they have accumulated. Decisions when participants choose whether or not to continue inflating the balloon involve activation of the ACC, DLPFC, anterior insula, and ventromedial prefrontal cortex (vmPFC) (Rao, Korkczykowski, Pluta, Hoang, & Detre, 2008; Schonberg, Fox, Mumford, Congdon, Trepel, & Poldrack, 2012). DLPFC, anterior insula, and ACC were activated when participants chose to continue with inflation, and vmPFC activity decreased (Schonberg et al., 2012). Similarly in a separate study (Rao et al., 2008), DLPFC, ACC, and anterior insula activation

was observed as participants continued to inflate the balloon, along with midbrain and striatal activation. Prefrontal regions such as the vmPFC have been suggested to underpin reward valuation, so reduced activation has been suggested to reflect decreasing expected value (Schonberg et al., 2012). Damage to the vmPFC is associated with heightened risk-taking (Bechara, Damasio, & Damasio, 1994). Thus, reduced vmPFC activation might also reflect poorer functionality in this region, in turn leading to heightened risk-taking.

In summary, risk-taking is associated with multiple psychiatric disorders and negative outcomes. Theoretical considerations and the association between ADHD and risk-taking indicate that risk-taking could be related to differences in attentional processing. However, studies have only recently begun to unpack the neural underpinnings of risk-taking. OFC/vmPFC and the ventral striatum appear to be key regions implicated in risk-taking.

Present Study

This study expanded upon previous research suggesting a link between attentional processing and impulsivity, anxiety, and risk-taking, by exploring associations between measures of these domains and neural activations during simple, selective, and divided attention. From the research outlined above, we hypothesized that specific brain regions would be differentially engaged during attentional tasks in relation to high or low impulsivity, anxiety, and risk-taking. However, whole-brain analyses were conducted given the exploratory nature of the study. Given the link between ADHD and hypoactivation of the frontoparietal network (Cortese et al., 2012; Rubia, 2011), we hypothesized that those with high impulsivity would show reduced activation in prefrontal regions involved in the top-down control of attention (DLPFC, ACC), as well as the IPL. Activation in the ventral striatum was hypothesized to correlate with impulsivity given previous research showing a link between impulsivity and ventral striatal structure and function (Carmona et al., 2009; Plichta et al., 2014).

Given previous research linking anxiety to differential functioning of the DLPFC during attentional tasks (Berggren & Derakshan, 2013; Bishop, 2009), we hypothesized that anxiety would modulate DLPFC activation. Some researchers (Berggren & Derakshan, 2013) suggest that high anxious individuals may display less DLPFC activation during simple tasks, with higher activation relative to low anxious participants in more strenuous tasks (e.g., divided attention). Hence, if the DLPFC showed heightened activation in highly anxious individuals during the divided attention task, this would be consistent with Berggren and Derakshan's (2013) model. However, the range of findings across different experimental tasks (Berggren & Derakshan, 2013; Bishop, 2009), most of which are very different to the present experiment, made firm predictions about the direction of DLPFC activation across specific conditions difficult. We therefore hypothesised that DLPFC activation would correlate with anxiety in the divided attention task, but did not specify the direction of this association. We also hypothesized that anxiety would relate to heightened activation of visual processing regions given prior findings (Berggren et al., 2014).

Finally, it was hypothesized that risk-taking would relate to differential engagement of the vmPFC/OFC and ventral striatum, given previous research linking activation of these

regions with risk-taking behaviour and risky decisions (Bechara et al., 2000; 1994; Rao et al., 2008).

Methods

Participants

Twenty-one adolescents were recruited from a longitudinal cohort to take part in this fMRI study. Participants had been recruited at birth and followed every 6 months thereafter. Exclusion criteria were mental or medical illness, use of psychotropic medications or medications that may influence autonomic responses, non-removable metal in the body, inability to tolerate scanning (e.g., due to claustrophobia or body weight), pregnancy, and the regular (daily or weekly) use of cigarettes or smokeless tobacco, and regular alcohol use. Participants with a positive urine toxicology test or positive breath alcohol test at either screening or study visits were not enrolled in the study. Two participants were excluded for technical reasons (excessive movement and use of a modified paradigm).

The final sample consisted of 19 adolescents (10 girls) between 12 and 18 years of age ($M=15.55$, $SD=1.53$). The Kaufman Assessment Battery for Children (KABC) composite score was used to determine IQ ($M=94.81$, $SD=14.28$, range=72.00-125.80) (Kaufman & Kaufman, 1983). Two participants did not complete the KABC in their study visits. Mental health was determined using the Schedule for Affective Disorders and Schizophrenia for School Aged Children (K-SADS) (Kaufman et al., 1997), a well-validated semi-structured interview for Axis-I DSM-IV disorders. No participants met the criteria for any Axis-I disorder.

