Delay and Probability Discounting: a Longitudinal Study of Neural, Cognitive, and Emotional Processes Contributing to Adolescent Development

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Dedication

To my parents, Les and Dot Olson, who taught me about the delayed rewards of higher education and the immediate rewards of interpersonal connection.

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Abstract

Adolescence is a time of rapid change in neurobehavioral characteristics, including emotional functioning, cognitive performance, and brain structure and function. The development of decision-making was examined in a group of adolescents (age 9-23) followed longitudinally over a two-year period. Delay and probability discounting tasks were used to assess decision-making. Change in discounting was examined in relation to baseline intelligence, working memory performance, personality factors, and internalizing and externalizing behaviors. In addition, contributions of brain structural features to the development of discounting behavior were analyzed. These included cortical thickness, white matter volume, subcortical volume, and diffusion tensor imaging (DTI) measures including fractional anisotropy and mean diffusivity. Delay discounting, but not probability discounting, showed significant maturation within individuals. Greater than expected maturation in delay discounting was seen in individuals with lower internalizing and externalizing psychopathology and higher positive emotionality. Brain structural factors predisposing toward greater than expected maturation included lower right frontal cortical thickness, larger cinguate and cuneate white matter volumes, larger hippocampal volumes, thicker parahippocampal gyrus cortical thickness, lower fractional anisotropy in the right temporal-parietal-occipital junction, and lower fractional anisotropy in the right amygdala/ pallidum/ hippocampus. Behavioral factors predisposing toward greater than expected change in probability discounting included female sex (for younger participants) and working memory performance (for males). Brain structural factors predisposing toward greater than expected change included

cingulate white matter volume and higher mean diffusivity in the left parieto-occipital area. Findings are discussed in terms of implications for development of decision-making processes during adolescence.

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1. Background

1.1 Overview

Adolescence is a time of significant change in social, emotional, cognitive, and neurobiological development, beginning with the physical and biological changes that mark the onset of puberty and ending when adult social roles and responsibilities are achieved (Dahl, 2004). Unfortunately, adolescence is also a period of increased vulnerability to harm, in part as a consequence of changes in decision-making (for a review, see Kelley, Schochet, & Landry, 2004); in fact, there is a 200% increase in morbidity and mortality rates from childhood to adolescence that is attributable to difficulties with emotional and behavioral control (Dahl, 2004). The purpose of the present work is to examine changes in decision-making behavior over the course of adolescence. Decisions involving rewards of various magnitudes available after different delays or with different likelihoods are studied in order to assess developmental changes. In this introduction, changes in behavior on delay and probability discounting tasks are examined in relation to aspects of cognition, behavior, personality, and brain development in order to identify factors associated with change in the discounting rate. First, basic information about delay and probability discounting is reviewed. Second, the adult discounting literature is examined in order to highlight relationships between discounting and aspects of psychopathology, personality, and cognition. Third, the child and adolescent discounting literature is reviewed. Fourth, the neuroimaging literature is reviewed in order to identify brain regions implicated in various aspects of discounting.

Fifth, changes in brain development during adolescence revealed by diffusion tensor imaging are reviewed. In this way, the introduction demonstrates that discounting is critically related to psychopathology and impulsive behavior and that discounting behavior is influenced by activity in brain regions that are rapidly developing over the course of adolescence. In order to examine the influence of demographic variables, cognitive performance, personality, internalizing/externalizing behaviors, and brain development (including white matter development) on the development of delay and probability discounting behavior during adolescence, participants age 9 to 23 were assessed longitudinally, with a two-year interval between sessions. Delay discounting showed significant maturation over a two-year period, but probability discounting did not. Both behavioral factors (internalizing/externalizing behavior and personality features) and brain structural factors (cortical thickness, white matter volume, subcortical volume, and diffusion tensor imaging measures) at time 1 were associated with greater than expected maturation in delay discounting from time 1 to time 2. Greater than expected change in probability discounting from time 1 to time 2 was associated with demographic factors (sex), cognitive performance, and brain structural factors (white matter volume and diffusion tensor imaging measures). Findings are discussed in terms of their implications for development of decision-making processes during adolescence, as well as for conceptualizing underlying processes contributing to behavior on discounting tasks.

1.2 Delay and Probability Discounting

Humans, like other animals, generally prefer to receive large rather than small rewards and to receive them immediately rather than after a delay. Unfortunately, there are many occasions during which the desire for the largest reward and the desire for the soonest reward may conflict. A choice is often faced between settling for a smaller sooner reward or waiting for a larger later reward. While waiting one additional hour for the larger reward may seem a reasonable tradeoff, waiting 10,000 hours for the larger reward may seem completely unreasonable. This intuition reflects the fact that the subjective value of a reward declines as the delay until its receipt increases, a process called delay discounting (Ainslie, 1975).

Individuals show considerable variability in the delay discounting rate, with some discounting delayed rewards sharply even at short delays and others showing hardly any discounting even at quite long delays. A number of paradigms have been developed to assess the delay discounting rate in humans, typically involving asking people to choose between a given amount of money immediately and a given amount of money after a specified delay (e.g., "Would you rather have $100 now, or $150 in 30 days?"). Varying the delay and one amount (typically of the immediate reward) allows for determination of an "indifference point," the value at which an individual is indifferent between the delayed and immediate reward. Indifference points can be plotted against time, and discounting can be quantified either by fitting an equation and using resulting variables as markers for the discounting rate or by considering the area under the discounting curve as a measure of the extent of discounting (Myerson, Green, & Warusawitharana, 2001). In

the latter case, a large area indicates less decline in subjective value over time (that is, less steep discounting).

A large body of literature indicates that delay discounting in both humans and other animals is best described by a hyperbolic rather than an exponential function (e.g. Bickel, Odum, & Madden, 1999; Green, Myerson, & McFadden, 1997; Kirby, 1997; Johnson & Bickel, 2002; Madden, Begotka, Raiff, & Kastern, 2003; Mazur, 1987; Myerson & Green, 1995; Rachlin, Raineri, & Cross, 1991; Reynolds & Schiffbauer, 2004; Richards, Zhang, Mitchell, & de Wit, 1999; Rodriguez & Logue, 1988; Vuchinich & Simpson, 1998). The hyperbolic function also accounts for preference reversals (Mazur, 1987). For instance, an individual might be asked whether she would prefer $100 in 1 year, or $150 in 1 year plus 30 days. She may choose to receive $150 in 1 year plus 30 days; waiting the extra 30 days seems like a trivial matter in exchange for another $50. However, 364 days later, when presented with the options of receiving $100 in 1 day or $150 in 31 days, she may well prefer to receive $100 the next day. In that case, her preference has reversed as she moved forward through time. Preference reversals are an instance of irrational choice, which is prevalent in human decision-making in contrast to economic models of human beings as rational actors (Kahneman & Tversky, 1979; Loewenstein & Prelec, 1992). (To some extent, delay discounting may be considered irrational even without preference reversals, since the declines in subjective value typically far exceed what would be reasonable given interest rates and the risk of future non-payment, etc.).

Probability discounting is a task that shares many features in common with delay discounting. In the probability discounting paradigm, preferences are assessed for smaller certain rewards versus larger uncertain rewards (e.g., "Would you rather have $100 now, or $150 with an 80% chance?"). Typically, risk-averse patterns of behavior are seen in humans on tasks assessing probability discounting of gains. The subjective value of a probabilistic gain is frequently lower than its expected value. For instance, a person may prefer to receive $300 for sure rather than $1000 with a 50% chance, even though the expected value of the latter option is $500. Again, indifference points can be calculated and plotted, in this case versus the odds against receiving the reward (rather than the delay to reward delivery). High discounting rates, or low areas under the discounting curve, reflect risk aversion, since subjective value declines rapidly with increasing odds against receiving the reward.

Several different explanations of possible relationships between delay and probability discounting have been proposed. Delay discounting has been described as a form of probability discounting; the delay to receiving the reward may be conceptualized as increasing the odds against reward delivery, because the likelihood of some event interfering with reward delivery increases with the length of the delay (Green & Myerson, 1996; Myerson, Green, Hanson, Holt, & Estle, 2003). Probability discounting also has been described as a form of delay discounting, since probabilistic rewards may be conceptualized as repeated gambles (Rachlin, Logue, Gibbon, & Frankel, 1986). For instance, when rewards are unlikely, it might take many more 'tries' (and thus a longer

time) to successfully receive them. Finally, both delay and probability discounting may reflect impulsivity (Green & Myerson, 2004). In this model, impulsive individuals are predicted to have high delay discounting rates (preferring smaller sooner rewards) and low probability discounting rates (as they are not risk-averse and may be in fact more prone to "chasing" risky gambles). However, delay and probability discounting rates tend to be uncorrelated or moderately positively correlated (Crean, de Wit, & Richards, 2000; Holt, Green, & Myerson, 2003; Mitchell, 1999; Ohmura, Takahashi, & Kitamura, 2005; Ohmura, Takahashi, Kitamura, & Wehr, 2006; Richards, Zhang, et al., 1999; Scheres et al., 2006) which does not support the latter model. In fact, while delay and probability discounting have some obvious common features, they have repeatedly been shown to have differential associations with a variety of features including psychopathology, demographic variables, cognitive performances, and personality measures, as discussed below.

1.3 Delay and Probability Discounting in Adults

1.3.1 Associations with psychopathology.

Discounting rates are associated with various forms of psychopathology, most notably substance use and other disorders of self-control. Below, the literature on delay discounting and psychopathology in adulthood is reviewed; the childhood and adolescent literature is reviewed in the following section.

Elevated delay discounting rates have been reported in adults with a variety of axis I and axis II diagnoses and subclinical syndromes, including ADHD hyperactive/ impulsive symptoms (Scheres, Lee, & Sumiya, 2008), schizophrenia and schizoaffective disorder diagnoses and negative symptoms (Heerey, Robinson, McMahon, & Gold, 2007), antisocial personality disorder features (Petry, 2002), impulse control disorders (Crean et al., 2000) including pathological gambling (Alessi & Petry, 2003; Dixon, Marley, & Jacobs, 2003; MacKillop, Anderson, Castelda, Mattson, & Donovick, 2006), social anxiety symptoms (Rounds, Beck, & Grant, 2007), and depressive symptoms (Yoon et al., 2007). Elevated delay discounting rates also have been reported in adults with other markers of possible impulsive behavior, including erotica users (Lawyer, 2008) and people who are obese (Weller, Cook, Avsar, & Cox; but see Epstein et al., 2003). In contrast, Takahashi et al. (2008) found lower overall delay discounting rates in individuals with unipolar or bipolar depression, although they had steeper rates than controls at the shortest delays. Lower rates of probability discounting are seen in gamblers (Holt et al., 2003). There have been few other studies of probability discounting and psychopathology, though those that have examined relationships have found no group differences (e.g. Crean et al., 2000).

Thus, there is evidence that altered delay discounting rates can be seen in individuals with either externalizing or internalizing forms of psychopathology; this is particularly interesting because internalizing syndromes are not necessarily characterized by impulsive behavior. The effect of psychopathology has been characterized as dose-

dependent, with increasing elevation in the delay discounting rate seen with increasing symptom counts (Alessi & Petry, 2003; Petry, 2001b; Petry, 2002) or increasing numbers of comorbid diagnoses (Bobova, Finn, Rickert, & Lucas, 2009; Petry & Casarella, 1999; Petry, 2001b; but see Dom, De Wilde, Hulstijn, van den Brink, & Sabbe, 2006). Though there is evidence that various forms of axis I and axis II psychopathology as well as acquired brain injuries (Dixon et al., 2005; McHugh & Wood, 2008) are associated with alterations in delay discounting, little information is available about the nature of the relationship between the alteration in the discounting rate and these forms of psychopathology. For instance, disturbance in valuation of future rewards might change as a result of the experience of having a psychiatric condition, or it might contribute to the development of the pathology.

The most robust associations between discounting and psychopathology have been reported for substance use disorders. This large body of literature has also begun to address possible mechanisms of association between psychopathology and discounting. Increased delay discounting rates are seen in chronic users of nicotine (Baker, Johnson, & Bickel, 2003; Bickel et al., 1999; Heyman & Gibb, 2006; Johnson, Bickel, & Baker, 2007; Jones, Landes, Yi, & Bickel, 2009; Ohmura et al., 2005; but see Petry, 2001b; Kollins, 2003), alcohol (Bjork, Hommer, Grant, & Danube, 2004; Dom, D'haene, Hulstijn, & Sabbe, 2006; Mitchell, Fields, D'Esposito, & Boettinger, 2005; Mitchell, Tavares, Fields, D'Esposito, & Boettinger, 2007; Petry, 2001a; but see MacKillop, Mattson, MacKillop, Castelda, & Donovick, 2007), cocaine (Bornovalova, Daughters,

Hernandez, Richards, & Lejuez, 2005; Coffey, Gudleski, Saladin, & Brady, 2003; Heil, Johnson, Higgins, & Bickel, 2006), amphetamines (Hoffman et al., 2006; Monterosso et al., 2007), opiates (Kirby, Petry, & Bickel, 1999; Madden, Petry, Badger, & Bickel, 1997), heroin (Kirby & Petry, 2004), and mixed substances (e.g. heroin or cocaine: Petry, 2002; Petry, 2004; Petry & Casarella, 1999). In chronic users, a dose-dependent effect of substance use on the discounting rate often is identified (Epstein et al., 2003; Field, Christiansen, Cole, & Goudie, 2007; Johnson et al., 2007; Mitchell et al., 2005; Ohmura et al., 2005; Reynolds, 2004; Vuchinich & Simpson, 1998; but see Sweitzer, Donny, Dierker, Flory, & Manuck, 2008), and greater disturbance in discounting is seen in individuals with poor prognostic markers such as early onset use (Dom et al., 2006; Kollins, 2003) or needle-sharing (Odum, Madden, Badger, & Bickel, 2000). Chronic substance users with comorbid conditions also affecting the discounting rate show a cumulative effect, with greater increases in delay discounting rates than are seen in chronic users without comorbidities (Bobova et al., 2009, Petry & Casarella, 1999; Petry, 2001b; but see Dom, De Wilde, et al., 2006).

For many substances, acute substance deprivation in chronic users changes the delay discounting rate (Giordano et al., 2002; Kirby & Petry, 2004), though this effect is not commonly seen for nicotine (e.g. Dallery & Raiff, 2007; Field, Rush, Cole, & Goudie, 2007; Mitchell, 2004; but see Field, Santarcangelo, Sumnall, Goudie, & Cole, 2006). Chronic users with lower delay discounting rates are more motivated to quit (Audrain-McGovern, Rodriguez, Epstein, Rodgers, et al., 2009) and are more likely to successfully

quit (Dallery & Raiff, 2007; Yoon et al., 2007; Yoon, Higgins, Bradstreet, Badger, & Thomas, 2009). Some studies suggest the possibility that quitting nicotine may also 'normalize' the delay discounting rate (Bickel et al., 1999; Sweitzer et al., 2008), though it is unclear whether this is because successful quitters are a self-selected population with lower discounting rates at baseline. For other substances, delay discounting rates remain elevated when substance-dependent individuals are abstinent (Heil et al., 2006; Hoffman et al., 2008; Passetti, Clark, Mehta, Joyce, & King, 2008). Finally, few studies have examined the relationship between probability discounting and substance use; most that have done so have found no association between the two (Ohmura et al., 2005; Reynolds, Patak, Schroff, Penfold, et al., 2007; but see Reynolds, Karraker, Horn, & Richards, 2003).

Although there are a few studies that provide some insight, the nature of the causal relationship(s) between psychopathology and discounting is not well understood. Several possibilities exist: individuals with high (delay) discounting rates may be more susceptible to developing various forms of psychopathology, the psychopathological experience may lead to alteration in the discounting rate, and/or discounting and psychopathology could also be linked through some third factor (such as a common genetic basis, early experiences, or general reactivity to rewards; Perry & Carroll, 2008). Although prospective studies in humans to address the first point are lacking, the animal literature suggests that high baseline delay discounting rates precede and predict drug

self-administration, suggesting that the delay discounting rate may serve as a marker for susceptibility to drug addiction (Anker, Perry, Gliddon, & Carroll, 2009).

1.3.2 Associations with demographic variables.

Most delay discounting studies have found no changes in the discounting rate with increasing age in adulthood (Baker et al., 2003; Bornovalova et al., 2005; Crean et al., 2000; de Wit, Flory, Acheson, McCloskey, & Manuck, 2007; Epstein et al., 2003; Green, Myerson, Lichtman, Rosen, & Fry, 1996; Kirby & Petry, 2004; Kirby et al., 1999; Ohmura et al., 2005; Petry, 2001a; Petry, 2001b; Reynolds et al., 2003; Sweitzer et al., 2008; Takahashi et al., 2008). For instance, Green et al. (1996) found no age-based differences between younger adults (mean age in the 30s) and older adults (mean age in the 70s), suggesting no change in discounting during adulthood. However, other studies have reported decreases in the discounting rate with increasing age (Bjork et al., 2004; Dom, D'haene, et al., 2006; Green, Fry, & Myerson, 1994; Yoon et al., 2007). This finding has a parallel in the rat literature: unlike young adult rats, older adult rats do not switch to smaller sooner rewards at increasing delays and instead prefer larger later rewards at any delay (Simon et al., 2008). Probability discounting has been studied less frequently but seems to be unrelated to age in adulthood (Ohmura et al., 2005; Reynolds et al., 2003).

Most studies have found no sex differences in delay discounting (Acheson, Richards, & de Wit, 2007; Ballard & Knutson, 2009; Bornovalova et al., 2005; de Wit, Enggasser, &

Richards, 2002; de Wit et al., 2007; Dom, D'haene, et al., 2006; Epstein et al., 2003; Heyman & Gibb, 2006; Kirby & Petry, 2004; Kollins, 2003; Lane, Cherek, Pietras, & Tcheremissine, 2003; McDonald, Schleifer, Richards, & de Wit, 2003; Mitchell et al., 2005; Ohmura et al., 2005; Reynolds et al., 2003; Reynolds, Richards, Dassinger, & de Wit, 2004; Reynolds, Patak, & Shroff, 2007; Reynolds, Patak, Schroff, Penfold, et al., 2007; Reynolds, Richards, & de Wit, 2006; Sweitzer et al., 2008; Takahashi et al., 2008; Vuchinich & Simpson, 1998; Wilson & Daly, 2006). While some studies have found steeper rates of delay discounting in women (Reynolds, Ortengren, Richards, & de Wit, 2006; Weller et al., 2008), men delay discounted more steeply in a study by Bobova et al. (2009). Most studies also find no sex differences in probability discounting (Acheson et al., 2007; de Wit et al., 2002; McDonald et al., 2003; Ohmura et al., 2005; Reynolds et al., 2003; Reynolds et al., 2007b).

Elevated delay discounting rates also have been noted in participants with lower educational levels (de Wit et al., 2007; Dom, D'haene, et al., 2006; Jaroni, Wright, Lerman, & Epstein, 2004; Yoon et al., 2007) or lower socioeconomic status (de Wit et al., 2007; Green et al., 1996). Cross-cultural differences also have been noted in both delay and probability discounting (Du, Green, & Myerson, 2002).

1.3.3 Associations with personality/self-report measures.

Delay discounting has been examined with respect to broad personality factors. Miller, Lynam, and Jones (2008) examined relationships between personality (assessed using the

NEO-PI-R) and delay discounting in undergraduates. Higher indifference points were associated with the Agreeableness factor and its components (Trust, Straightforwardness, Altruism, Compliance, Modesty, and Tender-Mindedness). Delay discounting was not associated with the Conscientiousness factor (except the Deliberation component). On other questionnaire measures, delay discounting was associated with a count of aggressive behaviors but not with variety of substance use, variety of antisocial behaviors, or engaging in riskier sex. Reynolds, Richards, Horn, and Karraker (2004) also examined delay discounting in relation to the Big 5 personality factors but found no relationship.

Higher delay discounting has been reported in association with high scores on a number of questionnaire-based measures of impulsivity, including various impulsivity measures on scales developed by Eysenck (Alessi & Petry, 2003; Eisenberg, Campbell, MacKillop, Lum, & Wilson, 2007; Kirby et al., 1999; Kirby & Petry, 2004; Madden et al., 1997; Petry, 2001b; Petry, 2002) (but see Coffey et al., 2003; Crean et al., 2000; MacKillop et al., 2007; Reynolds, Richards, Horn, et al., 2004; Vuchinich & Simpson, 1998). High probability discounting has been associated with high extraversion (Richards et al., 1999) and with low impulsivity (Mitchell, 1999) on the Eysenck measures (but see Crean et al., 2000).

High scores on the Barratt Impulsiveness Scale (BIS) also have been reported in steep delay discounters (Heyman & Gibb, 2006; Krishnan-Sarin et al., 2007; McHugh &

Wood, 2008; Sweitzer et al., 2008) (but see Bjork et al., 2004; Bjork, Momenan, & Hommer, 2009; Coffey et al., 2003; Dom, D'haene, et al., 2006; Dom, De Wilde, et al., 2006; Dougherty, Marsh-Richard, Hatzis, Nouvion, & Mathias, 2008; Eisenberg et al., 2007; Fellows & Farah, 2005; Reynolds, Ortengren, et al., 2006). Steeper delay discounters have been reported to have higher scores on the non-planning subscale (de Wit et al., 2007; Kirby et al., 1999; Kirby & Petry, 2004), the cognitive subscale (de Wit et al., 2007; Kirby et al., 1999), and the motor subscale (Kirby & Petry, 2004; but see Kirby et al., 1999). Lower delay discounting rates have been reported for individuals with high scores on the BIS cognitive complexity subscale (Reynolds, Richards, Dassinger, et al., 2004) and self control subscale (Reynolds, Richards, & de Wit, 2006).

Higher delay discounting rates also have been reported in association with higher scores on other aspects of externalizing, including on the Buss-Perry Aggression Questionnaire and the Life History of Aggression questionnaire (Bjork et al., 2004); and on the Thrill & Adventure Seeking, Experience Seeking, and Disinhibition subscales of the Zuckerman Sensation Seeking Scale (SSS: Richards, Zhang, et al., 1999; Vuchinich & Simpson, 1998). Perales, Verdejo-García, Moya, Lozano, and Pérez-García (2009) found no difference in delay discounting between women with high versus low trait impulsivity on the UPPS-P impulsivity scale. Richards, Zhang, et al. (1999) also found that high scorers on the SSS Disinhibition subscale had higher probability discounting rates.

1.3.4 Associations with cognition.

Higher overall intellectual functioning is associated with lower delay discounting rates (Baker et al., 2003; de Wit et al., 2007; Kirby & Petry, 2004) (but see Crean et al., 2000; Heerey et al., 2007; Kirby et al., 1999; Monterosso, Ehrman, Napier, O'Brien, & Childress, 2001; Petry, 2001a; Petry & Casarella, 1999; Weller et al., 2008); delay discounting rates also are lower in those with superior academic performance (Kirby, Winston, & Santiesteban, 2005; Silva & Gross, 2004). Discounting rates are also higher in those with lower years of education and in those whose parents had fewer years of education (even after controlling for the subject's educational background) (Sweitzer et al., 2008).

Behavioral inhibition refers to the ability to withhold or suppress a prepotent motor response. Although both delay discounting and behavioral inhibition are thought to be measures of different aspects of impulsivity, delay discounting typically has been found not to be related to performance on measures of behavioral inhibition, including Go-NoGo tasks and the Stop Task (Crean et al., 2000; de Wit et al., 2002; McDonald et al., 2003; Perales et al., 2009; Reynolds, Ortengren, et al., 2006). Probability discounting performance also is not associated with performance on these tasks (Crean et al., 2000; de Wit et al., 2002; McDonald et al., 2003). In contrast, Krishnan-Sarin et al. (2007) found that steep delay discounters made more errors on the Conners' Continuous Performance Test. In addition, Ortner, MacDonald, and Olmstead (2003) found that in intoxicated subjects, individuals with a high number of Go-NoGo commission errors also were steeper delay discounters.

