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A Multimodal Assessment of Reward Sensitivity and Its Relationship to Pubertal Development and Individual Differences in Impulsivity

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Abstract of the Dissertation

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Recent changes in NIMH funding priorities have led to a greater emphasis on the identification of common and unique neural substrates of psychiatric disorders. High comorbidity among disorders suggests common traits that predispose individuals to a variety of outcomes. For example, trait impulsivity has been associated with a number of childhood behavioral disorders including attention deficit hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), and conduct disorder (CD). Impulsivity can be characterized by alterations in reward processing, with a number animal and human studies pointing to attenuated sensitivity to rewards among impulsive populations. Impulsivity has also been long associated with early adolescence; as children undergo puberty they become more behaviorally impulsive. However, to date no study has simultaneously examined multiple markers of reward sensitivity and pubertal development in relation to impulsivity. Using factor analysis, we combined multiple physiological and behavioral measures of pubertal development and reward sensitivity, as well as self-reported impulsivity in a sample of over 150 adolescent girls. We predicted a final model which included bidirectional connections between reward sensitivity and pubertal development, and that both of these factors would predict impulsivity. Consistent with our hypotheses and previous research, model fit was excellent. Reward sensitivity was inversely related to impulsivity and positively correlated with pubertal development, while pubertal development was positively associated with impulsivity. Follow-up analyses revealed that reward sensitivity best predicted impulsivity while controlling for the effects of pubertal development. Construct validity was supported by expected associations between latent factors and measures of externalizing and internalizing

psychopathology. The findings of the current study strongly support previous models linking attenuated reward response to increased behavioral impulsivity. Furthermore, results indicate that puberty may suppress relationships between these measures, through modulatory effects on reward processing.

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Introduction

Over the past decade, researchers have increasingly recognized that a thorough understanding of psychopathology requires a collaborative effort across multiple domains of study. In particular, there has been an increased emphasis on the psychophysiological and neuro-biological/functional contributions to the etiology, course, and treatment of psychiatric disorders. In 2010, the National Institutes of Mental Health (NIMH) began the research domain criteria (RDoC) initiative – a comprehensive system of analysis that seeks to identify fundamental behavioral processes, which can be identified through basic behavioral and neuroscience research, and that cut across categorical definitions of psychopathology, providing new insights into the etiology, course and treatment of mental illness (Insel et al., 2010). Although authors of this initiative advocate for research that cuts across multiple levels of analysis, the RDoC initiative clearly states neither normative nor psychopathological processes can be understood without first understanding their neural substrates. Thus, advocates of RDoC seek to reframe psychopathology through a better understanding of basic neurobiological processes and their related psychological expression.

The RDoC are broadly organized into families of domains. For instance, positive valence systems describe domains that include approach motivation, reward responsiveness, and reward learning behaviors. Abnormalities in positive valence systems may be particularly relevant to the etiology of a number of currently-defined disorders including attention-deficit/hyperactivity disorder (ADHD), conduct disorder (CD), depression, schizophrenia, bipolar disorder, and borderline personality disorder (BPD) (Crow, 1980; Crowell, Beauchaine, & Linehan, 2009; Moeller et al., 2001; Nestler & Carlezon, 2006; Forbes & Dahl, 2012; Rubia, 2011). The consensus of these reviews suggests that attenuated sensitivity to rewards and signals of

impending reward result in behavioral symptoms including anhedonia, apathy, and irritability – symptoms which characterize a number of disorders including mood disorders (e.g., depression, bipolar), schizophrenia, and BPD. Likewise, reduced reward sensitivity may also be an important etiological factor among externalizing disorders – including ADHD, CD, anti-social personality disorder (ASPD), and substance use disorders (SUDs) – through the mediation of trait-like differences in impulsivity.

Numerous researchers have hypothesized that alterations in dopamine (DA) activity preceding and following reward delivery among these individuals results in reduced overall neural reactivity to rewards, which manifests behaviorally through disruptions in reward learning and increased reward seeking behaviors (Sagvolden et al., 2005; Tripp & Wickens, 2008; Luman, Tripp, & Scheres, 2010). The disruptions in neural response to rewards, and subsequent behavioral manifestations, may result in impulsivity, a trait-like tendency to seek out immediately available rewards irrespective of potential consequences. Impulsivity may be linked to a number of negative developmental outcomes, and appears to cuts across all disorders of the externalizing spectrum, leading some to argue that dysfunctions in reward processing represent a core etiological pathway to externalizing spectrum disorders (Beauchaine, Hinshaw, & Pang, 2010; Beauchaine & McNulty, in press; Neuhaus & Beauchaine, 2013).

Impulsivity and Risk for Externalizing Psychopathology

Externalizing spectrum disorders frequently co-occur (see, e.g., Armstrong & Costello, 2002; Jensen, Martin, & Cantwell, 1997; Kessler et al., 2006; Willcutt et al., 1999), and share a number of behavioral and developmental characteristics, including a tendency toward high activity level (i.e., impulsivity) and low empathy, coercive and inconsistent parenting, and low family socio-economic status (Mesman & Koot, 2000; Miller & Eisenberg, 1988; Deater-

Deckard et al., 1998). Although some of these factors also confer risk for internalizing psychopathology (Zimmerman & Katon, 2005; Ge et al., 1996), considerable evidence suggests that externalizing and internalizing spectrum disorders follow unique developmental pathways (Frick & Morris, 2004; Loeber & Burke, 2011; Lansford et al., 2006), suggesting a potential etiological risk factor that differentiates internalizing and externalizing disorder development within largely identical environmental context(s). One potential candidate may be temperamental differences in behavioral inhibition. For example, a number of studies have shown that children who are excessively shy or inhibited in early childhood are more likely to develop internalizing disorders such as anxiety and depression (Rosenbaum et al., 1993; Kampman & Poutanen, 2011). Conversely, those who are disinhibited or impulsive as children are more likely to develop externalizing disorders (Quay, 1997; Tarter et al., 2003; Young et al., 2009). These findings have led some to suggest that, in the context of environmental stressors, the development of externalizing psychopathology may be in part dependent on individual differences in trait impulsivity, with high levels of impulsivity *uniquely* conferring risk for externalizing psychopathology (see, e.g., Beauchaine & McNulty, in press). Thus, externalizing spectrum disorders may reflect a number of different phenotypic expressions of a single etiological risk factor (i.e., impulsivity).

This supposition is largely supported by factor analyses, which indicate a two-factor structure of common mental disorders, which largely differentiates internalizing and externalizing spectra (Krueger et al., 1998; Krueger, 1999; Krueger, McGue, & Iacono, 2001; Krueger et al., 2002). Although these factors are related to one another, presumably reflecting some common process predisposing individuals to all forms of psychopathology, factor analyses specifically examining externalizing psychopathology in both child and adult samples indicate

that risk for externalizing disorders is conferred by a single, highly heritable latent factor (Krueger et al., 2002; Tuvblad et al., 2009). Numerous researchers have argued that this latent factor may reflect trait impulsivity. For example, Beauchaine and colleagues have argued that this inherited tendency toward impulsivity represents the core etiological factor in numerous disorders, including ADHD, CD, ASPD and BPD (Crowell, Beauchaine, & Linehan, 2009; Beauchaine et al., 2009; Beauchaine, Hinshaw, & Pang, 2010). This assumption has largely been supported by the literature, which indicates a strong link between impulsivity and many forms of externalizing psychopathology, not internalizing disorders (Moeller et al., 2001). Longitudinal studies also indicate that early childhood impulsivity is associated with increased risk for SUDs, ADHD, and disruptive behavior (Berlin, Bohlin, & Rydell, 2004; Nigg et al., 1999; Tarter et al., 2003). Thus, impulsivity appears to reflect a core psychological process, which is highly heritable, and predisposes individuals to externalizing spectrum disorders.

The Biological Basis of Impulsivity

Personality theories. Early etiological models of impulsivity were largely derived from theories of personality development, and specifically the work of Gray (1987). According to Gray, motivated behavior results from the interaction of two fundamentally opposed systems. The behavioral activation system (BAS) reflects appetitive motivation, or the tendency to seek out rewards in the individual's environment. In contrast, the behavioral inhibition system (BIS) is sensitive to punishment cues, and serves to motivate the individual to avoid potential negative or even dangerous situations. Gray and others have suggested that the relative balance of these two systems serves to predict the individual's typical pattern of arousal to external stimuli, as well as their response selection tendencies (Pickering & Gray, 1999).

Importantly, Gray's theory explicates specific neurobiological substrates of the BIS/BAS. Gray argues that approach behaviors are mediated by dopaminergic (DA) neurons within the mesolimbic system, an area of the brain that is particularly sensitive to rewards and plays a significant role in reward learning (Gray, 1987; Robbins & Everitt, 1996; Schultz, 1998). Avoidance behavior (BIS) is in turn linked to activation within the serotinergically (5HT)-mediated septo-hippocampal system, which has been shown to activate differentially in response to uncertainty or unpredictability and plays an important role in fear conditioning (LeDoux, 2000; Holland & Gallagher, 1999; Whalen, 2007). Consistent with Gray's theory, evidence from both human and animal studies suggests that the mesolimbic and septo-hippocampal systems are anatomically and functionally linked, and that input from the septo-hippocampal system has a modulatory effect on DA neurons within mesolimbic system (Ambroggi et al., 2008; Grace et al., 2007; Cador, Robbins & Everitt, 1989; Everitt et al., 1999; Uylings et al., 1991). According to this model, differences in personality are thought to reflect the relative balance between these two systems. For example, researchers have suggested that extraversion or trait-like impulsivity results from excessive activation of DA neurons within the BAS system in the absence of corresponding inhibition by the BIS system in response to potential cues of threat/conflict (Pickering & Gray, 1999).

Gray's theory was quickly adopted by psychopathology researchers, and related models of ADHD/aggression quickly emerged (Gorenstein & Newman, 1980; Quay, 1988, 1993; Fowles, 1988). These theories proposed that pathological impulsivity results from excessive behavioral activation, specifically *hyper-reactivity* of the mesolimbic system. This conclusion was based on the face-valid assumption that excessive reward-seeking/approach behavior is related to excessive dopamine activity within the mesolimbic reward system, consistent with

Gray's original model. However, there are clear and consistent findings that conflict with these early models, which suggest that impulsivity instead reflects dopaminergic *hypo-activation* within the mesolimbic system.

Mesolimbic reward system and impulsivity. Both animal and human studies indicate that manipulations of the mesolimbic system that disrupt or attenuate DA neuron activity result in the development of behavioral characteristics that are consistent with impulsivity in humans. Lesions to the nucleus accumbens (NAcc) – a region of the mesolimbic system that is particularly responsive to reward stimuli such as food, sex and drugs (Hernandez & Hoebel, 1988; Damsma et al., 1992) – result in significant alterations in reward-related activity, including increased approach behavior to potential rewards and a failure to learn reward-behavior contingencies, as well as persistent impulsive behavior (Parkinson et al., 2002; Cardinal et al., 2001). Likewise, pharmacological manipulation of DA alters delay of gratification, which reflects preference for smaller immediate rewards over larger delayed rewards, and is a core feature of impulsivity-related psychopathology such as ADHD (Luman, Oosterlaan, & Sergeant, 2005). In rat studies, drugs that increase the availability of DA result in an increased delay of gratification, while drugs that decrease DA neuronal firing result in a decreased delay of gratification (Wade, de Wit, & Richards, 2000). Similar evidence exists in human studies. For example, psychopharmacological agents that increase striatal DA or improve DA receptor function result in decreased impulsivity, while drugs that block striatal DA receptors result in increased impulsivity (Cools et al., 2007; van Gaalen et al., 2006).

One potential explanation of the inverse relationship between impulsivity and mesolimbic reward response may reflect the role of DA in the experience of positive and negative affect. There is evidence to suggest that mesolimbic DA is associated with trait and state differences in

positive affectivity, such that higher levels of DA are associated with positive hedonic experience while low mesolimbic DA is associated with increases in trait irritability and negative mood (Forbes & Dahl, 2005; Laasko et al., 2003). This link between low DA and negative affective states has led to the suggestion that the hyperactivity and reward-seeking behaviors seen in impulsive individuals reflect attempts to up-regulate negative mood, which is accomplished through the phasic release of DA that accompanies the immediate receipt of reward (Sagvolden et al., 2005; Tripp & Wickens, 2008; Gatzke-Kopp & Beauchaine, 2007; Beauchaine & McNulty, *in press*). However, as illustrated by these authors, the hedonic value of these rewards is short-lived (especially over repetition of the same rewards), resulting in the search for ever larger and more abundant rewards – a key descriptive characteristic of impulsive individuals.