Measures of impulsivity, anxiety, and risk-taking

Impulsivity was assessed using the Barratt Impulsiveness Scale, 11th edition (BIS-11) (Patton, Stanford, & Barratt, 1995), a 30-item self-report instrument used to assess impulsive traits. The BIS-11 has displayed good reliability and validity and has also been used in adolescents (Li & Chen, 2007; Stanford et al., 2009). Total scores range from 30-120, with higher scores indicating greater impulsivity. Scores from 52-71 are considered to be in a normal range (Stanford et al., 2009).

Anxiety was assessed using the Multidimensional Anxiety Scale for Children (MASC) (March, 1997), a 39-item self-report scale designed for use in children and adolescents. The MASC displays satisfactory to excellent reliability and validity (March, Parker, Sullivan, Stallings, & Conners, 1997; March, Sullivan, & Parker, 1999). The top 10th percentile for community samples has been estimated at 66-68, with a mean of 47-48 (Baldwin & Daads, 2007).

The Balloon Analogue Risk Task (BART) (Lejuez et al., 2002), a computerized measure aiming to approximate real-world risk behavior, was used to assess risk-taking. Participants performing this task are presented with the image of a balloon and given the opportunity to earn money by inflating it with a click of the mouse. Each balloon pump increases the money earned but carries increasing risk of bursting the balloon and losing all the money earned for that trial. In this study, participants were informed that they would accumulate

money at a rate of 2 cents per pump, though all participants were paid at a flat rate irrespective of performance at the end of the study. Participants are not informed of the probability of the balloon bursting. Propensity to take risks was measured using the primary measure of BART performance: the adjusted average number of pumps. This number represents the average number of pumps for unexploded balloons. The BART has been shown to have good reliability and validity, and has been employed in adolescent samples (Crowley et al., 2009; Crowley, Raymond, Mikulich-Gilbertson, Thompson, & Lejuez, 2006; Lejuez et al., 2002; 2003; White, Lejuez, & de Wit, 2008).

fMRI task

Participants familiarized themselves with the scanning environment in a mock scanner. The scanning procedure was described in detail to all participants and their parents, and any participant or parent questions were answered.

The fMRI task, described previously (Jacobsen, Slotkin, Mencl, Frost, & Pugh, 2007; Shafritz, Marchione, Gore, Shaywitz & Shaywitz, 2004) was programmed in E-Prime (Psychology Software Tools, Pittsburgh, PA). Trials in each task lasted 5 seconds. In both simple and selective attention, trials began with a 500 msec visual cue depicting an eye or an ear, telling subjects to attend to either visual or auditory cues, respectively. After a 500 msec pause, a word or nonword was presented in the cued modality. For simple attention, non-linguistic stimuli (four diagonal lines or a tone stimulus) were presented simultaneously in the unattended modality. For selective attention, word or nonword stimuli were presented simultaneously in the unattended modality. Participants then responded with a button press to indicate whether they heard a word or nonword in the attended modality.

In the divided attention trials, word or non-word stimuli were presented for 500 msec simultaneously. Then, after a 500 msec pause, subjects were presented with a visual cue depicting both an eye and an ear, with a circle around the eye, the ear, both, or neither. The circling of a cue indicated that the computer ‘predicted’ the stimulus presented in that modality would be a real word. Participants were required to respond with an affirmative button press if the computer’s prediction matched the stimuli presented, and respond negatively if this was not the case. For example, if the eye was circled and the ear was not, and the stimuli presented were visual “boat” and auditory “nord”, then the participant should respond affirmatively as these match the cues. If neither cue was encircled, and the subsequent stimuli were the visual word “boat” and the auditory word “nord”, then the participant should respond in the negative, as the cues indicated that neither word should be a real word.

Participants received 42 trials each for auditory and visual simple and selective attention, and 48 trials for divided attention.

fMRI parameters and analysis

Image Acquisition—Data were collected using a 3.0 T Siemens Trio MRI system with a standard quadrature head coil. Foam padding and a forehead-restraining band were used to prevent head motion. Two localizer scans were first run, followed by a T1 fast low angle

shot (flash) anatomical scan with the repetition time (TR) = 300 ms, echo time (TE) = 2.47 ms, flip angle (FA) = 60°, Bandwidth = 300 Hz/pixel, FOV = 220×220, imaging matrix = 256 mm², axial oblique plane with 30 slices, slice thickness = 4 mm. Next, ten functional runs were acquired using a single-shot, gradient echo-pulse sequence with the following parameters: TR=2000ms, TE = 30 ms, FA = 80°, Bandwidth = 2520 Hz/pixel, FOV = 220×220, imaging matrix = 64 mm², axial oblique plane with 30 slices, 4 mm slice thickness and 120 measurements. Finally, a magnetization-prepared rapid acquisition with gradient echo (MPRAGE) was used to acquire high-resolution anatomic scans. It had the following parameters: TR=2530ms, TE = 2.77 ms, FA = 7°, Bandwidth = 179 Hz/pixel, FOV = 256×256, imaging matrix = 256mm², sagittal plane with 176 slices, slice thickness = 1 mm.

