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Developmental Differences in Reward Sensitivity and Sensation Seeking in Adolescence: Testing Sex-Specific Associations With Gonadal Hormones and Pubertal Development

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Sensation seeking has been found to increase, on average, from childhood to adolescence. Developmental scientists have hypothesized that this change could be driven by the rise of gonadal hormones at puberty, which affect reward-related processing in the brain. In a large, age-heterogeneous, population-based sample of adolescents and young adults (N = 810; ages 13–20 years), we tested for sex-specific associations between age, self-reported pubertal development, gonadal hormones (estradiol and testosterone) as measured in saliva, reward sensitivity as measured by a multivariate battery of in-laboratory tasks (including the Iowa gambling task, balloon analogue risk task, and stoplight task), and self-reported sensation seeking. Reward sensitivity was more strongly associated with sensation seeking in males than females. For both males and females, reward sensitivity was unrelated to age but was higher among those who reported more advanced pubertal development. There were significant sex differences in the effects of self-reported pubertal development on sensation seeking, with a positive association evident in males but a negative association in females. Moreover, gonadal hormones also showed diverging associations with sensation seeking—positive with testosterone but negative with estradiol. Overall, the results indicate that sensation seeking among adolescents and young adults depends on a complex constellation of developmental influences that operate via sex-specific mechanisms.

Keywords: reward, sensation seeking, adolescence, testosterone, estradiol

Adolescence is a time of elevated risk-taking. The teenage years are the peak developmental period for crime, injury to self and others (both accidental and deliberate), and the initiation of alcohol and drug use. *Sensation seeking* is a personality trait that increases, on average, from childhood to adolescence, and this developmental increase contributes to the emergence and escalation of risk-taking behavior in adolescence (Harden & Mann, 2015; Harden, Quinn, & Tucker-Drob, 2012; Harden & Tucker-Drob, 2011). One hypothesis that has been put forth in the developmental literature is that the rise in sensation seeking during adolescence is driven by rises in gonadal hormones during puberty through their effects on reward sensitivity (e.g., Peper & Dahl, 2013; Shulman, Harden,

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Data from this study were previously reported in Harden et al. (2017), which described (a) how the measures analyzed here converged with other self-report and behavioral measures relevant for adolescent risk-taking and (b) results from a behavioral genetic composition of variance in these measures. Data on self-reported sensation seeking and its relationship to antisocial behavior in these participants were also used in Mann, Engelhardt, et al. (2017); Mann, Kretsch, Tackett, Harden, and Tucker-Drob (2015); Mann et al. (2016); and Mann, Paul, Tackett, Tucker-Drob, and Harden (2017). Finally, a behavioral genetic decomposition of variation in salivary testosterone was reported in Harden, Kretsch, Tackett, and Tucker-Drob (2014).

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Chein, & Steinberg, 2015; Smith, Chein, & Steinberg, 2013). However, as we review in detail later, previous tests of this model have been limited by small sample sizes and by inconsistent measurement of key constructs. The current study addresses this gap in the literature by examining sex-specific associations between age, puberty, gonadal hormones, reward sensitivity, and sensation seeking, using a large population-based sample of U.S. teenagers who have been measured on a multivariate battery of self-report and behavioral measures.

To situate the current study within the extant empirical and theoretical literature, we first describe previous research related to three questions: (a) What is the relation between reward sensitivity and sensation seeking, and does this relation differ by sex? (b) How do reward sensitivity and sensation seeking change with age and pubertal development? and (c) Are developmental differences in reward sensitivity and sensation seeking due to increases in circulating levels of gonadal hormones, specifically testosterone and estradiol? For each question, we highlight what issues remain unresolved by previous research.

What Is the Relation Between Reward Sensitivity and Sensation Seeking?

Sensation seeking was defined by Zuckerman (1994), the originator of the construct, as a preference for "varied, novel, complex, and intense sensations and experiences, and the willingness to take physical, social, legal, and financial risks for the sake of such experiences" (p. 27). In other words, the construct of sensation seeking is centered on whether a person likes and wants certain types of experiences (e.g., physically arousing, intense, dangerous), rather than finding them aversive. In contrast, the construct of *reward sensitivity* centers on how much a person likes and wants rewards generally. These concepts are clearly dissociable, because one can respond strongly to rewards that are not thrilling in any way: A person settling into the couch to watch Netflix with a pint of ice cream is motivated by the rewarding properties of sugar and internet consumption but is not showing any evidence of sensation seeking. Yet sensation seeking and reward sensitivity are also clearly related: A person skiing quickly down a treacherous mountain slope is risking a broken leg (or worse) because she finds the experience rewarding. In the following sections, we consider the nature of the relation between sensation seeking and reward sensitivity, evidence for their association, and possible sex differences in that association.

Conceptual Model

The relation between reward sensitivity and sensation seeking is most commonly conceptualized as a hierarchical model: Reward sensitivity is seen as a more physiologically and phylogenetically basic dimension of individual differences that contributes to—but is not the sole determinant of—the higher order personality trait of sensation seeking. A corollary of this hierarchical model is that reward sensitivity and sensation seeking are often, but not always, measured at different levels of analysis. Reward sensitivity is often measured at a physiological level, for example, in terms of neural functioning (e.g., firing rate of dopaminergic neurons) or responses to a drug with known physiological effects on dopamine systems, whereas sensation seeking is often measured using self-report. For example, Chein, Steinberg, and colleagues (Shulman et al., 2016, p. 105; Smith et al., 2013) described reward sensitivity as a "neurobiological phenomen[on]" that is "measured in studies of brain structure or function," whereas sensation seeking is a "psychological manifestation" of reward sensitivity that is measured by "assessing psychological states or traits through the subjective reports of individuals." It is interesting, however, that even many of the studies that measured reward sensitivity using behavioral tasks (such as was the case for the current study) still conceptualized their relation hierarchically (e.g., Castellanos-Ryan, Rubia, & Conrod, 2011). That is, even when reward sensitivity and sensation seeking are measured at the same level of analysis (behavior), the former is presumed to partially underlie the latter.

Sensation Seeking and Dopamine

Mechanistic evidence concerning the relation between reward sensitivity and sensation seeking comes from research on linking sensation seeking to reward-relevant dopaminergic systems (DeYoung, 2013; Norbury & Husain, 2015). Dopamine is a neurotransmitter that is critical for the incentive salience of rewards (i.e., wanting rewards) and is also indirectly involved, via its interaction with the opioid system, in the liking of rewards (Berridge, Robinson, & Aldridge, 2009). The mesolimbic dopamine pathway is activated by rewards such as food, sex, love, and drugs (Berridge & Robinson, 1998).

Research using animal models of sensation seeking has found differences between high- and low-sensation-seeking animals in reward-relevant dopaminergic processes (reviewed in Norbury & Husain, 2015). For instance, rats with a high locomotor reactivity to novelty have shown higher levels of dopamine in the ventral striatum and more frequent firing of dopamine-releasing neurons in the midbrain than have rats with low response to novelty (Blanchard, Mendelsohn, & Stamp, 2009; Flagel et al., 2010). Human studies have reported that self-reported sensation and novelty seeking are associated with the availability of dopamine D2/D3 receptors in the striatum and midbrain (Gjedde, Kumakura, Cumming, Linnet, & Møller, 2010; Zald et al., 2008). Other research has found that adult participants who were administered cabergoline, a dopamine agonist, were more sensitive to information about potential rewards in a gambling task, but this effect was more pronounced among participants who were low in self-reported sensation seeking (Norbury, Manohar, Rogers, & Husain, 2013). These results were interpreted in light of Gjedde et al.'s (2010) results as suggesting that low sensation-seekers had lower endogenous dopamine levels and could thus experience greater dopamine gain from cabergoline.

Additional evidence for the relation between reward sensitivity and sensation seeking in humans has come from research on individual differences in responses to alcohol and drugs (Blanchard et al., 2009). Individuals high in self-reported sensation seeking have shown greater positive responses to amphetamine (Hutchison, Wood, & Swift, 1999; Kelly et al., 2006; Stoops et al., 2007) and have had a more positive subjective response to alcohol intoxication (Fillmore, Ostling, Martin, & Kelly, 2009). Overall, research in humans and animals has consistently found that sensation seeking is associated with differences in reward-relevant dopamine systems, in support of a conceptual model that positions reward sensitivity as a contributor to sensation seeking.