Alternative explanatory models of impulsivity. However, not all explanatory models of impulsivity focus solely on the mesolimbic reward system. A number of theories of impulsivity or behavioral disinhibition suggest that phenotypic expression of this trait may result from the interaction of multiple basic processes spanning several distinct neural systems (e.g., Nigg, 2000; Beauchaine & McNulty, *in press*). These models suggest that impulsivity and related psychopathology result from the interaction of phylogenetically old brain regions, including the mesolimbic dopamine system, with higher order prefrontal executive control regions. According to these models, impulsive tendencies result from alterations in reward-responding, as mediated by the mesolimbic system, but only in the context of deficient down regulation by prefrontal cortex circuits. This supposition is supported by research indicating reduced functional and anatomical connectivity between prefrontal and striatal regions among highly impulsive individuals with externalizing disorders (Shannon et al., 2009; Konrad & Eickhoff, 2010).

Although prefrontal cortices likely play an important role in the manifestation of impulsivity, numerous lines of evidence suggest that the core neural risk marker for impulsivity is mesolimbic dysfunction. First, animal models indicate that impulsive behavioral responding results from selective lesions to the mesolimbic system, but that similar lesions to executive control regions implicated in prefrontal models of impulsivity (e.g., anterior cingulate/prefrontal cortex) fail to produce the same effect (Cardinal et al., 2001). Second, psychopharmacological agents that are used to treat impulsivity-related externalizing disorders have been shown to have the greatest impact on DA activity within mesolimbic system, as opposed to prefrontal regions (Volkow et al., 2005). For example, methylphenidate – a psychostimulant used to treat ADHD and related disorders – preferentially binds DA transporters in the striatum at a rate of nearly 4:1 (Vles et al., 2003). Finally, prefrontal regions of the brain develop gradually through childhood, and are not fully developed until late adolescence (Halperin & Schulz, 2006). Thus, if executive control regions were principal in the manifestation of impulsivity, one might expect age-related decreases in trait impulsivity and related psychiatric disorders. Although rates of ADHD are lower in adolescents compared to young children (Polanczyk et al., 2007), evidence supporting discontinuation of the underlying phenotype is inconsistent, as many individuals with ADHD go on to develop other impulsivity-related problems (Willoughby, 2003).

Reward Dysfunction, Impulsivity, and Psychopathology

Dopamine. Several characteristics of externalizing disorders have been linked to reduced DA within the mesolimbic system, including high trait irritability, reduced motivational salience of rewards, and engagement in perseverative behavior (i.e. continued response despite change in reinforcement contingencies; Laakso et al., 2003; Berridge & Robinson, 1998; Wise, 2004; Sagvolden et al., 2005). Genetics studies have demonstrated a link between externalizing

disorders and a number of dopamine genes, including the dopamine receptor genes DRD4/5, and the dopamine transporter gene DAT1 (Li et al., 2006; Sharp et al., 2009). These genetic findings have been shown to influence neurobiology, with genetic variation of these candidate genes accounting for approximately 10% of the variance in striatal reactivity to rewards (Forbes et al., 2007; Nikolova et al., 2011). Likewise, high-risk alleles of the DAT1 gene (10-repeat) have been associated with differentially greater DA transporter (DAT) expression (VanNess, Owens, & Kilts, 2005). Increased DAT activity is associated with reduced amounts of available DA in the synaptic cleft, and increased DAT density is seen in both children and adults with ADHD (Dougherty et al., 1999; Vles et al., 2003; Krause et al., 2000).

The mechanism of action of psychopharmacological interventions for externalizing psychopathology, specifically with regard to psychostimulant medication, provides further evidence of a DA deficit. Psychostimulant medications, which are the most efficacious treatments for childhood externalizing disorders, (MTA Cooperative Group, 1999), serve to *increase* DA concentrations by blocking DA transporters (Vles et al., 2003; Volkow et al., 2005). This increase in DA is associated with increases in positive affect (Volkow et al., 1999), but only improves performance on cognitive tasks among individuals with an apparent DA deficit prior to treatment (Mattay et al., 2000; Cools et al., 2007; Clatworthy et al., 2009). Thus, there exists an inverted-"U"-shaped function that predicts the effects of DA on cognitive performance, such that those individuals with low baseline DA are helped by psychostimulant medication, while those with normative levels of baseline DA are impeded cognitively by psychostimulants (Cools & D'Esposito, 2011). In concert with substantial evidence linking psychostimulant treatment with improved cognitive performance in children and adults with externalizing psychopathology

(Bedard et al., 2004; Boonstra et al., 2005; Swanson et al., 1991), these studies further implicate DA deficits in individuals with externalizing disorder.

fMRI measures. Functional magnetic resonance imaging (fMRI) studies indicate that highly addictive substances such as cocaine, as well as normative rewards including food, erotic images, social and monetary rewards all selectively activate the mesolimbic reward system (Knutson et al., 2001; Pelchat et al., 2004; Demos, Heatherton & Kelley, 2012; Aharon et al., 2001). Consistent with the notion that externalizing psychopathology is associated with reduced response to rewards, individuals with externalizing disorders show consistent attenuation of reward responding within this region. A recent meta-analysis of fMRI and positron emission tomography (PET) studies indicates that both children and adults with ADHD show replicable deficits in striatal reactivity to rewards (Plichta & Scheres, 2013). Likewise, individuals with SUDs show decreased striatal reward reactivity, particularly among individuals reporting higher substance craving and impulsivity (Beck et al., 2009; Wrase et al., 2007). Similar findings are also seen among aggressive children, such as those with CD and ODD, as well as adults with personality disorders characterized by impulsive aggression (e.g., ASPD, BPD) (Gatzke-Kopp et al., 2009; Finger et al., 2011; Völlm et al., 2007). Several studies have also demonstrated a negative association between mesolimbic reward response and symptoms of externalizing psychopathology, such that individuals with greater externalizing symptoms and impulsivity show less reactivity to rewards (Stark et al., 2011; Scheres et al., 2007; Sauder et al., *in prep*).

Event-related potential (ERP) measures. There is also convincing evidence that reactivity to rewards can be successfully indexed using electroencephalography (EEG). ERP studies of reward processing typically focus on feedback negativity (FN), a fronto-centrally located component that is more positive for rewards then losses (Hajcak et al., 2006; Holroyd &

Coles, 2002; Miltner et al., 1997). Although originally it was unclear whether the FN reflected gain vs. loss-related reactivity (Holroyd, 2004), recent research suggests that the FN reflects a reward-related positivity that is absent in loss conditions. FN also correlates with self-report measures of reward sensitivity (Bress & Hajcak, 2013), and recent work utilizing principal components analysis (PCA) has localized the source of FN to an area of the mesolimbic system (Foti et al., 2011). Furthermore, multimodal imaging studies of adults indicate that FN magnitude is strongly associated with fMRI measures of striatal reactivity to rewards (Carlson et al., 2011; Foti et al., *under review*).

Although less robust than findings of fMRI studies, some evidence does link blunted FN to externalizing psychopathology. For example, research examining children and adults with ADHD suggests less reward-related modulation of the FN signal, although attenuated positivity to reward conditions relative to loss was found in some (van Meel et al. 2011; Ibanez et al., 2012) but not all (van Meel et al., 2005) studies. Thus, consensus work suggests that FN is sensitive to rewards, corresponds with self-report and fMRI measures of reward reactivity, and may serve as an additional biomarker of impulsivity and risk for externalizing psychopathology.

Behavioral measures. Behavioral measures of reward may index similar processes as ERP and fMRI measures. For example, tasks tapping reward-learning processes, such as Pizzagalli's signal detection task, have been shown to index anhedonia – a symptom of depression that reflects insensitivity to environmental rewards (Pizzagalli, Jahn, & O'Shea, 2005).

Pizzagalli signal detection task (Pizzagalli et al., 2005). In the Pizzagalli signal detection task (SDT), participants are shown an ambiguous stimulus and must make a forced choice between one of two options. One of these options is rewarded with high probability ("rich"),

whereas the other option is rewarded with low probability ("lean"). Individuals are typically compared on the acquisition of a response bias, or the tendency to preferentially select the richly reward response over the lean rewarded response, which is believed to index sensitivity to reward in a learning context (Pizzagalli, Jahn, & O'Shea, 2005; Bogdan & Pizzagalli, 2006).

In addition to the relationship between performance in the SDT and symptoms of anhedonia and depression, there is also evidence linking task performance with externalizing psychopathology. When compared to healthy controls, boys with ADHD completing a similar task showed an attenuated response bias, reflecting a failure to alter behavior in response to reward contingencies (Tripp & Alsop, 1999). Research also indicates a link between response bias and DA signaling. Individuals who are given a DA-agonist, a drug that results in reduced phasic DA response to rewards – a deficit believed to contribute to the etiology of ADHD (Sagvolden et al., 2005), - show reduced acquisition of response bias (Pizzagalli et al., 2007).

Finally, performance on the SDT has been linked to ERP and fMRI measures of reward response. Individuals with a greater bias toward rewards in the Pizzagalli task had larger FNs to reward in a gambling task (Bress & Hajcak, 2013). In addition, a direct examination FN during this task indicates that individuals who successfully acquire response bias showing greater positivity to feedback than non-learners, and also show greater striatal activity to rewards as measured by fMRI (Santesso et al., 2008). Thus, there is some evidence suggesting that behavioral response during the SDT may directly index reward sensitivity.

Progressive ratio task (Chelonis, Gravelin, & Paule, 2011). In addition to the signal detection task discussed above, we will include a progressive ratio (PR) task, which measures behavioral motivation to seek rewards. PR tasks progressively increase the amount of effort required to obtain the same amount of reward, and index reward sensitivity by evaluating the

response rate of the organism and the relative point at which it quits responding (break point). Importantly, a number of animal studies have indicated that performance on the PR task is moderated by DA. These studies indicate that DA depletion, particularly within the striatum, results in reduced response rate and decreased break point (Zhang, Balmadrid, & Kelley, 2003; Aberman, Ward, & Salamone, 1998). Although fewer studies have evaluated such tasks in humans, there is some evidence to suggest that this task also indexes reward preference in humans (Roane, 2008).

Defining reward sensitivity. Thus, collectively, research to date suggests that reward sensitivity can be operationalized via multiple biological and behavioral markers. Those individuals who exhibit stronger ventral striatal response to reward stimuli, who have greater ERP response to cues indicating gain vs loss, who are engaged in more effort and have higher break points in the PR task, and who show greater response bias in the SDT can be characterized as more reward sensitive. There is also evidence to suggest that these measures index self-reported sensitivity to rewards. For example, higher scores on the Behavioral Activation Scale (BAS; Carver & White, 1994), a measure which supposedly assesses reward sensitivity and reward approach traits, are associated with mesolimbic response to reward (Simon et al., 2010). Likewise, those with greater BAS scores show exaggerated P300 amplitude, a positive deflection in the waveform indexing cognitive processing, which is consistently attenuated in impulsive samples (Nijs, Franken, & Smulders, 2007). Thus, it is reasonable to expect that an omnibus measure of behavioral and biological sensitivity to reward will be positively associated with self-reported reward sensitivity, and importantly negatively associated with impulsivity.

Developmental Effects on Reward Processing

Although the studies reviewed above include both child and adult samples, evidence points to adolescence and pubertal development as a particularly important time in the development of reward-related neurobiology and function. For example, puberty is associated with increases in sensation seeking behavior, which is characterized by increased behavioral reward sensitivity and corresponding alterations in neurophysiology (Martin et al., 2002; Van Leijenhorst, Westenberg & Crone, 2008; Van Leijenhorst, Zanolie et al., 2010). During adolescence individuals become more sensitive to both social and non-social rewards, and a number of studies support the importance of this developmental period, demonstrating age-related differences in reward responsivity within the ventral striatum and other reward-related prefrontal regions (Blakemore, Burnett & Dahl, 2010). Although most studies examine these changes as a function of age, there is convincing evidence that behavioral and neural responding to rewards is more strongly mediated by hormonal changes associated with puberty, providing further evidence of the critical nature of this developmental period (Blakemore et al., 2010; Op de Macks et al., 2011). However, it is important to note that despite developmental changes in reward responding associated with puberty, neural response to rewards continues to be modulated by behavioral differences in risk-taking propensity across all age groups (Van Leijenhorst, Moor et al., 2010). Thus, individual differences in reward-seeking traits likely interact with age and pubertal changes in hormones, suggesting that reward responding is affected by both developmental and individuals differences factors.