Pre-Processing—All data were converted from Digital Imaging and Communication in Medicine (DICOM) format to analyze format using XMedCon (<http://xmedcon.sourceforge.net/>). During the conversion process, the first five images at the beginning of each of the ten functional series were discarded to restrict the dataset to images collected after a steady-state equilibrium between radio frequency pulsing and relaxation had been achieved, leaving 115 measurements for analysis. The functional data for each participant were corrected for slice time acquisition using sinc interpolation and corrected for motion using SPM99 (<http://www.fil.ion.ucl.ac.uk/spm>). Images were excluded from analysis if their displacement exceeded 1.5 mm and the rotation exceeded 2°. All subsequent analyses and registrations were completed using the tools within BioImageSuite (Joshi et al., 2011). Smoothing was carried out using a Gaussian filter of full width at half maximum (FWHM) of 6.88 mm. Five individual beta maps for each of the tasks (Visual Simple, Visual Selective, Auditory Simple, Auditory Selective, and Divided Attention) were generated for each subject by modelling blocks using regression-based methods implemented with the general linear model (GLM). Drift regressors were used to remove the mean time course, linear trend, quadratic trend, and cubic trend for each run resulting in a normalized beta map for each task in acquired resolution (3.438 × 3.438 × 4 mm³).

Image Registration—First, a linear registration was computed from the individual functional image to the corresponding individual participant 2D anatomical image. Second, a linear registration was computed from the 2D anatomical image to the individual participants' 3D structural image. Finally, a non-linear registration was computed between the individual 3D image and a standard reference 3D image (the Colin27 Brain; Woods et al., 1998). All three registrations were concatenated and applied as one registration to the normalized beta-maps in order to bring the data to a common reference space.

Group Analysis—To assess the group effects, each of the five maps was combined across subjects to generate group t-test maps. Individual scores on the BART, BIS-11 and anxiety measures were correlated with each voxel of the individual maps. Family-wise-error (FWE) correction for multiple comparisons was conducted using a Monte Carlo simulation with AlphaSim in AFNI. The AlphaSim parameters used were a smoothing kernel of 6.88mm, a connection radius of 6.296mm, and a resolution of 3.44×3.44×4 mm³. With these parameters, it was determined that an activation volume of 275 original voxels (7423 mm³) satisfied the $p < 0.05$ threshold. Subclusters have been generated to show the location and

extent of activation in different regions of the main cluster, but do not reflect separate statistical tests. Therefore, correlation values are only presented for each main cluster. This model did not include age or gender controls. In the supplementary materials we describe post-hoc tests that suggest these factors were unlikely to be confounds in our analyses, although BIS dropped to marginal significance ($p = .098$) in predicting mean beta values for the BIS visual selective attention cluster 1 when age, gender, and their interaction were added to the model (though these were non-significant predictors).

As it has been suggested that less stringent parameters may yield false-positive findings (Eklund, Nichols, & Knutsson, 2015), we present in the supplemental materials findings from analyses using stricter thresholds at which not all the results presented here survive.

Results

As participants were enrolled in part of a broader longitudinal study with a range of measures beyond those in the present study, not all participants completed all measures of impulsivity, anxiety, and risk-taking to prevent measurement fatigue in the study visits. Table 1 shows the group sizes, means, standard deviations, and ranges for measures used in each fMRI correlational analysis, along with demographic information for each group. The sample displayed a wide range of scores that might be expected from a population without any Axis-I disorder. Average BIS-11 scores were in approximately the middle of the normal range, with three participants scoring above and three below the normal range. MASC scores for the sample were lower than estimates from other community samples. There were no significant correlations between participants' scores on the BIS-11, BART scores, or total MASC scores (all $p > .1$).

fMRI results

Due to a technical error, task performance data during fMRI scanning were not available. The results below present correlations between fMRI blood oxygenation level dependent (BOLD) response and BIS-11, MASC, and BART scores. Scatter plots for each main cluster are presented in the supplementary materials.

Review of t -maps for activation in each condition (vs. the null hypothesis of no change in activation for that condition) showed that the tasks activated brain regions relevant to auditory and visual processing, such as primary auditory and visual cortices extending into further temporal (STG, MTG) and occipital (visual association area, peristriate area 19) regions. Additionally, frontoparietal activation consistent with engagement of attentional processing was observed (see Table S1 and Figures S1-S5). The following results focus on correlations between impulsivity, anxiety, and risk-taking scores, and brain activation in each condition.