Discounting performance also has been examined in relation to other decision-making and risk-taking tasks. One commonly used risk and decision-making task is the Balloon Analogue Risk Test (BART), a measure of decision-making and reward-related risk taking in which subjects earn money by inflating a computerized balloon; on each turn they have the option of 'banking' their earnings or continuing to inflate the balloon to earn more money; but if the balloon pops, all earnings are lost. Although Reynolds, Ortengren, et al. (2006) found that delay discounting loaded with the BART in a factor analysis into a component reflecting impulsive decision-making, others have reported no relationship between BART scores and delay discounting rates (Bornovalova et al., 2005; Reynolds, Richards, & de Wit, 2006).

Delay discounting performance also has been examined with respect to gambling tasks and has been found to be significantly associated with task performance after controlling for intelligence on the Cambridge Gamble Task and the Iowa Gambling Task (Monterosso et al., 2001). Individuals with high discounting rates also earned fewer points on a simulated group foraging task (Critchfield & Atteberry, 2003).

Delay discounting also has been examined in relation to working memory task performance. Delay discounting rates become steeper under conditions of increased working memory load (Hinson, Jameson, & Whitney, 2003). Although Shamosh et al. (2008) found no relationship between working memory and delay discounting after

controlling for the effects of intelligence, they found that prefrontal activity during a working memory task mediated the relationship between intelligence and delay discounting. Two studies have found no relationship between working memory and delay discounting performance (Heerey et al., 2007; McDonald et al., 2003).

There have been few other positive cognitive findings; no relationship has been found between delay discounting and performance on measures of simple auditory or visual attention (de Wit et al., 2002; McDonald et al., 2003) or visual-motor processing speed (de Wit et al., 2002; Heerey et al., 2007; McDonald et al., 2003). Probability discounting also is not associated with performance on measures of simple auditory or visual attention or visual-motor processing speed (McDonald et al., 2003). de Wit et al. (2002) found that delay and probability discounting are not associated with performance on a time estimation task.

Several studies have found associations between discounting and other cognitive measures in cognitively impaired subgroups only. For instance, poor performance on verbal learning tasks is not associated with elevated rates of delay discounting in healthy adults (de Wit et al., 2002; Heerey et al., 2007; McDonald et al., 2003), but is associated with delay discounting in individuals with schizophrenia/ schizoaffective disorder (Heerey et al., 2007) and methamphetamine dependence (Hoffman et al., 2006). Hoffman et al. (2006) also found that high delay discounting rates were associated with slower nondominant hand performance on the Grooved Pegboard test in methamphetamine-

dependent subjects, but there was no relationship between delay discounting and psychomotor performance in controls. Probability discounting is not associated with HVLT performance in healthy adults (McDonald et al., 2003), though it has not been studied in cognitively impaired populations.

1.4 Delay and Probability Discounting in Children and Adolescents

1.4.1 Associations with psychopathology.

There is a relatively small body of literature examining relationships between discounting and psychopathology (including substance use) in children and adolescents. The research that has been conducted has generally produced patterns consistent with those seen in the adult literature. Delay discounting rates are steeper in adolescents with attention-deficit/ hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD) (Barkley, Edwards, Laneri, Fletcher, & Metevia, 2001; but see Scheres et al., 2006 and Wilson & Daly, 2006). Adolescent substance use is associated with higher delay discounting rates, for nicotine (Audrain-McGovern, Rodriguez, Tercyak, Epstein, & Goldman, 2004; Reynolds, Patak, Schroff, Penfold, et al., 2007; but see Reynolds et al., 2003) and alcohol (Field, Christiansen, et al., 2007). Adolescent children of mothers who use substances also have higher delay discounting rates (Reynolds, Leraas, Collins, & Melanko, 2009). Delay discounting rates at baseline predict progressing severity in adolescent smoking patterns (Audrain-McGovern, Rodriguez, Epstein, Cuevas, et al., 2009), and delay discounting rates are lower in adolescent smokers than in adult smokers (Reynolds, 2004). As in adults, adolescents with lower delay discounting rates have greater success

in quitting (Krishnan-Sarin et al., 2007). Differences in discounting rates between adolescent nonsmokers and smokers have been shown to be attributable to differences in perceptions of the certainty of actually receiving the delayed reward (Reynolds, Patak, & Schroff, 2007). There have been few reported examinations of probability discounting in children and adolescents, though one study found that children (age 7 to 10) with ADHD were more likely to select large but unlikely rewards than same-aged peers (Drechsler, Rizzo, & Steinhausen, 2009). Reynolds et al. (2003) found that high-schoolers who had tried smoking had higher probability discounting rates than current smokers or those who had never tried smoking; that is, they were more risk-averse (but see Reynolds, Patak, Schroff, Penfold, et al., 2007).

1.4.2 Associations with demographic variables.

A number of studies have found changes in the delay discounting rate with age over the course of late childhood and adolescence. Sheres et al. (2006) found that children (age 6 to 11 years) have steeper delay discounting rates than adolescents (age 12 to 17 years). Green et al. (1994) found that sixth grade students have steeper delay discounting rates than college students. Steinberg et al. (2009) examined delay discounting cross-sectionally in a large sample of participants age 10-30 years ($N = 929$). Participants age 13 and younger had higher delay discounting rates than those aged 16 and older; this pattern did not vary based on sex or ethnicity. Olson, Hooper, Collins, and Luciana (2007) found lower delay discounting rates with increasing age in a sample of 9- to 23-year-olds. Other studies have found no association between delay discounting and age

(Audrain-McGovern, Rodriguez, Epstein, Cuevas, et al., 2009; Lamm, Zelazo, & Lewis, 2006; Reynolds et al., 2003). Patak and Reynolds (2007) demonstrated that, in adolescents aged 14-16 years, delay discounting rates were correlated with the extent to which participants endorsed uncertainty about receiving the delayed rewards. Three studies found no relationship between probability discounting and age (Drechsler et al., 2009; Olson et al., 2007; Sheres et al., 2006).

In terms of other demographic variables, most studies of adolescents report no gender differences in delay discounting (Olson et al., 2007; Steinberg et al., 2009; Reynolds et al., 2003; Wilson & Daly, 2006); one study that did report a difference failed to specify its direction (Audrain-McGovern et al., 2004). Audrain-McGovern, Rodriguez, Epstein, Cuevas, et al. (2009) reported that females had lower delay discounting rates at baseline. Audrain-McGovern et al. (2004) also noted a racial difference in discounting in their sample, though again this was not further characterized. Steinberg et al. (2009) found no differences in delay discounting based on socioeconomic status.

1.4.3 Associations with personality/self-report measures.

Few studies have examined relationships between questionnaire-based measures of personality or behavior and discounting in adolescents. Steinberg et al. (2009) found that delay discounting was related to scores on a measure of future orientation, including subscales assessing time perspective and anticipation of future consequences. Controlling for BIS-11 impulsivity scores did not account for the influence of age on delay

discounting, but entering BIS-11 scores together with future orientation did, which the authors interpret as suggesting that adolescents perform differently on DD tasks because of differences in future orientation not related to impulsivity. Olson et al. (2007) found no relationship between delay discounting and self-reported internalizing or externalizing behavior. However, individuals with higher self-reported externalizing symptoms showed less steep probability discounting. Similarly, Drechsler et al. (2009) found relationships between self-reported impulsivity and attention problems and probability discounting, although these were evident only in a subsample with ADHD and not in healthy controls.

1.4.4 Associations with cognition.

Higher IQ and higher academic performance are associated with lower delay discounting rates in adolescents (Audrain-McGovern, Rodriguez, Epstein, Cuevas, et al., 2009; Olson et al., 2007; Steinberg et al., 2009; but see Barkley et al., 2001); the effect may be specific to verbal IQ (Olson et al., 2007). Steinberg et al. (2009) found that, after controlling for IQ, there was little relationship between delay discounting rates and other executive functioning measures (including the Iowa Gambling Task and the Tower of London). Lamm et al. (2006) also found no relationship between delay discounting rates and performance on measures of executive functioning, including performance on the Iowa Gambling Task, Stroop task, digit span backwards, or Go/NoGo. Olson et al. (2007) found that there was no relationship between delay discounting and Iowa Gambling Task performance or Go-NoGo performance after controlling for the effects of IQ and age. Two studies have found no relationship between probability discounting and intelligence

(Drechsler et al., 2009; Olson et al., 2007). One study found that more conservative behavior on a probability discounting task was associated with better performance on measures of response consistency on a vigilance task and set-shifting task performance, although no attempt was made to control for intelligence or other factors that might explain these associations (Dreschsler et al., 2009).

1.5 Summary of the Behavioral Literature

To briefly summarize the behavioral literature, in both adolescents and adults delay discounting rates are generally found to be higher in individuals with psychopathology, with the most robust findings seen for substance use disorders. Delay discounting rates typically are found to decrease from adolescence to adulthood; they subsequently either remain stable or continue to decrease with increasing age. Elevated delay discounting rates are seen in individuals with lower levels of educational attainment and with lower socioeconomic status. High delay discounting rates also are associated with lower intelligence. Relationships between delay discounting and measures of executive functioning and other measures of risk-taking are occasionally reported, but findings have been somewhat inconsistent. High delay discounting rates have been found in association with questionnaire-based personality measures (e.g., lower agreeableness) and measures of impulsivity and externalizing behavior. Sex differences typically are not seen for delay or probability discounting. Probability discounting rates have been examined much less frequently. The association between probability discounting and substance use does not appear to be robust, and probability discounting does not typically change with age. High

probability discounting rates have been associated with questionnaire-based measures of high extraversion, disinhibition, and externalizing behavior. There have been few reported significant associations between probability discounting and any cognitive measure. Thus, delay and probability discounting have different sets of correlates, with delay discounting showing robust relationships with age and substance use and consistent relationships with intelligence. Probability discounting does not appear to be related to age, substance use, or cognitive performance but may be related to personality.

In subsequent sections, the imaging literature with respect to delay and probability discounting and with respect to adolescent brain development is reviewed in order to consider the possible neurobiological underpinnings of changes in discounting during adolescence.

1.6 Delay Discounting—Functional MRI in Adults

Functional MRI studies of delay discounting have focused exclusively on adults, and primarily on healthy college-aged adults without any known cognitive, neurological, or psychological impairment. In addition to six studies using a delay discounting paradigm in the fMRI scanner with this population (Ballard & Knutson, 2009; Bickel, Pitcock, Yi, & Angtuaco, 2009; Kable & Glimcher, 2007; McClure, Laibson, Loewenstein, & Cohen, 2004; McClure, Ericson, Laibson, Loewenstein, & Cohen, 2007; Wittman, Leland, & Paulus, 2007), there have been three studies examining fMRI activity on a *different* task in relation to (behavioral only) delay discounting data (Hariri et al., 2006; Hariri et al.,

2008, Shamosh et al., 2008). There also have been two studies using a delay discounting fMRI paradigm in individuals addicted to methamphetamine (Hoffman et al., 2008; Monterosso et al., 2007). Findings are summarized in table 1 and are discussed below.

Based on the healthy adult fMRI delay discounting literature, three groups of researchers have offered competing accounts of the neural systems underlying delay discounting. McClure et al. (2004, 2007) have argued for a two-system account, claiming that there is a set of predominantly limbic regions (including the OFC, medial PFC, subgenual cingulate, anterior cingulate, posterior cingulate, ventral striatum, posterior hippocampus, and precuneus) that are preferentially activated during choices involving an immediate reward option, and a set of higher reasoning regions that are activated during all reward-based decisions, including the visual cortex, premotor area, supplementary motor area, intraparietal cortex, DLPFC, VLPFC, and lateral OFC. In their model, the limbic regions are dubbed the "β regressor" regions, and the higher reasoning regions are called the "δ regressor" regions. When there is greater activity in the δ regressor regions than in the β regressor regions, the model predicts that delayed rewards will be selected.

Boettiger et al. (2007) mischaracterized their two-system account in which one system represents more proximate outcomes and one values delayed outcomes as being fundamentally different from McClure et al.'s model, which they described as being based primarily on a hot/ cool (limbic versus higher reasoning) distinction. In fact, McClure et al.'s model, like their model, is based primarily on distinction between

regions that value immediate rewards versus regions that do not. Boettiger et al. found that greater subject bias toward selecting immediate rewards was positively correlated with greater activation in the left dorsal PFC (superior frontal gyrus), right posterior parietal areas, right parahippocampal gyrus (near the amygdala), right middle and inferior temporal areas, and right cerebellum, while greater subject bias toward selecting delayed rewards was positively correlated with greater activation in orbitofrontal cortex.

In contrast, Kable and Glimcher (2007) argued that a single system involving the ventral striatum, medial PFC and posterior cingulate cortex tracks the subjective value of rewards, with each region individually integrating information about magnitude and delay. Because activation in these regions varies when the subjective value of the delayed reward changes, they argued that their data falsifies McClure et al.'s hypothesis that these regions primarily value immediate rewards.

Finally, Ballard and Knutson (2009) argued that McClure et al. have incorrectly characterized the nucleus accumbens and medial PFC as primarily valuing immediate rewards, since changes in the magnitude of the delayed reward also affect activity in these regions. However, they argue that Kable and Glimcher's account also is incomplete, since the nucleus accumbens, medial PFC, and posterior cingulate cortex are primarily sensitive to changes in reward magnitude (but not delay), and the DLPFC, temporal-parietal junction, and posterior parietal cortex are primarily sensitive to changes in reward delay (but not magnitude). Thus, they argued for a two-system approach in

which different systems are responding to different delayed reward attributes (magnitude versus delay), as opposed to McClure et al.'s two system approach in which one system values immediate rewards and one values delayed rewards. In addition, they identified one region (the inferior frontal gyrus) in which magnitude and delay information were integrated (i.e., the region was sensitive to the interaction of magnitude and delay).

Rather than focusing on which brain regions encode which aspects of the information involved in the delay discounting question, Wittman et al. (2007) identified regions that were more active when the individual was choosing the delayed reward. These included the bilateral posterior insular cortex, posterior cingulate, superior temporal gyrus, angular gyrus, inferior parietal lobule, and cuneus. There were no areas that were more active when individuals chose immediate rewards. The involvement of posterior (parietal and temporal) areas in the selection of delayed rewards is consistent with McClure et al.'s (2004, 2007) and with Ballard and Knutson's (2009) description of two-system models in which lateral and posterior regions tend to support the selection of delayed rewards. The involvement of the posterior cingulate in selecting delayed rewards in the Wittman et al. (2007) study, however, is somewhat more consistent with Kable & Glimcher's (2007) account; under the McClure et al. and Ballard & Knutson models, the posterior cingulate is expected to most heavily value reward magnitude.

Within individuals, there are several regions that show greater activity when choices are being made close to the indifference point (often characterized as 'difficult' or 'hard'

choices), including the OFC, DLPFC, VLPFC, supplementary motor area, insula, anterior cingulate cortex, posterior cingulate cortex, cuneus, and portions of the parietal lobe (Hoffman et al., 2008; McClure et al., 2004; Monterosso et al., 2007), suggesting that input from these regions may be particularly influential in 'tipping the balance' between the selection of immediate and delayed rewards when their subjective values are nearly equivalent. Wittman et al. (2007) found that steeper discounting was associated with greater differences in activation for short versus long delays in the posterior cingulate, lingual gyrus, cuneus, superior temporal gyrus, caudate, and inferior frontal gyrus, suggesting that individual differences in sensitivity to delays that affect decision-making behavior are reflected in these regions. In contrast, shallower discounting was associated with greater differences in activation for short versus long delays in the inferior frontal gyrus. Bickel et al. (2009) determined that a common set of regions was active during delay discounting for hypothetical gains, hypothetical losses, and real gains (where one choice was actually selected for payment), including the left and right lateral PFC, premotor area and supplementary motor area, posterior cingulate, bilateral insula, precuneus, visual cortex, striatum, and left and right parietal areas. Activity during the DD task in the DLPFC bilaterally, cerebellum, and cuneus was positively correlated with the discounting rate on a version of the task administered several weeks earlier, a finding which is somewhat unexpected given that DLPFC is typically found to be involved in promoting the selection of larger later rewards. The authors point out that this finding may reflect increased difficulty of responding to the task trials for those with higher discounting rates.

Data from fMRI investigations of brain activity during other reward-related tasks in relation to behavioral delay discounting data provide support for the involvement of prefrontal and striatal areas in delay discounting. Hariri et al. (2006) found that steep delay discounters showed greater ventral striatum and medial PFC activation than shallow delay discounters on a different reward-related decision-making task, particularly in response to positive (versus negative) feedback. In contrast, steep delay discounters showed less lateral OFC and DLPFC activation than shallow delay discounters. Prefrontal activation during a working-memory task also is associated with delay discounting behavior; individuals with high levels of activation during a 3-back task in the middle frontal gyrus/ anterior PFC had lower rates of delay discounting, while those with high levels of activation in the anterior cingulate and the right temporal lobe had high rates of delay discounting (Shamosh et al., 2008).

Finally, Bjork et al. (2009) conducted a structural MRI study examining the relationship between brain region volume and delay discounting. Inferolateral prefrontal cortex and DLPFC volumes were negatively correlated with delay discounting rates after controlling for age and brain volume. Delay discounting rate was not significantly associated with with medial frontal cortex or orbitolateral frontal cortex volumes, total cerebral brain volume, or intracranial volume.

Additionally, several neuroimaging studies have examined how individual differences in cognitive, neurological, or psychiatric functioning affect imaging findings pertaining to delay discounting. There is some evidence that the strength of the relationship between ventral striatal activity on other reward-based tasks and delay discounting may vary by genotype, with individuals with an allele (related to endocannabinoid signaling) conferring vulnerability to impulsivity showing a stronger association between ventral striatal activity and discounting (Hariri et al., 2008). Hoffman et al. (2008) found that individuals in treatment for methamphetamine dependence showed greater activation on catch trials (e.g. $60 in 10 days or $60 now) than did normal controls; this over-activation occurred in the right amygdala, superior frontal gyrus, middle cingulate cortex, putamen, posterior cingulate cortex, and temporal pole. Monterosso et al. (2007) also found that methamphetamine users had greater activation than controls during easy delay discounting choices; these group differences were evident in the left DLPFC and the right intraparietal sulcus. Boettiger et al. (2007) found that abstinent alcoholics had higher levels of activation in regions associated with choosing immediate rewards than did control subjects; in addition, individuals in that study with the Val/Val catechol-*O*-methyltransferase genotype also had higher activation in regions associated with choosing immediate rewards (though there was no group by genotype interaction).

1.7 Structural Imaging Studies in Children and Adolescents

Although a complete review of the adolescent structural neuroimaging literature is beyond the scope of this paper, key findings are reviewed below demonstrating extensive

developmental changes in white matter and gray matter over the course of adolescence. Many of these reports come from the Child Psychiatry Branch of the National Institute of Mental Health, which has conducted a massive longitudinal neuroimaging study of brain development during adolescence (e.g. Giedd et al., 1999; Gogtay et al., 2004; Lenroot et al., 2007; Paus et al., 1999; Shaw et al., 2006; Shaw et al., 2008).

White matter increases linearly with age from age 4 to age 22, with comparable rates of increase seen in frontal, temporal, parietal, and occipital lobes (Giedd et al., 1999; Paus et al., 1999; Pfefferbaum et al., 1994; Reiss, Abrams, Singer, Ross, & Denckla, 1996). Myelination follows a rostral to caudal pattern overall, as well as proceeding rostrally to caudally within a given region (Sampaio & Truwit, 2001). In contrast to linear and uniform patterns of white matter maturation, gray matter changes are nonlinear and vary by lobe, following an 'inverted U' developmental trajectory with an increase during childhood followed by a decrease during later adolescence (Giedd et al., 1999; Lenroot et al., 2007). For instance, Giedd et al. (1999) found that, in 4- to 22-year-olds, frontal gray matter increases until about age 11-12, after which it decreases; parietal gray matter increases until age 10-11, after which it decreases; the temporal lobe matures later, with peak levels of gray matter reached around age 16, followed by a slight decline; and occipital-lobe gray matter increases linearly from age 4 to age 22. Temporal gyri and prefrontal regions are among the final areas to mature (Gogtay et al., 2004).

Gray matter density (as opposed to volume) and cortical thickness also have been examined with respect to adolescent development. Gray matter density loss occurs well into the third decade of life, particularly in frontal areas, which may reflect both increased myelination and increased synaptic pruning (Sowell et al., 2001). Cortical thickness increases during childhood, decreases during adolescence, and stabilizes during adulthood; again, primary sensory cortices develop first, followed by secondary cortex, followed by association cortex (Shaw et al., 2008). In individuals with higher intellectual functioning, the developmental trajectory for cortical thickness is more protracted, with a longer and more rapid increase during childhood and decrease during adolescence (Shaw et al., 2006). This pattern was particularly evident in medial prefrontal cortex and in the left middle and inferior temporal gyri.

There is considerable evidence of sex differences in gray matter and white matter development. Males have more cortical gray matter (Giedd et al., 1999), though after controlling for overall brain volume, females have more gray matter in the frontal lobes (Lenroot et al., 2007). Gray matter development has an earlier peak in females than in males, by one to two years (Giedd et al., 1999; Giedd et al., 2006; Lenroot et al., 2007). Giedd et al. (1999) found a steeper rate of white matter increase in males than in females with age. Lenroot et al. (2007) also found significant interactions between sex and age; white matter volumes increase more rapidly in males during adolescence, across the frontal, temporal, parietal, and occipital lobes. Perrin et al. (2009) also found an interaction between sex and age on white matter volume; age was a stronger predictor of white matter volume in frontal, parietal, and temporal lobes in males than in females.

1.8 Diffusion Tensor Imaging as a Measure of White Matter Development

Because the present study uses diffusion tensor imaging (DTI) to examine white matter development during adolescence, in this section the DTI methodology is briefly reviewed. DTI is an imaging method that captures the degree and direction of random (Brownian) movement of water molecules over fixed time periods. In the absence of any barriers, water molecules are expected to diffuse evenly (i.e., isotropically) in three dimensional space. When diffusion is anisotropic, it is inferred that water molecules have encountered a structural barrier. Based on patterns of anisotropy, the extent and type of structural barriers can be reconstructed (Basser, Mattiello, & LeBihan, 1994).

DTI captures water motion by applying magnetic fields of increasing strength (i.e., gradients) along different directions (for example, along the Y axis of the magnetic bore). When a gradient is applied evenly along every part of the Y axis, the signal intensity given off by two water molecules at different positions along the Y axis will be equal. If the gradient is changed so that it is strong at one end and weak at the other, the water molecule further from the strong end of the gradient will resonate at a lower frequency. When an even gradient is once again applied, the two water molecules will resonate at the same frequency but will be out of phase with one another. Thus, the gradient pulse causes a phase difference that depends upon the location of the water molecules along the axis. The phase difference can be reversed by reversing the process of applying gradients, which should result in perfect refocusing of the phases. However, if the water molecules

have moved while the gradients have been applied, perfect refocusing will fail. Imperfect refocusing leads to signal loss. So by applying a pair of gradient pulses, the MR signal is able to quantify the water diffusion process (as described in Mori & Zhang, 2006).