Sensation Seeking and Novelty as Reward

In addition to having heightened sensitivity to the rewarding properties of drugs or "natural" reinforcers like food or sex, high sensation-seekers may preferentially experience novelty or uncertainty as rewarding in and of itself (DeYoung, 2013). Consistent with this hypothesis, Olsen and Winder (2009), using an operant conditioning paradigm, found that mice will acquire lever-pressing behavior only if reinforced by varied (i.e., novel) visual stimuli, a learning process that is disrupted by high doses of the dopamine antagonist *cis*-flupenthixol and that is eliminated by deletion of the dopamine D1 receptor gene. Norbury, Kurth-Nelson, Winston, Roiser, and Husain (2015) adapted this operant conditioning paradigm for humans. Participants had the option to choose between two visual images, one of which was associated with "stimulating but not painful" electric shock of the hand and one that also differed in reward values (specifically, points that were exchangeable for a cash bonus at the end of the task; Norbury et al., 2015, p. 3). It is interesting that some participants consistently chose the visual image associated with tactile stimulation, even when that image had a lower reward value, and the preference for receiving tactile stimulation was correlated with self-reported sensation seeking. This line of research suggests, then, that sensation seeking is associated with experiencing certain types of stimuli as rewarding.

Sensation-Seeking and Reward-Seeking Behavior

Yet another line of research has measured reward sensitivity using various behavioral tasks that measure an individual's responses under experimental conditions that vary the likelihood or magnitude of obtaining a reward (often, but not always, money). Performance on these behavioral tasks has consistently been found to be positively but modestly associated with self-reported sensation seeking in adolescent samples (Castellanos-Ryan et al., 2011). One study by Icenogle et al. (2017) is notable for its size: In over 3,000 adolescents, sensation seeking was positively correlated with reward sensitivity, as measured by learning to play from advantageous decks (i.e., decks of cards for which repeated play was expected to result in increasing monetary rewards) on the Iowa gambling task (IGT).

Contribution of Fear(lessness) to Sensation Seeking

Although sensation seeking is most commonly conceptualized in terms of appetitive motivation (gaining rewards), it might also be related to individual differences in avoidance motivation (avoiding punishments; Lissek et al., 2005). It is difficult for one to find a dangerous activity fun if it feels terrifying or if it is perceived as so frightening that it is avoided altogether. Consistent with this idea, previous studies of sensation seeking have found that high sensation-seekers report lower levels of trait anxiety and report feeling less fear (Franken, Gibson, & Rowland, 1992), show attenuated skin conductance and startle responses to aversive stimuli (Lissek et al., 2005; Lissek & Powers, 2003), show less selective memory for negative events (Toffalini, Bellavitis, & Cornoldi, 2015), and are less likely to develop posttraumatic stress disorder (PTSD) following exposure to trauma (Neria, Solomon, Ginzburg, & Dekel, 2000; Solomon, Ginzburg, Neria, & Ohry, 1995). Furthermore, neuroimaging research on reactivity to threat stimuli has found that young adolescents' self-reported sensation seeking and their self-reported anxiety problems were *both* predicted (in opposing directions) by an interaction between changes in the amygdala (which is critical for fear learning) and changes in the nucleus accumbens (which is critical for reward response; Spielberg, Olino, Forbes, & Dahl, 2014).

Possible Sex Differences

The role of fear–avoidance in shaping individual differences in sensation seeking also suggests possible sex differences in the relation between sensation seeking and reward sensitivity. Across the life span, males have reported higher sensation seeking than have females (see Cross, Cyrenne, & Brown, 2013, for meta-analysis), a sex difference that may underlie sex differences in vulnerability to externalizing psychopathology (e.g., substance use disorders), unintentional injuries (e.g., motor vehicle accidents), and behavioral risk-taking generally (J. P. Byrnes, Miller, & Schafer, 1999; Grunbaum et al., 2002; Johnston, O'Malley, Bachman, & Schulenberg, 2011; Martel, 2013). At the same time, females are more likely than males to experience disorders of fear and anxiety, including PTSD, a disorder for which high sensation seeking is protective (Neria et al., 2000; Solomon et al., 1995; Tolin & Foa, 2006). One hypothesis is that reward sensitivity in women is not as readily translated into a personality disposition of sensation seeking, because they are more likely to also experience strong countervailing threat responses.

Summary

Heightened reward sensitivity is thought to contribute to heightened sensation seeking. This model is supported by multiple lines of evidence from animal and human research that have used a variety of methods to examine reward sensitivity at multiple levels of analysis. Yet sensation seeking is not synonymous with reward sensitivity, and individual differences in fear responses also likely contribute to sensation seeking. The role of fear response in sensation seeking, combined with sex differences in mean levels of sensation seeking and in fear-related disorders, suggests the possibility of sex differences in the relation between reward sensitivity and sensation seeking. However, there remains a need for well-powered research that tests sex differences in the relation between reward sensitivity and sensation seeking.

How Do Reward Sensitivity and Sensation Seeking Change With Age and Pubertal Development?

Sensation Seeking

Average levels of self-reported sensation seeking increase from late childhood (age 10) through midadolescence (around age 16), an age-related mean trend that is evident in both cross-sectional (Steinberg et al., 2008) and longitudinal (Harden, Quinn, & Tucker-Drob, 2012; Harden & Tucker-Drob, 2011) data in both males and females (Shulman et al., 2015) and in both Western and non-Western nations (Steinberg et al., 2017). In animal models of sensation seeking, such as locomotor response to novelty, similar developmental increases in adolescence relative to childhood have

been observed (Adriani, Chiarotti, & Laviola, 1998; Laviola, Macrì, Morley-Fletcher, & Adriani, 2003; Stansfield & Kirstein, 2006).

Evolutionary psychologists have hypothesized that an increase in sensation seeking is typical during adolescence because it is an adaptive response to the challenges of reproductive maturity, such as competing for and attracting mates, establishing new territory, and separating from adult caregivers (Ellis et al., 2012). From an evolutionary perspective, therefore, sensation seeking is hypothesized to be functionally tied to reproductive development, rather than to age per se. Supporting this perspective, some studies have found that self-reported sensation seeking is correlated with pubertal development even after accounting for chronological age (Castellanos-Ryan, Parent, Vitaro, Tremblay, & Seguin, 2013; Kirillova et al., 2001; Martin et al., 2002). Moreover, sensation seeking peaks earlier in girls than in boys (Romer & Hennessy, 2007; Shulman et al., 2015), which is consistent with the earlier onset of puberty in girls compared to boys (Herman-Giddens, 2006). Other studies, however, have found associations between pubertal development and self-reported sensation seeking only for males but not for females (Steinberg et al., 2008). And yet another line of research using animal models of sensation seeking found no correlation between timing of pubertal development and novelty-directed behaviors in rats (Vetter-O'Hagen & Spear, 2012).

Reward Sensitivity

There have been more large-scale studies of developmental changes in sensation seeking, which can be measured using brief, self-report surveys that are easy to administer to many participants, than of reward sensitivity. The major exception is a cross-national study by Steinberg and colleagues (2017), which aggregated self-reported sensation seeking and two behavioral tests of reward sensitivity into a single composite score. This composite score was found to increase, on average, from age 10 to 20 in a sample of $N = 5{,}404$ individuals from 11 countries. In one additional small sample, self-reported pubertal development was not correlated with reward-driven behavior on a gambling task, with reward-related brain activation to that task, or with functional connectivity between the medial prefrontal cortex and the ventral striatum (van Duijvenvoorde et al., 2014).

Summary

Sensation seeking increases from childhood to adolescence. However, given the inconsistent results of previous studies, it remains an open question whether these developmental trends are better accounted for by age than by pubertal development. Data on developmental differences in reward sensitivity has been even sparser than data regarding sensation seeking.

Are Developmental Differences in Reward Sensitivity and Sensation Seeking Due to Increases in Gonadal Hormones?

Despite the inconsistencies in the literature that was reviewed in the previous sections, developmental researchers have hypothesized that developmental changes in sensation seeking are specifically due to increases in gonadal hormones at puberty, via the effects of these hormones on neural systems that respond to reward (Blakemore, Burnett, & Dahl, 2010; Forbes & Dahl, 2010; Peper & Dahl, 2013). Certainly, average levels of gonadal hormones, including testosterone and estradiol, change dramatically during puberty, increasing in both sexes. Moreover, androgen and estrogen hormone receptors are distributed throughout the central nervous system. Testosterone, in particular, has both organizational and activational effects on neuronal structure and function in adolescence (Cameron, 2004; Peper, Pol, Crone & Van Honk, 2011; Rubinow, & Schmidt, 1996; Sisk & Zehr, 2005; Welker, Gruber, & Mehta, 2015; Witt, 2007). Furthermore, animal research has suggested that adolescents may be particularly sensitive to the effects of testosterone on reward systems. For instance, in rodent models, repeated administration of testosterone sensitizes adolescent male animals, but not adult males, to the locomotor effects of cocaine, a process that is dependent on changes in dopamine neurotransmission (Engi, Cruz, Crestani, & Planeta, 2015). Neuroendocrine research in animal models, therefore, has supported the possibility that gonadal hormone change will impact reward sensitivity and consequently sensation seeking. In the following section, we evaluate the current state of research on associations between gonadal hormones and sensation seeking and/or reward sensitivity.