Impulsivity and attention

BIS-11 total scores were significantly negatively correlated with activation in one cluster involving the bilateral OFC and DLPFC, and the left caudate and ACC, during visual selective attention (Table 2; Figure S6). These findings partially support our hypothesis that impulsivity would be negatively correlated with frontoparietal activation: hypoactivation of

prefrontal control regions was observed, but there was no association between impulsivity and parietal activation.

During divided attention, BIS-11 total scores were significantly negatively correlated with brain activation in one cluster involving the brainstem, the left caudate, putamen, amygdala and hippocampus (Table 2; Figure S7). We had predicted differential engagement of the ventral striatum, which was not observed. Instead, correlations implicating the dorsal striatum, as well as other unpredicted regions (e.g., hippocampus), were found.

No significant correlations were found between BIS-11 scores and brain activation during simple auditory or visual attention, or auditory selective attention.

Anxiety and attention

During auditory selective attention, MASC total scores were significantly positively correlated with activation in one cluster involving the right primary motor cortex, as well as the premotor and anterior premotor cortices (Table 3, Figure S8).

During visual selective attention, significant positive correlations were found between MASC total scores and brain activation in three clusters (Table 3, Figure S9). Cluster 1 included the right hippocampus, parahippocampal gyrus, fusiform gyrus, visual association area, and brainstem. Cluster 2 included the left visual association area, peristriate area 19, and extended into the posterior cingulate cortex. Cluster 3 included the primary motor and premotor cortices, bilaterally. These findings partially corroborate our prediction that anxiety would be positively correlated with activation in visual processing regions, although multiple other regions were also implicated.

During divided attention, MASC total scores were significantly negatively correlated with activations in one cluster (Table 3, Figure S10). This cluster contained the visual association area, peristriate area 19, extending into the IPL and SPL, and the intraparietal sulcus. We did not observe the hypothesized association between DLPFC activation and anxiety in this – the most demanding – attentional task. Activation in a DLPFC cluster that did not reach the required cluster size was actually negatively correlated with anxiety scores, contrary to what might be expected given Berggren and Derakshan's (2013) model.

No significant correlations were found between brain activation and MASC total scores during visual or auditory simple attention.

Risk-taking and attention

During auditory simple attention, BART scores displayed a significant positive correlation with brain activation in two clusters (Table 4, Figure S11). Cluster 1 included the bilateral hypothalamus, caudate, nucleus accumbens, temporal poles, and amygdala, the left VLPFC, vmPFC and subgenual cingulate, and the right medial and superior temporal gyrus.

During auditory selective attention, BART scores were significantly positively correlated with activation in three clusters (Table 4, Figure S12). Cluster 1 included the cerebellum and the left primary visual cortex and visual association area. Cluster 2 included the

OFC and DLPFC bilaterally, as well as the right caudate. Cluster 3 included the left IPL and primary sensory cortex, and extended into the right thalamus.

During visual simple attention, BART scores were significantly positively correlated with activation in one cluster, composed of the caudate and ACC bilaterally, as well as the right OFC (Table 4, Figure S13).

Finally, during divided attention, BART scores showed significant positive correlations with brain activation in 3 clusters (Table 4, Figure S14). Cluster 1 included the right fusiform gyrus and IPL. Cluster 2 included the primary motor and premotor cortices, extending into the VLPFC. Cluster 3 included the left primary motor cortex, primary sensory cortex, and IPL.

No significant correlations were found between BART scores and brain activation during visual selective attention. Hence, across tasks we frequently observed the predicted differential engagement of the vmPFC/OFC. The hypothesised differential engagement of the ventral striatum was observed in auditory simple attention, but a more dorsal striatal region (caudate) was more consistently correlated with risk-taking across tasks.

Discussion

Consistent with previous research indicating possible links between attentional processing and impulsivity, anxiety, and risk-taking, this study found that these features were related to differential neural activation during an attention task in regions consistent with previous research. As impulsivity, anxiety, and risk-taking were not inter-correlated, these findings suggest independent associations between brain activity and these tendencies.

Notably, the anticipated associations between DLPFC and ACC hypoactivation and higher impulsivity were confirmed during visual selective attention. An inverse association between striatal activation and impulsivity scores was also found during visual selective attention and divided attention, although this was dorsal, involving the caudate, rather than the nucleus accumbens. Activity in visual processing regions was positively associated with anxiety during visual selective attention, which is in line with our predictions. Finally, risk-taking scores were positively correlated with vmPFC/OFC and ventral striatal activation in auditory simple and selective attention, as well as visual selective attention, consistent with our hypotheses.