Basser et al. (1994) described the derivation of the diffusion tensor, a 3 by 3 matrix that captures the movement of water molecules in each voxel along three axes. The measurements along each axis are fit to a 3D ellipsoid that characterizes the direction of water motion. The eigenvalues of the tensor ($\lambda 1$, $\lambda 2$, $\lambda 3$) reflect the length of the three axes. The eigenvectors of the tensor (V1, V2, V3) reflect the orientations of the axes. If gradients are applied in at least six different directions, these values can be calculated in order to compute the diffusion tensor. Measurements used in DTI include:

1) $\lambda 1$, or "axial diffusivity." This is a measurement of the extent of diffusion along the longest axis of the ellipsoid; it is also called longitudinal or parallel diffusivity.
2) $(\lambda 2+\lambda 3)/2$, or "radial diffusivity." This is a measurement of the averaged extent of diffusion along the two short axes of the ellipsoid; it is also called perpendicular or transverse diffusivity.
3) $\lambda 1+ \lambda 2 + \lambda 3$, or "trace." This is a measurement of the overall amount of water diffusion in the voxel (in all three directions).
4) $(\lambda 1+ \lambda 2 + \lambda 3)/3$, or "mean diffusivity (MD)." This is a measurement of the amount of water diffusion in the voxel, averaged over three directions.

5) "Fractional anisotropy" (FA: Pierpaoli & Basser, 1996) is essentially the sum of the squared differences of the eigenvalues and reflects the extent to which there are differences between them. Thus, FA will be high when the ellipsoid is pencil-shaped (reflecting anisotropic diffusion) and low when the ellipsoid is spherical (reflecting isotropic diffusion). It includes a correction factor to scale it from 0 (completely isotropic) to 1 (completely anisotropic). FA is computed as:

$$FA = \sqrt{\frac{1}{2}} \frac{\sqrt{\left((\lambda_1 - \lambda_2)^2 + (\lambda_2 - \lambda_3)^2 + (\lambda_3 - \lambda_1)^2\right)}}{\sqrt{\lambda_1^2 + \lambda_2^2 + \lambda_3^2}}$$

(Mori & Zhang, 2006)

Although these parameters are in common use, the cellular changes reflected by changes in these measures have not been confirmed (Ashtari et al., 2007). MD is thought to reflect tissue density (Schmithorst, Wilke, Dardzinski, & Holland, 2005). Higher FA may reflect increased density or packing of fiber tracts or increased parallel organization (Ashtari et al., 2007); it is influenced by the thickness of the myelin sheet as well as the thickness of axons, and is higher when axons are more directionally organized and lower when axons are crossing or branching within a voxel (Klingberg, Vaidya, Gabrieli, Moseley, & Hedehus, 1999). Lower radial diffusivity reflects increases in myelination (Eluvathingal, Hasan, Kramer, Fletcher, & Ewing-Cobbs, 2007; Qiu, Tan, Zhou & Khong, 2008). Axial diffusivity is related to increased "compact fiber coherence" (Ashtari et al., 2007); "intrinsic characteristics of axons" or extra-axonal or extracellular space (Eluvathingal et al., 2007); growth of extra-axonal structures including neurofibrils and glial cells, fiber

coherence, and/ or axonal injury (Qui et al., 2008); or axonal fiber organization (i.e. straightness versus tortuosity: Ashtari et al., 2007) rather than myelination. In the following section, changes in these measurements over the course of adolescence are reviewed.

1.9 DTI Studies of White Matter Development During Adolescence

As reviewed below, studies of white matter development during adolescence have typically found evidence for increases in fractional anisotropy throughout the brain. Often, decreases in mean diffusivity and radial diffusivity also are noted. Results have been more mixed for axial diffusivity, which may reflect the fact that diffusion parallel to fiber bundles may be influenced by a variety of factors that may both increase and decrease facility of parallel diffusion over the course of development. While some DTI-based imaging studies have inferred developmental changes during adolescence by comparing children to adults, others have directly examined development throughout the adolescent age range. Both region-of-interest (ROI)-based approaches, which specify anatomical boundaries and consider broad regions as homogenous structures, and voxel-wise approaches, which search throughout the entire brain without regard to anatomical or functional boundaries, have been used. Tractography also has been used to examine development of known white matter fiber pathways. Finally, several studies have examined DTI variables in relation to cognitive performance in adolescence.

DTI studies have indicated widespread changes in white matter structure during adolescence. Fractional anisotropy has been shown to increase in widespread cortical and

subcortical areas, including in frontal, temporal, and parietal areas; in regions of the basal ganglia including the caudate and putamen; in thalamus and internal and external capsules; in cerebral and cerebellar peduncles; in the cingulate gyrus, hippocampus, fornix, and corpus callosum; and in the brainstem (Ashtari et al., 2007; Berns, Moore, & Capra, 2009; Bonekamp et al., 2007; Klingberg et al., 1999; Lebel, Walker, Leemans, Phillips, & Beaulieu, 2008; Muetzel et al., 2008; Schmithorst, Wilke, Dardzinski, & Holland, 2002; Snook, Paulson, Roy, Phillips, & Beaulieu, 2005; Snook, Plewes, & Beaulieu, 2007; Qui et al., 2008). White matter tracts with increasing FA during adolescence include the corona radiata, superior longitudinal fasciculus, corticospinal tracts, arcute fasciculus, inferior longitudinal fasciculus, inferior fronto-occpital fasciculus, and uncinate fasciculus (Ashtari et al., 2007; Berns et al., 2009; Bonekamp et al., 2007; Eluvathingal et al., 2007; Lebel et al., 2008; Schmidthorst et al., 2002; Snook et al., 2007). FA may plateau in the late teens or early twenties, with fronto-temporal structures developing more slowly than others, with the uncinate continuing to develop past age 29 (Lebel et al., 2008).

MD decreases throughout adolescence in most of the aforementioned areas (Bonekamp et al., 2007; Eluvathingal et al., 2007; Lebel et al., 2008; Muetzel et al., 2008; Schmidthorst et al., 2002; Snook et al., 2005; Snook et al., 2007; Qui et al., 2008). Decreases in MD show a slightly more protracted pattern of development than is seen for increases in FA; the cingulum, superior fronto-occipital fasciculus, thalamus, superior longitudinal fasciculus, caudate, and corticospinal tract continue to develop past age 25 (Lebel et al.,

2008). Radial diffusivity tends to follow the same patterns as MD (Berns et al., 2009; Eluvathingal et al., 2007; Lebel et al., 2008; Qui et al., 2008) and follows a posterior-to-anterior pattern of development (Qui et al., 2008). Age changes in diffusion parameters have been reported to be more striking from late childhood to early adulthood than from middle to late childhood, suggesting that adolescence is a period of particularly significant change in white matter organization (Qui et al., 2008).

FA reflects a combination of the parameters that contribute to radial and axial diffusivity, and authors have disagreed about whether adolescent increases in FA are primarily attributable to decreases in radial diffusivity (e.g. Snook et al., 2005; Lebel et al., 2008) or to increases in axial diffusivity (e.g. Ashtari et al., 2007). This disagreement may stem in part from discrepant findings about developmental patterns of change in axial diffusivity, which has been noted to decrease with increasing age (Eluvathingal et al., 2007; Lebel et a., 2008), both increase and decrease (Qui et al., 2008), and increase (Ashtari et al., 2007).

DTI parameter values associated with greater maturation (higher FA, lower MD, and lower radial diffusivity) are associated with stronger cognitive performances after controlling for age in adolescents on a variety of cognitive and psychomotor tasks, including measures of working memory (Nagy, Westerberg, & Klingberg, 2004), bimanual task performance (Muetzel et al., 2008), native and non-native language reading (Qui et al., 2008), WAIS/WISC Information subtest performance (Ashtari et al.,

2007), IQ (full-scale, verbal, and performance: Schmithorst et al., 2005), and reaction time (Liston et al., 2006). Associations in the other direction typically are not seen, although Berns et al. (2009) found that high FA was associated with *increased* reckless behavior in adolescents. These authors speculated that adolescents who engage in reckless behavior are essentially demonstrating a more adult behavioral pattern, though there is no data to support or refute this claim, and replication of this finding is needed.

Sex differences in DTI parameters have been noted (Eluvathingal et al., 2007), and sex differences in the rate of change in DTI measures with age have also been reported for both FA and MD (Schmithorst, Holland, & Dardzinski, 2008), with the direction of sex differences and sex-by-age interactions varying regionally. This highlights the need to control for sex in aged-based DTI analyses. Finally, although voxelwise analyses tend to find more extensive patterns of associations than ROI-based approaches, they may miss significant findings in regions that are prone to problems with spatial normalization, particularly in developmental samples where structural variation may be greater (Snook et al., 2007).

We previously have reported findings related to delay discounting and diffusion tensor imaging in an adolescent sample using a cross-sectional design (Olson et al., 2007; Olson et al., 2009). In our behavioral study (Olson et al., 2007), we examined delay discounting in 9- to 23-year-olds and probability discounting in an overlapping sample of 11- to 23-year-olds. Inconsistent delay discounters had lower full-scale IQs (attributable to verbal

IQ) than consistent delay discounters; otherwise, discounting consistency was not related to age, gender, ethnicity, maternal/ paternal education, family income, IQ, self-reported internalizing, externalizing, total behavior problems, or delay or probability discounting AUCs. Consistent discounting data was retained for further analysis.

Delay and probability discounting AUCs were positively but not significantly correlated with each other; the correlation was higher in participants age 18 and up than in 11- to 17-year-olds. Delay discounting was positively correlated with age, verbal IQ, and good versus bad choices on the Iowa Gambling Task, and was negatively correlated with impulsive errors on the Go-NoGo task. Hierarchical linear regression models indicated that after controlling for the effect of verbal IQ, the associations between delay discounting and the IGT and Go-NoGo were no longer significant. Probability discounting AUC was positively correlated with externalizing problems but was unrelated to any other variable. Sex was unrelated to delay or probability discounting. Thus, both age and verbal IQ (though not their interaction) were significant predictors of delay discounting, while externalizing was a significant predictor of probability discounting.

In a voxelwise diffusion tensor imaging study focused on the delay discounting paradigm with a largely overlapping sample (Olson et al., 2009), we demonstrated that pubertal status did not contribute uniquely to the prediction of delay discounting after accounting for the effect of age. There were nine clusters where high FA was positively correlated

with larger delay AUCs. Two of these clusters remained significant after statistically controlling for the effects of age and IQ; these included a right frontal cluster with fibers from the anterior thalamic radiation, inferior fronto-occipital fasciculus, uncinate fasciculus, forceps minor, and superior longitudinal fasciculus in the region of the inferior frontal gyrus and the frontal pole, as well as a large left hemisphere cluster including fibers from the inferior fronto-occipital fasciculus, inferior longitudinal fasciculus, anterior thalamic radiation, and uncinate fasciculus, extending through the inferior and superior temporal gyri, the parahippocampal gyrus, and the temporal fusiform cortex. There also were nine clusters where high MD was negatively correlated with larger delay AUCs. Two of these clusters remained significant after statistically controlling for the effects of age and IQ. These included a large right frontal cluster including fibers from the superior longitudinal fasciculus, anterior thalamic radiation, and corticospinal tract near the precentral and middle frontal gyri, as well as a cluster in the right cerebral peduncle including fibers from the anterior thalamic radiation and corticospinal tract, near the amygdala and globus pallidus. To check for specificity of these findings, FA and MD in the significant clusters were checked for correlations with verbal IQ. No significant relationships between verbal IQ and the DTI parameters were seen. In addition, FA and MD in the significant clusters were examined in relation to probability discounting in the subset of individuals who completed that task. Probability discounting was related to FA in only one cluster; this was not one of the clusters that survived correction for age and IQ in the delay discounting analysis.

1.10 Summary of the Behavioral and Imaging Literature

The rate of discounting of delayed rewards is associated with various forms of psychopathology and has a particularly strong relationship with substance use disorders. Although delay discounting and executive functioning tasks measuring impulsive behavior and decision-making are often conceptualized as reflecting similar concepts, the lack of consistent correlation between these measures suggests that delay discounting taps a different function—one which is especially important in the prediction of risk-taking behavior such as substance abuse. Delay discounting rates show age-related trends, with rates continuing to decline into adulthood. Developmental changes also are occurring well into the third decade of life in the regions of the brain that support delay discounting, especially including prefrontal, temporal, and limbic regions, raising the question of how these changes in structure and connectivity may relate to changes in discounting behavior. While probability discounting and delay discounting have at times been characterized as essentially interchangeable, they are probably best characterized as at most partially overlapping, since they have different demographic, cognitive, and neurobiological correlates.

1.11 Specific Aims of the Present Study

The first aim of the present study is to examine the pattern of development of delay and probability discounting behavior using a two-year longitudinal follow-up design with participants age 9-23 years at baseline to evaluate changes in discounting within individuals over the course of adolescence. There has been one previous report of a

longitudinal study of delay discounting in adolescence (Audrain-McGovern, Rodriguez, Epstein, Cuevas, et al., 2009), which reported no change in the discounting rate in individuals followed longitudinally beginning in 10th grade and assessed at two additional time points in the first two years following high school. The present study is the first to examine delay discounting longitudinally throughout the complete adolescent age range and is the first longitudinal study of probability discounting in adolescence. Based on previous research demonstrating cross-sectional changes in the delay discounting rate with age but no changes in the probability discounting rate, it is hypothesized that delay discounting AUCs, but not probability discounting AUCs, will increase from time 1 to time 2. In addition, it is hypothesized that the degree of change in delay discounting behavior will be more marked in younger than in older participants.

The second aim of the present study is to determine whether individual differences in baseline cognitive performance predict greater change in delay and probability discounting rates. The tasks that will be the focus of this investigation measure intelligence, working memory, and reward-related decision-making (WASI, digits backward, and Iowa Gambling Task; see below). These processes were selected for comparison because of the literature relating discounting behavior to intelligence, working memory and reward sensitivity. It is hypothesized that better performance on cognitive measures at baseline will be associated with greater change in the discounting rate from time 1 to time 2.

The third aim is to assess how baseline differences in self-report measures of behavior and personality relate to longitudinal changes in delay and probability discounting. Although discounting has often been examined in relation to short questionnaire-based measures of problematic behaviors (such as impulsivity), there have been fewer studies relating discounting to broad facets of (normal) personality. Reynolds, Richards, Horn, and Karraker (2004) found no relationship between Big 5 personality factors and delay discounting, but Miller et al. (2008) found that lower delay discounting rates were associated with Agreeableness (but not Conscientiousness) on the NEO-PI-R. Broad personality factors have not been examined with respect to discounting in adolescents. The MPQ-Brief and ASEBA (see below) are used to assess personality and internalizing/ externalizing behavior in the present study. It is hypothesized that higher positive emotionality scores at baseline will be associated with greater change in the delay discounting rate from time 1 to time 2. Based on our previous finding that probability discounting AUCs are higher in high externalizers (Olson et al., 2007), it is also hypothesized that higher externalizing scores at baseline will be associated with greater change in the probability discounting rate from time 1 to time 2.

The fourth aim of the present study is to assess how baseline differences in white matter organization relate to longitudinal changes in delay and probability discounting, after controlling for age. In addition to DTI measures, measures of gray and white matter volume and cortical thickness also are examined, in order to capture developmental changes in pruning as well as in myelination. Additionally, ROI-based measures may

capture developmental changes that are obscured in regions near the ventricles due to normalization problems in voxelwise approaches (Snook et al., 2007). For delay discounting, it is hypothesized that individuals with a more mature pattern of development (that is, higher FA, lower MD, larger white matter volume, smaller gray matter volume, and thinner cortical thickness) in regions supporting discounting processes might show accelerated maturation in the discounting rate relative to same age peers. Analyses of relationships between probability discounting and white matter maturation are considered exploratory.

2. Methods

2.1 Participant Recruitment and Study Procedures

The sample in the present study overlaps and expands upon the sample examined in previous reports (Olson et al., 2007; Olson et al., 2009). At time 1, participants ages 9 to 23 years were recruited. Individuals age 9 through 17 were recruited using two methods. One involved the use of a parent volunteer database. Individuals in the target age range were identified, telephoned, and invited to participate in a study of adolescent brain development. The second recruitment method was to mail postcards to University staff members inviting children to participate. The use of this second method was initiated in an attempt to reach participants across a broader range of intellectual functioning, since the range of IQs in participants recruited from the volunteer database was somewhat restricted toward the upper end of the normal IQ distribution. Individuals ages 18 and up

were recruited through posted advertisements at the University. Inclusion criteria included being 9- to 23-years-old, being a native English speaker, having normal or corrected-to-normal vision and hearing, and having no history of neurological or psychological illness, mental retardation, or learning difficulties. All participants were right-handed and had no contraindications to MRI testing. These criteria were assessed through parent and child interviews using the Kiddie-SADS-Present and Lifetime Version (K-SADS-PL: Kaufman et al., 1997) and an in-house health questionnaire. Participants and parents provided informed consent or assent according to local IRB requirements.

At time 1, participants completed a three-hour screening session (semi-structured interview plus intelligence testing) on one day and completed the imaging protocol, neurocognitive battery, and questionnaire measures on a second day. Measures discussed here include the Delay/ Probability Discounting Task (Richards, Zhang, et al., 1999), the Iowa Gambling Task (IGT: Bechara, Damasio, Damasio, & Anderson, 1994), and Digit Span Backward (from the Wechsler Memory Scale, Third Edition: Wechsler, 1997). The DD/PD task and the IGT were programmed in E-prime; the Digit Span task was administered by a trained staff member. Tasks were administered in the same order to all participants. Age-appropriate self-report forms from the Achenbach System of Empirically Based Assessment (ASEBA: Achenbach & Rescorla, 2001) were administered. The Multidimensional Personality Questionnaire- Brief Form (MPQ-Brief: Patrick, Curtin, & Tellegen, 2002) was administered to participants age 11 and up.

Intelligence was measured using the four subtests of the Wechsler Abbreviated Scale of Intelligence (WASI: The Psychological Corporation, 1999).

Participants were contacted approximately two years after their initial assessment and were asked to return for a follow-up assessment. The mean time between assessments was 2.125 years (SD = .274 years). Again, participants completed the study protocol over the course of two visits. On the first day, they completed the WASI and K-SADS-PL, although this measure was not used for exclusionary purposes (i.e., no participants were screened out due to psychopathology at time 2). Only two WASI subtests (Vocabulary and Matrix Reasoning) were used to estimate Full-Scale IQ. On the second day, they completed the imaging protocol, neurocogntive battery, and questionnaires. The order of IGT decks was shuffled. The MPQ-Brief was administered to all participants regardless of age. Otherwise, study procedures and measures were comparable to those used at time 1.

2.2 Statistical Analytic Strategies

Initially, it was hoped that complete behavioral and imaging data sets from both time points could be analyzed. Unfortunately, it is not possible to analyze both time 1 and time 2 imaging data simultaneously at this time. The reason is that an upgrade to the scanner occurred between time 1 and time 2. The scanner upgrade resulted in differences in DTI parameter values (generally with greater clarity or differentiation between high and low values at time 2 than at time 1). However, the magnitude of the change in DTI variables

varied on a voxel-by-voxel basis and also with head size; this problem was discovered via scanning a separate pool of adult participants repeatedly before and after the scanner upgrade. Attempts to derive a correction strategy are ongoing but are complicated by the regional variations. Because of these complications, the present study focuses on the time 1 imaging data only and addresses the question of whether individual differences in FA and MD at time 1 are associated with the development of delay and probability discounting from time 1 to time 2. In order to connect parallel analyses for the behavioral data, only time 1 behavioral correlates are examined with respect to their associations with the development of discounting over time.

Several different data analytic approaches were considered. The first and most intuitive option is the use of change scores. However, change scores (i.e., subtracting time 1 performance from time 2 performance and using the resulting difference as a variable) have been heavily criticized (e.g. Cronbach & Furby, 1970; McArdle, 2009), for two primary reasons. The first is that change scores are much less reliable than either of the single time point measurements that contribute to them (Taris, 2000, pp. 59). The second is that change scores are often correlated with time 1 performance; high time 1 scorers tend to show less increase from time 1 to time 2 due to ceiling effects. Thus, when regression to the mean is occurring, the analysis of change scores is confounded by associations between time 1 performance and change (Cronbach & Furby, 1970). The second option that was considered was the "regressor variable method" (Allison, 1990), in which time 2 scores are regressed on time 1 performance as well as on other time 1

predictors, in order to address the question of which other time 1 predictors predict time 2 performance after adjusting for time 1 performance, which identifies factors associated with time 2 change that is larger or smaller than would have been predicted based on time 1 performance alone (Cronbach & Furby, 1970; Newton & Rudestam, 1999, pp. 218-219). Taris (2000, pp. 62) points out that the apparent conceptual differences between this approach and the change score approach are somewhat misleading, since both address the effects of other independent variables on the development of the dependent variable in time. The third approach that was considered was regressing the change score on time 1 performance as well as on the other variables of interest, in order to statistically correct for the association between change and time 1 performance. However, this approach produces the same results as the regressor variable method (Taris, 2000, pp. 63), and regressor variable approaches may be preferable to difference score approaches for examining interaction effects (Taris, 2000, pp. 67). Finally, the use of hierarchical linear modeling (HLM) was considered, but because data were available from only two time points and because the time interval between assessments was generally consistent amongst participants, the regressor variable method was selected. This approach is commonly used in the psychology literature to examine longitudinal changes (e.g. Stoeber, Otto, & Dalbert, 2009; Tessner, Mittal, & Walker, 2009; Timbremont & Braet, 2006).

To check the data for evidence of regression to the mean (verifying that the use of change scores would be problematic), participants scoring at least one standard deviation above

the mean delay AUC at time 1 were selected (N = 19). At time 1, the mean AUC for those participants was .8341, which was .3843 higher than the mean for the group as a whole (N = 85, M = .4498, SD = .2798). At time 2, the mean AUC for the 19 high-scoring time 1 participants was .6891, which was 0.1399 higher than the mean for the group as a whole (N = 85, M = .5492, SD = .2746). Thus, regression to the mean occurred, because participants who scored far above the group mean at time 1 were closer to the group mean at time 2. Regression to the mean also occurred in the group scoring at least one standard deviation below the mean delay AUC at time 1 (N = 20). Time 1 delay AUC for that group (M = .0913, SD = .0474) was .3585 lower than the mean for the group as a whole. Time 2 delay AUC for that group (M = .4336, SD = .2736) was only 0.1156 lower than the mean for the group as a whole. Thus, participants who had high delay AUCs at time 1 showed a mean decrease in AUC from time 1 to time 2, while those who had low delay AUCs at time 1 showed a mean increase in AUC from time 1 to time 2. For probability discounting, participants scoring at least one standard deviation above the mean probability AUC at time 1 were selected (N = 13). At time 1, the mean AUC for those participants was .7189, which was .2697 higher than the mean for the group as a whole (N = 91, M = .4492, SD = .1950). At time 2, the mean AUC for those participants was .6353, which was .1889 higher than the mean for the group as a whole (N = 91, M = .4464, SD = .1769). Thus, regression to the mean occurred, because participants who scored far above the group mean at time 1 were closer to the group mean at time 2. Regression to the mean also occurred in the group scoring at least one standard deviation below the mean probability AUC (N = 16). At time 1, the mean AUC

for those participants was .1678, which was 0.2813 lower than the mean for the group as a whole. Time 2 probability AUC for that group was .3400, which was only .1064 lower than the mean for the group as a whole.

For behavioral analyses, delay and probability discounting were first each examined separately using repeated measures ANOVA to determine whether cross-timepoint change had occurred. Subsequently, hierarchical regression analyses were conducted using the regressor variable method to identify factors associated with greater than predicted change from time 1 to time 2 discounting performance. For cognitive and personality measures, baseline discounting, age, and sex were entered in the first block. In the second block, the cognitive or personality variables of interest were added. In the third block, selected interactions were added (as described below). Regression analysis interactions were probed using the SPSS implementation of the Hayes & Matthes (2009) computational procedures to generate simple slopes and the Johnson-Neyman regions of significance. Partial correlations (controlling for age, sex, intelligence, baseline discounting, and total brain volume if appropriate) were used to examine relationships between discounting and Freesurfer variables. DTI variables were examined in SPM, first with only time 1 performance as a covariate, and second with time 1 performance, age, sex, and intelligence as covariates.