Adolescence is also an important period in the development of externalizing psychopathology. For example, antisocial behavior, which is associated with disorders such as ODD and CD, increases markedly in adolescence before declining again into adulthood (Moffit, 1993). Likewise, adolescence may be a critical period in the development of substance use

disorders (Kandel et al., 1997). Importantly, these problem behaviors tend to show a non-linear relationship with both child and adult problems, suggesting a peak that is specific to adolescence. This peak may result from adolescent-specific discontinuities in the neural development of reward circuitry and prefrontal regions, which some have argued underlie general increases in risk-taking behavior and impulsivity (Casey, Jones, & Hare, 2008; Van Leijenhorst, Moor et al., 2010). While such studies implicate pubertal effects on reward processing in the etiology of externalizing psychopathology, evidence indicates that this relationship may be mediated by individual differences in impulsivity. For example, increases in sensation seeking is associated with pubertal development, but also mediates the relationship between pubertal development and substance problems (Martin et al., 2002). Thus, a number of converging lines of evidence indicate that adolescence is a critical period in the development of neural circuitry of reward processing, impulsivity, and externalizing psychopathology.

Current Study

Despite significant evidence linking hyposensitivity to reward to individual differences in impulsivity and externalizing psychopathology, especially during adolescence, no study to date has examined multiple markers of reward response concurrently. Consistent with the RDoC initiative, the current study seeks to combine multiple biological and behavioral measures of reward sensitivity, and in turn relate these measures to measures of impulsivity and pubertal development. Exploratory factor analysis (EFA) was used to identify a single factor solution combing biological and behavioral measures of reward (reward sensitivity), hormone and self-report measures of pubertal development (pubertal development), and multiple self-report measures of impulsivity (impulsivity). The expected relationship(s) between these factors was subsequently tested via confirmatory factor analysis (CFA). Although a similar finding might be

expected in association with externalizing psychopathology, the normative nature of the sample in the current study precludes a thorough examination of this relationship given expected limited variance on measures of psychopathology.

The primary hypothesis in the current study is that reward sensitivity would be negatively associated with self-report measures of impulsivity. In addition, we hypothesized that reward sensitivity would be positively correlated with pubertal development, based on evidence indicating that reward sensitivity peaks during mid-adolescence (approximately at the upper end of the current study age range) and that these changes better mediated by pubertal development than chronological age (Van Leijenhorst, Moor et al., 2010; Van Leijenhorst, Zanolie et al., 2010; Op de Macks et al., 2011; Blakemore et al., 2010). Likewise, previous research suggests that impulsivity, in particular sensation seeking, rises dramatically in adolescence (Romer, 2010). Thus, we predicted that pubertal development would be positively associated with impulsivity. Finally, given the expected inverse relationships reward sensitivity and pubertal development with impulsivity, we predicted that pubertal development would moderate the relationship between reward sensitivity and impulsivity.

We also sought to assess the construct validity of our latent factors. Previous studies have suggested that childhood depression is associated with attenuated response to rewards, while pubertal development is a risk factor in the development of depression, particularly among girls (Forbes & Dahl, 2012; Angold, Costello, & Worthman, 1998). Based on this work we hypothesized that pubertal development would be positively associated with depression, while reward sensitivity would be negatively related. In contrast, while externalizing disorders such as ADHD are thought to be associated with reduced reward reactivity (Neuhaus & Beauchine, 2013), there is no reason to suspect that symptoms of ADHD would increase through puberty –

particularly given that the diagnostic criteria of the disorder require the development of symptoms prior to the age of 7 (APA, 2000). Thus we hypothesized that reward sensitivity would be negatively associated with symptoms of ADHD, but that there would be no effect of puberty. Finally, we predicted that reward sensitivity and pubertal factors should both predict other measures of impulsivity not included in the model, consistent with the primary hypotheses above.

METHOD

Participants

The final sample included 153 girls aged 8-15 (M = 12.32, SD = 1.78) who completed at least one biological measure (e.g., EEG/fMRI). The sample self-identified, 85% Caucasian, 5.9% African American, 3.3% Hispanic, 5.9% other. The sample was part of a larger study examining pubertal development and risk for depression, which utilized a general community sample recruited from Suffolk County, New York. To be included in the study, children must live with at least one biological parent and both child and parent must speak English well enough to complete study assessments. Children with significant developmental or medical disability were excluded, as were those using psychotropic medications. Additionally, children with previous head trauma or other fMRI contraindications were excluded.

Procedure

Participants completed a laboratory visit at Stony Brook University consisting of semi-structured interviews, self-report measures, and the collection of behavioral, hormonal, fMRI, and EEG data. Participants will complete visit tasks in a pseudo-random order, counter balancing the order of EEG and fMRI recording. Prior to fMRI data collection, participants will have an opportunity to acclimate to the MR environment by undergoing a mock-scanning procedure,

during which participants will complete training for the doors reward task described below. In accordance with institution review board (IRB) policies, informed consent (from the child's parent) and assent will be obtained.

Measures of Psychopathology

Conners' Parent Rating Scale – Revised (CPRS-R; Conners et al., 1998). The CPRS-R is a parent report measure of childhood behavior problems across seven empirically validated domains (cognitive problems, oppositionality, hyperactivity-impulsivity, anxious-shy, perfectionism, social problems, and psychosomatic complains). T-scores are calculated for each subscale based on number of symptoms endorsed in relation to an age/gender-matched healthy control sample. In the current study we will utilize the ADHD index in order to assess external validity of the expected impulsivity and reward-sensitivity measures.

Child Depression Inventory (CDI; Kovacs, 1985). The CDI is a short self-report scale for children that assesses symptoms of depression. The CDI has strong internal consistency and reliability, as well as construct and discriminant validity (Kovacs, 1985; Carey et al., 1987). In the current study, the CDI was included only to assess the validity of our purported reward and puberty factors. Previous research has shown that decreased reward sensitivity is associated with depression in both fMRI and ERP measures (Forbes, 2006; Foti & Hajcak, 2009), and that the onset of puberty is associated with increased depression, especially among girls (Angold et al., 1998). Thus, the inclusion of the measure will allow us to examine the external validity of our latent factor(s).

Measures of Impulsivity

Personality Inventory for DSM-V (PID-V; Krueger et al., 2012). The PID-V is a 220 item self-report measure designed to assess personality based on conclusions from the

Personality Disorders Workgroup for DSM-V. The measure includes 220 items that load on a total of 25 empirically validated facets and a total of five broad based factors (negative affect, detachment, antagonism, disinhibition, and psychoticism), which show strong internal reliability (Krueger et al., 2012). In the current study we will utilize the impulsivity and risk taking factors. Scores on each factor are normed on a 4-point scale, with a value of 2 representing a moderate level of each factor.

UPPS Impulsive Behavior Scale (UPPS; Whiteside & Lynam, 2001). The UPPS contains 45 items and assesses a number of impulsivity-related traits including: negative urgency, lack of premeditation, lack of perseverance, and sensation seeking. These subscales have strong internal consistency, and have been shown to reliably index externalizing psychopathology (Whiteside et al., 2005; Miller et al., 2010). A subset of 11 items from this scale, which most strongly load on the above mentioned impulsivity-related traits, was utilized in the current study, with the average across all items reflecting overall impulsivity on a scale from 1-4.

Behavioral Approach / Behavioral Inhibition Scales (BAS/BIS; Carver & White, 1994). The BAS/BIS measures two general motivation systems thought to underlie behavioral approach and inhibition, consistent with the theory of Gray (1987). In the current study we evaluated one of three potential BAS scales: BAS Fun Seeking. Carver & White's own work (1994) demonstrated a strong, unique relationship with the BAS Fun Seeking scale and the Tridimensional Personality Questionnaire (TPQ) Novelty Seeking scale (Cloninger, 1987), which in turn has been linked to abnormalities in reward centers of the brain, symptoms of hyperactivity/impulsivity, and substance abuse (Epstein et al., 1996; Johnson, Waid, & Anton, 1997; Gomez & Corr, 2010). The BAS Fun Seeking subscale has also been more strongly

associated with behavioral impulsivity than other BAS subscales, which tend instead to index sensitivity to rewards (Franken & Muris, 2006). The BAS Fun Seeking scale shows strong test-retest reliability (r = .69). Possible scores on the BAS Fun Seeking items (summed) range from 4 to 16, with lower scores reflecting greater "Fun Seeking".

Pubertal Measures

Pubertal Development Scale (PDS; Peterson, Crockett, & Richards, 1988). We will obtain adolescent and parent report of pubertal development using the PDS, which assesses several physical indicators of puberty (e.g., body hair, breast development, growth spurt). The measure consists of 5 items (1-4 scale reflecting level of development), creating a continuous outcome measure for both adolescent and parent report. Previous studies indicate that PDS scores are highly correlated with pubertal stages as assessed by both physical exam and hormone levels (Shirtcliff, Dahl, & Pollak, 2009).

Picture-Based Interview about Puberty (PBIP; Shirtcliff et al., 2009). Both parent and adolescent will complete the PBIP, a picture based interview of pubertal development. The PBIP includes a script-driven examination of several color photos of adolescent girls through various stages of pubertal development, including both breast and pubic hair images. After the PBIP is explained by a female research assistant, parent/adolescent will complete PBIP ratings (1-5 based on level of breast/pubic development) through use of a self-driven PowerPoint presentation. Previous studies indicate the scores on the PBIP and PDS are highly correlated (Op de Macks et al., 2011).

Hormone Collection. We will collect saliva samples to assess testosterone and estradiol hormones. Testosterone and estradiol levels both increase during pubertal development, and have been linked to reward-related brain activity in adolescent girls (Op de Macks et al., 2011). Two

saliva samples will be obtained for each participant by passive drool (see Shirtcliff et al., 2001) and a consensus measure will be calculated for both testosterone and estradiol, consistent with previous studies (Op de Macks et al., 2011). Samples will be stored in a freezer and sent together to be assayed by Salimetrics (http://www.salimetrics.com/), a commercial service utilized by saliva researchers across the world.

Behavioral Tasks

Signal Detection Task (SDT; Pizzagalli et al., 2005). The SDT is designed to assess bias toward frequently rewarded responses. On each trial, participants are presented with a fixation cross (1400ms) followed by a line drawing of a face without a mouth (500ms). Subsequently, either a short (11.5mm) or long (13mm) straight line representing a mouth appeared for 100ms, followed by another drawing of a face without a mouth (remaining on the screen until participant response). Participants are then asked to differentiate between the short and long mouth type by button press. Following response, either a blank screen is presented, or feedback indicating participants guessed correctly and won five cents.

A total of 160 trials will be presented across 2 blocks, with an equal number of "short" and "long" mouth conditions in pseudo-random order. For each participant, one type of correct response (short or long) will be rewarded with greater frequency. Previous research indicates that participants develop a response bias (RB) toward the more richly rewarded response over the course of the task, reflecting the degree to which participants monitor and modify their behavior to obtain rewards (Pizzagalli et al., 2005).

Progressive Ratio Task (Chelonis, Gravelin, & Paule, 2011). In the current study we will use an adapted version of the progressive ratio (PR) task described by Chelonis and colleagues (2011). PR tasks index motivation to obtain reward by asking participants (or

animals) to engage in increasing amounts of effort to obtain the same reward (Roanne, 2008; Chelonis et al., 2011). In our adaptation, participants receive a monetary reward ($0.10) in response to pressing a spacebar on a standard keyboard. The number of presses required to obtain a monetary reward increases by a total of 10 presses with each successive reward (e.g., participants receive a reward for 1, 11, 21, 31, etc. presses). Participants are told that they can continue to play the game as long as they would like to keep receiving additional dimes, but that they may quit at any point (there is a 10 minute time limit to the task unbeknownst to the participant). We will examine a number of outcome measures including speed of button press, number of presses, length of time before quitting, and effort (reflecting speed, number of presses, and time played).