However, analyses implicated additional brain regions during phase-specific attentional processing that were associated with impulsivity, anxiety, and risk-taking. Many of these regions (e.g. visual processing regions, auditory processing regions, IPL and SPL) might be anticipated in tasks requiring attentional control over auditory and visual cues. Associations with subcortical regions, notably the hippocampus and amygdala in association with BIS-11, MASC, and BART scores in different attentional tasks, were not hypothesized but are not unexpected given their contributions to emotional and motivational processes (Cardinal, Parkinson, Hall, & Everitt, 2002; Phelps, 2004; Phelps & LeDoux, 2005). These results are discussed in more detail below.

Impulsivity and attention

Our finding of reduced activation in the DLPFC and ACC in those with high impulsivity during visual selective attention builds upon previous studies indicating that those with ADHD – a disorder often characterized by high-levels of impulsivity – show hypoactivation of regions in the frontoparietal network (Cortese et al., 2012; Rubia, 2011). This study suggests that when attention must be directed to visual cues with the inhibition of auditory cues, individuals without ADHD but showing higher levels of impulsivity display hypofunctionality of key prefrontal cortical regions in the frontoparietal network. However, because in-scanner performance data were not available, it is unclear whether this reduced activation might also be linked with poorer performance, or whether it, for example, may reflect different attentional processing strategies among those with high impulsivity.

During both visual selective attention and divided attention, hypoactivation in relation to impulsivity was also found in the caudate. Studies of self-reported impulsivity in those with ADHD (Carmona et al., 2009; Scheres et al., 2007) and healthy populations (Plichta & Scheres, 2014) have suggested differential processing in the ventral striatum in those with higher impulsivity during reward anticipation, and it has been suggested that hyporesponsivity might specifically relate to impulsivity in ADHD or other diagnostic groups (e.g., those with alcohol-use or gambling disorders; Balodis et al., 2012; Beck et al., 2009), with ‘normal’ impulsivity being associated with ventral striatal hyperactivity (Cortese et al., 2012). We observed hypofunctionality in the dorsal striatum. As the present study used attentional tasks rather than reward tasks, the finding of dorsal striatal hypofunctionality in this psychiatrically healthy sample does not contradict previous studies. Differential functionality of the dorsal as well as ventral striatum has been noted in previous studies (Carmona et al., 2009). Rather than contradicting previous research, our findings suggest that even in tasks outside of reward processing, striatal abnormalities may be observed in relation to impulsivity.

Impulsivity was also related to hypofunctionality of other subcortical regions, namely the amygdala and hippocampus, during divided attention. Whilst hippocampal regions were not initially hypothesized to be involved in the attentional tasks, research in animals has found that lesions to the hippocampus can induce impulsive behavior (Cheung & Cerdinal, 2005; Mariano et al., 2009), possibly due to hippocampal connections with the nucleus accumbens (Pattij & Vanderschuren, 2008). Amygdala dysfunction has also been implicated in impulsive or poor decision-making (Bechara et al., 1999; Matsuo et al., 2009), although Matsuo and colleagues (2009) did not find an association between amygdalar gray-matter volume and BIS-11 scores. It is therefore possible that activation of these subcortical regions is associated with impulsivity during attentional tasks, although this should be investigated in future studies with larger sample sizes.

It is notable that no associations between brain activation and impulsivity were found during either simple attention task. As suggested in previous studies of anxiety (Eysenck et al., 2007; Berggren & Derakshan, 2013), it may be that differential processing associated with these possible risk factors is most apparent when more demanding tasks (i.e. selective and particularly divided attention) are performed.

Anxiety and attention

We expected that our findings might help elucidate previous research regarding DLPFC function and anxiety during attentional tasks. In one study, Bishop (2009) found that high trait anxiety was linked with reduced DLPFC activation. However, drawing on other studies, Berggren and colleagues (Berggren & Derakshan, 2013) suggested that the DLPFC might be more activated in cognitively demanding tasks. Divided attention was the most demanding of the tasks used in the present study, yet we did not observe increased DLPFC activation in participants with high anxiety during this task. One subthreshold cluster in the DLPFC actually showed a negative association between anxiety and BOLD response during divided attention. Hence, our findings do not support Berggren and Derakshan (2013). However, it is possible that the difficulty of the divided attention task was not sufficient to produce the expected differential engagement of the DLPFC, even if it was more difficult than the other experimental conditions. Future research might specifically investigate at what difficulty levels the putative neural functioning abnormalities in those with high anxiety arise.