Previous Research on Testosterone

Most studies that have directly measured gonadal hormonal concentrations in humans have focused on testosterone. In humans, studies of adult men have reported positive correlations between testosterone and self-reported sensation seeking or self-reported novelty seeking (Aluja & García, 2005; Aluja & Torrubia, 2004; Campbell et al., 2010; Määttänen et al., 2013; the study by Määttänen & colleagues, 2013, is particularly noteworthy because of its large sample size, with more than 800 participants). Also, using adult men as participants, research has found that salivary testosterone was correlated with quantity of hot sauce voluntarily consumed in an in-laboratory meal (Bègue, Bricout, Boudesseul, Shankland, & Duke, 2015), a behavior that was interpreted as an indicator of sensation seeking (N. K. Byrnes & Hayes, 2013). Other studies, however, have failed to find an association between testosterone and sensation seeking personality in adult men (e.g., Tsuchimine, Kaneda, Nakamura, & Yasui-Furukori, 2015). Finally, there is currently little direct evidence that testosterone is associated with self-reported sensation seeking in females.

A few studies have examined testosterone in relation to reward-related behavioral tasks. In a sample of 8- to 25-year-olds, salivary testosterone predicted reward-driven behavior (specifically, greater average pumps of the balloon) on the youth version of the balloon analogue risk task (BART-Y) in both males and females; in males, this association was statistically mediated by smaller gray matter volume in the medial orbitofrontal cortex (Peper, Koolschijn, & Crone, 2013). In adult women, administering testosterone (vs. a placebo control) and endogenous variation in testosterone were both associated with behavior on the IGT (Stanton, Liening, & Schultheiss, 2011; van Honk et al., 2004). However, the direction of the association was opposite to what might be expected based on research on pubertal development: Icenogle et al. (2017) found that more advanced puberty was associated with choosing more advantageous decks on the IGT, whereas the stud-

ies of testosterone in adult women found that testosterone was associated with choosing fewer advantageous decks and more disadvantageous decks.

The functional neuroimaging literature on the relation between testosterone and reward-related neural activity has yielded, in addition to these behavioral findings, mixed results. The largest study to-date on this question ($N > 250$) found that testosterone did not predict reward-related activity in the nucleus accumbens after accounting for the effects of age (Braams, van Duijvenvoorde, Peper, & Crone, 2015). Other studies have used both correlational and experimental designs to assess the effect of testosterone on reactivity to rewards in the striatum (Forbes & Dahl, 2010; Hermans et al., 2010; Op de Macks et al., 2011). Unfortunately, the sample sizes of these studies (Ns ≤ 50) were insufficient to yield reliable estimates, particularly regarding sex-specific effects.

One additional study is notable, despite its small sample size ($N = 61$, reduced to 38 after excluding for head motion), because it used 2-year longitudinal data to assess the effects of longitudinal change in testosterone on task-related brain activity (Spielberg et al., 2014). Drawing on the idea that individual differences in sensation seeking reflect both reward-relevant and threat-relevant processes, this study examined brain responses to threatening stimuli (angry and fearful faces, as opposed to geometric shapes), rather than to monetary rewards. It is interesting that average levels of testosterone were unrelated to neural activity; rather, *change* in testosterone predicted increases in amygdala and the nucleus accumbens reactivity to threat, even controlling for age. This association was reported as significant for boys but not for girls (Spielberg et al., 2014).

Previous Research on Estradiol

Compared to testosterone, estradiol and its relation with self-reported sensation seeking has been examined less, particularly in adolescence, although estradiol has been found to be positively correlated with sensation-seeking-related behaviors such as alcohol use (Martin, Mainous, Curry, & Martin, 1999). Animal studies have found evidence that estradiol enhances dopamine synthesis and release (Becker, 2000; Di Paolo, 1994; Pasqualini, Olivier, Guibert, Frain, & Leviel, 1995; Thompson & Moss, 1994), suggesting that rising levels of estradiol may increase reward sensitivity. Consistent with this idea, rapid withdrawal from ovarian hormones reduces rodents' preference for sucrose (a natural reward), an effect that can be mitigated with estradiol treatment (Brummelte & Galea, 2010). Yet animal research has also found that estradiol dampens both sensation seeking and reward sensitivity: Estradiol treatment of ovariectomized animals decreased novelty seeking and increased fearful behavior in mice (Morgan & Pfaff, 2001) and decreased cocaine self-administration in rats (Grimm & See, 1997).

Research on the effects of estradiol on reward-related decision-making and reward-related brain activity in humans has also yielded contradictory findings. Some studies have suggested that estradiol enhances reward response. In a placebo-controlled intervention study of healthy adult women, administration of goserelin, a gonadatrophin-releasing hormone agonist, resulted in the down-regulation of estradiol and testosterone levels; this reduction in estradiol and testosterone, in turn, was associated with reduced amygdala reactivity to large monetary rewards in a gambling task (Macoveanu et al., 2016). Consistent with these results, in young adolescent girls, estradiol correlated positively with reward response in the dorsal striatum, dorsolateral prefrontal cortex, and medial prefrontal cortex (Op de Macks et al., 2011).

In contrast, other studies examining naturally occurring fluctuations in ovarian hormones across the menstrual cycle have found evidence that estradiol is negatively associated with reward response. Specifically, nucleus accumbens activation during monetary reward anticipation (Ossewaarde et al., 2011) and preference for immediate rewards (Smith, Sierra, Oppler, & Boettiger, 2014) were found to be lower during the late follicular period, while estradiol levels are rising, than in the days before or during menstruation, when estradiol levels are low. Similarly, reactivity in the orbitofrontal cortex to monetary gains was found to be lower in the luteal period, when both estradiol and progesterone are high, than in the early follicular period, when both are low (Bayer, Bandurski, & Sommer, 2013). Considering the inconsistencies across studies, it is possible that the effect of exogenous changes in estradiol does not necessarily mimic the effects of endogenous fluctuations and that neither necessarily mimics the effects of developmental increases in estradiol in adolescence or of temporally stable individual differences (Harden, Kretsch, Moore, & Mendle, 2014).

Summary

Overall, research on gonadal hormones in relation to reward sensitivity and sensation seeking has yielded mixed results. The neuroimaging literature on testosterone and reward sensitivity has yielded an inconsistent picture, with some studies finding effects of testosterone in both sexes, some finding effects only in boys, and some finding no effects after controlling for age. Results from estradiol have been even more inconsistent, with some studies suggesting positive associations with reward sensitivity and some suggesting negative associations. The small sample sizes characteristic of this literature undoubtedly contribute to the inconsistency in results.

Goals of the Current Study

In summary, previous research has hypothesized that (a) reward sensitivity contributes to, but is not synonymous with, sensation seeking; (b) reward sensitivity and sensation seeking increase from childhood to adolescence; (c) this change may be driven by pubertal development and/or gonadal hormones; and (d) these relations may be sex-specific. However, studies on this topic, particularly research including measures of gonadal hormones, have generally suffered from several limitations, including small sample sizes and univariate measurement of key constructs. This study aimed to clarify the sex-specific associations between age, pubertal development, gonadal hormones, reward sensitivity, and sensation seeking using a large, diverse, population-based sample of adolescents. Drawing on factor analyses of a larger battery of measures in this sample (Harden et al., 2017), we combined three in-laboratory behavioral measures—the balloon analogue risk task, the Iowa gambling task, and the stoplight task—to estimate a latent reward-sensitivity factor. We then applied structural equation modeling techniques, including multiple-group analyses, to test the

relations among reward sensitivity, developmental differences (age, puberty, gonadal hormones), and sensation seeking.

Method

This study received ethical approval from the Institutional Review Board at the University of Texas at Austin (Protocol 2016–01-0004: "Genetic and Hormonal Influences on Adolescent Decision Making").

Participants

Data for the current study were drawn from an ongoing, in-laboratory study of adolescent twins that forms one of the core components of the Texas Twin Project (Harden, Tucker-Drob, & Tackett, 2013). Participants were identified and recruited from public school rosters in the Austin and Houston metropolitan areas in Texas. The analytic sample consisted of 810 adolescents nested within 398 families. Participants ranged in age from 13.6 to 20.1 years (M = 15.9, SD = 1.4). Most participants (89%) were between the ages of 14 and 18. Participants' self-reported race–ethnicity was as follows: 57% non-Hispanic White, 18% Hispanic–Latino, 15% Black–African American, 5% Asian–Asian American, and the remaining 5% another race–ethnicity.

Measures

All measures were completed in the laboratory. Surveys were completed using REDCap (Research Electronic Data Capture), a secure, web-based application designed to support data collection for research studies (Harris et al., 2009). The Iowa gambling task (IGT) was programmed and administered using E-Prime Version 2.0 (Psychology Software Tools, http://www.pstnet.com). The youth version of the balloon analogue risk task (BART–Y) and the stoplight task were distributed by the task authors as free-standing executable programs.