Structured Clinical Interview

Both the primary care-giver and child will be interviewed by an advanced graduate student using the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS-PL; Kaufman et al., 1997), 2009 revision. For each childhood DSM-IV (American Psychiatric Association [APA], 2000) diagnosis, information from both child and parent is collected on all diagnostic symptom criteria. Consensus ratings are then created by the interviewer, in conjunction with other interviewers and supervisors. The K-SADS-PL has demonstrated high inter-rater and test-retest reliability, and good concurrent validity (Kaufman et al., 1997).

We anticipate rates of externalizing disorders consistent with female population estimates, approximately 4-8% for ADHD with rates of ODD and CD ranging from 2-15% (Cohen et al., 1993; Maughan et al., 2004; Pastor & Reuben, 2008). Due to expectations that only a small proportion of the sample will meet diagnostic criteria for externalizing psychopathology, symptom counts, reflecting the number of diagnostic criteria met for each

disorder, will serve as the outcome measure. Symptom counts will be examined for childhood externalizing disorders, including ADHD, ODD, and CD, as well MDD.

Doors Reward Task

The current study will utilize a variant of the "Doors" gambling task used in previous studies utilizing ERP and fMRI measures of reward in adults and children (Carlson et al., 2011; Foti et al., 2011; Bress & Hajcak, 2013; Bress et al., 2012). During each trial participants are instructed to select, via a button press with their dominant hand, one of two doors presented on the screen. Participants are told that if they guess correctly they will win $.50, while incorrect guesses result in a loss of $.25, with values selected based on evidence indicating that the subjective value of loss is greater (Tversky & Kahneman, 1992). Despite the illusion of control, reward contingencies are fixed with an equal number of gain (green arrow pointing up) and loss (red arrow pointing down) trials.

ERP and fMRI versions of the task differ slightly. In the ERP version of the task, each trial begins with a fixation cross presented for 1,000ms followed by an image of two doors, which remained on the screen until the participant makes a response. Response is followed by a 1000ms fixation, and feedback lasting 2000ms indicating reward or loss. After feedback another 1500ms fixation is presented, prior to a screen allowing participants to continue when ready (by button press). The task includes three blocks of trials, each with a total of 20 trials and a break (duration determined by participant) between.

During fMRI acquisition, a total of 60 trials will also be presented, with alterations in timing to allow for the dynamics of the hemodynamic response function (HRF). Each trial begins with 600ms fixation, followed by the presentation of two doors for 3000ms. Participants are instructed that if they do not select a door while the doors remain on the screen, a door will be

selected for them. A subsequent fixation cross is presented for 600ms prior to feedback, which lasts 1000ms. Each trial ends with a fixation cross, with a jittered duration between 1100 and 11600ms (average 3200ms). Finally, a long fixation of 21000ms will be included at the start and end of acquisition, for a total task time of 9.1 minutes.

Psychophysiological Recording and Data Reduction

Consistent with standard lab procedures, continuous EEG will be recorded using a 34-channel ActiveTwo BioSemi system (BioSemi, Amsterdam, Netherlands). EEG signal will be sampled at a rate of 1024 Hz with at 24-bit resolution and a low-pass fifth-order since filter using a half-power cutoff of 204.8 Hz. Offline-analysis will be completed in Brain Vision Analyzer software (Brain Products, Munich, Germany). All data will be referenced to the average of two mastoids electrodes and bandwidth filtered with .1 and 30 Hz cutoffs. EEG will be segmented for each trial, from -200ms to +800ms relative to feedback onset, and each trial will be corrected for blinks and eye movements consistent with Gratton, Coles, & Donchin (1983). Semi-automated channel rejection will be used for each trial (step of > 50 μV between samples, within trial signal Δ > 300 μV, or less than 0.5 μV maximal signal difference within 100-ms intervals), with additional identification of physiological artifacts by visual inspection.

Principal Components Analysis of Feedback Negativity

Feedback-locked ERPs will be averaged separately for gain and loss trials (-200ms to +800ms) for each participant using the average activity from 200ms to feedback onset as the baseline. Feedback related ERPs are then calculated using temporospatial PCA, a data reduction technique that identifies unique combinations of temporal and spatial factors that are believed to reflect discrete ERP components (Dien, 2010a; Foti et al., 2009). This approach produces a more accurate estimate of feedback related ERP components, in part due to its ability to isolate these

components from other overlapping ERP components. Previous research indicates that this process allows for more accurate estimation of the relationship between FN and other neural measures of reward, for example fMRI measures of striatal response (Foti et al., 2011). PCA will be conducted using ERP PCA Toolkit, version 3 (Dien, 2010b). First, a temporal PCA will be performed on average data for each trial type (gain/loss) across all participants to isolate unique factors across time points. Due to expected covariance between temporal factors, a direct oblimin rotation allowing for non-orthogonality will be used. This method is expected to significantly reduce the number of temporal dimensions (1024 observations for each trial type, for each participant), as demonstrated in previous research with adults (Foti et al., 2011). Following rotation, a parallel test will be conducted on the resulting Scree plot, comparing PCA eigenvalues from the current dataset to those expected from a random dataset (Horn, 1965; Cattell, 1966). As explained by Dien (2010a), the number of factors retained will reflect the number of factors accounting for a greater proportion of the variance than the random dataset.

Temporal factors, which retain all spatial information (e.g., full scalp topography), will then be further reduced by a subsequent spatial PCA. A spatial PCA will be conducted on each temporal factor retained from the previous step, using an Infomax rotation consistent with previous work (Dien, 2010a; Dien et al., 2007). A similar parallel test comparing spatial PCA factors from the study dataset to a random dataset will be conducted to determine the number of spatial factors retained. Thus, the number of temporal-spatial factors retained will be equal to the number of temporal factors x the number of spatial factors (based on the results of the parallel tests) (Dien, 2010b). Factors that fail to account for at least 1% of the variance will be excluded, and remaining factors will be reconstructed into μV-scaled waveforms by multiplying the factor loadings, scores, and standard deviations (Dien, 1998). Finally, remaining temporal-spatial

factors will be submitted to a one-way ANOVA to identify factors that differentiate loss and gain conditions. In the advent that more than one temporal-spatial factor combination differentiates these two conditions, a visual inspection will be used to select the factor combination that best conforms to the expected FN waveform based on previous studies.

MRI Acquisition

fMRI acquisition. Data will be acquired using a Siemens Magnetom Trio 3T system. A single functional run (consisting of 260 volumes, approximately 9 minutes) will be collected using a T2*-weighted echo-planner imaging (EPI) sequence with 37 continuous slices (thickness = 3.5mm), TR = 2100ms, TE = 23ms, FOV = 224 x 224mm, 83° F.A.

Anatomical acquisition. A high resolution magnetization prepared rapid gradient-echo (MPRAGE) anatomical scan will be acquired for use during normalization of the functional data to standard Montreal Neurological Institute (MNI) coordinate space. The scan acquisition will last 4 minutes 26 seconds, TR=1900ms, TE=2.53ms, collecting a total of 176 slice 1mm slices and an in-plane resolution of 1x1mm (1mm iso-voxel).

B0 field map acquisition. Field maps will be acquired for use during motion correction. Acquisition will last approximately 1 minute, with parameters matched to EPI acquisition (see above), TE-short = 4.92ms, TE-long = 7.38ms.

fMRI Data Analysis

Preprocessing. Data will be processed using SPM8 (http://www.fil.ion.ucl.ac.uk/spm/software/spm8/) software. Initial preprocessing will consist of slice-time correction, and motion correction and deformation of field nonhomogeneity using pre-collected field maps and SPM's unwarping procedure. Data will subsequently be examined using ArtRepair5, an artifact detection and repair tool (Mazaika et al., 2009). Due to potential spin-

effects from rapid movement, data with greater than 1mm between-scan rapid movement will be interpolated (nearest-neighbor replacement, with data de-weighting occurring at first-level estimation – see below). Participant data will then be re-analyzed using interpolated data (slice-time and motion correction as described above). At this stage, participants with inter-EPI motion (>1.5mm rapid movement between scans) that exceeds 15% of the total EPI scans acquired will be excluded. Data will then be normalized to the MNI template using Diffeomorphic Anatomical Registration Through Expotentiated Lie Algebra (DARTEL; Ashburner, 2007), a high dimensional warping process. Finally, data will be smoothed using an 8mm Gaussian kernel.

Post-processing. A general linear model (GLM) will be created with reward *minus* fixation and reward *minus* loss modeled at the first level for each participant, and excluded scans and motion parameters from preprocessing included as covariates. Subsequently, a second-level mixed-effects GLM will be created using a one-way *t*-test for the reward vs. fixation and reward vs. loss conditions, separately, to examine the effects across participants. Resultant statistical maps will be corrected for multiple comparisons using a family-wise error (FWE) correction of $p < .05$. Based on previous studies with adults (Carlson et al., 2011), we anticipate that reward conditions will produce an area of significant activation within the ventral region of the striatum. Beta-value estimates for each participant will be extracted from this functional region of interest (ROI) using SPM's principal eigenvariate. Eigenvalues for each participant will be included in exploratory factor analysis (EFA; see below).

Data Analysis

Exploratory factor analysis (EFA). Three separate EFAs were conducted using AMOS software (http://www-03.ibm.com/software/products/en/spss-amos). The first model, representing reward sensitivity, included behavioral indices of reward sensitivity from the

Pizzagalli and progressive ratio task, striatal reactivity to rewards as measured by fMRI, and feedback negativity as indicated by the best fitting factor(s) from the temporal-spatial ERP factor analysis. The second model reflected trait-like differences in impulsivity by combining child report measures (UPPS, PID-V, and BAS Fun Seeking). The third model of pubertal development was created from parent and adolescent report measures (PDS; PBIP) as well as pubertal hormones (estradiol and testosterone). In all cases, we attempted to extract a single factor using all available predictors. However, in the advent of poor model fit, individual predictors that accounted for less than 10% of the total variance in the latent factor, or which are the least predictive factors, were dropped until model fit (or just-identification) was achieved. In addition, whenever possible (i.e., provided sufficient *df* for model identification), residuals of common metrics were allowed to covary. Thus, we sought the most parsimonious and best fitting factor representation, while restricting modeling to a single factor solution.

Confirmatory factor analysis (CFA). A CFA was conducted to test the study hypotheses: 1) pubertal development is positively associated with reward sensitivity, 2) pubertal development is positively associated with impulsivity, and 3) reward sensitivity is negatively associated with impulsivity. Final models from the three EFAs described above were combined in a single model, with modelled covariance between the reward sensitivity and pubertal development factors, and unidirectional relationships between both reward sensitivity/pubertal development and impulsivity. Final model fit was examined via Chi-squared statistic, root mean square error of approximation (RMSEA), comparative fit index (CFI), and the Tucker-Lewis index (TLI). Model fit was considered to be achieved if RMSEA < .05, CFI & TLI > .95, and Chi-squared was non-significant.

Assessment of moderation effects and construct validity. Scores for each factor were imputed for each participant and analyzed to examine hypothesized relationships between these latent factors, and their relationship with external measures. Reward sensitivity factor scores were regressed against impulsivity factor scores, alternative measures of impulsivity, ADHD measures, depression measures, and age. Using independent and multiple linear regression, we also examined the relationship between pubertal development, independently and conjointly with reward sensitivity, in the prediction of the same measures. A series of correlations were also conducted to assess the relationship between impulsivity factor scores, which was a dependent variable in the previous analyses, with external measures, including CRPR-R ADHD scores and scores from the CDI. Finally, we examined potential moderating effects of puberty on reward sensitivity and impulsivity. Consistent with the work of Baron and Kenny (1986), we first conducted a hierarchical linear regression with the two main effects at the first step, and the interaction term at the second step. Results indicating a significant change in R-squared and a significant effect of the interaction term over and above the main effects are consistent with a moderation effect.

Supplementary analyses. Follow-up analyses examined the relationship between all three latent factors and symptoms of depression, ADHD, ODD, and CD endorsed during the KSADs interview. We predicted similar relationships, with increased impulsivity and decreased reward sensitivity predicting increased ADHD, ODD, and CD. Likewise, we predicted that increased pubertal development and decreased reward sensitivity will also predict symptoms of depression.