Participants reporting greater levels of anxiety showed reduced activation in the parietal components (IPL, SPL) of the frontoparietal attention network previously implicated in divided attention tasks (Vohn et al., 2007). Reduced recruitment of this frontoparietal network, used to direct attentional resources, in those with high anxiety could also explain reduced activation of visual (visual association area, peristriae area 19) and auditory/language (BA44, BA45) processing regions observed during divided attention. With increasing task demands, those with anxiety may struggle to cope and thus display hypoactivation of regions integral to the task.

During visual selective attention, individuals with high anxiety displayed the predicted increased activation of some visual processing regions (visual association area bilaterally and left peristriae area 19), although increased activation in the primary visual cortex was not observed. Anxiety has been frequently linked to heightened processing of visual cues, such as the modulation of early visual waveforms in ERP studies (Berggren et al., 2014). The present finding is consistent with this previous work, with the caveat that under more demanding attentional conditions, modulation of visual processing regions may break down, resulting in reduced activation. To further elucidate the mechanism behind such modulation of visual processing, functional connectivity analyses and diffusion tensor imaging might be conducted in future studies to indicate whether links between the amygdala and visual cortex could underpin visual processing differences in relation to anxiety, as suggested by previous researchers (Amaral et al., 2003; Berggren et al., 2014).

Contrary to expectations, MASC total anxiety scores were also associated with heightened activation of motor and premotor cortices during both auditory and visual selective attention, as well as increased hippocampal and parahippocampal gyral activation during visual selective attention. Hippocampal regions have been implicated in disorders of memory associated with anxiety, and it has been suggested that the ventral hippocampus may be particularly involved in anxiety-related behaviors (Bannerman et al., 2004). Our study suggests that differential engagement of the hippocampus may occur in relation to anxiety in contexts beyond memory. Differences in motor and premotor cortex activation in relation to anxiety are also a novel finding. One possibility is that performance anxiety may lead highly

anxious participants to prepare their responses more effortfully, thus potentiating motor regions of the brain. However, with the present findings this remains conjectural, and future studies should be used to assess the robustness of the associations between anxiety and hippocampal and motor activation during attentional tasks.

Risk-taking and attention

Consistent with previous research and our hypotheses (Bechara et al., 2000; Rao et al., 2008; Schonberg et al., 2012), scores on the BART were positively correlated with activation in the vmPFC or OFC in both simple and selective auditory attention, as well as visual selective attention. Risk-taking was also positively correlated with ventrostriatal activation during auditory simple attention, and dorsal striatal activation during simple and selective auditory attention. These findings are of interest in showing that differences in the function of frontostriatal circuitry implicated in risk-taking behavior are apparent in those with a propensity to take risks even in contexts not directly related to risk. Structural differences in frontostriatal circuitry have been implicated in risk-taking and related disorders (e.g. substance abuse), and it may be that the function of these regions are impaired across a range of tasks (Franklin et al., 2002; Peper et al., 2013). Such differences in activation may be particularly relevant in adolescence, a time when risk-taking is increased and frontostriatal circuitry is maturing (Galvan, 2013; Steinberg, 2007). It would be interesting to assess whether adults who are less risk averse also display altered functionality of these regions in attentional tasks, or whether this finding is specific to adolescent samples.

Brain regions previously implicated in selective and divided attention (Hahn et al., 2008; Johnson & Zatorre, 2006; Vohn et al., 2007) were also significantly more active in those with higher risk-taking scores. During auditory selective attention, high risk-taking was associated with increased activation in frontoparietal regions (DLPFC, IPL) involved in top-down attentional control, and with bilateral IPL activation during divided attention. Whilst little previous research has examined the relationship between risk-taking and attention, the fact that regions involved in attentional control were differentially active dependent upon risk-taking is suggestive of differences in attentional processing in relation to risk-taking status.

A broader range of brain regions than hypothesized was more active in those with high risk-taking, and may suggest difficulties in coping with the demands of the attentional tasks. For example, during auditory simple attention, heightened activation was evident in superior temporal regions implicated in auditory processing (Johnson & Zatorre, 2005; 2006). Increased activation in task-relevant regions during a low-demand task (e.g. simple attention) has previously been interpreted as reflecting reduced efficiency of the function they underpin (Kaladjian et al., 2011). Furthermore, those with high risk-taking also displayed heightened activation in some visual-processing regions (e.g. primary visual cortex, visual association area, peristriate area 19) during auditory selective attention, when attention to visual cues ought to be inhibited – an ability previously linked to decreased activation in visual regions (Johnson & Zatorre, 2005). Taken together, these findings suggest either impaired ability to focus attention to auditory cues, reduced efficiency in doing so, or different strategies to complete these tasks in those characterised by high risk-

taking. Further research examining performance in relation to brain activity could provide further insight.