2.3 Tasks and Measures

Delay/ Probability Discounting Task (Richards, Zhang, et al., 1999): On each trial, participants chose between an immediate amount of money or $10 available after a delay (i.e., "Would you rather have $2 now or $10 in 30 days?"). The immediate amount was determined by a random adjusting-amount procedure (Richards, Zhang, et al., 1999) that randomly selected a value for the immediate amount within a fixed interval that depended on previous choices. On probability trials, the choice was between a certain amount of money or $10 available with a given probability. Discounting was assessed at six delays (1, 2, 10, 30, 180, and 365 days later) and five probabilities (25, 50, 75, 90, and 95% chance of winning). Task instructions appeared onscreen and were read aloud. Afterwards, the computer 'randomly' selected one trial and the participant received a cash payment based on that trial. (Although participants were told that all trials had an equal chance of being selected, selections were constrained without their knowledge to choices involving immediate payoffs for pragmatic reasons). Although it has been demonstrated that adults discount real and hypothetical rewards to similar degrees (Madden et al., 2004), provision of a real reward appeared to increase participants' attention and task enjoyment. Participants ages 11 through 23 completed both tasks; at time 1, 9 and 10 year-olds completed the delay task only, because the overall protocol was too lengthy for them to complete in a reasonable timecourse.

The adjusting-amount procedure (Richards, Zhang, et al., 1999) results in the establishment of one indifference point for each delay/probability interval. Indifference points reflect the subjective value of the delayed or probabilistic amount at a given delay.

Indifference points were established within participants at each interval and plotted against time (delay) or odds against (probability). Following Myerson et al. (2001), the areas under these discounting curves (AUC) were calculated by summing the resulting trapezoids. The subjective value of $10 at time = 0 or odds against = 0 was presumed to be $10 for all participants. The AUC method of quantifying discounting behavior is frequently used (e.g. Acheson & de Wit, 2008; Dixon et al., 2003; Du et al., 2002; Jones & Rachlin, 2009; Ohmura et al., 2005; Perkins et al., 2008; Scheres et al., 2008) and provides a measure of discounting that is not linked to any theoretical framework (Myerson et al., 2001).

Participants may produce inconsistent or erratic discounting behavior due to inattention and/or poor motivation; such 'nonsystematic' discounting data is typically eliminated from further analysis (Dixon et al., 2005; Johnson & Bickel, 2008; Reynolds, Richards, & de Wit, 2006; Reynolds & Schiffbauer, 2004). Indifference points should decrease as the time until or odds against reward delivery increases. Otherwise, discounting behavior is not occurring and individuals are using some other decision-making metric to guide their choices. As Johnson & Bickel (2008) point out, many authors have used R^2 cutoffs in order to eliminate data with a poor fit to a specified discounting function from further consideration. However, as these authors illustrate, this approach has several limitations. One is that it requires fitting a particular theoretical model. Given that it may not be possible at this time to justify a particular model on empirical grounds (McKerchar et al., 2009), an atheoretical approach such as the AUC method is preferable. An additional

limitation of the R^2 cutoff is that R^2 is correlated with k (i.e., the fit of the model is better for higher discounters; Johnson & Bickel, 2008); therefore, using this approach introduces bias in culling nonsystematic data. An approach based on expected patterns of increase/ decrease in indifference points is an alternative that avoids these pitfalls (Johnson & Bickel, 2008). In the present study, consistent discounting behavior was defined as having at least two decreases in subjective value (indifference point) and not more than one increase in subjective value as time or odds against increased (Dixon et al., 2005; Dixon et al., 2003). In adults, AUCs derived using the adjusting-amount computerized algorithm have high test-retest reliability (.96 to .98: Dallery & Raiff, 2007); test-retest reliability may be lower for discounting of abstract goods (e.g. health) (Johnson et al., 2007).

Iowa Gambling Task (Bechara et al., 1994): Participants selected from four decks of cards varying in rewards and punishments. At time 1, Decks 1 and 2 yielded $0.25 for each selection; over 20 selections, a net loss of $1.25 was incurred. Decks 3 and 4 yielded $0.10 or $0.15 for each selection; over 20 selections, a net gain of $1.25 was incurred. Thus, Decks 3 and 4 are advantageous ('good'), yielding smaller immediate rewards and long-term gains. Decks 1 and 2 are disadvantageous ('bad'), yielding larger immediate rewards and long-term losses. Deck 1/3 and 2/4 differ in punishment frequency: Decks 1 and 3 yield frequent (50%) small losses and Decks 2 and 4 yield infrequent (10%) large losses. Participants received feedback with each trial; those with positive net earnings were paid that amount. Task performance reflects decision-making based on implicit

contingencies in the context of personally relevant motivational influences on behavior; the extent to which individuals prefer the high immediate reward decks may reflect risk-taking or impulsivity (Bechara et al., 1994; Bechara, Tranel, & Damsasio, 2000). At time 2, the positions of the good and bad decks were switched, in order to mitigate potential practice effects. Overall bad choices were subtracted from overall good choices at each time point to yield a measure of task performance.

Digit Span Backwards (Wechsler Memory Scale, Third Edition: Wechsler, 1997): In the digit span task, participants are first required to repeat series of orally presented digits verbatim. Two items are presented for each length of the digit string; the series length increases until the participant can no longer accurately perform either item at a given length or until the task ends (at 9 digits forward). In the digits backward task, participants must repeat the sequence in reverse order. Digits backward is considered a measure of verbal working memory due to the mental manipulation of items that must occur in order to repeat the sequence backwards. The total number of correctly repeated sequences in the reverse direction was the score used in the present analysis.

ASEBA: Participants ages 17 and under completed the Youth Self-Report; those ages 18 and up completed the Adult Self-Report (Achenbach & Rescorla, 2001). The ASR and YSR include eight subscales assessing symptoms of DSM-IV psychopathology and an "other problems" scale. On the YSR and the ASR, Internalizing is computed by summing scores on the Withdrawn, Somatic Complaints, and Anxiety/ Depression subscales;

however, because different items contribute to these subscales in the adult and child versions, the maximum scores differ. On the YSR, Externalizing is computed by summing scores on the Delinquency and Aggression subscales, while on the ASR, Externalizing is the sum of the Delinquency, Aggression, and Intrusive Behavior subscales. (The latter subscale is not part of the YSR). Again, even for scales with equivalent names, the maximum scores differ. In order to combine the versions to allow for statistical comparisons, raw scores on each version were divided by the maximum score available for that scale or subscale on that version to yield a score reflecting the percentage of items endorsed out of the total available items. Internalizing and externalizing were transformed by taking the square root to normalize the distributions. Participants' scores generally fell in the average range compared to normative samples of same-age peers (table 2).

Multidimensional Personality Questionnaire-Brief (MPQ-Brief) (Patrick et al., 2002): Participants ages 11 and up completed the MPQ-Brief. (Participants ages 9 and 10 did not complete this measure, because the overall protocol was too lengthy for them to complete in a reasonable timecourse.) The MPQ includes 3 broad factors measuring Positive Emotionality (PEM), Negative Emotionality (NEM), and Constraint (CON). The 11 primary trait scales load onto these factors as follows: Wellbeing, Social Potency, Achievement, and Social Closeness (PEM); Stress Reaction, Alienation, and Aggression (NEM); Control, Harm Avoidance, and Traditionalism (CON); and Absorption. Adolescent norms for the MPQ are not available. Scores were compared to adult norms

in order to identify gross deviations from expected means (table 2). Mean scores fell within a standard deviation of the expected adult norms. Separate analyses of MPQ-Brief data by this author have indicated adequate internal reliability and replication of the basic factor structure in adolescent samples (unpublished analyses).

2.4 Imaging Acquisition and Analysis

MRI image acquisition was performed on a Siemens 3 Tesla Trio scanner (Siemens Medical Systems, Erlangen, Germany) using an 8-channel array head coil, at the University of Minnesota Center for Magnetic Resonance Research. A three dimensional T1 weighted volume was obtained using a coronal magnetization prepared gradient echo (MPRAGE) sequence (TR = 2530 ms, TE = 3.65 ms, TI = 1100 ms, 240 slices, voxel size = 1.0 x 1.0 x 1.0, flip angle = 7 degrees, FOV = 256 mm). An axial hyper-echo turbo spin echo (TSE) sequence was used to collect proton density (PD) images (TR = 8550 ms, TE = 14 ms, 80 slices, voxel size= 1.0 x 1.0 x 2.0 mm, flip angle = 120, FOV = 256 mm). DTI data were acquired axially, aligned with the TSE images, using a dual spin echo, single shot, pulsed gradient, echo planar imaging (EPI) sequence (TR = 12.5s, TE = 98ms, 64 slices, voxel size = 2.0 x 2.0 x 2.0 mm, 0 mm skip, FOV = 256 mm, 2 averages, b value = 1000 s/mm^2). Thirteen unique volumes were collected to compute the tensor: a b = 0 s/mm^2 image and 12 images with diffusion gradients applied in 12 non-collinear directions: (Gx,Gy,Gz) = [1.0,0.0,0.5], [0.0,0.5,1.0], [0.5,1.0,0.0], [1.0,0.5,0.0], [0.0,1.0,0.5], [0.5,0.0,1.0], [1.0, 0.0,-0.5], [0.0,-0.5, 1.0], [-0.5, 1.0, 0.0], [1.0,-0.5, 0.0], [0.0, 1.0,-0.5], [-0.5, 0.0, 1.0]. Field maps were acquired and used to correct the DTI data

for geometric distortion (TR = 700 ms, TE = 4.62 ms/7.08 ms, flip angle = 90°, voxel parameters identical to the DTI, magnitude and phase difference contrasts).

2.4.1 Image processing.

Image processing was performed using tools from the Oxford Centre for Functional Magnetic Resonance Imaging of the Brain's (FMRIB) Software Library (www.fmrib.ox.ac.uk/fsl; Smith et al., 2004). The Brain Extraction Tool (BET) was used to remove skull and other non-brain areas from the T1, PD, and DTI b = 0 images. T1 and PD volumes were co-registered using an affine transformation with trilinear interpolation.

2.4.2 DTI image processing.

The diffusion tensor was computed using the Diffusion Toolbox (FDT) from the FMRIB library (Smith et al., 2004) (FSL version 4.0.1, http://www.fmrib.ox.ac.uk/). A linear affine transformation was applied to diffusion weighted images to correct for the distortions caused by eddy currents (Haselgrove & Moore, 1996). PRELUDE and FUGUE were used to unwarp the fieldmaps, in order to correct for geometric distortion due to magnetic field inhomogeneity. Six maps of the apparent diffusion coefficient (ADC) were computed using the single b=0 image and the twelve eddy-current-corrected and geometric-distortion-corrected diffusion weighted images. The diffusion tensor was then derived, and fractional anisotropy (FA) and mean diffusivity (MD) maps were created.

Normalization of imaging data required several steps. We used a sub-sample of subjects from the larger study to create a study-average template (n = 72). FA maps for 72 subjects were aligned to the FMRIB (FSL) 58 average FA map, yielding a study-average FA map template.

For each subject in the present study, FA images were registered to the study-average FA map template using an affine transformation (in FSL 4.0.1 using areg from IRTK: (Studholme, Hill, & Hawkes, 1999). The linear transformation matrix was then used to initiate the nonlinear warp sequence in order to register each subject to the study-average template using a nonlinear transform (in FSL 4.0.1 using nreg from IRTK) (Rueckert et al., 1999; Schnabel et al., 2001). The linear transformation and nonlinear warp were concatenated and then applied to the remaining DTI images (MD, axial, radial).

The images were then smoothed with a Gaussian kernel of 8 mm full width at half maximum. Absolute threshold masking of 0.2 was applied to FA maps to restrict the statistical analysis to white matter, and an explicit mask was applied to restrict analyses to the cerebrum. MD images were masked to the same voxels as in the FA analysis by applying a sample-average mask of voxels above the threshold of 0.2 in FA images. Voxel-wise analyses of association between FA or MD values and AUCs were carried out using random-effects multiple regression procedures as implemented in Statistical Parametric Mapping software (SPM5; Wellcome Department of Cognitive Neurology,

London, UK). All analyses were thresholded at $p < .001$ uncorrected for multiple comparisons, with an extent threshold of 20 voxels to eliminate isolated small clusters from further consideration. Voxel clusters were identified using FSL 4.0's FSLView with integrated brain atlases including the Harvard-Oxford cortical and subcortical structural atlases, the ICBM-DTI-81 white matter atlas, the JHU white matter tractography atlas, and the MNI structural atlas. Since the rate of brain development in numerous regions varies by sex (Giedd et al., 2006) and by intelligence (Shaw et al., 2006), age, sex, and IQ were entered as covariates in the DTI analyses. It should be noted that an SPM cluster is merely a set of contiguous voxels with a statistical association with variables of interest above a specified threshold; clusters therefore may cross anatomical and functional boundaries.

2.4.3 Freesurfer image processing.

Cortical and subcortical volumes and cortical thickness were computed using Freesurfer image analysis software (http://surfer.nmr.mgh.harvard.edu/; Dale, Fischl, & Sereno, 1999; Fischl, Sereno, & Dale, 1999). For each subject, the T1 image is automatically registered to the Talairach atlas using an affine registration. Segmentation of the subcortical white matter and deep gray matter structures is performed. Likely white matter points are selected based on signal intensity, local neighborhood intensity, and location in Talairach space. An inner surface ("white surface") is built for each hemisphere by tiling (tessellating) the outside of the white matter mass and is refined to follow intensity gradients between gray and white matter. A second surface ("pial

surface") is then built to follow intensity gradients between gray matter and CSF. For each subject, the pattern of folding on the cortical surface is aligned with a spherical cortical surface-based atlas based on cortical folding patterns using a probabilistic high-dimensional nonlinear registration algorithm to match cortical geometry across subjects. The cortex is then automatically parcellated based on gyral and sulcal structures. The cortical thickness is calculated as the closest distance from the white surface to the pial surface at each vertex. Cortical thickness, white, and gray matter volumes were examined by running partial correlations with discounting at time 2 after controlling for discounting at time 1, age, sex, and intelligence. (This partial correlation strategy is equivalent to the regressor variable approach).

3. Results

3.1 Behavioral Results

3.1.1 Demographics and demographic changes.

Demographics of the sample at time 1 and at time 2 are presented in table 3. At time 1, several recruitment strategies were used. Due to concerns that the use of a different mix of recruitment strategies in the younger versus the older age group might result in demographic differences between the groups, younger and older age groups (median split based on time 1 age) were compared at each time point. Aside from the expected age difference, at time 1 the younger group had higher maternal years of education than the older group. However, the practical significance of this difference is likely to be quite

small, as both group means reflected the fact that on average the mothers were college-educated. At time 2, aside from the expected age difference, there was a group difference in Matrix Reasoning T-scores and, consequently, in full-scale IQ. Older participants had higher scores than younger participants. However, there was less than a 6-point full-scale IQ difference between the groups, which is likely to have little functional influence. Aside from the aforementioned differences, the groups did not differ at either time point in sex composition, parental education, median income, or intelligence. Among participants with valid delay or probability discounting at time 1, racial/ ethnic backgrounds were Caucasian (118), African American (2), Hispanic (3), Asian/ Pacific Islander (7), Native American (0), other (including multiracial) (6), and not reported (1).

Most of the demographic variables tended to remain stable over time. Among individuals with valid delay or probability discounting at time 1 and at time 2 (N= 116), paired-samples t-tests revealed no significant change from time 1 to time 2 in maternal education, $t(105) = -.241$, $p = .810$, paternal education, $t(103) = .877$, $p = .383$, Vocabulary T score, $t(113) = -.367$, $p = .714$, Matrix Reasoning T score, $t(113) = -1.338$, $p = .183$, or Full-Scale IQ, $t(113) = -1.238$, $p = .218$. (Degrees of freedom are less than 115 due to missing data). In contrast, a Wilcoxon signed ranks test indicated that there was a significant increase in family income from time 1 to time 2 (N = 87), $Z = -3.756$, $p < .001$ (a nonparametric test was used because income data is significantly skewed).

3.1.2 Consistent and inconsistent discounters.

141 participants completed the DD task at time 1, 122 of whom discounted consistently (table 4). 9- and 10-year-olds were excluded from participating in the PD task at time 1, and one 18-year-old was accidentally administered this abbreviated version of the task. Therefore, there were 120 participants who completed the PD task at time 1. Of those, 110 discounted consistently. 120 participants returned for testing at time 2. Of these, 98 discounted consistently on the DD task, and 116 discounted consistently on the PD task. Differences between inconsistent and consistent discounters are explored in this section. Following this section, the remainder of the analyses in this paper are conducted for consistent discounters only.

At time 1, inconsistent delay discounters had higher delay AUCs (M = .697, SD = .324) than consistent delay discounters (M = .478, SD = .290), $t(139) = -3.026$, $p = .003$, likely because individuals who do not discount at all are classified as inconsistent discounters. Otherwise, after adjusting as appropriate for inequality of variances, inconsistent and consistent delay discounters were comparable in age, gender, IQ, and self-reported internalizing and externalizing. Again, at time 2, inconsistent delay discounters had higher delay AUCs (M = .711, SD = .321) than consistent delay discounters (M = .550, SD = .287), $t(118) = -2.319$, $p = .022$. In addition, there were more females than males at time 2 who showed inconsistent delay discounting, $\chi^2(1, N = 120) = 6.203$, $p = .017$. There were no group differences on any of the remaining variables. At time 1, inconsistent probability discounters had lower Vocabulary T-scores (M = 55.10, SD = 6.540) than consistent probability discounters (M = 59.87, SD = 7.225), $t(118) = 2.014$, p

= .046. There were no significant differences between consistent and inconsistent probability discounters at time 2.

Inconsistent discounting data were excluded from further analysis. Data were inspected for normality and were transformed as necessary to achieve normal distributions (tables 5, for delay discounting, and 6, for probability discounting). Correlations among study measures are presented in tables 7 and 8.

3.1.3 Delay and probability discounting associations.

At time 1, 95 participants had consistent discounting behavior on both tasks. Delay and probability discounting AUCs were positively correlated with each other, $r(93)= .268$, $p = .009$. At time 2, 94 participants had consistent discounting behavior on both tasks; however, the correlation between delay AUC and probability AUC was no longer significant, $r(92) = .152$, $p = .145$.

3.1.4 First study aim: change and stability in discounting rates.

3.1.4.1 Delay discounting.

Analyses discussed in the following section were conducted for individuals with consistent delay discounting data at both time points ($N = 85$; for demographics, see table 9). A repeated measures ANOVA was conducted for delay discounting, entering AUC at time 1 and time 2 as within-subjects variables. There was a significant effect of time, $F(1,84) = 8.545$, $p = .004$. Delay discounting AUCs were larger at time 2 ($M = .564$, *SD*

= .029) than at time 1 (M = .455, SD = .030), reflecting less steep discounting at time 2 (figure 1). Although the mean level of delay discounting changed from time 1 to time 2, there was significant rank-order stability of delay discounting scores, Spearman's *rho*(83) = .356, p = .001. Therefore, while the group mean shifted from time 1 to time 2, there was a significant tendency for individuals to maintain their relative position in the group (though the moderate magnitude of the correlation indicates some tendency for shifting within the group as well).

Hierarchical regression analyses were conducted to examine the contributions of age, sex, and time 1 delay discounting to time 2 delay discounting (table 10). The dependent variable was time 2 discounting AUC. Time 1 discounting was entered in the first block. Age and sex were added to time 1 discounting in the second block, and interactions between age, sex, and time 1 discounting were added in the third block. In the first block, time 1 discounting explained a significant proportion of the variance in time 2 discounting, R^2 = .130, $F(1, 83) = 12.425$, p = .001. Age and sex were added in the second block. Age and delay discounting at time 1, but not sex, explained a significant proportion of the variance in delay discounting at time 2 after controlling for delay discounting at time 1; the addition of these variables did not significantly improve the model fit, although the improvement in fit reached a trend level, ΔR^2 = .061, $\Delta F(2, 81)$ = 3.029, p = .054. In the third block, the two-way and three-way interactions (between time 1 delay discounting, age, and sex) were added. The addition of the interactions significantly improved the model fit, ΔR^2 = .126, $\Delta F(4, 77) = 3.543$, p = .010, although

none of the interactions individually explained a significant proportion of the variance in delay discounting at time 2 after controlling for age, sex, and time 1 discounting. Again, age and delay discounting at time 1 were significant predictors of delay discounting at time 2. To summarize, age had a significant effect on time 2 discounting after controlling for delay discounting at time 1 (with younger participants showing steeper discounting than older participants at time 2). The effect of sex was not significant, and neither were any of the interactions.

In order to confirm that both age and delay discounting at time 1 independently predict delay discounting at time 2, a second regression analysis was conducted entering age in the first block, and age and delay discounting at time 1 in the second block. In the first block, age explained a significant proportion of the variance in time 2 discounting, R^2 = .132, $F(1, 83) = 12.663$, $p = .001$. When delay discounting at time 1 was added in the second block, it significantly improved the model fit, $\Delta R^2 = .055$, $\Delta F(1, 82) = 5.524$, p = .021. Delay discounting at time 2 was significantly predicted by both age ($\beta = .261$; t = 2.396, $p = .019$) and delay discounting at time 1 ($\beta = .256$; $t = 2.350$, $p = .021$). Thus, reversing the order of the predictors does not affect the finding that both age and time 1 delay discounting independently contribute to the prediction of delay discounting at time 2 (figure 2).

3.1.4.2 Probability discounting.

Analyses in the following section were conducted for individuals with consistent probability discounting data at both time points (N = 91; for demographics, see table 9). A repeated measures ANOVA was conducted for probability discounting, entering AUC at time 1 and time 2 as within-subjects variables. The main effect of time was not significant, $F(1,90) = .021$, $p = .884$ (figure 1). Unlike delay discounting, probability discounting showed mean level stability, given that there was no effect of time. In addition, probability discounting showed rank-order stability; scores at time 1 were correlated with scores at time 2, Spearman's *rho* (89) = .554, $p < .001$.

Hierarchical regression analyses were conducted to examine the contributions of age, sex, and time 1 probability discounting to time 2 probability discounting (table 11). The dependent variable was time 2 discounting AUC. Time 1 discounting was entered in the first block. Age and sex were added to time 1 discounting in the second block, and interactions between age, sex, and time 1 discounting were added in the third block. In the first block, time 1 discounting explained a significant proportion of the variance in time 2 discounting, $R^2 = .269$, $F(1, 89) = 32.748$, $p < .001$. Age and sex were added in the second block, and the addition of these variables significantly improved the model fit, $\Delta R^2 = .092$, $F(2, 87) = 6.242$, $p = .003$. Sex and probability discounting at time 1, but not age, explained a significant proportion of the variance in probability discounting at time 2. Probability discounting AUCs at time 2 were higher in males (M = .513, SD = .171) than in females (M = .375, SD = .155). In the third block, the two-way and three-way interactions (between time 1 probability discounting, age, and sex) were added. The

addition of the interactions did not significantly improve the model fit, $\Delta R^2 = .055$, $F(4, 83) = 1.955$, $p = .109$, although the interaction between age and sex was a significant predictor of probability discounting at time 2 after controlling for probability discounting at time 1, age, and sex. Again in the third block, sex and probability discounting at time 1 were significant predictors of probability discounting at time 2. To explore the interaction, simple slopes analysis was used. The relationship between time 2 probability discounting and sex varied based on age (after controlling for all other main effects and interactions); the effect of sex was significant for participants aged one standard deviation below the mean, $t(83) = -3.7869$, $p = .0003$, and for participants at the mean age, $t(83) = -3.3048$, $p = .0014$, but not for participants aged one standard deviation above the mean, $t(83) = -.8033$, $p = .4241$. The Johnson-Neyman region of significance for the effect of sex is age 18.6 or younger; there is no significant effect of sex on time 2 discounting above that cutoff.