Results

Preliminary Analyses

PCA of ERP Data. 95% of the final sample completed the ERP doors task. A temporal-spatial PCA analysis of FN data revealed a total of 23 temporal-spatial factors that accounted for at least 0.5% of the overall variance, and which accounted for, in sum, 76.8% of the total variance. Of these 23 factors, four temporal-spatial factor combinations were identified that differentiated win and loss conditions, were maximal at either Cz or FCz, and occurred between 200 and 400ms post stimulus onset. The two factor combinations that accounted for the greatest amount of variance of the four potential factors, PCA_FN_301 and PCA_FN_601 (accounting for 3.4% and 2.4% of the total variance, respectively), were further examined. PCA_FN_301 and PCA_FN_601 were maximal at 241 and 339ms post stimulus onset and located at Cz and FCz, respectively (see Figure 1).

fMRI Analysis. 53% of the total sample completed the fMRI doors paradigm. Second-level analyses compared reward vs fixation across the sample. An examination of within-group *t*-maps, thresholded at $p < .05$, *FWE* corrected, revealed a single large cluster of activation (voxel extent =1088) that spanned both the left and right VS (see Figure 2). Areas of maximal activation were found within both the left (MNI: -9, 19, 7; *Z*-score = 6.45) and right (MNI: 9, 16, 10; *Z*-score = 7.37) striatum. Data were extracted from both regions utilizing a 6mm sphere centered at the voxel of maximal activation (indicated above), for each participant. Importantly, an examination of the effect of loss vs fixation revealed no activity within this region.

Signal Detection Task. 96% of the total sample completed the Pizzagalli signal detection task; however, only 76% of the total sample provided valid data. A total of 30 participants' data were excluded according to thresholds set forward in previous publications utilizing this task (e.g., Pizzagalli et al., 2005), based upon the number of statistical outliers in response time (RT) and accuracy, which together produced unreliable data. Estimates of the participants' response

bias, which reflects the tendency to select the more richly rewarded of two ambiguous stimuli, was calculated for the 116 participants providing valid data (M = .17, SD = .13), and was consistent with previous studies with adults (Pizzagalli et al., 2005).

Progressive Ratio Task. 93% of the final sample completed the PR task. For each participant, an estimate of effort was calculated as follows:

PR_Effort = npress / RT * Time

Such that, npress = the number of presses during the game, RT = the average time between presses (in ms), and Time = the length of time the child engaged in the game. This estimate was strongly correlated with the amount of money earned during the task (r = .91, p < .001) and the age of the child (r = .20, p < .05), consistent with previous developmental studies of the PR task (Chelonis et al., 2011).

Puberty measures. PDS and PBIP scores were highly correlated across measure (r = .90, p < .001). In addition, scores were strongly correlated across informant (parent vs. child) on the PDS (r = .90, p < .001), PBIP pubic development (r = .77, p < .001), and PBIP breast development (r = .80, p < .001) measures. Given this, child and parent report were combined for both the PDS and PBIP, and measures of breast and pubic development on the PBIP were combined. The PDS and PBIP averages were in turn were highly correlated with both testosterone (r > .51, p < .001) and estrogen (r > .46, p < .001).

Self-report of impulsivity. All four measures of impulsivity showed relatively normal distributions with skewness ranging from 0.03 (UPPS) to .681 (PID-V Impulsivity), and kurtosis ranging from -0.02 (BAS Fun Seeking) to 1.16 (UPPS). The UPPS (M = 1.99, SD = 0.55), PID-V Impulsivity (M = 0.88, SD = 0.64), PID-V Risk Taking (M = 1.35, SD = .51), and BAS Fun Seeking (M = 12.21, SD = 2.39) all fell within expected values. The normative sample in the

current study reported moderate impulsivity according to the UPPS, below average impulsivity and risk-taking according to the PID-V, and slightly below average on the BAS Fun Seeking measure.

Self/parent report of psychopathology. Measures of psychopathology in the current sample were both skewed and showed high kurtosis. Self/parent report measures were moderately positively skewed (skewness > 1.50), with non-normatively kurtosis, greater than 2.68. Scores on both the CPRS-R ADHD index (M = 5.40, SD = 6.09) and CDI (M = 5.67, SD = 5.64) were within the normative range. Symptom counts on the KSADs were all extremely skewed, ranging from 2.52 to 7.59, with kurtosis values from 5.73 to 64.82. Thus, the measures of psychopathology in this sample were, as expected, non-normally distributed. This was particularly true of KSADs symptom counts, where over 80% of the sample reported no symptoms of ADHD, ODD, CD, or MDD. Thus, KSADs symptom counts were not included in regression analyses (below), although CDI and Conners' scores were, as they were more closely normative.

Exploratory Factor Analysis

Separate EFAs were conducted for each predicted latent construct: reward sensitivity, impulsivity, and pubertal development.

Reward sensitivity. Reward sensitivity EFA began with two PCA factors representing FN, estimates of fMRI activity for the left and right VS, PR effort, and response bias estimates from the signal detection task. Residuals were correlated for the two FN and fMRI measures separately. Initial EFA failed to achieve model fit (RMSEA > .05). PCA_FN_601 accounted for less than 4% of the variance in the latent factor and was subsequently dropped. The following model achieved model fit; however, response bias measures were not strongly predictive of the

final latent factor (accounting for approximately 4% of the variance), and so a subsequent model was created without this measure. This final model achieved good fit with four predictors: PCA_FN_301, left and right VS activity (fMRI), and PR effort – all accounting for more than 10% of the total variance in the latent factor (see Table 1, Figure 3).

Impulsivity. The initial impulsivity model included BAS Fun Seeking, UPPS total score, PID-V Impulsivity and PID-V Risk Taking. The initial model failed to achieve good model fit. BAS Fun Seeking was the least predictive variable and was dropped. The subsequent model achieved model fit, with strong factor loadings (see Table 1, Figure 4).

Pubertal development. A total of four predictors were included in the prediction of pubertal development: testosterone levels, estradiol levels, average rating of the parent and child on the PDS, and average ratings of both child and parent on the PBIP. Due to insufficient *dfs*, only one set of variables (testosterone/estradiol; vs. PBIP/PDS) could be modeled with correlated residuals. The best fitting model allowed correlated residuals between the PBIP and PDS scores, and showed moderate to strong factor loadings (see Table 1, Figure 5).

Confirmatory Factor Analysis

A CFA was conducted to assess model fit of the combined puberty development, impulsivity, and reward sensitivity factor models. Consistent with our hypotheses, pubertal development and reward sensitivity factors were permitted to covary, while impulsivity was in turn predicted by both reward sensitivity and pubertal development factors. The CFA model showed excellent fit (RMSEA = .000), with expected relationships between latent factors. In addition, the factor loadings of each predictor variable remained larger than .33 across individual factors (see Table 1; Figure 6).

Moderation and Construct Validity Analyses

Factor scores were imputed for the reward sensitivity, impulsivity, and pubertal development latent factors, and were subsequently compared with each other and with other measures. A series of linear regressions were conducted, with reward sensitivity and pubertal development regressed separately and together against impulsivity factor scores. As expected from the final CFA model, impulsivity factor scores were positively associated with pubertal development and negatively associated with reward sensitivity. Although there were no moderation effects, impulsivity was most strongly predicted be each factor when controlling for the other (see Table 2).

Identical analyses to those described above were also conducted to assess the independent and combined predictive effect of the reward sensitivity and pubertal development factors on alternative measures of impulsivity, as well as measures of both internalizing and externalizing psychopathology. Reward sensitivity and pubertal development factors were similarly associated with BAS Fun Seeking, both predicting greater BAS Fun Seeking, although only when controlling for one another. Likewise, reward sensitivity and pubertal development both predicted CDI scores, with pubertal development associated with higher scores on the CDI and reward sensitivity associated with lower. Reward sensitivity alone was predictive of ADHD symptoms, as expected. Finally, when regressed independently, both pubertal development and reward sensitivity predicted child age, but when regressed simultaneously only pubertal development remained a significant predictor, thus indicating that the variance accounted for in age by reward sensitivity was attributable to amplifying effects of pubertal development on reward sensitivity (see Table 2).

In addition, impulsivity factor scores were positively correlated with CPRS-R ADHD scores ($r = .24$, $p < .01$), BAS Fun Seeking ($r = .28$, $p < .001$), and CDI scores ($r = .28$, $p <$

.001). Although the latter association was unexpected, it is important to note that CPRS-R ADHD and CDI scores were correlated as well (r = .35, p < .001).

Supplementary Analyses

Factor scores were examined in relationship to symptoms endorsed on the KSADs. Given high positive skew for all clinical measures, Spearman's correlations were calculated. Impulsivity was positively correlated with past symptoms of depression (r_s = .20, p < .05), and equally with both past and current ADHD (r_s = .19, p < .05). Reward sensitivity was negatively associated with current MDD symptoms (r_s = -.19, p < .05), both past and current ADHD (r_s = -.28, p < .01), and past ODD r_s = -.18, p < .05). Finally, puberty was positively correlated with past MDD (r_s = .26, p < .01), and negatively correlated with both past and current ADHD (r_s = -.20, p < .05), as well as past ODD (r_s = -.17, p < .05). CD showed very little variance in this normative sample, and thus was expectedly unrelated to any measure.

Discussion

In the current study, we sought to characterize the relationship between biological and behavioral sensitivity to rewards and self-reported impulsivity in the context of pubertal development. Exploratory factor analyses (EFAs) were conducted to identify single factor solutions for reward sensitivity, pubertal development, and impulsivity. A confirmatory factor analysis (CFA) model was then constructed to test the primary hypothesis that both pubertal development and reward sensitivity predict impulsivity. The CFA model showed excellent fit, confirming the validity of this predictive model. Consistent with our hypotheses, reward sensitivity predicted less impulsivity. Likewise, as expected, pubertal development and reward sensitivity were positively correlated, and pubertal development predicted greater impulsivity (see Figure 6).

To test the hypothesis that pubertal development would moderate the relationship between reward sensitivity and impulsivity, we extracted factor scores for each latent factor and conducted a series of linear and multiple-linear regressions. However, contrary to our predictions, pubertal development did not moderate the relationship between reward sensitivity and impulsivity. Instead, we found that the covariance between the reward sensitivity and pubertal development factors resulted in decreased predicative utility of each. Thus, when controlling for pubertal development, the variance accounted for in impulsivity by reward sensitivity increased substantially (from 9% to 40%). This was also true of pubertal development's influence on impulsivity, which increased from 3% to 30%. Thus, although pubertal development did not alter the manner in which reward sensitivity predicted impulsivity, it is clear that puberty influences reward sensitivity, and that these effects may suppress the relationship between baseline reward sensitivity (i.e., reward sensitivity outside of the increasing influence of pubertal development) and impulsivity.

The link between attenuated reward sensitivity and impulsivity, although perhaps not a face-valid assumption, has substantial empirical and theoretical support. For example, Sagvolden and colleagues (2005) proposed a model of impulsivity in ADHD based on animal models indicating that reduced DA response to rewarding stimuli in the striatum is associated with impaired reward learning, which in turn predicts impulsive reward seeking behaviors. Likewise, among externalizing youth, theories suggest that attenuated reactivity to rewards results in increased reward seeking behavior to up-regulate an underactive reward system, and that these behaviors are characterized by impulsivity (Beauchaine & McNulty, *in press*). Our results are supportive of both models, although it is important to note that our sample was normative and thus findings may not extend to externalizing youth. Interestingly, the association between

reward sensitivity was most pronounced when controlling for pubertal development. In part this is not surprising given that pubertal development was positively associated with both reward sensitivity and impulsivity in the current model, while reward sensitivity was negatively associated with impulsivity. Previous studies have demonstrated that reward sensitivity peaks in middle adolescence, approximately at the upper limit of the study age range, and that reward sensitivity is best accounted for by pubertal development not age (Van Leijenhorst, Moor et al., 2010; Van Leijenhorst, Zanolie et al., 2010; Op de Macks et al., 2011; Blakemore et al., 2010). Thus, increasing reward sensitivity as a function of pubertal development may have masked the baseline or true relationship between reward sensitivity and impulsivity in this model.