Limitations

A main limitation of the present study was the loss of behavioral performance data as a result of technical error. Hence, it cannot be determined whether differential engagement of brain regions in relation to impulsivity, anxiety, and risk-taking are not also associated with differential performance at varying levels of these characteristics. However, the task *t*-maps do suggest that each condition elicited processing in appropriate brain regions (e.g. visual and auditory processing areas as well as frontoparietal activation associated with attentional processing), and further contrasts between conditions suggest that, for example, visual processing regions were more strongly recruited for visual selective than auditory selective attention, and auditory processing regions were more strongly recruited for auditory selective than visual selective attention, indicating that participants appeared engaged in the respective tasks. An additional limitation is the relatively small sample. Additionally, we employed relatively lenient thresholds given the exploratory nature of this study. Thus, the reported findings must be considered preliminary until replicated.

A further consideration is whether subscales of the BIS-11 and MASC might be associated with different neural substrates. Little research has considered the BIS-11 and MASC in relation to the neural processing of attentional tasks, so this exploratory study aimed to determine whether an association would be present in a non-clinical sample. Having established that BIS-11 and MASC scores are related to neural responses during attentional tasks, future studies could use this research as a foundation for more specific analyses looking at independent contributions of different subscale scores. Moreover, this study suggests a number of brain regions that could be used as regions of interest in less exploratory analyses of attention and impulsivity, anxiety, and risk-taking. From previous research and the results of this study, the DLPFC and ventral striatal regions appear of particular interest in relation to impulsivity. For anxiety, regions in the DLPFC and visual processing regions (e.g. primary visual cortex, visual association area, peristriae area 19) may be particularly fruitful. Motor and premotor cortices were also differentially activated in relation to anxiety in this study and warrant consideration. Finally, studies of risk-taking in relation to attention might focus on vmPFC/OFC, ventral striatum, as well as visual and auditory (e.g., occipital and temporal gyral areas) processing regions.

Conclusion

This study expanded upon previous research and theory by showing that, in healthy adolescents, differences in three putative risk domains (impulsivity, anxiety, and risk-taking) were related to differential activation of brain regions during an attentional task. Our finding of frontostriatal hypofunctionality in those with high impulsivity shows that differences in attentional processing associated with impulsivity are not necessarily dependent upon full-fledged disorders of inattention and disinhibition. Differential activation of visual processing regions dependent upon anxiety scores corroborate research indicating a relationship between anxiety and attention in affectively neutral contexts, though we did not find support for an association between DLPFC activation and anxiety (Berggren & Derakshan, 2013;

Bishop, 2009; Eysenck et al., 2007). Finally, we found evidence to suggest that risk-taking was associated with patterns of activation in frontoparietal and sensory processing regions that may indicate impairments in attentional control in high risk takers. The findings of this exploratory study can serve to guide future research probing the relationship between these risk factors and attention in more detail.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Descriptive statistics for impulsivity, anxiety, and risk-taking measures

Measure	n	Mean	SD	Range	M:F	Age M(SD)	IQ M (SD)
BIS-11	17	59.1	9.8	45-75	7:10	15.4 (1.5)	94.8 (14.6)
MASC	19	31.6	11.2	13-48	9:10	15.6 (1.5)	94.8 (14.3)
BART	16	28.2	10.9	10.6-45.5	8:8	15.7 (1.6)	94.4 (12.3)

BIS-11 = Barratt Impulsiveness Scale version 11; MASC = Multidimensional Anxiety Scale for Children; BART = Balloon Analogue Risk Task M:F = Male:Female ratio; M = Mean; SD = Standard deviation

Table 2

Regions with significant correlations between brain activation and BIS-11 total scores during visual selective and divided attention

Regions	Coordinates					Mean <i>r</i>
	Lat	BA	X	Y	Z	
<i>BIS-11 visual selective attention</i>						
OFC, DLPFC / Caudate, ACC / -	B/L/R	9/32, 46/10	-17	31	16	11726
Caudate	L	-	-16	22	7	1895
DLPFC, OFC	R	9, 10	8	43	20	1231
DLPFC, OFC, ACC	L	9, 10, 32, 46	-23	35	22	5145
<i>BIS-11 divided attention</i>						
Midbrain / Caudate, putamen, amygdala, hippocampus	B/L	-	-19	-1	-8	9265
Midbrain	B	-	-3	-13	-17	1131
Amygdala, hippocampus	L	-	-24	-6	-22	1397
Putamen	L	-	-24	2	-3	4420
Caudate	L	-	-14	9	6	959

ACC = anterior cingulate cortex; B = bilateral, BA = Brodmann area; DLPFC = dorsolateral prefrontal cortex; L = left; OFC = orbitofrontal cortex; R = right. Coordinates in MNI space.