3.1.5 Second study aim: cognitive factors at time 1 associated with change in discounting.

3.1.5.1 Delay discounting.

Hierarchical linear regressions were used to examine whether performance on other cognitive measures at time 1 is associated with delay discounting at time 2, after controlling for delay discounting at time 1. Variables examined included WASI-derived IQ scores, the overall number of choices from 'good' decks minus choices from 'bad' decks on the Iowa Gambling Task (IGT), and digit span backward total score. Of the 85

participants with valid discounting data at both time points, two were missing IGT data and one was missing IQ data, so the final *N* for this analysis was 82. Time 2 AUC was the dependent variable. In the first block, delay discounting at time 1, age, and sex were entered; the model fit was significant, $R^2 = .182$, $F(3, 78) = 5.798$, $p = .001$, and time 1 discounting and age were significant predictors. In the second block, estimated Full-Scale IQ, good versus bad IGT choices, and digit span backward were entered to examine the effects of these variables after controlling for baseline delay discounting, age, and sex. None of the cognitive correlates at time 1 significantly predicted time 2 delay discounting (table 12), and adding these variables did not significantly improve the model fit, $\Delta R^2 = .042$, $\Delta F(3, 75) = 1.368$, $p = .259$. Time 1 discounting remained a significant predictor, though the addition of the cognitive correlates resulted in the reduction of the influence of age to a trend level ($\beta = .214$, $t = 1.690$, $p = .095$). Given the association between age and delay discounting, interactions between age and these cognitive measures were examined by adding them in the third block. Adding the interactions did not significantly improve the model fit, $\Delta R^2 = .015$, $\Delta F(3, 72) = .486$, $p = .693$. As in the second block, delay discounting at time 1 was the only significant predictor of delay discounting at time 2, and the significance of age was reduced to a trend level ($\beta = .228$, $t = 1.721$, $p = .090$).

3.1.5.2 Probability discounting.

A parallel analysis was conducted for probability discounting (table 13). Of the 91 participants with valid probability discounting data at both time points, one was missing IGT data, so the final *N* for this analysis was 90. In the first block, probability

discounting at time 1, age, and sex were entered; the model fit was significant, $R^2 = .357$, $F(3, 86) = 15.937$, $p < .001$, and sex and time 1 discounting were significant predictors of time 2 discounting. In the next block, the cognitive correlates at time 1 were added. Adding these variables did not significantly improve the model fit, $\Delta R^2 = .007$, $\Delta F(3, 83) = .323$, $p = .809$; sex and time 1 discounting remained the only significant predictors. Because there were sex differences in probability discounting, interactions between sex and these cognitive measures also were entered in the third block. Adding these variables did not significantly improve the model fit, $\Delta R^2 = .033$, $\Delta F(3, 80) = 1.475$, $p = .228$. The interaction between sex and digits backward on probability discounting at time 2 after controlling for age, sex, and probability discounting at time 1 was significant. Simple slopes analysis indicated that males with lower time 1 digits backward scores had higher time 2 probability discounting AUCs at a trend level, $t(80) = -1.9561$, $p = .0539$; the effect of digits backward was not significant in females, $t(80) = .8897$, $p = .3763$.

3.1.6 Third study aim: behavioral/personality factors at time 1 associated with change in discounting.

3.1.6.1 Internalizing/externalizing.

3.1.6.1.1 Delay discounting.

The ASEBA's raw internalizing and externalizing scores were prorated to adjust for different maximum raw scores on the adult and child versions of the forms and were subsequently combined. Scores were transformed to adjust for skew. Two individuals who had consistent discounting data at both time points had incomplete ASEBA data; the

final *N* for this analysis is 83. Hierarchical linear regressions were used to examine which variables at time 1 were associated with delay discounting AUC at time 2, after controlling for delay discounting at time 1 (table 14). In the first block, delay discounting at time 1, age, and sex were entered; the model fit was significant, R^2 = .192, $F(3, 79)$ = 6.240, p = .001. In the next block, internalizing and externalizing scores were entered to examine the effects of these variables after controlling for baseline delay discounting, age, and sex. Neither of the behavioral correlates at time 1 significantly predicted change in delay discounting, and adding these variables did not significantly improve the model fit, ΔR^2 = .038, $\Delta F(2, 77)$ = 1.882, p = .159. Because there were age differences in the change in delay discounting over time, interactions between age and internalizing/ externalizing were added in the third block. Adding the interactions resulted in a trend-level improvement in the model fit, ΔR^2 = .049, $\Delta F(2, 75)$ = 2.525, p = .087.

There was a significant interaction between age and time 1 internalizing as well as between age and time 1 externalizing on time 2 delay discounting after controlling for baseline discounting, sex, and age. To explore these interactions, simple slopes analysis was conducted. The relationship between time 2 discounting and internalizing varied based on age (after controlling for all other main effects and interactions); the effect of internalizing was significant for participants aged one standard deviation below the mean, $t(75)$ = 2.5452, p = .0130, but not for participants at the mean age, $t(75)$ = 1.3270, p = .1885, or aged one standard deviation above the mean, $t(75)$ = -.6053, p = .5468. In the younger participants, high internalizers had larger delay discounting AUCs at time 2 after

controlling for the other main effects and interactions, and the effect was significant; in the older age group, the direction of the effect was reversed but nonsignificant. The Johnson-Neyman region of significance for the effect of internalizing is age 14.2 or younger; there is no significant effect of internalizing on time 2 discounting above that cutoff. The relationship between time 2 discounting and externalizing varied based on age (after controlling for all other main effects and interactions); the effect of externalizing was nonsignificant but at a trend level for participants aged one standard deviation above the mean, $t(75) = 1.9173$, $p = .0590$, but not for participants at the mean age, $t(75) = .6613$, $p = .5104$, or aged one standard deviation below the mean, $t(75) = -1.3227$, $p = .1900$. In the older participants, high externalizers had larger delay discounting AUCs at time 2 after controlling for the other main effects and interactions (at a trend level); in the younger participants, the direction of the effect was reversed but nonsignificant. The Johnson-Neyman region of significance for the effect of externalizing is age 20.6 or older; there is no significant effect of externalizing on time 2 discounting below that cutoff. Thus, the significant interactions reflect the fact that high time 2 delay discounting AUC is associated with high internalizing at time 1 in younger (but not older) participants and with high externalizing at time 1 in older (but not younger) participants.

3.1.6.1.1 Probability discounting.

A parallel analysis was conducted for probability discounting (table 15). Three individuals who had consistent discounting data at both time points had incomplete ASEBA data; the final *N* for this analysis is 88. In the first block, probability discounting

at time 1, age, and sex were entered; the model fit was significant, $R^2 = .360$, $F(3, 84) = 15.764$, $p < .001$, and sex and time 1 discounting were significant predictors. In the next block, internalizing and externalizing scores were entered to examine the effects of these variables after controlling for baseline probability discounting, age, and sex. Neither of the behavioral correlates at time 1 significantly predicted change in probability discounting, and adding these variables did not significantly improve the model fit, $\Delta R^2 = .011$, $\Delta F(2, 82) = .722$, $p = .489$. Sex and time 1 discounting remained significant predictors. Because there are sex differences in the change in probability discounting over time, interactions between sex and internalizing/ externalizing were added in the third block. Adding the interactions did not significantly improve the model fit, $\Delta R^2 = .005$, $\Delta F(2, 80) = .292$, $p = .747$. The interaction between sex and internalizing was not associated with change in probability discounting after controlling for initial probability discounting, age, and sex; nor was the interaction between sex and externalizing. Sex and time 1 discounting remained significant predictors.

3.1.6.2 Personality.

3.1.6.2.1 Delay discounting.

MPQ raw scores at time 1 were available for participants age 11 and up at time 1 ($N = 71$; one participant had incomplete MPQ data, so the final N for these analyses is 70). Raw scores were natural log transformed (or reflected and natural log transformed) as appropriate to address skew. Hierarchical linear regression was used to examine whether Positive Emotionality (PEM), Negative Emotionality (NEM), and/ or Constraint (CON)

at time 1 was associated with delay discounting AUC at time 2, after controlling for delay discounting at time 1 (table 16). In the first step, delay discounting AUC at time 1, age, and sex were entered. The model fit was significant, $R^2 = .257$, $F(3, 66) = 7.619$, $p < .001$, and time 1 discounting and age were significant predictors. In the second block, the MPQ factors PEM, NEM, and CON were entered to examine the effects of these variables after controlling for baseline delay discounting, age, and sex. None of the personality correlates at time 1 significantly predicted change in delay discounting, and adding these variables did not significantly improve the model fit, $\Delta R^2 = .038$, $\Delta F(3, 63) = 1.120$, $p = .348$. However, PEM at time 1 predicted delay discounting at time 2 at a trend level, $t(63) = 1.796$, $p = .077$. Age and time 1 discounting remained significant predictors. Because there were age differences in the change in delay discounting over time, interactions between age and PEM, NEM, and CON were added in the third block. Adding these variables did not significantly improve the model fit, $\Delta R^2 = .062$, $\Delta F(3, 60) = 1.911$, $p = .137$, though the interaction of age and positive emotionality was a significant predictor of time 2 discounting, $t(60) = 2.095$, $p = .040$. Age and time 1 discounting remained significant predictors. To examine the interaction, a simple slopes analysis was conducted. The relationship between time 2 discounting and PEM varied based on age (after controlling for all other main effects and interactions); the effect of PEM was nonsignificant for participants aged one standard deviation below the mean, $t(60) = -.4322$, $p = .6672$, at a trend level of significance for participants at the mean age, $t(60) = 1.8179$, $p = .0741$, and significant for participants aged one standard deviation above the mean, $t(60) = 2.8323$, $p = .0063$. The Johnson-Neyman region of significance

for the effect of PEM is age 16.8 or older; there is no significant effect of positive emotionality on time 2 delay discounting below that cutoff, with individuals with higher PEM scores having higher time 2 delay AUCs.

Since PEM is composed of two separate factors, communal positive emotionality and agentic positive emotionality, the contribution made by these factors to delay discounting at time 2 was examined (table 17). The addition of these factors significantly improved the model fit over the prediction made by delay discounting at time 1, age, and sex, ΔR^2 = .130, $\Delta F(2, 64) = 6.757$, $p = .002$. There was a significant contribution of agentic PEM to the prediction of delay discounting at time 2, $t(64) = 3.662$, $p = .001$, but the contribution of communal PEM was not significant, $t(64) = -1.544$, $p = .127$. In addition to agentic PEM, time 1 discounting was a significant predictor in the second block; the contribution of age was no longer significant. In the third block, interactions between age and agentic and communal PEM were added. The addition of these interactions did not significantly improve the model fit, $\Delta R^2 = .025$, $\Delta F(2, 62) = 1.309$, $p = .278$. Time 1 discounting and agentic PEM remained the only significant predictors.

There are four first-order subfactors that contribute to agentic PEM: wellbeing, social potency, achievement, and absorption; the contribution to the prediction of delay discounting at time 2 made by these subfactors was examined (table 18). The addition of these factors improved the model fit at a trend level over the prediction made by delay discounting at time 1, age, and sex, $\Delta R^2 = .101$, $\Delta F(4, 62) = 2.443$, $p = .056$. There was a

significant contribution of achievement to the prediction of time 2 delay discounting, $t(62) = 3.010$, $p = .004$, but the contributions of wellbeing, social potency, and absorption were not significant. Time 1 delay discounting also remained a significant predictor. Thus, individuals with higher levels of agentic positive emotionality at time 1 achieved higher delay discounting scores at time 2 after accounting for baseline delay discounting, age, and sex; this effect was driven by the achievement subfactor. In the third block, the interactions between age and the agentic PEM subfactors (wellbeing, social potency, achievement, and absorption) were added. The addition of the interactions significantly improved the model fit, $\Delta R^2 = .098$, $\Delta F(4, 58) = 2.616$, $p = .044$. Time 1 delay discounting and achievement remained significant predictors, and the interaction of age and wellbeing also significantly predicted delay discounting at time 2, $t(58) = 2.387$, $p = .020$. The relationship between time 2 discounting and wellbeing varied based on age (after controlling for all other main effects and interactions); the effect of wellbeing was significant for participants aged one standard deviation below the mean, $t(58) = -2.1156$, $p = .0387$, nonsignificant for participants at the mean age, $t(58) = -.7119$, $p = .4794$, and nonsignificant for participants aged one standard deviation above the mean, $t(58) = 1.3273$, $p = .1896$. The Johnson-Neyman region of significance for the effect of wellbeing is age 14.0 or younger (in which group high wellbeing at time 1 is associated with low delay discounting AUCs at time 2).

3.1.6.2.2 Probability discounting.

Comparable analyses for probability discounting were conducted to examine whether PEM, NEM, and CON were associated with probability discounting AUC at time 2, after controlling for probability discounting at time 1 (table 19; N = 91 participants with valid probability discounting at both time points, but one participant had incomplete MPQ data for a final N of 90). In the first step, probability discounting AUC at time 1, age, and sex were entered; time 1 discounting and sex were significant predictors. Addition of the MPQ factors in the second step did not significantly improve the model fit, ΔR^2 = .025, $\Delta F(3, 83) = 1.134$, p = .340. Time 1 discounting and sex remained significant predictors, and NEM at time 1 predicted probability discounting at time 2 at a trend level, $t(83)$ = 1.699, p = .093. Given the sex differences in probability discounting, interactions between sex and PEM, NEM, and CON were added in the third block. The addition of the interactions did not significantly improve the model fit, ΔR^2 = .008, $\Delta F(3, 80) = .354$, p = .786, and none of the interactions was a significant predictor of time 2 probability discounting. Time 1 discounting and sex remained the only significant predictors.

Since NEM is can be decomposed into alienated negative emotionality and agentic negative emotionality, the contribution made by these factors was examined (table 20). The addition of these factors did not significantly improve the model fit over the prediction made by probability discounting at time 1, age, and sex, ΔR^2 = .029, $\Delta F(2, 84)$ = 1.991, p = .143. Time 1 discounting and sex remained the only significant predictors. There was no significant contribution of alienated NEM to the prediction of probability discounting at time 2, $t(84) = -.094$, p = .926, but the contribution of agentic NEM was

significant at a trend level, $t(84) = 1.808$, $p = .074$. In the third block, the interactions between sex and agentic or alienated NEM were added. The addition of the interactions did not significantly improve the model fit, , $\Delta R^2 = .002$, $\Delta F(2, 82) = .148$, $p = .863$, and time 1 discounting and sex remained the only significant predictors.

There are two primary first-order factors that contribute to agentic NEM: stress reactivity and aggression; the contribution to the prediction of probability discounting at time 2 made by these subfactors was examined (table 21). The addition of these subfactors did not significantly improve the model fit over the prediction made by probability discounting at time 1, age, and sex, $\Delta R^2 = .037$, $F(2, 84) = 2.582$, $p = .082$, and neither stress reaction nor aggression at time 1 significant contributed to the prediction of probability discounting at time 2. Time 1 discounting and sex remained the only significant predictors. The interactions between sex and stress reactivity or aggression were added in the third block. The addition of the interactions did not significantly improve the model fit, $\Delta R^2 = .019$, $F(2, 82) = 1.308$, $p = .276$.

3.2 Fourth Study Aim: Brain Structural Features at Time 1 Associated with Change in Discounting

3.2.1 Freesurfer analyses.

Following a consensus process to identify problematic imaging data, four of the participants in the present study were excluded from further Freesurfer analyses due to

structural brain abnormalities (including a temporal lobe cyst, frontal lobe damage, frontal hyperintensity, and asymmetrical lateral ventricles).

3.2.1.1 Delay discounting.

3.2.1.1.1 Cortical thickness.

After controlling for time 1 delay discounting, age, sex, and intelligence, delay discounting at time 2 was significantly associated with cortical thickness in the left parahippocampal gyrus and frontal pole, and in the right caudal middle frontal gyrus, frontal pole, inferior frontal gyrus (pars opercularis, pars orbitalis, pars triangularis), precentral gyrus, and rostral middle frontal gyrus (table 22; figure 3). Individuals with thinner gray matter in the frontal regions at time 1 had higher delay AUCs at time 2, reflecting less steep discounting, after accounting for age, baseline discounting, intelligence, and sex. (This is the predicted pattern in which more mature structural development is associated with lower rates of delay discounting). In contrast, thicker gray matter in the left parahippocampal gyrus at time 1 was associated with higher delay discounting AUCs at time 2 (figure 3).

3.2.1.1.2 White matter volume.

After controlling for time 1 delay discounting, age, sex, intelligence, and total brain volume, delay discounting at time 2 was significantly associated with white matter volumes in several right hemisphere regions, including the right caudal anterior cingulate gyrus, isthmus of the cingulate gyrus, and cuneate gyrus (table 22; figure 4). There were

no significant left hemisphere associations. Larger white matter volumes in the cingulate and cuneate gyri delay were associated with greater change in delay discounting from time 1 to time 2.

3.2.1.1.3 Subcortical volume.

There was a significant association between hippocampal volumes bilaterally and delay discounting at time 2 after controlling for baseline discounting, age, sex, intelligence, and total brain volume (table 23; figure 5). Larger hippocampal volumes at time 1 were associated with higher delay AUCs (less steep discounting) at time 2 after adjusting for baseline discounting.

3.2.1.2 Probability discounting.

3.2.1.2.1 Cortical thickness

After controlling for time 1 probability discounting, age, intelligence, and sex, probability discounting at time 2 was not significantly associated with cortical thickness in any of the regions examined (table 24).

3.2.1.2.2 White matter volume

After controlling for time 1 probability discounting, age, intelligence, and sex, probability discounting at time 2 was significantly associated with white matter volume in the left rostral anterior cingulate gyrus (table 24; figure 6). Individuals with larger white matter

volumes in that region at time 1 had higher probability AUCs at time 2 after adjusting for baseline probability discounting.

3.2.1.2.3 Subcortical volume

After controlling for time 1 probability discounting, age, intelligence, and sex, probability discounting at time 2 was not significantly associated with subcortical volume in any of the regions examined (table 25).

3.2.2 SPM analyses.

For delay discounting, two brains were excluded from SPM analysis due to structural abnormalities (temporal lobe cyst, frontal lobe damage). A third case was considered for exclusion due to large lateral ventricles but was ultimately held in, because its inclusion did not cause a significant change in search volume. One delay discounting participant also was missing IQ data; the final N for delay discounting analyses was 82. For probability discounting, two brains were excluded from SPM analysis due to structural abnormalities (frontal lobe damage, large lateral ventricles). The final N for probability discounting analyses was 89, as the individual with missing IQ data did not have consistent probability discounting data.

3.2.2.1 Delay discounting.

In the first set of analyses, the relationship between time 1 DTI variables (FA and MD) and time 2 delay discounting was examined, controlling for baseline levels of discounting

by entering time 1 AUC as a covariate. There was a cluster in the left posterior thalamus/ hippocampus including parts of the fornix, stria terminalis, and anterior thalamic radiation where low time 1 MD was associated with higher time 2 delay AUCs after adjusting for baseline discounting (table 26; figure 7). There were no clusters where high time 1 MD was associated with higher time 2 delay AUCs, and there were no clusters in either direction for FA.

Because different patterns of association in younger versus older participants might obscure existing associations, this analysis was repeated separately for participants age 9-15 ($N = 45$) versus age 16 and up ($N = 37$). In the younger age group, higher time 2 delay AUCs were associated with high time 1 FA in a cluster involving fibers from the left inferior fronto-occipial fasciculus/ anterior thalamic radiation/ uncinate fasciculus/ superior longitudinal fasciculus in the inferior frontal gyrus (pars opercularis and triangularis)/ middle frontal gyrus (table 27; figure 8). There were no other significant FA or MD clusters in either direction (positive or negative) for either age group.

In the second set of analyses, voxelwise analyses were repeated while including age, sex, and intelligence as covariates; since these factors affect in DTI variables, failing to control for their influence might mask significant associations between delay discounting and the imaging variables. Again, baseline levels of delay discounting were controlled for by entering time 1 delay AUC as a covariate (along with sex, age, and IQ). There was a cluster including fibers from the right anterior thalamic radiation/ corticospinal tract in

the region of the amygdala/ pallidum/ hippocampus where high FA at time 1 was associated with *lower* delay discounting AUCs at time 2 after adjusting for baseline discounting (table 28; figure 9). There were no clusters where high time 1 FA was associated with higher time 2 delay AUCs, and there were no clusters in either direction for MD.

The previous analysis was repeated for each age group separately. In the younger age group, there was a cluster where high time 1 FA was associated with lower time 2 delay AUCs after adjusting for baseline discounting (table 29; figure 10). This cluster included fibers from the right superior longitudinal fasciculus/ inferior longitudinal fasciculus/ inferior fronto-occipital fasciculus/ anterior thalamic radiation in the region of the lateral occipital cortex/ angular gyrus/ precuneus/ superior parietal lobule. There were no significant clusters in the older age group.

3.2.2.1 Probability discounting.

In the first set of analyses, the relationship between time 1 DTI variables (FA and MD) and time 2 probability discounting was examined, controlling for baseline levels of discounting by entering time 1 AUC as a covariate. There were no significant clusters in either direction (positive or negative) for either variable (FA or MD) in the sample as a whole. Repeating the analysis in each age group separately (age 11-15: $N = 40$; age 16 and up: $N = 49$) also resulted in no significant clusters.

In the second set of analyses, the relationship between time 1 DTI variables and time 2 probability discounting was examined, controlling for baseline levels of discounting by entering time 2 AUC as a covariate and also controlling for age, sex, and intelligence. There was a cluster in the left inferior longitudinal fasciculus/ inferior fronto-occipital fasciculus/ forceps major in the lateral occipital cortex/ cuneus/ precuneus where high MD at time 1 was associated with lower time 2 probability AUC (table 30). There were no areas where low MD at time 1 was associated with low probability discounting AUCs at time 2, nor were there any significant associations (in either direction) between time 1 FA and time 2 probability discounting after controlling for baseline discounting, age, and sex. Data were analyzed separately for participants age 11 through 15 versus 16 and up at time 1. When the age groups were analyzed separately, there were no significant clusters in either direction for either DTI variable.

4. Conclusions

4.1 Summary of Behavioral Findings

Within individuals, delay discounting rates developed over the course of a two-year span during adolescence, with less steep delay discounting seen with maturation (figure 3). Although a number of previous cross-sectional studies have demonstrated changes in discounting with increasing age, this is the first longitudinal study to find significant changes within individual adolescents in the delay discounting rate over the course of adolescence. Greater age also was associated with lower time 2 discounting rates (higher

AUCs) after controlling for baseline levels of delay discounting. Consistent with much of the delay discounting literature, there was no effect of sex on delay discounting. The lack of a significant sex by age interaction indicates that the relationship between age and delay discounting is comparable for both sexes.

None of the cognitive measures, including measures of intelligence, decision-making aspects of executive functioning, and working memory, were significant predictors of time 2 delay discounting after controlling for time 1 delay discounting, age, and sex. This finding was somewhat surprising, since several studies have demonstrated lower delay discounting rates in individuals with stronger overall intellectual abilities. One interpretation of the present findings is that intellectual ability influences relative delay discounting rates at baseline but does not influence the developmental trajectory of changes in the discounting rate after controlling for baseline discounting. The lack of relationship between changes in delay discounting and baseline performance on the IGT and digits backward is not entirely surprising, since performance on these and other measures of executive functioning and working memory is at best inconsistently related to delay discounting performance in children and adults.