Pubertal development was associated with both greater impulsivity and greater reward sensitivity, and although these findings are consistent with the literature linking puberty to impulsivity and increased reward sensitivity (Van Leijenhorst, Zanolie, et al., 2010; Forbes & Dahl, 2010), they are inconsistent with the general model proposed, which suggests that lower reward sensitivity predicts greater impulsivity. In part this inconsistency may reflect differential effects of pubertal development on neural activity. For example, in a recent study advanced pubertal development was associated with more prefrontal activation during reward outcome, but less striatal activation (Forbes et al., 2006). Although there were significant differences in sample between this study and the current study (including gender and age range), the results suggest that pubertal development may inversely affect mesolimbic and cortical activation to reward. However, to date very few studies have examined the functional correlates of pubertal development markers (for review, see Blakemore et al., 2010), and thus interpretation of our findings in the context of neural development is necessarily limited.

An alternative yet related possibility is that pubertal development is accounting for variance in impulsivity that is not related to reward sensitivity. For example, previous studies have shown that there is substantial change in neural development throughout puberty, particularly within the prefrontal cortex (Halperin & Schulz, 2006). A number of alternative of the biological basis of impulsivity suggest a deficit in executive control and inhibition, which are believed to largely be mediated by prefrontal regions (Logan, Schachar, & Tannock, 1997; Schachar & Logan, 1990; Congdon & Canli, 2005; Nigg, 2000). Thus, it is possible that pubertal development is exerting its effects on impulsivity through neural circuits that underlie inhibitory control, which may be largely distinct from the regions underlying reward processing. However, the lack of substantial evidence to support either claim implies the need for future research to better understand the complex relationship between pubertal influences on neural development and impulsivity.

Individual Factors

Reward Sensitivity. Reward sensitivity in the current study was comprised of left and right striatal reactivity to rewards (fMRI), a single temporal-spatial PCA factor representing FN, and a measure of effort from the PR task. Loadings were relatively consistent with the literature. For example, fMRI activity was positively associated with the reward sensitivity factor, which is consistent with previous studies showing that individuals who score higher on reward sensitivity and approach motivation measures have greater striatal reactivity to rewards (Simon et al., 2010). However, it is important to note that for both biological measures, activity was measured related to reward only, not in comparison to loss. We found that reward vs. fixation provided much superior predictive validity than did reward relative to loss. Importantly, an examination of the loss condition revealed no striatal activity in fMRI, and ERP response to loss was did not well

predict reward sensitivity, producing unacceptable model fit. Thus, the results of the current study suggest that the reward vs. fixation contrast was not spurious (e.g., reflecting general task vs fixation, etc.). It is, however, unclear why reward *minus* loss conditions provided worse model fit. Additionally, reward vs. fixation produced similarly large areas of activation in other regions including the medial prefrontal cortex (MPFC). However, these regions were not tested in the current study. The selection of only VS regional activation to rewards was based on previous research indicating the importance of this region, but also on the specificity of this region to rewards. For example, while activation in the MPFC and other regions was present for both loss and reward conditions relative to fixation, VS activity was only found for reward vs. fixation.

Results were similar for the single behavioral measure contributing to factor identification. Level of effort was calculated for the PR task, consistent with previous animal studies which have examined rate of response and length of time until discontinuation. PR effort loaded positively on reward sensitivity, indicating that those children who engaged in the task longer had greater sensitivity to rewards. This is consistent with previous animal studies, which indicate that DA antagonists and other alterations that reduce DA reward response in the striatum are associated with reduced effort on PR tasks (Aberman et al., 1998; Zhang et al., 2003). In addition, although there are few studies of PR tasks in human adolescents (Chelonis et al., 2011), our findings were consistent: PR effort was also positively associated with pubertal development (via reward sensitivity) and age

In the final model, gain related ERP response from a single temporal-spatial factor was utilized. Although gain related activity was examined for two temporal-spatial factor combinations, only an early breaking spatial-temporal factor provided adequate factor loadings to be included in the final model. The relationship between the temporal-spatial factor and

reward sensitivity was consistent with previous work, suggesting that greater reward sensitivity is associated with greater FN (Bress & Hajcak, 2013). However, the spatial and temporal characteristics of the factor utilized were not ideal. FN typically peaks around 300ms post stimulus onset and is most often maximal at FCz spatial location (Hajcak et al., 2006). However, in the current study, the temporal-spatial factor combination utilized in the final factor analysis showed an earlier latency (241ms) and a more posterior maximal channel (Cz), although both FCz and Cz showed similar signal patterns (see Figure 1). In addition, this factor combination had a relatively small difference between reward and loss conditions. However, the PCA temporal-spatial factor combination did account for the greater proportion of variance in ERP signal than any other combination that differentiated gain and loss. Thus, although the relationship between this temporal-spatial factor combination and reward sensitivity is consistent with the literature, results should be interpreted with caution.

Finally, although we hypothesized the response bias from the SDT would predict increased reward sensitivity, this measure failed to adequately load on the reward sensitivity factor and was not included in the final model. In addition, factor loadings were in the opposite of the expected direction. Less response bias was associated with higher reward sensitivity factor scores, although the reward sensitivity factor only accounted for a very small (< 5%) and perhaps non-significant proportion of the variance in this measure. This finding is inconsistent with previous research, which indicates that populations associated with reduced reward sensitivity (e.g., depressed or anhedonic individuals) show reduced response bias (Pizzagalli et al., 2005). Likewise, while there is a scarcity of studies using the SDT in children, similar studies have found that depressed adolescents are less likely to augment their response to select highly rewarded stimuli (Forbes, Dahl, & Shaw, 2007). While our findings do not comport with existant

studies, it is important to note that the current study used a much abbreviated version of the original task, with approximately half the total trials. In addition, approximately one quarter of the sample were excluded due to excessive outliers and inconsistent responding. Thus, it is unclear to what extent these null findings reflect true differences between our hypotheses and outcomes vs. method or data collection problems.

Pubertal Development. We utilized four predictors of pubertal development, including two hormone measures and two scales that assess the physical changes associated with pubertal development. Testosterone has been consistently linked to pubertal changes in reward sensitivity in both male and female adolescents (Forbes et al., 2006; Op de Macks et al., 2011). Thus, it was not surprising that testosterone was most strongly associated with the latent pubertal development factor (although this relationship was somewhat attenuated when allowing correlated residuals among estradiol and testosterone). However, estradiol and child/parent report of physical development also loaded strongly on pubertal development. Previous studies have indicated that estradiol may play an important role in the neural restructuring that occurs during adolescence, and that these effects may be gender specific (Peper, Hulshoff Pol, Crone, & van Honk, 2011). In addition, while little data exists on the relationship between pubertal changes in reward sensitivity and estradiol, there is some evidence to suggest that estradiol influences reward processing. For example, reward processing has been shown to vary in concordance with estradiol fluctuations associated with menstrual phase cycle (Dreher et al., 2007).

However, it has been argued that hormone measures may not relate particularly well with physical development given the potential confounds of monthly and circadian cycles, as well as method effects (Blakemore et al., 2010). Despite this, the current study found relative consistency in these measures. The PBIP and PDS more strongly correlated with testosterone

than estradiol, however all correlations were greater than 0.45. Although these associations are not as strong as the within measure associations (hormones: $r = .73$; physical development: $r = .92$), hormone levels did account for approximately 25% of the variance in physical development. Likewise, factor loadings suggest that the omnibus puberty factor accounts for approximately 25% of the variance in physical development and between 52% (estradiol) and 72% (testosterone) of the variance in hormone measures, which suggest that the pubertal development factor in the current study was fairly representative of both hormonal and physical changes.

Impulsivity. The impulsivity factor was strongly associated with all individual measures of impulsivity. All three predictors (UPPS, PID-V Impulsivity, and PID-V Risk Taking) had factor loadings ranging from 0.68 to 0.75. Previous studies have shown that PID-V Impulsivity and Risk Taking subscales load together with measures of irresponsibility and lack of constraint (Anderson et al., 2013); however, there is relatively little data on the discriminant validity of these scales. In contrast, the UPPS has been shown to adequately differentiate healthy controls from impulsive samples such as alcoholic and borderline individuals (Whiteside, Lynam, Miller, & Reynolds, 2005). Likewise, children with ADHD have also score higher on the UPPS subscales (Miller et al., 2010). While the latent factor model appeared to have well characterized impulsivity, it is important to note that the impulsivity factor was just identified, and therefore we were unable to test model fit. Another potential issue relates to the measures utilized in the current study, which were nearly all designed for adults. Only one measure, the UPPS, was specifically designed for children, and even then only for children 12+. Therefore, it remains a possibility that the association between puberty and impulsivity may in part reflect more accurate scores among older children, who were better able to understand measure items.

Factor Score Validity

Although the factor structure extracted was consistent with our hypotheses, a contrast of these factor scores against measures not included in the CFA is necessary to demonstrate construct validity. These measures included an impulsivity scale (e.g., BAS Fun Seeking), in addition to measures of psychopathology that are believed to be linked to increased impulsivity and reduced reward sensitivity (e.g., ADHD), as well as those linked to reward sensitivity and pubertal development, but not impulsivity (e.g., depression).

Depression. As expected, measures of depression were associated with reduced reward reactivity and greater pubertal development. Thus, children with lower reward reactivity had higher self-reported symptoms of depression, as did those who had greater pubertal development, although only when controlling for each other. These results are consistent with previous studies indicating reduced reward activity among depressed adolescents, as well as studies indicating an increased risk for depression as a function of pubertal development (Angold et al., 1998; Forbes & Dahl, 2012). However, contrary to expectations, impulsivity was also associated depression. Although these findings may in part reflect correlations among measures of internalizing and externalizing psychopathology, which were generally positive and significant, it is important to note that comorbidity between ADHD and MDD is common (Biederman et al., 2008; Jensen et al., 1997). Thus, the overlap between impulsivity and depression may reflect diagnostic overlap among children with ADHD. Unfortunately, despite the fact that comorbidity is common, little research has examined potential common neural substrates of ADHD and MDD. Both disorders are associated with attenuated reward sensitivity, both in the current study and in previous studies. Thus, future studies may wish to examine the extent to which attenuated reward

sensitivity can account for the overlap between impulsivity, externalizing, and internalizing psychopathology.

Impulsivity and ADHD. Reward sensitivity and pubertal development showed expected relationships with measures of externalizing pathology, in this case ADHD, and with other measures of impulsivity. Reward sensitivity was negatively correlated symptoms of ADHD, although this relationship was much stronger when controlling for pubertal development. This is consistent with numerous studies indicating that ADHD is associated with reduced FN (van Meel et al. 2011; Ibanez et al., 2012) and decreased striatal activity to rewards (Plitcha & Scheres, 2013). As expected, no effect of pubertal development on ADHD was found, even when controlling for reward sensitivity. Thus, consistent with research suggesting that ADHD begins early in childhood and persists through adolescence (Riddle et al., 2013; Willoughby, 2003), changes in pubertal development did not influence symptoms of ADHD, despite being associated with changes in reward sensitivity. This finding is particularly important given the fact that both the impulsivity factor scores and other measures of impulsivity (e.g., BAS Fun Seeking) were positively associated with pubertal development (especially when controlling for reward sensitivity). Thus, findings suggest that ADHD may be more strongly linked to reward sensitivity itself, than trait impulsivity, although the latter did show a moderate positive relationship with ADHD as well.

Strengths/Limitations and Conclusions

Factor analytic findings in the current study are largely consistent with our hypotheses and with previous research. However, it is important to note that there were a number of potential confounds in the current study. Of particular concern is the amount of missing data. Almost half of the sample did not complete fMRI procedures. In part, this is due to changes in

imaging protocol that lead to the exclusion of fMRI data from nearly over 30 study participants. However, previous simulations have demonstrated that the original factor structure and factor weights are largely retained with up to 50% missing data (Kamakura & Wedel, 2000). To ensure that missing data from the fMRI measures did not unfairly bias our analysis, we also examined the same factor structure excluding those subjects who completed a non-compatible version of the fMRI task. Factor weights and structure were largely similar, although fit was slightly attenuated. Thus, despite a number of missing data points, the factor model appears to be valid. In addition, a number of the individual factors within the CFA may not be adequately identified. For example, although the reward sensitivity factor had adequate fit indices, factor loadings were relatively low, ranging from 0.36 – 0.55. Thus, it may be that a single factor solution for reward sensitivity is not accurate. Alternatively, although impulsivity showed strong factor loadings, the model was just identified and therefore it was impossible to assess fit indices.