Pubertal development. Participants reported on their pubertal development using the Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988). All participants rated growth in height, growth of body hair, and skin changes on a 4-point scale ranging from 1 (*Not Yet Begun to Change*) to 4 (*Finished Changing*). Male participants rated, in addition to these three items, growth of facial hair and deepening of voice on the same 4-point scale. Female participants also rated growth of breasts and whether they had begun to menstruate. The menstruation item was coded to be consistent with the 4-point scale (1 = No, 4 = Yes). Scores were taken as the average across the five items. As expected, girls reported more advanced pubertal development than did boys (see Table 1 for sample statistics).

Gonadal hormones. A saliva sample collected via passive drool during the lab visit was assayed to determine testosterone and estradiol concentrations. Samples were collected at one of three appointment times: 9:00–10:00 a.m. (29% of participants), 11:00 a.m. to 1:00 p.m. (49% of participants), or 1:00–3:00 p.m. (22% of participants). Participants were instructed to avoid eating or drinking anything 2 hr prior to beginning the experiment, to avoid flossing the morning of the experiment, and to avoid smoking 4 hr prior to coming in. Participants provided salivary samples in a 2-ml vial after approximately 15 min of completing consent forms and answering questions relevant to the salivary samples (e.g., food eaten that day). Immediately following collection, saliva samples were frozen at ≤−30 °C prior to being shipped on dry ice to Clemens Kirschbaum's laboratory at the Technical University of Dresden for analyses. Commercially available chemiluminscense immunoassays (IBL International, Hamburg, Germany) were used to measure testosterone and estradiol concentrations with high sensitivity. The lower limits of sensitivity for

Table 1
Descriptive Statistics and Zero-Order Correlations by Sex for Focal Study Constructs

Sex and variable	*M*	*SD*	1	2	3	4	5	6	7
Female									
1. Age (years)	15.91	1.45	—						
2. Pubertal development	3.38	.82	**.41**	—					
3. Testosterone (pg/mL)	26.48	12.11–57.90	**.13**	**.14**	—				
4. Estradiol (pg/mL)	3.61	1.85–7.07	**.14**	.07	**.42**	—			
5. Stoplight (proportion intersections)	.43	.20	.09	.03	**.10**	−.03	—		
6. BART–Y (pumps)	29.13	12.30	−.01	.00	−.02	**−.12**	**.25**	—	
7. IGT[a]	.76	.13	.04	**.22**	−.03	−.04	**.12**	**.21**	—
8. Sensation seeking	2.74	.60	.06	**−.14**	−.09	.06	**.15**	.04	−.01
Male									
1. Age (years)	15.83	1.31	—						
2. Pubertal development	2.91	.71	**.54**	—					
3. Testosterone (pg/mL)	81.42	37.73–175.73	**.45**	**.44**	—				
4. Estradiol (pg/mL)	3.20	1.41–7.25	**.15**	**.13**	**.37**	—			
5. Stoplight (proportion intersections)	.44	.18	.05	**.15**	.04	.09	—		
6. BART–Y (pumps)	30.82	12.14	−.01	.06	−.03	−.07	.05	—	
7. IGT[a]	.80	.13	**.18**	**.15**	.01	.05	**.24**	.09	—
8. Sensation seeking	2.95	.53	**.12**	**.18**	.10	−.01	**.13**	**.21**	.09

Note. All analyses of testosterone and estradiol were conducted on log-transformed and winsorized scores. This table presents exp(M) of the log-transformed variables in the M column and exp(M − 1 SD) − exp(M + 1 SD) in the SD column to characterize the sample in terms of the original units of hormonal concentrations. Correlations significantly different from 0 at $p < .05$ are in bold type. BART–Y = youth version of the balloon analogue risk task; IGT = Iowa gambling task; exp = exponential function.
[a] Summary statistics are reported in the proportion of good deck trials that the participant chose to play.

the assays were .3 pg/mL for estradiol and 1.8 pg/mL for testosterone; extremely high values were estimated from standard curves. The intra-assay and interassay coefficients of variation were <8% and <11%, respectively, for both testosterone and estradiol.

Of the 810 participants, 697 had a usable measurement of testosterone, 680 had a usable measurement of estradiol, and 679 had both. Common reasons for missing hormonal concentrations were insufficient volume of saliva and saliva contamination. Individuals with missing hormone values were included in all analyses, and missing data were handled using full information maximum likelihood in Mplus (Little, Jorgensen, Lang, & Moore, 2014).

Quality control procedures for salivary hormone data, including information on menstrual cycle variability, have been reported in detail elsewhere (Grotzinger et al. 2017). Briefly, hormonal concentrations were log-transformed and then residualized (separately by sex) for time since waking and study site. Additionally, for females who reported regular menstrual cycles with 28–35 days' duration, values were residualized for menstrual cycle phase (menstrual phase [Days 0–5] vs. follicular phase [Days 6–14] vs. luteal phase [Days 15–35]). Outliers were replaced, for males and females separately, using a winsorizing procedure that replaced extreme values by the highest observed scores within 3 standard deviations of the mean. Finally, the winsorized residuals were standardized within sex.

Sensation seeking. The UPPS Impulsive Behavior scale (Whiteside & Lynam, 2001) is a 45-item self-report survey tapping four dimensions of impulsivity: *urgency* (e.g., "Sometimes when I feel bad, I can't seem to stop what I am doing, even though it is making me feel worse"), *premeditation* (e.g., "I like to stop and think things over before I do them"), *perseverance* (e.g., "I finish what I start"), and *sensation seeking* (e.g., "I generally seek new and exciting experiences and sensations"). Items are rated on a 1–4 scale. This study used the mean score of items on the sensation-seeking scale (12 items; Cronbach's alpha = .84). The UPPS was developed based on factor analyses of numerous personality inventories related to the umbrella construct of "impulsivity" (Whiteside & Lynam, 2001), which found that the Sensation Seeking Scale (Zuckerman, 1994), NEO-PI Excitement Seeking (Costa & McCrae, 1992), EASI Sensation Seeking (Buss & Plomin, 1975), and I-7 Venturesomeness (Eysenck, Pearson, Easting, & Allsopp, 1985) were all indicators of a sensation-seeking factor. Items across the different scales that had similar content were identified, and items that had the highest item–factor correlations were retained (Whiteside & Lynam, 2001). Thus, UPPS Sensation Seeking distills multiple other commonly used sensation-seeking scales and includes items such as "I'll try anything once" and "I sometimes like doing things that are a bit frightening."

Iowa gambling task. We used a modified version of the IGT (Cauffman et al., 2010). On each trial, participants are given the opportunity to "play" or "pass" from one of four decks of cards. Two decks were advantageous ("good"), in that repeated play will ultimately result in winning money, whereas two decks were disadvantageous ("bad"), in that repeated play will result in losing money. Because all participants were offered the same number of opportunities to play from good decks and bad decks, this version of the IGT can quantify two separable and potentially orthogonal parameters: (1) the extent to which participants learn to play from good decks versus (2) the extent to which participants learn to avoid bad decks. The former has been conceptualized as a measure of reward sensitivity or approach behavior, whereas the latter has been conceptualized as a measure of punishment sensitivity or avoidance behavior (Cauffman et al., 2010). Developmental studies have found that these two IGT parameters are only modestly correlated ($r \sim .10$; Harden et al., 2017), load on different factors when submitted to exploratory factor analyses along with a battery of other behavioral and self-report measures relevant to risk-taking (Harden et al., 2017), show diverging mean age trends (Cauffman et al., 2010; Harden et al., 2017; Icenogle et al., 2017), and are differentially related to pubertal development (Harden et al., 2017; Icenogle et al., 2017). The version of the IGT used in the study can be contrasted with versions that allow participants to choose freely among all four decks of cards on each trial (e.g., van Honk et al., 2004). This collapses individual differences in approach and avoidant behavior, because choosing a good deck necessarily involves not choosing a bad deck (and vice versa).

The dependent variable used in the current study was play on good decks (see the supplement to Harden et al., 2017), because this parameter has been associated with pubertal development and with self-reported sensation seeking in previous studies (e.g., Icenogle et al., 2017). Specifically, mixed-effects models were used to model the proportion of trials that a person decided to play (vs. pass) over the course of the six task blocks. Separate models were estimated for good and bad decks. The effect of block was coded (−5 to 0) such that the intercept represents play at the final block (attained level), and random effects for intercept and slope were estimated (see Cauffman et al., 2010, for more details on mixed effects modeling of IGT performance). Models were fit using the lme4 package (Version 1.1–10; Bates et al., 2015) in (R R Core Team, 2015).

On average, play on good decks increased, and play on bad decks decreased, over the course of the IGT (Harden et al., 2017). For good decks, there was a positive average slope for block ($b = .03$, $SE = .002$, $t = 15.5$), whereas for bad decks, there was a negative average slope for block ($b = -.02$, $SE = .002$, $t = -9.94$). Random intercepts and slopes were substantially and positively correlated for both good ($r = .61$) and bad ($r = .95$) decks. Therefore, we used the empirical Bayes estimates for random intercepts for subsequent analyses; these estimates were output from the mixed-effects models and standardized (pooling across sex).