Self-reported internalizing and externalizing behavior at time 1 did not significantly predict time 2 delay discounting after controlling for time 1 delay discounting, age, and sex. However, there were significant interactions between these variables and age. Younger participants who were high in internalizing behavior at time 1 had larger delay

discounting AUCs at time 2. Older participants who were high in externalizing at time 1 had larger delay discounting AUCs at time 2 (at a trend level). Thus, greater decrease in the discounting rate is seen over a two-year period for younger participants who are high internalizers at baseline and for older participants who are high externalizers at baseline. This type of pattern might be seen if adolescents experiencing a transient spike in internalizing or externalizing behavior also show transient elevation in the delay discounting rate; a return to baseline may occur in both behavioral symptoms and in the decision-making processes underlying delay discounting. The fact that different behavioral patterns are associated with greater than expected change at different developmental stages (internalizing in younger adolescents and externalizing in older adolescents) could occur if spikes in internalizing symptoms are more prevalent in younger adolescents and spikes in externalizing symptoms are prevalent in older adolescents. If this explanation were true, larger decreases in internalizing or externalizing from time 1 to time 2 should predicted larger decreases in the rate of delay discounting. Examination of the full longitudinal data set (both measures at both time points) could ultimately clarify this issue.

The general personality dimension of positive emotionality at time 1 was related to time 2 delay discounting rates after controlling for time 1 delay discounting, age, and sex at a trend level. There was no relationship between the general dimensions of negative emotionality or constraint and delay discounting. Decomposing positive emotionality into agentic versus communal subfactors revealed a significant relationship between agentic

positive emotionality at time 1 and time 2 discounting but no relationship between discounting and communal positive emotionality. Individuals high in agentic positive emotionality at time 1 had lower rates of delay discounting after controlling for baseline discounting, age, and sex. Examination of the first-order factors suggested that this finding was driven by scores on the achievement factor; high time 1 achievement was associated with lower time 2 discounting rates. There also was a significant interaction between wellbeing and age on time 2 discounting rates; younger participants with high wellbeing scores at time 1 had higher time 2 discounting rates after controlling for time 1 discounting, but there was no relationship between wellbeing and discounting in older participants.

Unlike delay discounting, probability discounting rates did not change within individuals over the two-year timeframe of this study. Although we did not find a sex difference in probability discounting in our previous cross-sectional report (Olson et al., 2007), sex predicted a significant amount of the variance in time 2 discounting after adjusting for baseline probability discounting. Male sex was associated with an increase in probability discounting AUC (i.e., decreased risk aversion) from time 1 to time 2, while female sex was associated with a decrease in probability discounting AUC from time 1 to time 2. Although adding the interactions did not improve the prediction of time 2 discounting, there was a significant interaction between age and sex. Female sex was associated with a greater decrease in probability discounting AUC from time 1 to time 2 in younger participants only. The greater decrease from time 1 to time 2 in female participants

persisted until over age 18 years. Aside from our previous research, only one other group has studied probability discounting in adolescence. Sex was not examined in that study, likely due to the small number of female participants. This is the first demonstration of a sex difference in the change in probability discounting in adolescence; sex differences are not typically seen in probability discounting studies in adults. This finding suggests that probability discounting may be a sensitive marker for types of risk-taking behavior that differ by sex during adolescence.

None of the cognitive measures had a significant main effect on probability discounting rates at time 2 after controlling for baseline probability discounting, age, and sex. However, there was a significant interaction between a measure of verbal working memory (digits backward) and sex on probability discounting. Lower digits backward scores were associated with higher time 2 probability AUCs (at a trend level) in males only; the relationship was not significant in females. This finding provides another piece of evidence that the probability discounting task captures different aspects of behavior in males than in females. Males with poorer working memory performance are less risk-averse on the probability discounting task, while working memory and risk aversion are not related in females. The association between poor working memory performance and risk-seeking behavior in adolescent males could reflect decreased ability to hold in mind past experiences when making decisions, resulting in poorer integration of past (negative) outcomes with present or future behavior. This pattern may place these adolescent males

at increased risk for experiencing harmful consequences as a result of the 'double blow' of disturbance in working memory and in risk-seeking.

Self-reported internalizing and externalizing behavior at time 1 did not significantly predict time 2 probability discounting after controlling for time 1 probability discounting, age, and sex. There also were no significant sex by internalizing or externalizing interactions. We previously found lower probability discounting rates associated with higher externalizing scores. Externalizing may influences relative probability discounting rates at baseline, but it does not influence the developmental trajectory of changes in the discounting rate after controlling for baseline discounting.

The general personality dimensions of positive emotionality, negative emotionality, and constraint at time 1 were not significantly related to time 2 probability discounting rates after controlling for time 1 probability discounting, age, and sex at a trend level. While there was a trend for negative emotionality, decomposing NEM into agentic versus alienated components and decomposing agentic NEM into the first-order factors continued to reveal only trend-level associations. Interactions with sex also were not significant. In general, significant relationships between personality factors and change in probability discounting were not seen.

Delay and probability discounting AUCs were significantly yet moderately positively correlated with one another at time 1 (r = .268) and were positively yet non-significantly

correlated with one another at time 2 (r = .152). This does not support a model positing that both tasks capture a general trait of impulsivity, since this model predicts low probability discounting (or low risk-aversion) in the context of high delay discounting (or impatience). These findings also do not support models that suggest that delay discounting is merely a form of probability discounting or vice-versa. The weak to moderate correlations between the two measures suggests that the tasks capture at least partially different aspects of choice behavior.

The higher AUCs of inconsistent versus consistent delay discounters reflect the fact that failure to discount at all is classified as inconsistent discounting. Such "non-discounting" was seen for delay discounting but not probability discounting. Although the small number of participants involved precluded adequate statistical comparisons of "non-discounters" to "erratic" and consistent discounters, there were over twice as many female non-discounters as male non-discounters, raising the possibility of a sex difference in non-discounting. There are no published analyses examining differences between individuals who adopt these three styles of approaching the delay discounting task; this is an important future direction for research.

4.2 Summary of Imaging Findings

4.2.1 Delay discounting.

In many areas, larger delay discounting AUCs at time 2, controlling for baseline discounting, were associated with more mature patterns of brain development at time 1.

For instance, larger change in delay discounting AUCs was seen in individuals with thinner cortical thickness measurements in the frontal pole bilaterally, right caudal middle frontal gyrus, right inferior frontal gyrus, right precentral gyrus, and right rostral middle frontal gyrus (controlling for baseline discounting, age, sex, and intelligence). The middle and inferior frontal gyri have been implicated in delay discounting fMRI studies in adults, with activity in the middle frontal gyrus generally associated with less steep discounting (Shamosh et al., 2008) and with the inferior frontal gyrus tracking both reward magnitude and delay (Ballard & Knutson, 2009).

Larger change in delay discounting AUCs also was seen in individuals with larger white matter volumes in the right caudal anterior cingulate gyrus, right isthmus of the cingulate gyrus, and right cuneate gyrus (controlling for baseline discounting, age, sex, intelligence, and total brain volume). The anterior cingulate may be particularly sensitive to immediate rewards (McClure et al., 2004, 2007) and increased activation in this area is associated with steeper discounting (Shamosh et al., 2008). Although few investigators predicted a priori that occipital cortex would be involved in delay discounting, there have been several replications of this finding in the adult fMRI literature, although the direction of influence has varied (e.g. Wittman et al., 2007; Bickel et al., 2009).

Larger change in delay discounting AUCs also was seen in individuals with larger hippocampal volume (controlling for baseline discounting, age, sex, intelligence, and

total brain volume). Adult fMRI studies have indicated that the hippocampus plays a role in the evaluation of immediately available rewards (McClure et al., 2004, 2007).

In terms of the DTI findings, larger change in delay discounting AUCs also was seen in individuals with lower MD in a cluster involving fibers from the fornix, stria terminalis, and anterior thalamic radiation in the left posterior thalamus/ hippocampus (without controlling for age, sex, or intelligence). Again, the hippocampus may play a role in evaluating immediately available rewards (McClure et al., 2004, 2007). Additionally, greater change in delay discounting was seen in individuals in the younger age group with higher FA in a cluster involving fibers from the left inferior fronto-occipital fasciculus/ anterior thalamic radiation/ uncinate fasciculus/ superior longitudinal fasciculus in the inferior frontal gyrus (pars opercularis and triangularis)/ middle frontal gyrus (without controlling for age, sex, or intelligence). Again, activity in the middle frontal gyrus is generally associated with less steep discounting (Shamosh et al., 2008), and the inferior frontal gyrus tracks both reward magnitude and delay (Ballard & Knutson, 2009).

On the other hand, several analyses revealed areas where less mature patterns of brain development at time 1 were associated with larger delay discounting AUCs at time 2, controlling for baseline discounting. Larger delay discounting AUCs (controlling for baseline discounting) were seen in individuals with thicker cortical thickness in the left parahippocampal gyrus (controlling for age, sex, and intelligence). The parahippocampal

gyrus may play a role in biasing decisions in favor of smaller sooner rewards (Boettiger et al., 2007). Larger changes in delay discounting AUCs also were seen in individuals with lower FA in a cluster including fibers from the right anterior thalamic radiation/ corticospinal tract in the region of the amygdala/ pallidum/ hippocampus (controlling for age, sex, and intelligence). Regions of the parahippocampal gyrus near the amygdala are implicated in biasing decisions toward smaller sooner rewards (Boettiger et al., 2007). Greater changes in delay discounting AUCs also were seen in individuals in the younger age group with lower FA in a cluster involving fibers from the right superior longitudinal fasciculus/ inferior longitudinal fasciculus/ inferior fronto-occipital fasciculus/ anterior thalamic radiation in the region of the lateral occipital cortex/ angular gyrus/ precuneus/ superior parietal lobule (controlling for age, sex, and intelligence). The occipital cortex and precuneus have previously been identified as areas that are active during decision-making on delay discounting tasks (Bickel et al., 209; McClure et al., 2004, 2007); the precuneus is active when immediate rewards are available (McClure et al., 2004, 2007). On the other hand, the angular gyrus is more active when choosing the delayed reward (Wittman et al, 2007).

4.2.1 Probability discounting.

Larger time 2 probability AUCs after controlling for baseline discounting were associated with more mature patterns of brain development at time 1, including larger white matter volume in the left rostral anterior cingulate gyrus (controlling for age, sex, intelligence, and total brain volume). The anterior cingulate may be particularly sensitive to immediate

rewards (McClure et al., 2004, 2007) and increased activation in this area is associated with steeper delay discounting (Shamosh et al., 2008).

On the other hand, DTI analyses revealed areas where less mature patterns of brain development at time 1 were associated with larger probability discounting AUCs at time 2, controlling for baseline discounting. Larger probability discounting AUCs (controlling for baseline discounting) were seen in individuals with higher MD in a cluster involving fibers from the left inferior longitudinal fasciculus/ inferior fronto-occipital fasciculus/ forceps major in the lateral occipital cortex/ cuneus/ precuneus (controlling for age, sex, and intelligence). The occipital cortex and precuneus have previously been identified as areas that are active during decision-making on delay discounting tasks (Bickel et al., 209; McClure et al., 2004, 2007); the precuneus is active when immediate rewards are available (McClure et al., 2004, 2007).

4.2.3 Discussion of directionality of imaging findings.

As described above, the regressor variable approach identifies factors associated with time 2 change that is larger or smaller than would have been predicted based on time 1 performance alone, essentially identifying variables at time 1 that are associated with greater change after adjusting for baseline performance. Thus, the imaging analyses allow the identification of time 1 structural brain features that are associated with a greater increase in time 2 delay AUC (i.e., more patient performance) than would have been predicted based solely on time 1 delay AUC. These features included thinner cortex in a

variety of right prefrontal and frontal areas, larger white matter volumes in the cingulate and cuneate gyri, and larger hippocampal volumes (as well as *thicker* cortical thickness in the left parahippocampal gyrus).

Before controlling for age, sex, and intelligence, larger than predicted time 2 delay AUCs also were associated with lower MD (reflecting increased tissue density) in a left hippocampal/ thalamic cluster including fibers from the fornix, stria terminalis, and anterior thalamic radiation; and with higher FA (reflecting increased density or packing of fiber tracts or increased parallel tract organization) in a cluster in the left middle and inferior frontal gyri involving fibers from the left IFOF, ATR, UF, and SLF. These findings are in the predicted direction and are consistent with the Freesurfer findings, which (with one exception) indicate that greater than predicted maturation of delay discounting behavior is found in individuals with more mature patterns of brain development at time 1.

However, after controlling for age, sex, and intelligence, the direction of the DTI findings reversed, with larger than predicted time 2 delay AUCs associated with *less* mature patterns of organization at time 1. Specifically, larger than predicted time 2 delay AUCs were associated with *lower* FA in a cluster in the right amygdala/ pallidum/ hippocampus including fibers from the right ATR and CST. In the younger age group, larger than predicted time 2 delay AUCs were associated with *lower* FA in the region of the right

lateral occipital cortex/ angular gyrus/ precuneus/ superior parietal lobule (right temporal-parietal-occipital junction) in a cluster involving fibers from the SLF/ ILF/ IFOF/ ATR.

Examining relationships between DTI variables and time 2 discounting controlling for baseline discounting but without controlling for age, sex, and intelligence allows for the identification of clusters where age-related differences in FA or MD contribute to changes in discounting. Controlling for baseline discounting allows for the identification of clusters where non-age-related differences in FA or MD contribute to changes in discounting. Findings were generally in the predicted direction for the analyses that did not control for age, and were in the reversed direction for the analyses that did control for age, suggesting opposing influences of age-related and non-age-related individual differences in white-matter organization on discounting.

There are two possible explanations for the reversed-direction findings in the analyses controlling for age. One is that if these regions support underlying processes that would favor the selection of immediate rewards (rather than larger delayed rewards), then their immaturity may bias decisions in favor of larger delayed rewards. An alternate explanation is that these analyses may be identifying individuals who have 'room to grow' from time 1 to time 2; in essence, immature individuals at time 1 may experience less of a ceiling effect on their delay discounting behavior and may be more apt to make large gains from time 1 to time 2. Eventually, consideration of imaging data from both time points may allow for differentiation of these explanations. If the first explanation

were true (and the identified immature regions promote selection of larger later rewards), then low FA/ high MD at time 2 also should correlate with higher time 2 delay AUCs. If the second explanation were true (and the immaturity at time 1 is associated with greater 'room to grow' from time 1 to time 2), then the amount of increase in FA (or decrease in MD) in these regions from time 1 to time 2 should correlate with the amount of increase in delay AUC.

Although these explanations cannot be definitively teased apart without examination of two-timepoint imaging data, several pieces of evidence suggest that the first explanation may account for the findings. If the 'room to grow' explanation were true for DTI data, it might also be expected to hold for the Freesurfer findings. However, those findings are most consistent with the opposite pattern, where greater time 1 brain structural maturity predicts greater growth in delay discounting AUCs. In addition, the regions identified in the DTI analyses where less maturity at time 1 is associated with greater change in discounting from time 1 to time 2 are regions that have been identified in the adult fMRI literature as promoting the selection of smaller sooner rewards. The first cluster is near the hippocampus and amygdala; parahippocampal regions near the amygdala play a role in biasing decisions in favor of smaller sooner rewards (Boettiger et al., 2007). The second cluster is in the temporal-parietal-occipital junction; parietal regions may bias decisions in favor of smaller sooner rewards (McClure et al., 2004, 2007). Immaturity of these regions may therefore reflect less strong biasing in favor of smaller immediate reward selection.

4.2.4 Discussion of regions and pathways implicated by imaging findings

In our previous report of single time point associations between DTI measures and discounting performance in adolescents (Olson et al., 2009), we found that larger delay AUCs were associated with high FA and low MD in a right frontal cluster (inferior frontal gyrus/ frontal pole) containing fibers from the ATR, IFOF, UF, forceps minor, and SLF; with high FA in a large left hemisphere temporal cluster including fibers from the IFOF, ILF, ATR, and UF; and with low MD in a cluster near the right amygdala and globus pallidus including fibers from the ATR and CST.

In the present study, we again found evidence that right prefrontal areas are involved in delay discounting performance in adolescents and extended this finding to demonstrate that the baseline maturity of these regions predicts the extent of change in delay discounting over time. Thinner cortical thickness in right prefrontal regions at time 1 was associated with a larger change in delay discounting AUC from time 1 to time 2. In addition, higher FA in the right inferior frontal gyrus was associated with a larger change in delay discounting AUC from time 1 to time 2 in the younger age group (although this finding did not hold after additionally controlling for age, sex, and intelligence). The right inferior frontal gyrus tracks the interaction of delay and reward magnitude in adults (Ballard & Knutson, 2009); higher baseline levels of maturation in this region may precede and predict maturation of delay discounting behavior in adolescents.

In contrast, the present study indicates only limited evidence of relationships between left temporal lobe maturation and delay discounting performance, which were striking in the previous single time point study. Associations were evident between change in delay discounting and time 1 hippocampal volume (bilaterally), cortical thickness in the left parahippocampal gyrus, and MD in a region overlapping the hippocampus and thalamus including fibers from the fornix, stria terminalis, and ATR. However, there were no relationships between delay discounting and more lateral temporal regions such as the inferior and superior temporal gyri. The discrepancy between the previous and the present findings might be interpreted as suggesting that left (lateral) temporal lobe functioning influences overall delay discounting rates through a process not associated with development or change in this age range. The present study did implicate the right temporal-parietal-occipital junction in developmental change in delay discounting; in the younger age group, lower FA in the right SLF, ILF, IFOF, and ATR in this region was associated with greater change in delay discounting.

The TPO junction is an association area with complex functions, including the integration of spatial and motion information and in exploring space (Karnath, 2001). Additionally, this is a region where a number of long cortico-cortical pathways (including the SLF, ILF, IFOF, and ATR) are in close proximity. The SLF has four components. These connect the superior parietal cortex to the DLPFC (playing a role in regulating complex motor behavior); the angular gyrus to the lateral prefrontal cortex (regulating visual spatial attention); the supramarginal gyrus to ventral premotor and prefrontal regions

(regulating motor actions); and the superior temporal gyrus to the lateral PFC (this latter branch is also called the arcuate fasciculus, and is involved in auditory spatial processing) (Makris et al., 2005). The ILF is an occipito-temporal pathway that may be involved in memory for visual information and in visual object ('what') processing (Catani, Jones, Donato, & ffytche, 2003; Qui et al., 2008), while the IFOF is an occipito-frontal pathway (Catani et al., 2003). The ATR projects from the dorsomedial nucleus of the thalamus to prefrontal regions (Wakana, Jiang, Nagae-Poetscher, van Zijl, & Mori, 2004).

The present study also implicates additional areas in the maturation of delay discounting behavior, including the cuneate gyrus, cingulate gyrus (isthmus and anterior portions), and amygdala/pallidum. Involvement of occipital cortex (cuneate gyrus) in delay discounting processes has been previously reported, although no coherent theoretical framework has been offered to explain this association (e.g. Wittman et al., 2007; Bickel et al., 2009). The anterior cingulate plays a role in a variety of functions that are closely related to delay discounting, including influencing future behavior based upon past histories of reinforcement and deciding a course of action based on past outcomes (Kennerley, Walton, Behrens, Buckley, & Rushworth, 2006). It may be particularly sensitive to immediate rewards and may bias decisions toward steeper delay discounting (McClure et al., 2004, 2007; Shamosh et al., 2008). Additionally, MD in fibers from the ATR and CST in the region of the amygdala and pallidum was associated with change in discounting; as discussed above, the ATR relays information between the thalamus and prefrontal regions, and the CST is involved in motor output.

As in our previous single time point report, findings were much more limited for probability discounting than for delay discounting. Again, there was no relationship between changes in probability discounting and right frontal brain structure, providing additional evidence that delay discounting and probability discounting reflect at least partially different processes. Larger time 2 probability discounting AUCs (reflecting lower risk aversion) were seen in individuals with larger anterior cingulate white matter volumes at time 1, after controlling for baseline probability discounting. This is consistent with the view that the anterior cingulate is implicated in reward-seeking behavior (Kennerley et al., 2006). In addition, individuals with larger time 2 probability discounting AUCs had higher PD in a left occipito-parietal cluster involving fibers from the left ILF, IFOF, and forceps major (which connects the two occipital lobes). This cluster is in a similar location to the right TPO junction cluster that was significantly associated with change in delay discounting in the younger group, though it is in the left rather than the right hemisphere.

4.3 Developmental Implications

This study has identified patterns of change and stability in delay and probability discounting over the course of adolescence and has identified factors associated with greater change in discounting rates after adjusting for baseline performance. For delay discounting, these factors include symptoms of psychopathology (internalizing for younger participants; externalizing for older participants); the personality trait of positive

emotionality (particularly related to agentic behavior and achievement, as well as wellbeing in younger participants); right frontal cortical thickness; cingulate and cuneus white matter volumes; hippocampal volumes; parahippocampal thickness; FA in the TPO junction, and FA in the right amygdala/ pallidum/ hippocampus. This set of factors indentifies participants who might be expected to have higher-than-average decreases in the delay discounting rate over a two-year period during adolescence. These findings suggest that personality and psychopathology, as well as brain maturation, are related to intra-individual changes in delay discounting during adolescence. A larger number of factors predicting greater than expected change in delay discounting were identified in younger than in older participants. Interestingly, cognitive performance was not related to intra-individual changes, even though it was related to single time point delay discounting behavior in adolescents in a previous study (Olson et al., 2007). This raises the possibility that cognitive factors are more related to the establishment of baseline discounting rates prior to adolescence than to developmental changes, while personality functioning and psychopathology, which were not related to single time point performance, may be related to development.

For probability discounting, factors associated with change from time 1 to time 2 included female sex (in the younger group of participants), working memory performance on the digits backward task (in males only), cingulate gyrus white matter volumes, and mean diffusivity in left parieto-occipital regions. Importantly, probability discounting did not show any evidence of overall age-related changes, so these factors may reflect non-

developmental influences on change in the probability discounting rate. That is, some individuals did show evidence of change in the probability discounting rate from time 1 to time 2, and these individuals can be identified using the factors described above. The finding of sex differences in change in the probability discounting rate in adolescents is notable, particularly since single time point cross-sectional studies have generally not revealed sex differences in probability discounting. Again, the factors associated with change in the probability discounting rate may differ from the factors involved in establishing a pre-adolescent probability discounting baseline. Directly examining single time point versus cross time point influences within the same sample is an important direction for future research, though current findings provide some support for the idea that for both delay and probability discounting, there may be separate sets of factors influencing the baseline discounting rate versus change during adolescence.

Although delay discounting was once conceptualized as essentially an operationalization of the concept of impulsivity (Ainslie, 1975), recent work suggests that delay discounting reflects other underlying processes as well. For instance, multiple studies have demonstrated its weak relationship to measures of behavioral inhibition and stronger relationship to intelligence (e.g. Olson et al., 2007; Steinberg et al., 2009), which would not be expected if it were merely a straightforward measure of impulsivity. Similarly, its relationship to other measures of reward-seeking behavior (including tasks such as the Iowa Gambling Task and the Balloon Analogue Risk Task) is typically weak and often explained by relationships with intelligence (e.g. Olson et al., 2007; Steinberg et al.,

2009). Delay discounting most likely reflects impulsivity but also future orientation (Mitchell et al., 2007; Steinberg et al., 2009), or the tendency to consider future outcomes in decision-making processes. The present study indicates that delay discounting also is related to positive emotionality, which is consistent with the finding that Agreeableness is associated with lower delay discounting rates (Miller et al., 2008). Delay discounting rates may be low when positive future outcomes (of future reward delivery) are considered or expected, and they may be high when future outcomes are anticipated to be negative or are not considered at all. The present study suggests that the development of the process of turning one's attention to the future and anticipating positive or negative outcomes is shaped by a variety of factors during adolescence, including personality, brain maturation, and internalizing/externalizing behavior. Probability discounting is less well understood but most likely reflects risk-seeking versus risk aversion. Unlike valuation of future outcomes, risk aversion does not appear to develop during adolescence and may instead reflect a more stable and longstanding trait. The personality, cognitive, and brain structural factors that affect discounting processes may interact with one another, and the outcomes of reward-related decisions may affect future decisions, so that the experience of success with waiting for and receiving delayed rewards may ultimately feed back and influence subsequent decisions, producing the adolescent decline in the delay discounting rate. The present study suggests that positive emotionality, lack of internalizing/externalizing behavioral problems, and greater brain maturation (and/or perhaps less maturation in regions that bias decisions in favor of smaller sooner rewards) may help to accelerate this process within individuals.