Regions with significant correlations between brain activation and MASC total scores during auditory and visual selective attention, and divided attention

Table 3

Regions	Coordinates						Mean <i>r</i>
	Lat	BA	X	Y	Z	Volume(mm ³)	
<i>MASC auditory selective attention</i>							
M1, premotor and anterior premotor cortices	R	4, 6, 8	22	3	57	8003	0.53
<i>MASC visual selective attention</i>							
Hippocampus, parahippocampal gyrus, brainstem, fusiform, visual association area	R	-	39	-56	-3	17619	0.53
Hippocampus, parahippocampal gyrus	R	-	26	-30	-19	223	
Visual association area	R	-	39	-57	-3	17108	
Midbrain	B	-	8	-27	-22	283	
Visual association area, peristriate area 19, PCC	L	19, 23, 31	-35	-68	14	8450	0.51
M1, premotor cortex	B	4, 6	10	-22	62	13017	0.54
M1, premotor cortex	L	4, 6	-6	-20	57	2415	
M1, premotor cortex	R	4, 6	15	-21	63	9635	
<i>MASC divided attention</i>							
Visual association area, peristriate area 19, SPL, IPL, IPS	L	7, 19, 39	-24	-78	23	9891	-0.52

B = bilateral; BA = Brodmann area.; IPL = inferior parietal lobule; IPS = intraparietal sulcus; L = left; M1 = primary motor cortex; PCC = posterior cingulate cortex; R = right; SPL = superior parietal lobule. Coordinates in MNI space.

Table 4

Regions showing significant correlations between brain activation and BART scores during auditory simple and selective attention, visual simple attention, and divided attention

Regions	Coordinates						
	Lat	BA	X	Y	Z	Volume(mm ³)	Mean <i>r</i>
<i>BART auditory simple attention</i>							
Hypothalamus, caudate, NAcc, temporal poles, amygdala / VLPFC, vmPFC, subgenual cingulate / MTG, STG	B/L/R	38/11, 25, 47/21, 22	17	4	-17	19264	0.59
Hypothalamus	B	-	1	2	-15	1038	
Caudate, Nacc	B	-	-2	11	-9	1427	
Amygdala	L	-	-17	0	-26	564	
vmPFC, VLPFC, subgenual cingulate	L	11, 25, 47	-21	23	-18	4264	
Temporal pole, MTG, STG	R	21, 22, 38	55	-10	-15	7609	
Amygdala	R	-	29	8	-24	201	
V1, peristriae area 19, visual association area, cerebellum, cerebellar vermis	L	17, 19	-18	-67	-7	7893	0.57
V1, peristriae area 19, visual association area	L	17, 19	-17	-69	-3	6042	
Cerebellum, cerebellar vermis	L	-	-22	-59	-19	1851	
<i>BART auditory selective attention</i>							
Cerebellum / V1, visual association area	B/L	17	-21	-70	-21	9013	0.54
V1, visual association area	L	17	-16	-71	-1	2312	
Cerebellum	B	-	-23	-70	-28	6701	
OFC, DLPFC / - / caudate	B/L/R	9, 10/11/-	12	49	5	17823	0.58
Caudate	R	-	15	21	10	696	
OFC, DLPFC	R	9, 10	24	48	9	11837	
OFC, DLPFC	L	9, 10, 11	-15	56	-5	5010	
IPL, primary sensory cortex / Thalamus	L/R	1, 2, 3, 39, 40	-31	-36	30	11818	0.57
Thalamus	R	-	9	-30	12	179	
IPL, primary sensory cortex	L	1, 2, 3, 39, 40	-40	-36	35	8224	
<i>BART visual simple attention</i>							
Caudate, ACC / OFC	B/R	24-Oct	2	19	17	10825	0.56
Caudate	R	-	12	19	11	434	

Coordinates						
Regions	Lat	BA	X	Y	Z	Mean <i>r</i>
OFC, ACC	R	10, 24	20	40	7	3713
Caudate	L	-	-17	9	23	445
ACC	L	24	-1	21	18	344
<i>BART divided attention</i>						
Fusiform, IPL	R	39, 40	53	-39	22	7473
M1, premotor cortex, VLPFC	R	4, 6, 44	45	-4	31	10537
M1, IPL, primary sensory cortex	L	1, 2, 3, 4, 39, 40	-53	-32	33	10460

ACC = anterior cingulate cortex; BA = Brodmann area; DLPFC = dorsolateral prefrontal cortex; IPL = inferior parietal lobule; L = left; M1 = primary motor cortex; MTG = medial temporal gyrus; NAcc = nucleus accumbens; OFC = orbitofrontal cortex; R = right; STG = superior temporal gyrus; V1 = primary visual cortex; VLPFC = ventrolateral prefrontal cortex; vmPFC = ventromedial prefrontal cortex. Coordinates in MNI space.