Balloon analogue risk task—youth version. In the BART–Y (Lejuez et al., 2007), individuals decide how much air to "pump" into a balloon on the computer screen. For each successful pump of air, more points are accrued; however, at some point, the addition of more air causes the balloon to burst, leading the participant to lose all points accrued during that trial. The dependent variable was the average number of pumps on trials in which the balloon did not explode (i.e., average adjusted pumps).

Stoplight. In the stoplight task (Steinberg et al., 2008), individuals "drive" a car to a destination under time pressure. Along the way are a series of crossroads, and at each one the person must decide whether to run a yellow light, which turns red after a variable amount of time, or to stop and wait for the light to turn red and then green. Time is saved if the person successfully runs the yellow light, whereas time is lost when the light turns red and the person crashes into another car at the intersection. The dependent

variable was the proportion of crossroads at which the person failed to stop.

Analytic Plan

All analyses were conducted using structural equation modeling in Mplus Version 7.1 (Muthén & Muthén, 1998–2015). Model fit was primarily evaluated using the root-mean-square error of approximation (Steiger, 1990), with values <.05 indicating good model fit. All analyses treated each individual (rather than a twin pair) as a case; therefore, standard errors and model fit statistics were corrected for nesting within nuclear families using the TYPE = COMPLEX option in Mplus (McNeish, Stapleton, & Silverman, 2017). Sex differences were tested using multiple-group models. Models were estimated using maximum likelihood with robust standard errors. Models were compared using the Satorra-Bentler scaled chi-square test (Satorra & Bentler, 2001).

Two sets of analyses were conducted. First, we fit a latent factor model that used the three behavioral tasks as indicators of a latent reward-sensitivity factor and then conducted measurement invariance analyses to examine whether this latent factor model was equivalent in males versus females and in older versus younger adolescents. Measurement invariance analyses are a critical step in developmental research that aims to make conclusions about sex differences and age-related differences in latent constructs, because such differences are meaningful only if the observed indicators of the latent constructs reflect the latent factors in the same way across development and across sexes (Widaman, Ferrer, & Conger, 2010).

Second, we conducted a series of multiple-group structural equation models (SEMs) that tested whether associations among study constructs differed by sex. Specifically, these models tested for (a) sex differences in the association between reward sensitivity and sensation seeking and (b) sex differences in the associations with each of the developmental correlates (age, pubertal development, and gonadal hormones), which were considered separately. Results from these models informed our final comprehensive model, which tested whether any of the developmental correlates independently predicted reward sensitivity and sensation seeking.

Results

Sample Statistics

Table 1 describes summary statistics for measures and the correlations among all measures by sex.

Measurement Invariance of the Reward-Sensitivity Factor Across Sexes and Ages

Informed by previous factor analyses of data from this sample (Harden et al., 2017), we modeled performance on the IGT, BART–Y, and stoplight tasks as indicators of a reward-sensitivity factor and tested the measurement invariance (MI) of this factor across sex and age (see Figure 1).

Sex invariance. MI models that tested sex differences used multiple-group modeling. First, a model in which the factor loadings, intercepts, and residual variances for the observed indicators were freely estimated in each sex (Model 1: noninvariance) was compared to a model in which the factor loadings (paths labeled λ in Figure 1) were constrained to be equal in males and females (Model 2: metric invariance). This model comparison tests whether the associations between the latent construct (reward sensitivity) and performance on the behavioral tasks were equivalently strong in both sexes. The metric invariance model did not fit significantly worse than did the noninvariance model—Model 2: $\chi^2(3) = 5.10$, scaling correction factor (c) = 1.075, $p = .16$; Model 1 (fully saturated model): $\chi^2(0) = 0$, $c = 0$, $p = .00$; Model 2 vs. Model 1: Satorra-Bentler scaled $\chi^2(3)$ difference test = 5.10, $p = .16$—indicating that metric invariance was supported.

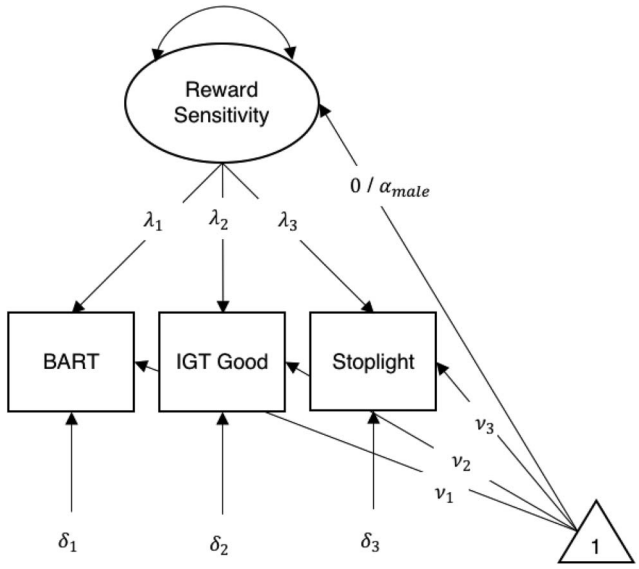

Figure 1. Measurement model of reward sensitivity among males and females ages 13–20. Reward-sensitivity factor standardized ($M = 0$, $SD = 1$). Factor mean for males (α_{male}) estimated only in models in which indicator intercepts (ν) were fixed to equality in males and females. BART = balloon analogue risk task; IGT = Iowa gambling task.

Next, a model in which both the factor loadings and the intercepts (parameters labeled λ 1-3 and ν 1-3, respectively in Figure 1) for the factor indicators were fixed to be equal across sex (Model 3: scalar invariance) was compared to Model 2. This model comparison tests whether sex differences in performance on the behavioral tasks is mediated entirely through sex differences in the latent factor or whether there are sex differences in any individual task that do not reflect sex differences in the underlying construct of reward sensitivity. The scalar invariance model did not fit worse than did a metric invariance model—Model 3: $\chi^2(5) = 10.10$, $c = .97$, $p = .07$; Model 2: $\chi^2(3) = 5.10$, $c = 1.08$, $p = .16$; Model 3 vs. Model 2: Satorra-Bentler scaled $\chi^2(2)$ difference test = 5.30, $p = .07$—indicating that scalar invariance was supported.

Finally, a model in which both the factor loadings, intercepts, and residual variances (parameters labeled with δ in Figure 1) for the factor indicators were fixed to be equal across sex (Model 4: strict invariance) was compared to Model 3. This model comparison tests whether there are sex differences in the magnitude of the error variances in the behavioral tasks. Strict invariance implies that any sex differences in the variances of the behavioral tasks are

due to sex differences in variability at the latent factor level. The strict invariance model did not fit worse than did a scalar invariance model—Model 4: $\chi^2(8) = 12.06$, $c = 1.049$, $p = .15$; Model 3: $\chi^2(5) = 10.10$, $c = .974$, $p = .07$; Model 4 vs. Model 3: Satorra-Bentler scaled $\chi^2(3)$ difference test = 2.40, $p = .49$—indicating that scalar invariance was supported. Consequently, a reward sensitivity measurement model in which parameters were constrained to be equal in both sexes (Model 4: strict invariance) was carried forward for subsequent models.

Age invariance. MI models that tested age differences treated age as a continuous variable (rather than arbitrarily binning age and using multiple-group models; Tucker-Drob, 2009, 2013). The first comparison found that a model in which the three indicators were each regressed on age, $\chi^2(2) = 6.441$, $c = .77$, $p = .04$, did not fit significantly better than did a model in which only the reward-sensitivity factor was regressed on age, $\chi^2(4) = 8.679$, $c = .99$, $p = .07$; Satorra-Bentler scaled $\chi^2(2)$ difference test = 2.98, $p = .23$. Next, we allowed the factor loadings to vary as a continuous function of age, and this model found that none of the age interactions on the factor loadings were significantly different from 0 at $p < .05$.

Summary of measurement invariance results. In summary, results from MI models indicated that the reward-sensitivity factor was invariant across ages and sex. The standardized loadings of the three behavioral tasks on the reward-sensitivity factor were small to moderate in magnitude (all ps $< .01$): IGT = .58 (*SE* = .12), BART–Y = .41 (*SE* = .10), stoplight = .24 (*SE* = .09). There were substantial sex differences in the mean of the reward-sensitivity factor, with males nearly .5 *SD* higher than females (.45, *SE* = .13, $p < .001$).