4.4 Limitations

4.4.1 Study participants.

The ability to assess relationships between cognitive factors, behavioral factors, and demographic factors may be limited by nature of the sample, which is predominantly white, of relatively high socioeconomic status, and of above-average intelligence. These associations between discounting, age, intelligence, personality, and white matter organization should be examined in groups with average or low-average intelligence to determine their generalizability. Steinberg et al. (2009) found similar patterns of association in a very large cross sectional study with a more socioeconomically diverse sample of adolescents; delay discounting rates were higher in younger participants and in those with lower IQ.

4.4.2 Statistical analyses.

Given that data were available from only two time points and that only one set of imaging data could be analyzed at a time, the analyses in the current study focused on the prediction of time 2 discounting based on behavioral and brain structural factors available at time 1, adjusting for baseline discounting rate. Examining the relationship between change in discounting and change in imaging variables is an important future direction. Analysis of relationships between change scores may be best conducted using hierarchical linear modeling (HLM), a technique which permits modeling of data that is

missing from one timepoint and has greater power when working with data from multiple timepoints.

4.4.3 Imaging analyses.

The SPM imaging analysis was not stringently corrected for multiple comparisons (e.g. by using the False Discovery Rate technique). A slightly more lenient threshold in terms of cluster size was used than was used in the previous single-timepoint report. It was felt that the cross-timepoint analysis warranted a slightly less stringent approach. However, the statistical parameters ($p < .001$, extent threshold ≥ 20 voxels) remain more stringent than those used in some previous published analyses of DTI variables (e.g. Snook et al., 2007: $p < .05$, extent threshold ≥ 10 voxels, no correction for multiple comparisons). In addition, we did not use tractography to more precisely localize significant clusters in relation to known white matter pathways in order to reduce the possibility of type I error. Restricting the SPM analysis only to those voxels where FA exceeds .2 in every participant's scan additionally helps to restrict the analysis to white matter and thereby reduce the risk of type I error. Visual inspection of these clusters indicates that they are resting on white matter versus areas with higher concentrations of gray matter or cerebrospinal fluid. The large number of correlations involved in the analysis of the Freesurfer data also was not corrected for multiple comparisons, due to the exploratory nature of these analyses.

Unfortunately, methodological limitations related to a scanner upgrade caused substantial difficulties in analyzing the DTI imaging data longitudinally. Although a small sample of adults was scanned pre- and post-upgrade, attempts to derive a correction factor have proven extremely complicated. Additionally, a correction factor derived in adults may or may not adequately correct for differences in signal intensity in children. Attempts to address this issue are ongoing but are beyond the scope of the present work.

4.5 Unanswered Questions

As discussed above, the present study does not directly address the question of whether separate sets of factors influence the baseline discounting rate versus intra-individual change during adolescence. Additionally, due to methodological limitations, the relationship between *change* in DTI measures and change in discounting could not be addressed; because of this limitation, the analysis of behavioral and neuroimaging data focused only on the identification of factors that predict greater than average change from the discounting baseline. Relationships between delay discounting and the onset of psychopathology (including substance use) during adolescence also were not addressed. Finally, the study does not address whether there is a cumulative (additive or multiplicative) effect of the factors that are associated with change in discounting, and/or which factors have the greatest influence. Identifying which factors are most influential and examining the interactions amongst them may help to clarify which forces contribute most strongly to adolescent change in delay and probability discounting.

4.6 Future Directions

Expanding the age range, demographic diversity, range of psychopathology, and number of longitudinal assessments would extend and further develop the present study's findings. Extending the sample to include younger participants would be particularly interesting, since probability discounting has rarely been examined in children under age 11. The sex differences in probability discounting observed in the present study should be examined with respect to pubertal development; it would be interesting to assess whether or not these differences in probability discounting are present prior to puberty. Assessing the relationships between discounting and personality, cognition, and brain development in populations with a greater range of racial and ethnic diversity, with lower socioeconomic status, and/ or with lower intellectual capacity is important in order to assess whether demographic factors may have influenced the present findings. Examining relationships between discounting and neurocognitive and personality development in adolescent populations with significant psychopathology is another important future goal. Additionally, it would be ideal to follow participants throughout three or more longitudinal assessments over the course of adolescent development in order to examine the shape of the change curves using hierarchical linear modeling.

Extending the range of discounting paradigms assessed with respect to neurodevelopmental changes in adolescence to include social discounting paradigms is another future goal. Social discounting tasks (Jones & Rachlin, 2006, 2009) ask individuals to imagine developing a list of 100 people they know, with the person in the

first slot being the person they know best (or at the closest "social distance"). They are then asked whether they would prefer to receive a small amount of money themselves or for an acquaintance at a given "social distance" to receive a larger amount of money. For instance, questions would be framed as, "Would you rather have $5 for yourself or $50 for person #10?" In this way, discounting curves can be constructed. Steep social discounting rates reflect less social generosity while shallow rates reflect greater generosity. Contrary to researchers' expectations, preferences on the social discounting task often become extremely "irrational" at very close social distances, so that individuals may prefer for close friends and family to receive money rather than to receive money themselves (Jones & Rachlin, 2006, 2009). Social discounting AUCs are positively correlated with delay and probability discounting AUCs in adults (Jones & Rachlin, 2009). Jones & Rachlin also found higher rates of social discounting in older adult participants versus undergraduates, suggesting a possible age effect (although the older participants also were MBA students and may have therefore differed from the undergraduates along important dimensions besides age). Social discounting has not been examined in adolescence. Given adolescent changes in delay discounting as well as the rapid and dramatic changes in social behavior that occur during adolescence, examining social discounting in adolescents is another important future goal.

From a theoretical perspective, it also is interesting that others have found that delay, probability, and social discounting AUCs are positively correlated in adults (Jones & Rachlin, 2009). The present study illustrated that delay discounting is related to positive

emotionality in adolescents, and others have found relationships between other personality traits related to positivity and delay discounting. One possible explanation is that all three types of discounting may relate to aspects of optimism. Discounting rates will be low when individuals have faith that the desired outcome (delivery of delayed rewards, winning improbable rewards, rewards delivered via social reciprocity) will come to pass. Although delay discounting and probability discounting are incompletely overlapping, some of the overlap that does exist may relate to positive expectancy.

4.7 Summary

Adolescence is a time of rapid change in neurobehavioral characteristics, including emotional functioning, cognitive performance, and brain structure and function. Delay and probability discounting tasks were used to assess factors contributing to change in decision-making, using a two-year longitudinal design, in adolescents age 9 to 23 at baseline. Delay discounting, but not probability discounting, showed significant maturation within individuals over a two-year period. Behavioral factors predisposing toward greater than expected maturation in delay discounting included lower internalizing and externalizing psychopathology and higher positive emotionality. Brain structural factors predisposing toward greater than expected maturation included lower right frontal cortical thickness, larger cinguate and cuneate white matter volumes, larger hippocampal volumes, thicker parahippocampal gyrus cortical thickness, lower fractional anisotropy in the right temporal-parietal-occipital junction, and lower fractional anisotropy in the right amygdala/ pallidum/ hippocampus. Behavioral factors

predisposing toward greater than expected change in probability discounting included female sex (for younger participants) and working memory performance (for males). Brain structural factors predisposing toward greater than expected change included cingulate white matter volume and higher mean diffusivity in the left parieto-occipital area. Findings support the conceptualization of delay and probability discounting as incompletely overlapping processes. Given discrepancies with previous single time point research, it is hypothesized that separate sets of factors may influence baseline discounting versus change in the discounting rate during adolescence.

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6. Appendix: Tables and Figures

Table 1. Summary of delay discounting fMRI/ sMRI findings in healthy adults.

Region	**Adult fMRI/ sMRI literature**	
OFC	Active when an immediate reward is available (β system). Lateral: active during decision-making (δ system).	McClure et al., 2004, 2007
	Biases toward larger later rewards	Boettiger et al., 2007
	More active during 'hard' choices	
	Lateral: Steep discounting is associated with less activation on another reward-related task	Hariri et al., 2006
medial PFC	Active when an immediate reward is available (β system)	McClure et al., 2004, 2007
	Tracks subjective value	Kable & Glimcher, 2007
	Primarily tracks reward magnitude	Ballard & Knutson, 2009
	Steep discounting is associated with more activation on another reward-related task	Hariri et al., 2006
DLPFC	Active during decision-making (δ system).	McClure et al., 2004, 2007
	Primarily tracks reward delay	Ballard & Knutson, 2009
	More active during 'hard' choices	
	Greater activity corresponds to steeper discounting	Bickel et al., 2009
	Lateral: Steep discounting is associated with less activation on another reward-related task	Hariri et al., 2006
	Steep discounting associated with smaller volume	Bjork et al., 2009
VLPFC	Active during decision-making (δ system).	McClure et al., 2004, 2007
	More active during 'hard' choices	
lateral PFC	Active when making choices	Bickel et al., 2009
inferolateral PFC	Steep discounting associated with smaller volume	Bjork et al., 2009
dorsal PFC (sup. frontal gyrus)	Biases toward smaller sooner rewards	Boettiger et al., 2007
inferior frontal gyrus	Tracks both reward delay and reward magnitude	Ballard & Knutson, 2009
	If more active for short versus long delays, steeper discounting	Wittman et al, 2007
	If more active for short versus long delays, shallower discounting	Wittman et al., 2007
middle frontal gyrus	Steep discounting is associated with less activation on a working memory task	Shamosh et al., 2008
premotor area	Active during decision-making (δ system).	McClure et al., 2004, 2007
	Active when making choices	Bickel et al., 2009

supplementary motor area	Active during decision-making (δ system).	McClure et al., 2004, 2007
	More active during 'hard' choices	
	Active when making choices	Bickel et al., 2009
subgenual cingulate	Active when an immediate reward is available (β system)	McClure et al., 2004, 2007
anterior cingulate	Active when an immediate reward is available (β system)	McClure et al., 2004, 2007
	More active during 'hard' choices	
	Steep discounting is associated with more activation on a working memory task	Shamosh et al., 2008
posterior cingulate	Active when an immediate reward is available (β system)	McClure et al., 2004, 2007
	Tracks subjective value	Kable & Glimcher, 2007
	Primarily tracks reward magnitude	Ballard & Knutson, 2009
	More active when choosing the delayed reward	Wittman et al., 2007
	More active during 'hard' choices	
	If more active for short versus long delays, steeper discounting	Wittman et al., 2007
	Active when making choices	Bickel et al., 2009
cingulate		
insula	(Posterior): More active when choosing the delayed reward	Wittman et al., 2007
	More active during 'hard' choices	
	Active when making choices	Bickel et al., 2009
ventral striatum (nucleus accumbens)	Active when an immediate reward is available (β system)	McClure et al., 2004, 2007
	Tracks subjective value	Kable & Glimcher, 2007
	Primarily tracks reward magnitude	Ballard & Knutson, 2009
	Steep discounting is associated with more activation on another reward-related task	Hariri et al., 2006
striatum	Active when making choices	Bickel et al., 2009
caudate	If more active for short versus long delays, steeper discounting	Wittman et al., 2007
posterior hippocampus	Active when an immediate reward is available (β system)	McClure et al., 2004, 2007
parahippocampal gyrus (near the amygdala)	Biases toward smaller sooner rewards	Boettiger et al., 2007
middle and inferior temporal areas	Biases toward smaller sooner rewards	Boettiger et al., 2007
superior temporal gyrus	More active when choosing the delayed reward	Wittman et al., 2007
	If more active for short versus long delays, steeper discounting	Wittman et al., 2007

temporal lobe	Steep discounting is associated with more activation on a working memory task	Shamosh et al., 2008
temporal-parietal junction	Primarily tracks reward delay	Ballard & Knutson, 2009
angular gyrus	More active when choosing the delayed reward	Wittman et al., 2007
intraparietal cortex	Active during decision-making (δ system).	McClure et al., 2004, 2007
inferior parietal	More active when choosing the delayed reward	Wittman et al., 2007
parietal	More active during 'hard' choices	
	Active when making choices	Bickel et al., 2009
posterior parietal areas	Biases toward smaller sooner rewards	Boettiger et al., 2007
	Primarily tracks reward delay	Ballard & Knutson, 2009
precuneus	Active when an immediate reward is available (β system)	McClure et al., 2004, 2007
	Active when making choices	Bickel et al., 2009
cuneus	More active when choosing the delayed reward	Wittman et al., 2007
	More active during 'hard' choices	
	If more active for short versus long delays, steeper discounting	Wittman et al., 2007
	Greater activity corresponds to steeper discounting	Bickel et al., 2009
lingual gyrus	If more active for short versus long delays, steeper discounting	Wittman et al., 2007
visual cortex	Active during decision-making (δ system).	McClure et al., 2004, 2007
	Active when making choices	Bickel et al., 2009
cerebellum	Biases toward smaller sooner rewards	Boettiger et al., 2007
	Greater activity corresponds to steeper discounting	Bickel et al., 2009

Table 2. Personality and psychopathology variables: T scores based on MPQ adult normative sample and ASEBA age-appropriate norms for subjects with valid time 1 probability or delay discounting data age 11 and up at time 1.

	N	Mean	Standard deviation
MPQ: wellbeing	116	55.51	8.637
MPQ: social potency	116	55.37	7.382
MPQ: achievement	116	50.56	8.517
MPQ: social closeness	116	56.19	9.223
MPQ: stress reaction	116	43.90	8.743
MPQ: alienation	116	52.53	8.169
MPQ: aggression	116	51.80	9.741
MPQ: control	116	49.14	11.110
MPQ: harm avoidance	116	42.14	9.796
MPQ: traditionalism	116	45.17	7.178
MPQ: absorption	116	50.69	9.595
MPQ: PEM	116	56.31	9.371
MPQ: NEM	116	48.90	9.899
MPQ: CON	116	43.18	9.318
PEM agentic	116	53.51	8.546
PEM communal	116	57.47	10.089
NEM agentic	116	47.99	10.956
NEM alienated	116	49.32	9.838
ASEBA withdrawal	115	52.96	5.259
ASEBA somatic complaints	115	52.43	4.132
ASEBA anxiety/ depression	115	52.79	4.802
ASEBA intrusivity	50	53.84	5.765
ASEBA thought problems	115	52.18	4.299
ASEBA social problems	65	53.69	5.181
ASEBA attention problems	115	53.70	4.935
ASEBA delinquency	115	52.55	3.943
ASEBA aggression	115	52.12	3.885
ASEBA internalizing	115	45.87	9.680
ASEBA externalizing	115	46.74	8.516
ASEBA total problems	115	45.70	9.025

[1]Variables transformed prior to analysis to normalize

Table 3. Demographics: Entire Sample

Characteristic	Time 1: Total	Time 1: Younger	Time 1: Older	Group Diff.: Time 1	Time 2: Total	Time 2: Younger	Time 2: Older	Grp Diff: Time 2
*N**	137	68	69		120	65	55	
% female	51.8	45.6	58.0	χ^2= 2.103, *ns*	53.3	46.2	61.8	χ^2= 2.103, *ns*
Age (years)	16.18 (3.91)	12.92 (2.26)	19.40 (2.10)	*t*(135) = -17.411***	17.88 (3.89)	14.85 (2.22)	21.45 (1.87)	*t*(118) = -17.440***
Mo.'s Ed. (yrs)	15.74 (2.09)	16.16 (1.56)	15.30 (2.47)	*t*(105.733) = 2.391*	15.81 (2.07)	16.08 (1.51)	15.46 (2.60)	*t*(74.456) = 1.498, *ns*
Fa.'s Ed. (yrs)	16.10 (2.84)	16.15 (2.60)	16.05 (3.09)	*t*(127) = .207, *ns*	16.18 (2.80)	16.28 (2.55)	16.06 (3.09)	*t*(97.149) = .419, *ns*
Income (median)	80,000	80,000	87,500	*U*= 1539.5, *ns*	97,500	100,000	92,500	*U* = 1048.5, *ns*
WASI Voc. T	59.76 (7.178)	59.55 (7.36)	59.97 (7.05)	*t*(134) = -.339, *ns*	59.64 (6.91)	58.70 (7.62)	60.73 (5.86)	*t*(117) = -1.604, *ns*
WASI MR T	55.49 (7.168)	56.79 (6.98)	54.24 (7.18)	*t*(133) = 2.085, *ns*	56.19 (6.81)	54.30 (7.10)	58.40 (5.76)	*t*(117) = -3.425**
WASI FSIQ (est.)	113.47 (10.59)	114.45 (11.02)	112.52 (10.15)	*t*(133) = 1.060, *ns*	114.00 (9.97)	111.45 (10.82)	116.96 (7.80)	*t*(114.515) = -3.186**

** N reflects number of participants having valid delay and/ or probability discounting data at that time point.*

Values represent percentages or means (and standard deviations). Degrees of freedom vary because some participants omitted responses.

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

Table 4. Number of consistent and inconsistent respondents for delay discounting (above) and probability discounting (below) at each time point

	Time 2: No Data	Time 2 DD: Consistent	Time 2 DD: Inconsistent	Total
Time 1 DD: Consistent	19	85	18	122
Time 1 DD: Inconsistent	2	13	4	19
Total	21	98	22	141

	Time 2: No Data	Time 2 PD: Consistent	Time 2 PD: Inconsistent	Total
Time 1 PD: Consistent	16	91	3	110
Time 1 PD: Inconsistent	3	7	0	10
Time 1 PD: Not Administered	2	18	1	21
Total	21	116	4	141

Table 5. Descriptive statistics for study variables for delay discounting analyses (all are time 1 except for time 2 delay discounting). N=122 with valid time 1 discounting.

	N	Mean	Standard deviation	Skewness	SE skewness	Kurtosis	SE kurtosis
Delay AUC: T1	122	.478	.290	.099	.219	-1.230	.435
Delay AUC: T2	98	.550	.287	-.198	.244	-1.257	.483
Age	122	16.051	3.976	-.046	.219	-.933	.435
Sex	122	1.51	.502	-.033	.219	-2.032	.435
Vocab	121	60.05	6.709	-.021	.220	.106	.437
Matrix Reas.	120	55.76	6.828	-1.040	.221	2.181	.438
IQ (2 subtests)	120	113.90	9.827	-.281	.221	.312	.438
IGT: good-bad	119	4.08	29.096	.382	.222	.300	.440
IGT: freq-infreq	119	-24.740	26.547	-.179	.222	-.287	.440
Digits back.	122	7.07	2.525	.626	.219	-.178	.435
ASEBA internal.[1]	120	.306	.146	.329	.221	.192	.438
ASEBA extern.[1]	120	.288	.141	.114	.221	-.248	.438
MPQ PEM[1]	103	2.031	.586	.137	.238	-.354	.472
MPQ NEM[1]	103	3.436	.402	.140	.238	-.537	.472
MPQ CON	103	75.42	15.363	-.346	.238	-.576	.472
PEM agentic[1]	103	1.410	.342	.161	.238	-.524	.472
PEM communal[1]	103	1.819	.443	-.522	.238	-.418	.472
NEM agentic[1]	103	3.747	.321	.297	.238	-.494	.472
NEM alienated[1]	103	3.485	.337	.500	.238	-.283	.472

[1]Variables transformed prior to analysis to normalize

Table 6. Descriptive statistics for study variables for probability discounting analyses (all are time 1 except for time 2 probability discounting). N=110 with valid time 1 discounting.

	N	Mean	Standard deviation	Skewness	SE skewness	Kurtosis	SE kurtosis
Probability AUC: T1	110	.443	.193	.166	.230	.082	.457
Probability AUC: T2	116	.440	.178	.390	.225	.242	.446
Age	110	17.142	3.331	.110	.230	-.872	.457
Sex	110	1.48	.502	.074	.230	-2.032	.457
Vocab	110	59.87	7.225	-.488	.230	.498	.457
Matrix Reas.	110	54.73	7.201	-1.335	.230	2.799	.457
IQ (2 subtests)	110	112.86	10.498	-.798	.230	1.210	.457
IGT: good-bad	108	8.04	30.763	.153	.233	1.239	.461
IGT: freq-infreq	108	-25.352	25.900	-.392	.233	.103	.461
Digits back.	110	7.45	2.565	.477	.230	-.364	.457
ASEBA internal.[1]	107	.302	.143	.266	.234	.449	.463
ASEBA extern.[1]	107	.308	.131	.144	.234	-.065	.463
MPQ PEM[1]	108	1.995	.586	.133	.233	-.400	.461
MPQ NEM[1]	108	3.417	.408	.218	.233	-.499	.461
MPQ CON	108	74.66	15.398	-.351	.233	-.601	.461
PEM agentic[1]	108	1.407	.344	.212	.233	-.517	.461
PEM communal[1]	108	1.787	.466	-.466	.233	-.580	.461
NEM agentic[1]	108	3.746	.316	.244	.233	-.610	.461
NEM alienated[1]	108	3.469	.332	.667	.233	.139	.461

[1]Variables transformed prior to analysis to normalize

Table 7. Pearson correlations among study variables for delay discounting (all are time 1 except for time 2 delay discounting). N=122 with valid time 1 discounting.

	Delay AUC: T1	Delay AUC: T2	Age	Sex	Vocab	Matrix Reasoning	IQ (2 subtest)	IGT: good-bad	IGT: frequent-infreq.	Digits backwards	ASEBA internalizing	ASEBA externalizing	MPQ PEM	MPQ NEM	MPQ CON	PEM agentic	PEM communal	NEM agentic	NEM alienated
Delay AUC: T1	1.0 (122)																		
Delay AUC: T2	.361** (85)[2]	1.0 (98)																	
Age	.344**	.315**	1.00 (122)																
Sex	.084	-.074	.085	1.00 (122)															
Vocab	.071	.215*	.031	.054	1.00 (121)														
Matrix Reas.	.003	.131	-.251**	.074	.288**	1.00 (120)													
IQ (2 subtests)	.040	.199	-.146	.080	.806**	.798**	1.00 (120)												
IGT: good-bad	.204*	.276**	.348**	-.133	.152	-.049	.062	1.00 (119)											
IGT: freq-infreq	-.159	-.165	-.102	-.172	-.113	.102	-.012	.063	1.00 (119)										
Digits back.	.201*	.239*	.462**	.143	.188*	-.031	.094	.218*	-.121	1.00 (122)									
ASEBA internal.[1]	-.046	.073	-.194*	-.018	.006	.096	.064	.093	.114	-.146	1.00 (120)								
ASEBA extern. [1]	.072	.148	.120	-.225*	-.009	-.212*	-.138	.226*	.008	.008	.510**	1.00 (120)							
MPQ PEM[1]	.031	.201	.349**	.250*	.007	-.182	-.109	-.040	-.131	.238*	-.351**	-.118	1.00 (103)						
MPQ NEM[1]	-.161	-.152	-.366**	-.202*	-.032	-.054	-.049	.066	-.034	-.245*	.492**	.523**	-.218*	1.00 (103)					
MPQ CON	-.071	-.090	.048	.183	-.095	.114	.008	-.036	.124	-.061	-.065	-.513**	.121	-.085	1.00 (103)				

PEM agentic[1]	-.004	.345**	.405**	.272**	.073	-.043	.020	-.056	-.056	.309**	-.194	-.102	.836**	-.246*	.109	1.00 (103)			
PEM communal[1]	.022	-.059	.159	.160	-.015	-.280**	-.183	.021	-.140	.143	-.399**	-.123	.736**	-.184	.068	.337**	1.00 (103)		
NEM agentic[1]	-.073	-.071	-.239*	-.259**	.020	-.036	-.005	.058	.011	-.137	.399**	.555**	-.243*	.847**	-.171	-.239*	-.213*	1.00 (103)	
NEM alienated[1]	-.197*	-.196	-.399**	-.128	-.038	-.013	-.028	.041	-.061	-.277**	.440**	.335**	-.213*	.823**	-.024	-.233*	-.200*	.421**	1.00 (103)

[1]Variables transformed prior to analysis to normalize
[2]N= 85 with valid time 1 and time 2 DD data
* $p < .05$ ** $p < .01$

Table 8. Pearson correlations among study variables for probability discounting (all are time 1 except for time 2 probability discounting). N=110 with valid time 1 discounting.