The potential concerns raised above can typically be addressed through EFA via parallel analysis. Typically, a parallel analysis suggests the extraction of factors up until the point that additional factors are no longer accounting for more variance than could be expected by chance (Hayton, Allen, & Scarpello, 2004). Thus, the factor structure retained is the most parsimonious, which also accounts for substantially more variance in the predictors than could expected by chance. However, we were unable to use this measure in the current study largely to method covariance. The inclusion of multiple measures from the same construct (e.g., left and right striatal activity to rewards) resulted in a factor structure that appeared to represent method effects only (e.g., in the case of reward sensitivity, a factor for fMRI, a factor for FN, and a factor for behavioral data). Thus, exploratory analyses were conducted in AMOS, which allowed us to address method effects. Alternatively, the factor structure could also be confirmed through a

split-half analysis. However, the relatively small sample size in the current study also precluded this option. Therefore, research examining the application of this model to alternative normative and non-normative samples is necessary to verify our results.

However, despite these limitations, the current study provides a number of important findings. First and foremost, the results of the current study provide substantial evidence confirming the purported relationship between puberty, reward sensitivity, and impulsivity. Although previous studies have illustrated similar effects with individual behavioral or biological measures, the current study is the first to examine multiple markers of each construct. Our results extend upon this research, and further validate the reward insensitivity model of impulsivity. In addition, the results of the study suggest a relatively strong inverse relationship between puberty and reward sensitivity in the prediction of impulsivity. Thus, our findings suggest that the relationship between reward sensitivity and impulsivity is strongly influenced by the pubertal development of the individual, and that it is important to control for pubertal markers when examining these relationships. Finally, although our study was based on a normative sample, the results of the current study provide further evidence linking trait impulsivity and attenuated reward sensitivity to increased externalizing psychopathology. Girls with higher greater reported symptoms of ADHD showed less reactivity to rewards.

Although the results of the current study are compelling, future study is needed to confirm these findings. The current sample was relatively small, and had a number of missing data points. In addition, although our model appeared to have relatively good construct and convergent validity, the current sample had relatively limited range in measures of internalizing and externalizing psychopathology. Thus, both larger and more representative samples are needed to test this model. However, despite these limitations, the current study strongly supports

the existent theoretical models and empirical data linking attenuated reward sensitivity to increased impulsivity, and illustrates the importance of assessing the influence of pubertal development on these relationships.

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Table 1. Fit indices for the Pubertal Development, Impulsivity, and Reward Sensitivity exploratory factor analyses, and the integrative confirmatory factor analysis.

Model	χ^2	*df*	CFI	TLI	RMSEA	90% CI on RMSEA
Pubertal Development[a]	1.79	2	1.00	1.00	0.00	0.00-0.16
Impulsivity[b]	--	--	--		--	--
Reward Sensitivity	0.09	2	1.00	1.00	0.00	0.00-0.15
Integrative CFA	**34.55**	**38**	**1.00**	**1.00**	**0.00**	**0.00-0.05**

[a] Puberty model varied slightly from that in the integrative model. Due to modeling constraints, covariance between the estradiol and testosterone measures could not be modelled.

[b] Impulsivity model was just identified, not allowing for estimation of fit indices.

Table 2. Multiple regression models, fit indices, and regression coefficients for Reward Sensitivity and Pubertal Development, regressed separately and conjointly.

Predicted Variable	Model 1			Model 2			Model 3				
	R^2	Reward		R^2	Puberty		R^2	Reward		Puberty	
		B (SE)	β		*B* (SE)	β		*B* (SE)	β	*B* (SE)	β
Impulsivity (Factor)	.09	-.008 (.002)	-.30***	.03	.186 (.084)	.18*	.29	-.018 (.002)	-.63***	.579 (.089)	.55***
BAS Fun Seeking	.01	-.014 (.013)	-.09	.01	.437 (.472)	.08	.03	-.033 (.016)	-.21*	1.16 (.759)	.20*
CPRS-R ADHD	.04	-.080 (.032)	-.20*	.00	-.741 (1.20)	-.05	.05	-.105 (.039)	-.27**	1.60 (1.46)	.11
CDI	.01	-.041 (.030)	-.11	.01	1.65 (1.11)	.12	.07	-.103 (.036)	-.28**	3.94 (1.34)	.29**
Age	.17	.048 (.009)	.42***	.49	3.01 (.254)	.70***	.48	.000 (.008)	.00	3.00 (.317)	.69***

Notes. CDI = Child Depression Inventory (Kovacs, 1985); BAS = Behavioral Activation Scale (White & Carver, 1994); CPRS-R ADHD = Conners' Parent Rating Scales, Revised, Attention Deficit Hyperactivity Disorder subscale (Conners et al., 1998); Age is defined as number of days between birth and completion of study protocols, converted to years. n = 153 for all models with the exception of BAS Fun Seeking (n = 152).

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

Figure 1. Principal Components Analysis of Feedback Negativity factor 301 (A) and 601 (B), waveforms and headmaps. Loss (red), Win (Blue), and Black (Difference).

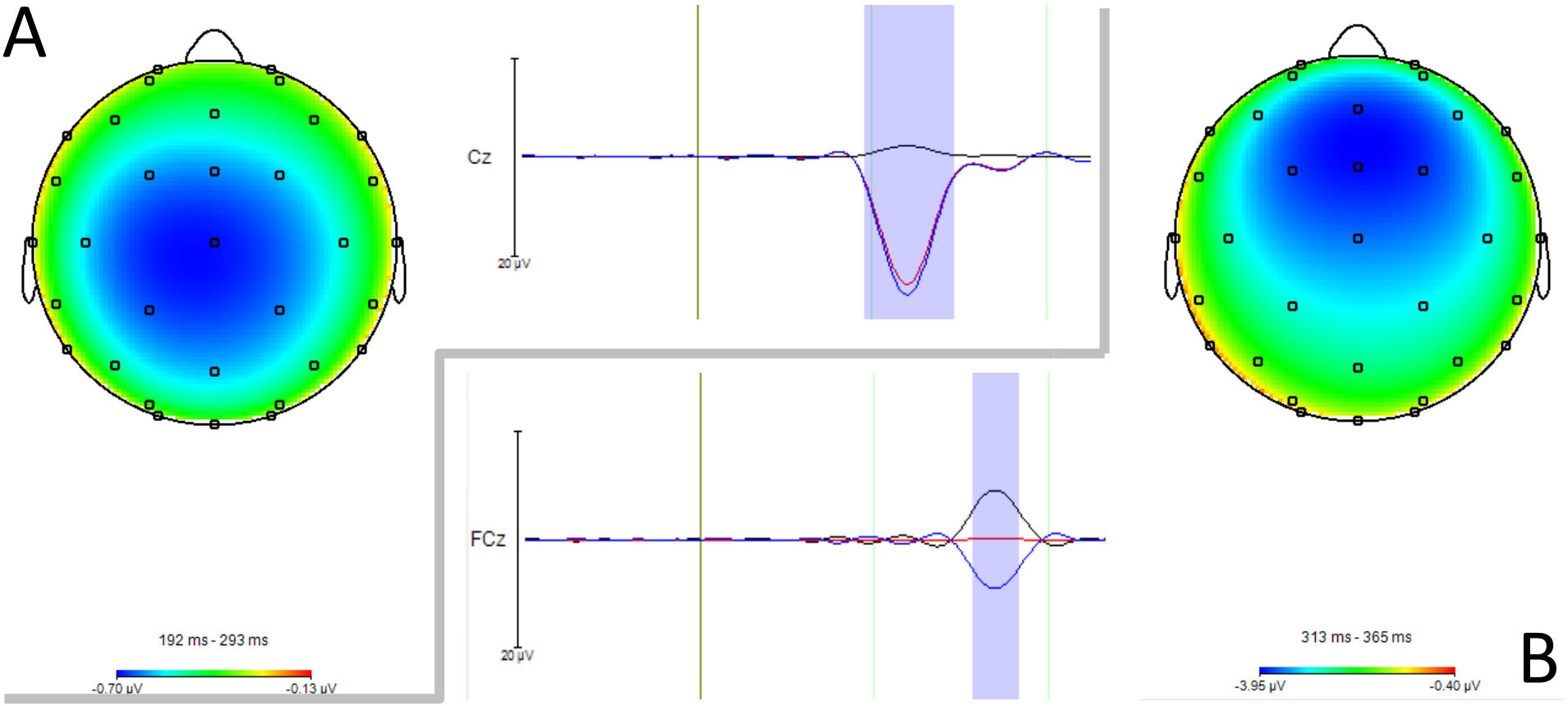


Figure 2. Functional magnetic resonance imaging activation for Win vs Fixation, with ventral striatal activity circled in red.

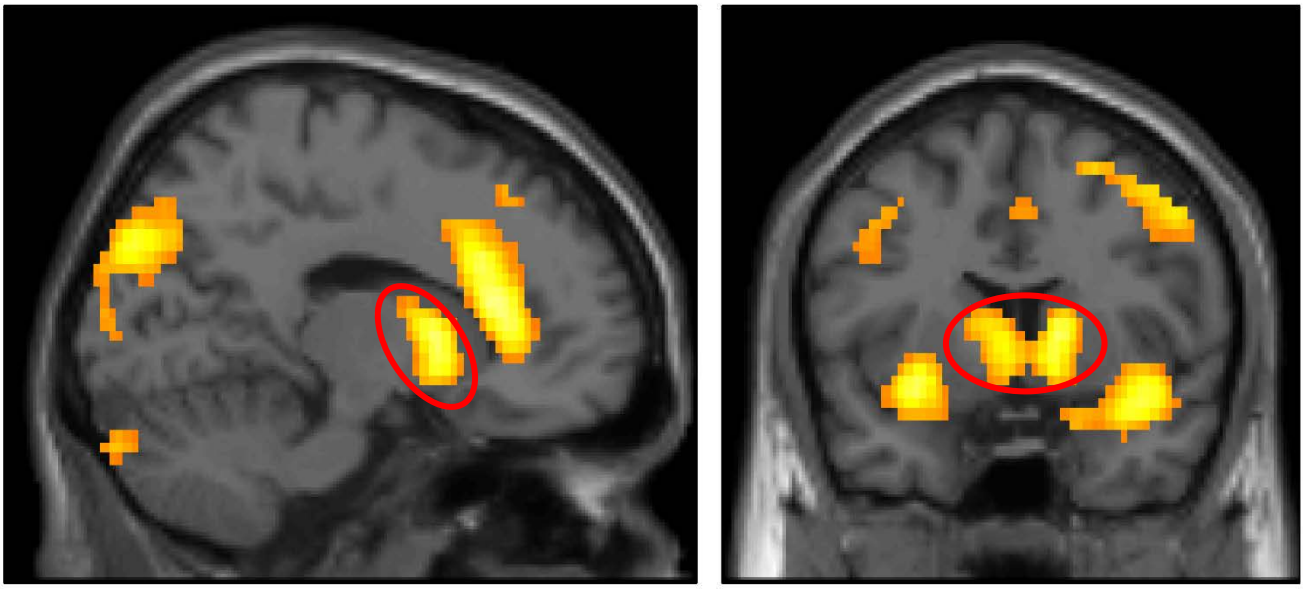
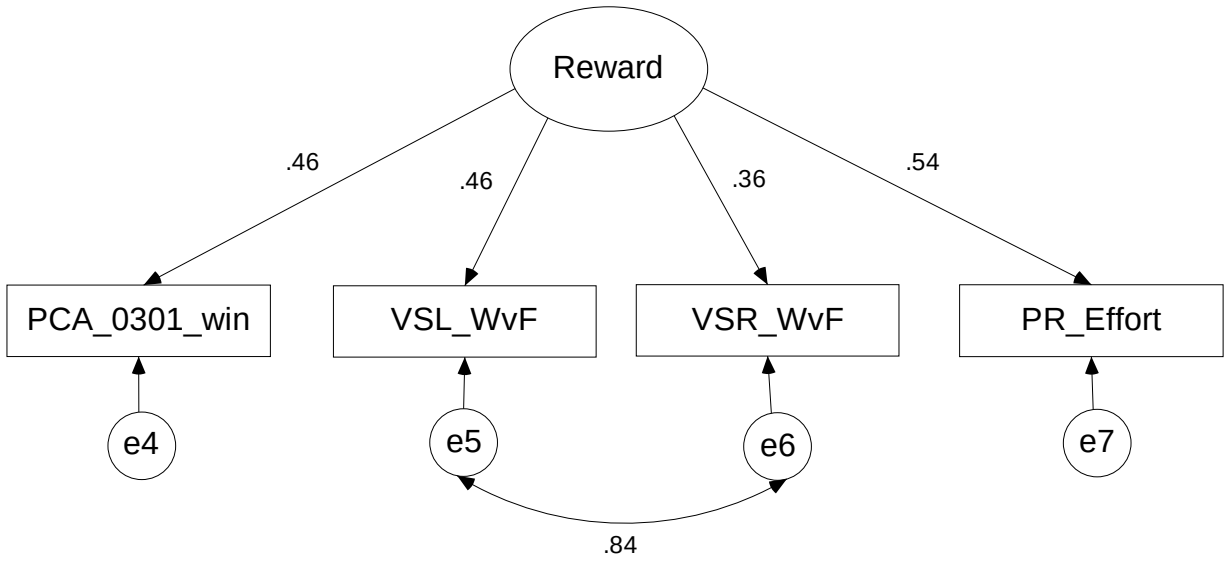
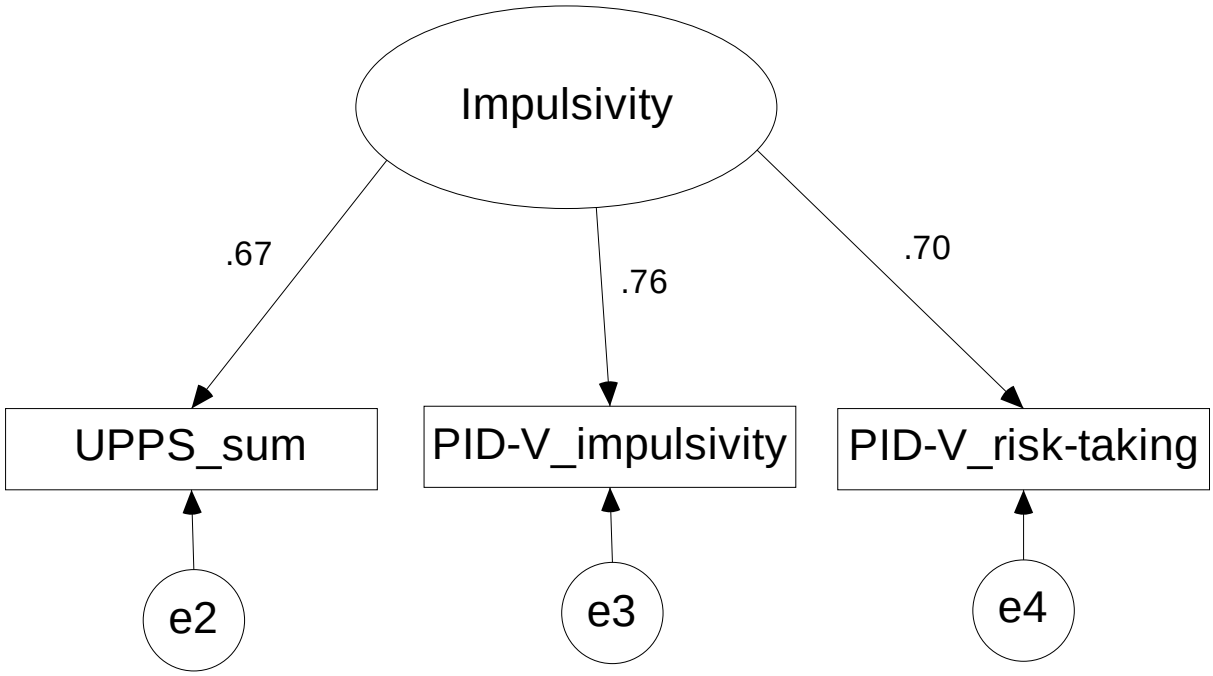