Sex Differences in the Reward Sensitivity–Sensation Seeking Association

To test the hypothesis that the relation between reward sensitivity and sensation seeking was stronger in males than in females, we tested a multiple-group SEM, as shown in Figure 2. In a sex-differences model, the measurement model for reward sensitivity was constrained to be equal across sexes, but the path from reward sensitivity to sensation seeking and the residual variance in sensation seeking were allowed to differ between sexes. This model fit the data significantly better, $\chi^2(12) = 28.55$, $c = 1.02$, $p = .005$, than did a more constrained model, $\chi^2(14) = 36.06$, $c = 1.03$, $p = .001$, which equated the association between reward sensitivity and sensation seeking, and the residual variance in sensation seeking, in males and females, Satorra-Bentler scaled $\chi^2(2)$ difference test = 7.40, $p = .02$ (mean levels of sensation seeking were allowed to vary by sex in both models). Consistent with our hypothesis, reward sensitivity was more strongly related to sensation seeking in male adolescents ($\beta = .25$, *SE* = .10, $p = .01$) than in female adolescents ($\beta = .09$, *SE* = .09, $p = .32$). The path from reward sensitivity to sensation seeking, and the residual variance in sensation seeking, was freely estimated across sex for all remaining models.

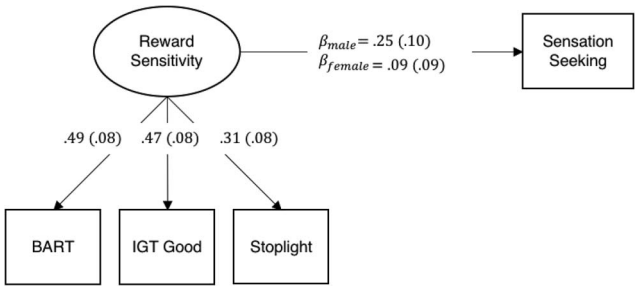

Figure 2. Sex-specific associations between reward sensitivity and sensation seeking among males and females ages 13–20. Standardized parameters, with standard errors in parentheses, are shown. Parameters that are presented separately by sex could not be equated without a significant loss of model fit. BART = balloon analogue risk task; IGT = Iowa gambling task.

Age Differences in Reward Sensitivity and Sensation Seeking

We next extended the model to include age as a predictor of both reward sensitivity and sensation seeking (not shown). Age was centered at 15 years. A model that constrained the paths from age to reward sensitivity and sensation seeking, as well as residual variance in reward sensitivity, to be equal across sex did not fit significantly worse, $\chi^2(18) = 39.18$, $c = 1.04$, $p = .003$, than did a model that allowed these parameters to differ between males and females, $\chi^2(15) = 40.10$, $c = .96$, $p < .001$; Satorra-Bentler scaled $\chi^2(3)$ difference test = 1.49, $p = .68$. Notably, age was not significantly associated with either reward sensitivity (*b* [per year of age] = .02, *SE* = .02, $p = .15$, $\beta = .12$) or sensation seeking (*b* [per year of age] = .02, *SE* = .02, $p = .08$, $\beta = .07$).

Puberty-Related Differences in Reward Sensitivity and Sensation Seeking

As with age, we compared the fit of a model that allowed the paths from self-reported pubertal development to reward sensitivity and sensation seeking to differ between sexes to a more constrained model that equated these paths, and the residual variation in reward sensitivity, in males and females (see Figure 3). In contrast to what was observed for age, a constrained model fit significantly worse, $\chi^2(18) = 58.29$, $c = .99$, $p < .0001$, than did a model allowing puberty associations to differ between males and females, $\chi^2(15) = 43.68$, $c = .91$, $p = .0001$; Satorra-Bentler scaled $\chi^2(3)$ difference test = 12.89, $p = .005$. However, a follow-up model that constrained the path from pubertal development to reward sensitivity (and the residual variance in reward sensitivity), but not the path from pubertal development to sensation seeking to be equal across sexes, did not fit significantly worse than did a model allowing both paths to differ, $\chi^2(17) = 41.21$, $c = .99$, $p = .0009$; Satorra-Bentler scaled $\chi^2(2)$ difference test = 0.75, $p = .69$, suggesting a differential relation between puberty and sensation seeking, but not between puberty and reward sensitivity, across sexes.

Results from this final model are shown in Figure 3. For both males and females, pubertal development was positively associated with reward sensitivity ($\beta = .28$, *SE* = .08, $p < .001$). In contrast, pubertal development was positively associated with sensation seeking in males ($\beta = .16$, *SE* = .07, $p = .02$) but negatively associated with sensation seeking in females ($\beta = -.18$, *SE* = .06, $p = .005$).

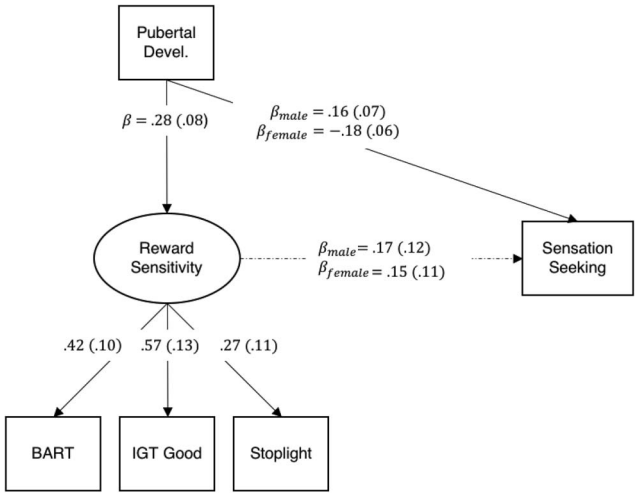

Figure 3. Puberty-related differences in reward sensitivity and sensation seeking among males and females ages 13–20. Standardized parameters, with standard errors in parentheses, are shown. Parameters that are presented separately by sex could not be equated without a significant loss of model fit. Dashed lines represent parameters that are not significantly different from 0 at $p < .05$.

After we controlled for pubertal development, the residual association between reward sensitivity and sensation seeking in males was no longer significantly different from 0 ($\beta = .17$, $SE = .12$, $p = .15$; the relation also remained nonsignificant in females). In a related vein, the indirect path from pubertal development to sensation seeking via reward sensitivity was also not significantly different from 0 ($\beta = .04$, $SE = .03$, $p = .16$). That is, although both reward sensitivity and sensation seeking were positively associated with self-reported pubertal development, the latter relationship was not statistically mediated via the former. This result was contrary to our hypothesis, although the usual caveats regarding cross-sectional tests of mediation apply here (Maxwell & Cole, 2007).

Gonadal Hormone Associations With Reward Sensitivity and Sensation Seeking

Following the same logic as used in the previous model comparisons, we compared the fit of a model that allowed the paths from gonadal hormones to reward sensitivity and sensation seeking to differ between sexes to a more constrained model that equated these paths and the residual variation in reward sensitivity to be equal in males and females (see Figure 4); these equality constraints did not result in a significant change in model fit: no sex differences, $\chi^2(24) = 41.65$, $c = 1.033$, $p = .01$; sex differences, $\chi^2(19) = 39.91$, $c = .967$, $p = .0003$; Satorra-Bentler scaled $\chi^2(5)$ difference test = 3.43, $p = .63$. It is interesting that neither hormone had a significant association with reward sensitivity, and the direction of the hormone associations sensation seeking diverged: Testosterone was positively associated with sensation seeking, but estradiol was negatively associated.

We then tested, as a follow-up analysis, the associations with each hormone individually. The pattern of results for testosterone was unchanged: Testosterone was not significantly associated with reward sensitivity ($\beta = -.08$, $SE = .07$, $p = .24$) but was positively associated with sensation seeking ($\beta = .09$, $SE = .04$, $p = .02$). When we were not simultaneously controlling for testosterone, however, estradiol was no longer significantly associated with either reward sensitivity ($\beta = .06$, $SE = .07$, $p = .40$) or sensation seeking ($\beta = -.06$, $SE = .04$, $p = .18$). That is, estradiol is a better predictor of (lower) sensation seeking when considered against the backdrop of another gonadal hormone. The mechanisms underlying this observation are not yet apparent.

Combining Self-Reports of Pubertal Development and Gonadal Hormones

When considered separately, self-reported pubertal development and gonadal hormones were each related to either or sensation seeking. Our final model, therefore, combined these developmental indicators into a single model (see Figure 5). The model was specified such that we could test whether associations with self-reported pubertal development were statistically mediated through observed levels of gonadal hormones. Sex differences that were significant in previous models were carried forward, and the relations between self-reported puberty and gonadal hormones were also allowed to differ by sex.

Results are shown in Figure 5. Among female adolescents, there were five main results. First, self-reported pubertal development was minimally associated with concentrations of gonadal hormones measured in saliva. Second, self-reported puberty was positively correlated with reward sensitivity but negatively with sensation seeking. Third, puberty-related differences in reward sensitivity and sensation seeking were not statistically mediated via measured hormones. Fourth, gonadal hormones were associated with sensation seeking but in different directions depending

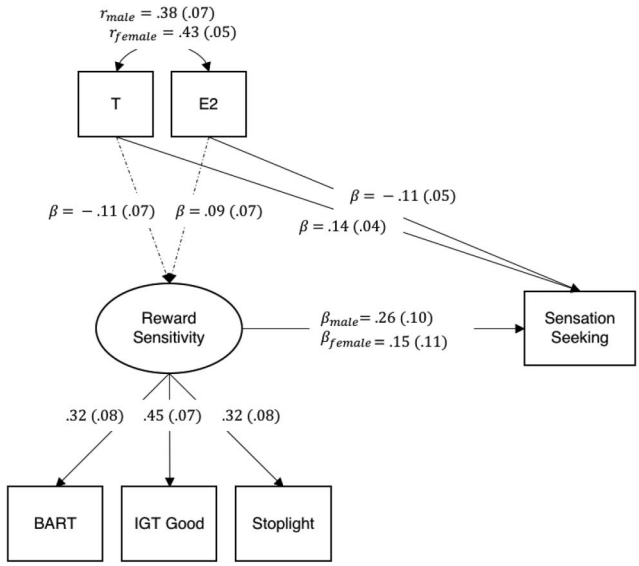

Figure 4. Gonadal hormone associations with reward sensitivity and sensation seeking among males and females ages 13–20. Standardized parameters, with standard errors in parentheses, are shown. Parameters that are presented separately by sex could not be equated without a significant loss of model fit. Dashed lines represent parameters that are not significantly different from 0 at $p < .05$.

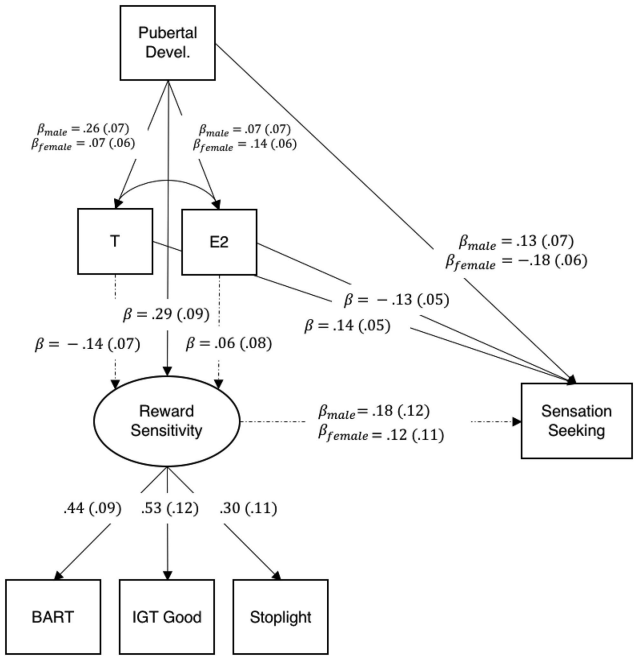

Figure 5. Associations among self-reported puberty, gonadal hormones, reward sensitivity, and sensation seeking among males and females ages 13–20. Standardized parameters, with standard errors in parentheses, are shown. Parameters that are presented separately by sex could not be equated without a significant loss of model fit. Dashed lines represent parameters that are not significantly different from 0 at $p < .05$.

on the hormone, and these associations were independent of self-reported puberty and of reward sensitivity. Five, reward sensitivity and sensation seeking were not associated with each other. Overall, the pattern of results was considerably more complex and dissociated in female adolescents than has been proposed in much of the developmental literature.

The picture was somewhat clearer among male adolescents. Two results supported predictions from extant developmental theory. First, self-reported pubertal development was positively correlated with measured testosterone levels and with reward sensitivity. Second, testosterone was positively associated with sensation seeking and statistically mediated part of the puberty–sensation-seeking association (indirect effect of puberty on sensation seeking via testosterone: β = .04, *SE* = .02, *p* = .02; direct effect of puberty on sensation seeking: β = .13, *SE* = .07, *p* = .05; total effect: β = .20, *SE* = .06, *p* = .001).

Two additional results in male adolescents were inconsistent with our hypotheses. The puberty–reward sensitivity association was not statistically mediated via testosterone (indirect effect of puberty on reward sensitivity via testosterone: β = −.04, *SE* = .02, *p* = .09; direct effect: β = .29, *SE* = .09, *p* = .001). And, although both reward sensitivity and sensation seeking were positively associated with pubertal development in male adolescents, the effect of pubertal development on sensation seeking was not statistically mediated by reward sensitivity (indirect effect of puberty on sensation seeking via reward sensitivity: β = .05, *SE* = .03, *p* = .08).

Discussion

In an effort to identify the developmental mechanisms that underlie adolescents' propensity for risk-taking, personality researchers have investigated patterns of age-related changes in disinhibited traits. This line of research has found that average levels of sensation seeking increase from childhood to midadolescence, especially in male adolescents, paralleling the developmental trends in the incidence of risk-taking behavior (Harden & Tucker-Drob, 2011; Shulman et al., 2015; Steinberg et al., 2008) and that individual differences in changes in delinquency and substance use are yoked to changes in sensation seeking (Harden, Quinn, & Tucker-Drob, 2012; Quinn & Harden, 2013). At the same time, developmental cognitive neuroscience has begun to elucidate how neural and behavioral responses to rewards differ in adolescents versus children and adults. Both the personality and the developmental cognitive neuroscience literatures have hypothesized that these developmental changes in sensation seeking and reward sensitivity are linked to each other and linked to rising levels of gonadal hormones that accompany puberty. In the current article, we presented data from a large sample of adolescents, ages 13–19, to unpack the relation between reward sensitivity and sensation seeking and investigate the sex-specific effects of age, pubertal development, and gonadal hormones on both constructs.

Sex Differences in the Association Between Reward Sensitivity and Sensation Seeking

Reward sensitivity—as measured by three behavioral tasks—was more strongly associated with self-reported sensation seeking in males than in females. In fact, in females, the reward-sensitivity–sensation-seeking association was not significantly different from 0. It is important to note that measurement invariance analyses indicated that the behavioral tasks were measuring the latent construct equivalently in males and females, indicating that the dissociation between reward sensitivity and sensation seeking in females cannot be attributed to measurement problems in females.

This sex difference in the reward-sensitivity–sensation-seeking association is consistent with results of previous neurobiological studies on reward responses to addictive substances, such as amphetamine and nicotine, which have found that sensation seeking predicts the rewarding effects of substances more strongly in men than in women (Perkins et al., 2008; Riccardi et al., 2006). It is also consistent with the well-established sex difference in vulnerability to fear and anxiety (Kessler et al., 2012), which are inversely related to sensation-seeking tendencies (Lissek et al., 2005). That is, the association between sensation seeking and reward sensitivity might be weaker in women, because they are more likely to experience stronger avoidance motivations, which operate as countervailing influences on sensation seeking.

Even in male adolescents, the residual variances in sensation seeking and reward sensitivity were not significantly associated with one another after accounting for their common relationship with pubertal development. This result can be considered in relation to two other previously reported results. First, in a quantitative genetic analysis of this data set (Harden et al., 2017), we found that genetic influences on sensation seeking (collapsing across both sexes) were largely distinct from genetic influences on a reward-

sensitivity factor. Second, at least one study has reported that self-reported sensation seeking was uncorrelated with neural activity to reward among adolescents ages 12–16 (Hawes et al., 2017). Much of the neuroscience research on sensation-seeking and risk-taking behavior has focused on reward-related neural systems (e.g., dopaminergic neurotransmission, activity in the ventral striatum). But, such systems are likely only one contributor to the neurobiology of sensation seeking. Researchers would do well to look beyond reward-related processes, brain regions, and neurotransmitter systems in explaining individual differences and developmental trends in sensation seeking.

In contrast to the present results, those of a previous meta-analysis did not find that the sex composition of the sample significantly moderated the association between sensation seeking and performance on the BART–Y; however, there may have been too few studies to test sex moderation with adequate power: There were only two studies with predominantly male samples and five with predominantly female samples, and study-specific sex interactions were not themselves meta-analyzed (Lauriola, Panno, Levin, & Lejuez, 2014). Additionally, as indicated by the moderate factor loading of BART–Y on the reward-sensitivity factor, there is a substantial amount of unique task-specific variance in BART–Y performance that is not shared with other popular measures of the reward-sensitivity construct. Our previous behavioral genetic decomposition of the variance in this battery of tasks (Harden et al., 2017) found that task-specific variance in the BART–Y was nonshared environmental in origin (i.e., not systematically correlated across either identical or fraternal twins), a quantity that includes measurement error. Thus, aggregating multiple behavioral measures into a latent factor for reward sensitivity, as we did in the current study, is likely to produce a more reliable estimate of its relation to sensation seeking.

What Construct Is Measured by the Behavioral Tasks?