Figure 3. Final exploratory factor analysis model for Reward Sensitivity.



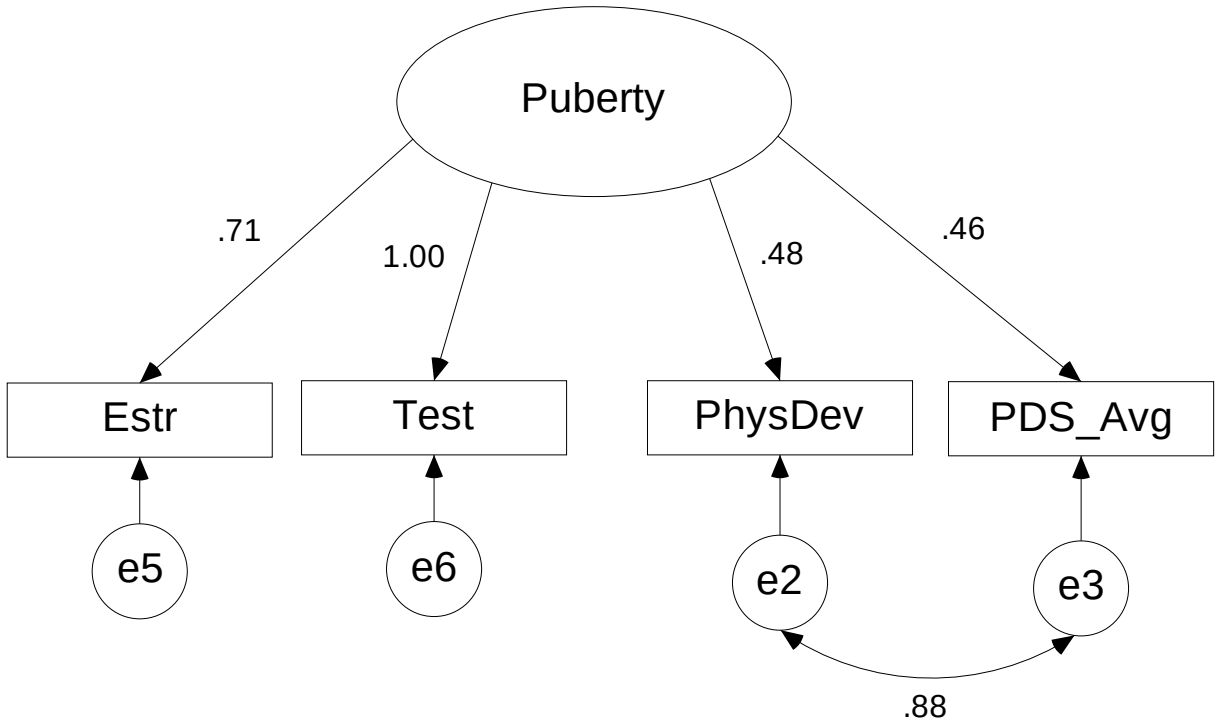
Notes. PCA_0301_win = Principal components analysis of feedback negativity factor 301, reward activity; VSL_WvF = Left ventral striatal activity for win vs fixation; VSR_WvF = Right ventral striatal activity for win vs fixation; PR_Effort = Effort estimate from the progressive ratio task (Chelonis et al., 2011).

Figure 4. Final exploratory factor analysis model for Impulsivity.



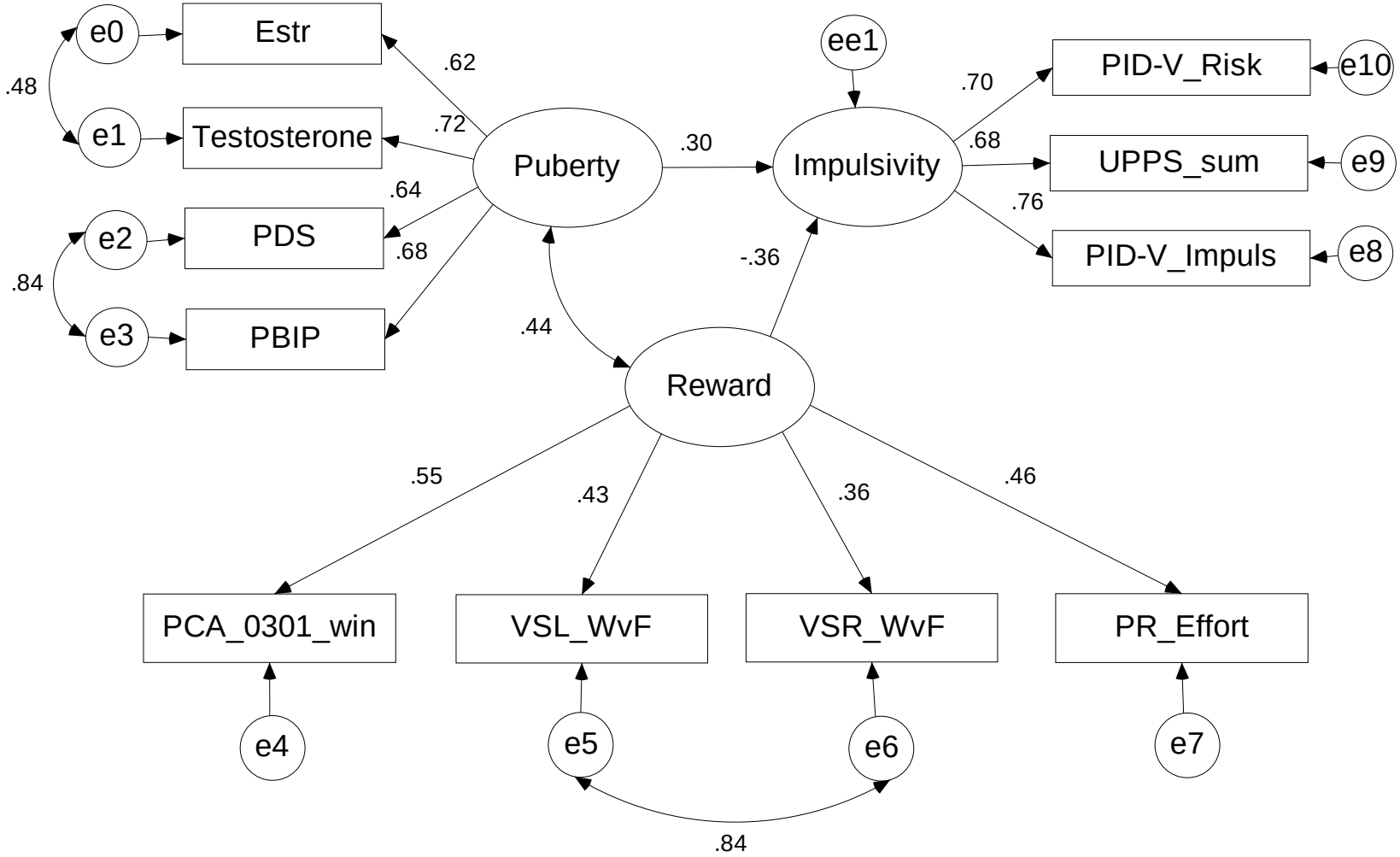
Notes. UPPS_sum = Total subscale scores of the *UPPS Impulsive Behavior Scale* (Whiteside & Lynam, 2001); PID-V_impulsivity = Impulsivity subscale of the Personality Inventory for DSM-V; PID-V_risk-taking = Risk taking subscale of the Personality Inventory for DSM-V (Krueger et al., 2012) \

Figure 5. Final exploratory factor analysis model of Pubertal Development.



*Note*s. Estr = salivary hormone estimates for estradiol. Test = salivary hormone estimates for testosterone. PhsyDev = average of child and parent ratings of pubertal and breast development on the Picture Based Inventory about Puberty (PBIP; Shirtcliff et al., 2009). PDS_Avg = average of child and parent scores on the Pubertal Development Scale (PDS; Peterson, Crockett, & Richards, 1988).

Figure 6. Final confirmatory factor analysis model, including Pubertal Development, Reward Sensitivity, and their relationship with Impulsivity.



Notes. Estr = salivary hormone estimates for estradiol. Test = salivary hormone estimates for testosterone. PhsyDev = average of child and parent ratings of pubertal and breast development on the Picture Based Inventory about Puberty (PBIP; Shirtcliff et al., 2009). PDS_Avg = average of child and parent scores on the Pubertal Development Scale (PDS; Peterson, Crockett, & Richards, 1988). PCA_0301_win = Principal components analysis of feedback negativity factor 301, reward activity; VSL_WvF = Left ventral striatal activity for win vs fixation; VSR_WvF = Right ventral striatal activity for win vs fixation; PR_Effort = Effort estimate from the progressive ratio task (Chelonis et al., 2011).