We have labeled the variance common to performance on the IGT, stoplight, and BART–Y as *reward sensitivity* rather than *risk-taking*. This nomenclature is supported by the observation that not only were the measured behaviors (pumping the balloon higher on the BART–Y, running through the intersection on the stoplight, playing the hand on the IGT) motivated by the possibility of earning a monetary reward but engaging in these behaviors did, in fact, result in higher rewards. For example, earnings are maximized on the BART–Y when pumps equal 64, whereas the mean pumps in our sample was around 31, and only five participants out of over 600 showed "risk-taking" on the BART–Y that was actually disadvantageous (pumps >64). Our sample is not atypical in this regard. A meta-analysis of studies documenting a correlation between self-reported sensation seeking or impulsivity and BART performance found that, across 22 studies, mean BART performance ranged from 24.6 to 44.1 pumps ("there were not studies in which the majority of participants made consistent risk-disadvantageous choices from a normative point of view"; Lauriola et al., 2014, p. 28). Similarly, the good decks of the IGT are structured such that repeated play leads to earning, rather than losing, money. Overall, play on these tasks was "risky" only in the sense that each trial involved an (unknown) chance of loss, but the expected reward values of risk-taking on these tasks were positive.

The reward-sensitivity tasks may have also included an element of arousal (beyond the arousal inherent in receiving a reward), because trials that a participant lost contained the higher intensity stimuli (e.g., a loud sound as the balloon burst in BART–Y, the sound of screeching brakes as the car crashed in stoplight). Experiencing these relatively intense stimuli was possible only if a participant engaged in the reward-sensitivity behavior, thus potentially contributing to the observed association between sensation seeking and reward sensitivity. This issue speaks to the continued methodological challenges involved in separating out how strongly one is motivated by rewards from how much one prefers intense or arousing stimuli.

Methodological Challenges in Identifying Hormonal Mechanisms for Pubertal Associations

This study found evidence that self-reported pubertal development was associated with greater reward sensitivity in both sexes and with higher sensation seeking in male adolescents. The puberty–sensation-seeking association was statistically mediated, in part, via higher testosterone levels in male adolescents. However, contrary to our hypothesis, the puberty–reward sensitivity association was not statistically mediated through salivary testosterone.

Why might self-reported pubertal development be a better predictor of reward sensitivity behavior than is measured testosterone concentration? We can think of at least three potential explanations, each of which suggests avenues for future research. First, the association between pubertal development and reward sensitivity may, indeed, be independent of hormones and may be due to another mechanism, such as the differing social experiences of early maturing versus later maturing adolescents (e.g., Ge & Natsuaki, 2009) or to genetic differences between early and late maturers (Harden & Mendle, 2012; Harden, Mendle, & Kretsch, 2012).

Second, several previous studies have found that testosterone is associated with elevated externalizing and aggressive behaviors, with risk-taking, and with social status–seeking and status-maintaining behaviors only when cortisol, the primary output of the hypothalamic–pituitary–adrenal axis, is low (Mehta & Josephs, 2010; Mehta & Prasad, 2015; Tackett, Herzhoff, Harden, Page-Gould, & Josephs, 2014). If the relation between testosterone and reward sensitivity depends on cortisol, then the main effect estimated here might be misleading.

Third, self-reports of pubertal development may, counterintuitively, be a better marker for the body's cumulative previous exposure to gonadal hormones, and of its sensitivity to those hormones, than is a single measure of current testosterone levels. For male adolescents, the PDS asks about masculine secondary sex characteristics (voice changes, facial hair) that are known to be the result of exposure to androgens. Consider two male adolescents, Jacob and Esau. They have equivalent testosterone levels at age 15, but Esau reports he can grow a beard and that his voice has finished changing, whereas Jacob reports that he has barely begun to grow hair on his face and that his voice is still cracking. One possibility is that the discrepancy between self-reports and hormonal measurement is due to bias or error in reporting, and the self-reports should be interpreted entirely in terms of an adolescent's subjective self-perceptions (Dorn, Dahl, Woodward, &

Biro, 2006). Another possibility, however, is that Esau really *is* a bearded bass and Jacob a hairless tenor and that these differences in secondary sex characteristics are an indication that Esau has been exposed to his currently high levels of testosterone for longer and/or that his body is more sensitive to the effects of androgens. Distinguishing between these alternatives is not possible using only single, cross-sectional "snapshots" of hormonal concentrations and would require repeated hormonal measurements over the course of pubertal development. The collection of such longitudinal data on pubertal and hormonal development and behavioral risk in large representative samples is an exciting prospect for future work.

Finally, it is important to mention that pubertal development was indexed by self-reports of pubertal development and not by a Tanner examination by a medical professional. In fact, we have advocated in previous reviews for using self-reports rather than Tanner staging in research requiring large numbers of participants (Harden, Kretsch, Moore, & Mendle, 2014): Compared to self-report measures, Tanner staging is not only more intrusive and more expensive (and thus prohibitive for large-sample studies), but it also shows poor interrater reliability in large-scale studies (Albert, Hunsberger, & Biro, 1997), fails to capture individual differences in the development of secondary sex characteristics in mid- to late adolescence, when nearly all participants are at Tanner Stage 5 (Susman et al., 2010), shows worse convergence with measured hormonal concentrations than do self-reports (Shirtcliff, Dahl, & Pollack, 2009), and is frequently a poorer predictor of behavioral and psychological outcomes than are self-report measures (Harden, Kretsch, Moore, & Mendle, 2014; Harden, Mendle, & Kretsch, 2012).

Need for Further Research on Estradiol

We observed a negative association between estradiol levels and self-reported sensation seeking. When interpreting the negative correlation with estradiol found here, it is important to keep in mind an important limitation of this study—that hormones were measured on only one occasion. There are (at least) five temporal dimensions that could contribute to observed variation in sex hormones measured on a single occasion: (1) acute reactivity to stress and immediate environmental circumstances; (2) diurnal rhythm, such that sex hormone concentrations are typically higher in the morning and lower in the evening; (3) menstrual cycle–related variation in females; (4) developmental change due to advancing puberty; and (5) temporally stable individual differences in average circulating levels.

We attempted to minimize menstrual cycle–related variation (Temporal Dimension 3) by collecting data within the first 2 weeks of the menstrual cycle and controlling for menstrual cycle phase. Additionally, the estradiol–sensation-seeking association could be equated in females and males, for whom menstrual variation is obviously not a concern. Nevertheless, menstrual cycle variation remains a potential consideration that could be eliminated with more repeated hormonal sampling.

Although sex hormone levels measured in adolescent samples are typically presumed to index developmental change due to advancing puberty (Temporal Dimension 4), we found minimal correspondence between gonadal hormone concentrations and self-reported pubertal development in female adolescents. The poor correspondence is likely due to the age range of the current sample. By age 15 (the average age of participants in the full sample), most female adolescents are postmenarcheal; they have finished, or nearly finished, the breast and pubic hair changes captured by the Tanner stages (Susman et al., 2010); and levels of estradiol and testosterone have been rising for several years. We would expect stronger correspondence between estradiol and pubertal development in samples of younger female adolescents, in which there is more variation in the early stages of pubertal development. For instance, in a sample of 11- to 13-year-old girls, salivary estradiol was correlated with both PDS scores ($r = .53$) and age ($r = .44$; Op de Macks et al., 2016). In contrast, within the current study the correlations among age, self-reported pubertal development, and hormones were substantially stronger in male adolescents, who began puberty later (on average) and thus had greater variability in pubertal development within the age range sampled. More generally, the pattern of associations examined in the current study should be investigated in a sample spanning late childhood to early adolescence, because relations might differ earlier in the process of pubertal development (e.g., Hawes et al., 2017).

With that in mind, it is intriguing to consider that estradiol levels measured in the current study likely tapped, at least in part, temporally stable individual differences in hormone levels and that estradiol was inversely associated with sensation seeking (but not reward sensitivity). This finding is consistent with results of one line of animal research, which found that estradiol treatment of ovariectomized animals decreased novelty seeking and increased fearful behavior in mice (Morgan & Pfaff, 2001). One direction for future research will be to test the hypothesis that this negative effect of estradiol on sensation seeking is mediated through its effects on fear and/or anxiety.

Conclusions

This study examined developmental differences in sensation seeking and reward sensitivity due to age, puberty, and gonadal hormones. Consistent with our hypotheses, self-reported pubertal development was associated with greater reward sensitivity in both sexes. However, inconsistent with our hypotheses, this effect was not statistically mediated by measured testosterone or estradiol. In male adolescents, pubertal development was also associated with self-reported sensation seeking, an effect that was partially mediated via higher testosterone levels. We found evidence that reward sensitivity is more closely yoked to sensation seeking in male adolescents than in female adolescents and found evidence for negative associations between estradiol and sensation seeking. These results suggest new avenues for future research on the sex-specific developmental mechanisms that underlie adolescents' elevated propensity for risk-taking behavior.

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