	Prob. AUC: T1	Prob. AUC: T2	Age	Sex	Vocab	Matrix Reasoning	IQ (2 subtest)	IGT: good-bad	IGT: frequent-infreq.	Digits backwards	ASEBA internalizing	ASEBA externalizing	MPQ PEM	MPQ NEM	MPQ CON	PEM agentic	PEM communal	NEM agentic	NEM alienated
Prob. AUC: T1	1.0 (110)																		
Prob. AUC: T2	.519** (91)[2]	1.0 (116)																	
Age	-.024	-.111	1.00 (110)																
Sex	-.254**	-.238**	.181	1.00 (110)															
Vocab	-.216*	-.084	.072	.042	1.00 (110)														
Matrix Reas.	-.174	-.091	.037	.052	.386**	1.00 (110)													
IQ (2 subtests)	-.226*	-.099	.059	.053	.838**	.823**	1.00 (110)												
IGT: good-bad	-.089	-.097	.205*	-.092	.292**	.202*	.299**	1.00 (108)											
IGT: freq-infreq	.105	.083	-.136	-.039	-.139	.003	-.081	-.062	1.00 (108)										
Digits back.	-.143	-.176	.372**	.177	.260**	.168	.254**	.164	-.172	1.00 (110)									
ASEBA internal.[1]	-.094	.078	-.236*	.004	.007	.058	.045	-.017	.144	-.098	1.00 (107)								
ASEBA extern.[1]	.177	.190*	-.111	-.164	.004	-.144	-.077	.094	.018	-.105	.499**	1.00 (107)							
MPQ PEM[1]	-.116	-.204*	.326**	.178	-.015	-.151	-.106	.034	-.149	.241*	-.350**	-.156	1.00 (108)						
MPQ NEM[1]	-.023	.168	-.369**	-.233*	-.123	-.138	-.145	-.075	.057	-.238*	.498**	.476**	-.244*	1.00 (108)					
MPQ CON	-.125	-.128	.064	.174	-.137	.080	-.037	-.032	.124	-.073	-.075	-.514**	.131	-.085	1.00 (108)				
PEM agentic[1]	-.215*	-.271**	.401**	.199*	.062	.013	.041	.047	-.125	.331**	-.200*	-.132	.826**	-.284**	.112	1.00 (108)			

PEM communal[1]	.029	-.088	.112	.120	-.021	-.262**	-.173	.062	-.097	.104	-.392**	-.141	.722**	-.183	.088	.326**	1.00 (108)		
NEM agentic[1]	.113	.259**	-.249*	-.278**	-.043	-.135	-.093	-.036	.105	-.152	.384**	.522**	-.212*	.877**	-.165	-.228*	-.181	1.00 (108)	
NEM alienated[1]	-.164	.035	-.404**	-.149	-.153	-.077	-.129	-.102	.005	-.270**	.503**	.324**	-.313*	.844**	-.030	-.326*	-.252*	.506**	1.00 (108)

[1]Variables transformed prior to analysis to normalize
[2]N= 91 with valid time 1 and time 2 PD data
* $p < .05$ ** $p < .01$

Table 9. Demographics: Samples with Valid Discounting Data at Both Time Points

Characteristic	DD sample	PD sample
N	85	91
% female	54.1	54.1
Age (years)	15.55 (3.91)	16.93 (3.28)
Mother's Education (years)	15.67 (2.17)	15.68 (2.25)
Father's Education (years)	16.08 (3.11)	15.92 (2.87)
Income (median)	90,000	90,000
WASI Vocabulary T	60.21 (6.67)	59.82 (7.10)
WASI Matrix Reasoning T	55.62 (7.01)	54.43 (7.51)
WASI Full Scale IQ (est.)	113.93 (9.85)	112.58 (10.62)

Values represent percentages or means (and standard deviations).

Table 10.
Hierarchical Regression Analysis: Demographic Correlates at Time 1 Predicting Delay Discounting at Time 2

Step and Variable	B	SE B	β
Step 1			
Time 1 Delay AUC	.354	.100	.361**
Step 2			
Time 1 Delay AUC	.255	.107	.260*
Age	.019	.008	.265*
Sex	-.033	.055	-.060
Step 3			
Time 1 Delay AUC	.253	.108	.257*
Age	.018	.008	.252*
Sex	-.068	.057	-.125
Age * Time 1 Delay AUC	.042	.026	.163
Sex * Time 1 Delay AUC	-.182	.108	-.185

Age * Sex	-.010	.008	-.138
Age * Sex * Time 1 Delay AUC	.040	.026	.168

Note: R^2 = .130 for Step 1 (p = .001), ΔR^2 = .061 for Step 2 (p = .054), ΔR^2 = .126 for Step 3 (p = .010) * p<.05 ** p<.01

Table 11.

Hierarchical Regression Analysis: Demographic Correlates at Time 1 Predicting Probability Discounting at Time 2

Step and Variable	B	SE B	β
Step 1			
Time 1 Probability AUC	.471	.082	.519**
Step 2			
Time 1 Probability AUC	.414	.079	.457**
Age	-.004	.005	-.076
Sex	-.100	.032	-.283**
Step 3			
Time 1 Probability AUC	.375	.086	.413**
Age	-.004	.005	-.069
Sex	-.103	.031	-.291**
Age * Time 1 Probability AUC	-.030	.026	-.115
Sex * Time 1 Probability AUC	-.052	.086	-.056

Age * Sex	.010	.005	.188*
Age * Sex * Time 1 Probability AUC	-.025	.026	-.096

Note: R^2 = .269 for Step 1 (p < .001), ΔR^2 = .092 for Step 2 (p = .003), ΔR^2 = .055 for Step 3 (p = .109) * p<.05 ** p<.01

Table 12.

Hierarchical Regression Analysis: Cognitive Correlates at Time 1 Predicting Delay Discounting at Time 2 (N = 82)

Step and Variable	B	SE B	β
Step 1			
Time 1 Delay AUC	.259	.109	.264*
Age	.018	.008	.248*
Sex	-.038	.056	-.069
Step 2			
Time 1 Delay AUC	.236	.109	.241*
Age	.015	.009	.214
Sex	-.022	.058	-.041
Full-Scale IQ (estimate)	.003	.003	.093
IGT good minus bad	.002	.001	.186
Digit span backward total	-.005	.013	-.049
Step 3			

Time 1 Delay AUC	.251	.112	.256*
Age	.016	.009	.228
Sex	-.029	.059	-.053
Full-Scale IQ (estimate)	.002	.003	.074
IGT good minus bad	.002	.001	.160
Digit span backward total	-.005	.013	-.044
Age * FSIQ	.000	.001	-.079
Age * IGT	.000	.000	.090
Age * Digits backwards	.001	.004	.016

Note: $R^2 = .182$ for Step 1 ($p = .001$), $\Delta R^2 = .042$ for Step 2 ($p = .259$), $\Delta R^2 = .015$ for Step 3 ($p = .693$)

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

Table 13.

Hierarchical Regression Analysis: Cognitive Correlates at Time 1 Predicting Probability Discounting at Time 2 (N = 90)

Step and Variable	B	SE B	β
Step 1			
Time 1 Probability AUC	.414	.080	.458**
Age	-.004	.005	-.069
Sex	-.099	.032	-.280**
Step 2			
Time 1 Probability AUC	.398	.084	.440**
Age	-.002	.005	-.031
Sex	-.099	.033	-.280**
Full-Scale IQ (estimate)	.000	.002	-.016
IGT good minus bad	.000	.001	-.038
Digit span backward total	-.005	.007	-.072
Step 3			

Time 1 Probability AUC	.398	.085	.440**
Age	.000	.005	-.007
Sex	-.102	.033	-.290**
Full-Scale IQ (estimate)	.000	.002	-.010
IGT good minus bad	.000	.001	-.052
Digit span backward total	-.005	.007	-.073
Sex * FSIQ	-.001	.002	-.066
Sex * IGT	.000	.001	-.036
Sex * Digits backward	.014	.007	.191*

Note: $R^2 = .357$ for Step 1 ($p < .001$), $\Delta R^2 = .007$ for Step 2 ($p = .809$), $\Delta R^2 = .033$ for Step 2 ($p = .228$)

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

Table 14.

Hierarchical Regression Analysis: Behavioral Correlates at Time 1 Predicting Delay AUC at Time 2 (N = 83)

Step and Variable	B	SE B	β
Step 1			
Time 1 Delay AUC	.254	.109	.260*
Age	.019	.008	.266*
Sex	-.028	.056	-.050
Step 2			
Time 1 Delay AUC	.225	.108	.230*
Age	.022	.008	.309*
Sex	-.022	.057	-.041
Internalizing	.369	.254	.178
Externalizing	.074	.247	.037
Step 3			
Time 1 Delay AUC	.209	.107	.213

Age	.028	.009	.397**
Sex	-.057	.059	-.102
Internalizing	.325	.251	.157
Externalizing	.171	.246	.085
Age * Internalizing	-.144	.069	-.290*
Age * Externalizing	.169	.082	.323*

Note: R^2 = .192 for Step 1 (p = .001), ΔR^2 = .038 for Step 2 (p = .159), ΔR^2 = .049 for Step 2 (p = .087)

* p<.05 ** p<.01

Table 15.

Hierarchical Regression Analysis: Behavioral Correlates at Time 1 Predicting Change in Probability AUC (N =88)

Step and Variable	B	SE B	β
Step 1			
Time 1 Probability AUC	.420	.082	.460**
Age	-.004	.005	-.080
Sex	-.096	.033	-.269**
Step 2			
Time 1 Probability AUC	.424	.085	.465**
Age	-.003	.005	-.050
Sex	-.096	.033	-.268**
Internalizing	.123	.135	.098
Externalizing	.026	.148	.019
Step 3			

Time 1 Probability AUC	.439	.088	.482**
Age	-.002	.005	-.046
Sex	-.095	.033	-.266**
Internalizing	.124	.136	.099
Externalizing	.026	.149	.019
Sex * Internalizing	.081	.134	.064
Sex * Externalizing	-.107	.148	-.077

Note: R^2 = .360 for Step 1 (p < .001), ΔR^2 = .011 for Step 2 (p = .489), ΔR^2 = .005 for Step 3 (p = .747)

* p<.05 ** p<.01

Table 16.

Hierarchical Regression Analysis: Personality Correlates at Time 1 Predicting Delay AUC at Time 2 (N = 70)

Step and Variable	B	SE B	β
Step 1			
Time 1 Delay AUC	.363	.114	.355**
Age	.023	.010	.275*
Sex	-.066	.059	-.122
Step 2			
Time 1 Delay AUC	.382	.115	.374**
Age	.021	.010	.249*
Sex	-.081	.061	-.148
Positive Emotionality	.100	.056	.201
Negative Emotionality	.028	.076	.043
Constraint	.000	.002	-.031
Step 3			

Time 1 Delay AUC	.423	.115	.414**
Age	.024	.011	.288*
Sex	-.102	.062	-.188
Positive Emotionality	.100	.055	.200
Negative Emotionality	.041	.074	.063
Constraint	.000	.002	-.038
Age * PEM	.043	.020	.236*
Age * NEM	.031	.021	.160
Age * CON	.000	.001	-.118

Note: $R^2 = .257$ for Step 1 ($p < .001$), $\Delta R^2 = .038$ for Step 2 ($p = .348$), $\Delta R^2 = .062$ for Step 3 ($p = .137$).

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

Table 17.

Hierarchical Regression Analysis: Components of Positive Emotionality at Time 1 Predicting Delay AUC at Time 2 (N = 70)

Step and Variable	B	SE B	β
Step 1			
Time 1 Delay AUC	.363	.114	.355**
Age	.023	.010	.275*
Sex	-.066	.059	-.122
Step 2			
Time 1 Delay AUC	.377	.106	.369**
Age	.010	.010	.123
Sex	-.082	.056	-.151
Positive Emotionality (Agentic)	.349	.095	.417**
Positive Emotionality (Communal)	-.100	.065	-.165
Step 3			
Time 1 Delay AUC	.418	.109	.409**

Age	.009	.010	.110
Sex	-.070	.056	-.129
Positive Emotionality (Agentic)	.335	.101	.400**
Positive Emotionality (Communal)	-.085	.066	-.140
Age * Agentic PEM	.030	.032	.106
Age * Communal PEM	.023	.023	.101

Note: $R^2 = .257$ for Step 1 ($p < .001$), $\Delta R^2 = .130$ for Step 2 ($p < .001$), $\Delta R^2 = .025$ for Step 3 ($p = .278$)

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

Table 18.

Hierarchical Regression Analysis: Components of Agentic Positive Emotionality at Time 1 Predicting Delay AUC at Time 2 (N = 70)

Step and Variable	B	SE B	β
Step 1			
Time 1 Delay AUC	.363	.114	.355**
Age	.023	.010	.275*
Sex	-.066	.059	-.122
Step 2			
Time 1 Delay AUC	.359	.189	.352**
Age	.013	.010	.158
Sex	-.084	.058	-.155
Wellbeing	-.006	.014	-.058
Social Potency	-.001	.011	.010
Achievement	.033	.011	.345**
Absorption	.006	.011	.064

Step 3			
Time 1 Delay AUC	.393	.109	.384**
Age	.012	.010	.146
Sex	-.066	.057	-.121
Wellbeing	-.008	.014	-.071
Social Potency	.000	.011	.004
Achievement	.036	.011	.375**
Absorption	.006	.010	.059
Age * Wellbeing	.038	.016	.291*
Age * Social Potency	.001	.004	.022
Age * Achievement	.000	.004	-.014
Age * Absorption	.001	.003	.048

Note: R^2 = .257 for Step 1 (p < .001), ΔR^2 = .101 for Step 2 (p = .056), ΔR^2 = .098 for Step 3 (p = .044)

* p<.05 ** p<.01

Table 19.

Hierarchical Regression Analysis: Personality Correlates at Time 1 Predicting Probability AUC at Time 2 (N = 90)

Step and Variable	B	SE B	β
Step 1			
Time 1 Probability AUC	.403	.081	.442**
Age	-.005	.005	-.084
Sex	-.102	.032	-.290**
Step 2			
Time 1 Probability AUC	.422	.083	.462**
Age	.000	.005	-.018
Sex	-.090	.033	-.254**
Positive Emotionality	-.011	.028	-.036
Negative Emotionality	.070	.041	.166
Constraint	.000	.001	.000
Step 3			

Time 1 Probability AUC	.429	.086	.470**
Age	.000	.005	-.005
Sex	-.089	.033	-.252**
Positive Emotionality	-.016	.030	-.051
Negative Emotionality	.079	.043	.188
Constraint	.000	.001	.025
Sex * PEM	.007	.030	.022
Sex * NEM	.007	.041	.017
Sex * CON	.001	.001	.093

Note: $R^2 = .360$ for Step 1 ($p < .001$), $\Delta R^2 = .025$ for Step 2 ($p = .340$), $\Delta R^2 = .008$ for Step 2 ($p = .786$)

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

Table 20.

Hierarchical Regression Analysis: Components of Negative Emotionality at Time 1 Predicting Probability AUC at Time 2 (N = 90)

Step and Variable	B	SE B	β
Step 1			
Time 1 Probability AUC	.403	.081	.442**
Age	-.005	.005	-.084
Sex	-.102	.032	-.290**
Step 2			
Time 1 Probability AUC	.403	.084	.441**
Age	-.002	.005	-.043
Sex	-.090	.032	-.255**
Negative Emotionality (Agentic)	.101	.056	.185
Negative Emotionality (Alienated)	-.005	.056	-.010
Step 2			
Time 1 Probability AUC	.402	.085	.440**

Age	-.003	.005	-.055
Sex	-.089	.033	-.253**
Negative Emotionality (Agentic)	.096	.059	.176
Negative Emotionality (Alienated)	.000	.057	-.002
Sex * Agentic NEM	-.015	.058	-.027
Sex * Alienated NEM	.029	.054	.057

Note: R^2 = .360 for Step 1 (p < .001), ΔR^2 = .029 for Step 2 (p = .143), ΔR^2 = .002 for Step 3 (p = .863)

* p<.05 ** p<.01

Table 21.

Hierarchical Regression Analysis: Components of Agentic Negative Emotionality at Time 1 Predicting Probability AUC at Time 2 (N = 90)

Step and Variable	B	SE B	β
Step 1			
Time 1 Probability AUC	.403	.081	.442**
Age	-.005	.005	-.084
Sex	-.102	.032	-.290**
Step 2			
Time 1 Probability AUC	.429	.085	.469**
Age	-.002	.005	-.032
Sex	-.088	.032	-.249**
Stress Reactivity	.034	.027	.132
Aggression	-.030	.025	.121
Step 3			

Time 1 Probability AUC	.431	.085	.472**
Age	-.001	.005	-.024
Sex	-.091	.032	-.257**
Stress Reactivity	.034	.024	.133
Aggression	.024	.025	.098
Sex * Stress Reactivity	.027	.023	.105
Sex * Aggression	-.033	.024	-.128

Note: R^2 = .360 for Step 1 (p < .001), ΔR^2 = .037 for Step 2 (p = .082), ΔR^2 = .019 for Step 3 (p = .276)

* p<.05 ** p<.01

Table 22. Partial correlations between Freesurfer regions of interest at time 1 and delay discounting at time 2, controlling for delay discounting at time 1, age at time 1, sex, IQ, and (for white matter volumes but not thickness) total brain volume, n = 81.

	Left Hemisphere-Cortical Thickness	Left Hemisphere-White Matter Volume	Right Hemisphere-Cortical Thickness	Right Hemisphere-White Matter Volume
Banks of the superior temporal sulcus	-.005	-.012	-.027	.120
Caudal anterior cingulate	-.098	.041	.070	**.284*[1]**
Caudal middle frontal	-.136	-.061	**-.282***	-.086
Corpus callosum	.038	-.067	.068	.040
Cuneus	.095	-.076	.070	**.228***
Entorhinal	-.059	-.007	.004	.054
Frontal pole	**-.238***	.206	**-.227***	.076
Fusiform	-.007	.022	-.019	.080
Inferior parietal	-.098	.195	.041	-.027
Inferior temporal	-.023	.059	-.079	.107
Isthmus cingulate	-.033	.026	.086	**.286***
Lateral occipital	-.030	-.076	.024	.064
Lateral orbitofrontal	-.140	.186	-.163	.119
Lingual	.079	.198	-.006	.082
Medial orbitofrontal	-.180	.166	-.106	.090
Middle temporal	.027	-.067	-.085	.113
Paracentral	-.082	.002	-.186	-.062
Parahippocampal	**.281***	.163	-.019	.052
Pars opercularis	-.030	-.094	**-.233***	-.099
Pars orbitalis	.011	.027	**-.252***	.154
Pars triangularis	-.055	.054	**-.342****	.183

Pericalcarine	.159	.053	.060	.030
Postcentral	.046	.012	.017	.177
Posterior cingulate	-.029	.220	-.082	.043
Precentral	-.194	-.075	**-.244***	.092
Precuneus	.075	-.078	.009	-.091
Rostral anterior cingulate	.137	-.059	.184	-.025
Rostral middle frontal	-.200	.222	**-.305****	.060
Superior frontal	-.082	-.133	-.187	-.084
Superior parietal	-.124	.062	-.090	.204
Superior temporal	-.215	-.062	-.187	.142
Supramarginal	-.066	-.218	-.059	-.221
Temporal pole	-.026	.014	-.158	.054
Transverse temporal	-.107	.010	-.023	.000

[1]Correlation reported after removal of one influential outlier.

Table 23. Partial correlations between subcortical volumes in Freesurfer regions of interest at time 1 and delay discounting at time 2, controlling for delay discounting at time 1, age at time 1, sex, IQ, and total brain volume, n = 81.

	Left	Right
Nucleus accumbens	-.115	.004
Amygdala	.117	.214
Caudate	-.111	-.088
Cerebellum (cortex)	-.054	-.044
Cerebellum (white matter)	-.065	-.154
Hippocampus	**.306****	**.297***[1]
Pallidum	.056	-.002
Putamen	.187	.159
Thalamus	.107	.090
Ventral diencephalon	.064	.049

[1]Correlation reported after removal of one influential outlier.

Table 24. Partial correlations between Freesurfer regions of interest at time 1 and probability discounting at time 2, controlling for probability discounting at time 1, age at time 1, sex, IQ, and (for white matter volumes but not thickness) total brain volume, n = 90.

	Left Hemisphere-Cortical Thickness	Left Hemisphere-White Matter Volume	Right Hemisphere-Cortical Thickness	Right Hemisphere-White Matter Volume
Banks of the superior temporal sulcus	.072	.001	-.192	.088
Caudal anterior cingulate	.183	.017	.034	.078
Caudal middle frontal	-.029	.113	-.167	.099
Corpus callosum	-.035	.170	-.017	.142
Cuneus	.024	-.009	-.181	.079
Entorhinal	.010	.093	.044	-.022
Frontal pole	-.051	-.085	.028	-.056
Fusiform	-.183	-.112	-.172	.031
Inferior parietal	-.112	.081	-.128	-.044
Inferior temporal	-.106	.023	-.028	.111
Isthmus cingulate	.037	.091	.185	.036
Lateral occipital	-.161	-.033	-.113	.078
Lateral orbitofrontal	-.151	-.007	-.149	.111
Lingual	-.059	-.071	-.051	-.089
Medial orbitofrontal	-.093	-.047	-.115	.002
Middle temporal	-.104	.002	-.103	-.063
Paracentral	-.044	-.113	-.178	-.092
Parahippocampal	.127	.071	.075	.066
Pars opercularis	.046	.169	-.011	.037

Pars orbitalis	-.022	-.055	.011	-.013
Pars triangularis	.027	-.101	-.048	-.037
Pericalcarine	-.055	.121	-.096	-.013
Postcentral	-.075	-.120	-.079	.035
Posterior cingulate	-.053	.030	-.042	-.106
Precentral	-.099	-.018	-.142	.127
Precuneus	-.033	.079	-.104	.088
Rostral anterior cingulate	.088	**.221*[1]**	.070	-.022
Rostral middle frontal	-.045	.040	.022	-.176
Superior frontal	-.059	-.034	-.081	-.069
Superior parietal	-.186	.160	-.134	.107
Superior temporal	-.211	.186	-.159[1]	-.111
Supramarginal	-.060	-.021	-.157	.125
Temporal pole	-.070	.042	-.062	.072
Transverse temporal	-.116	.047	-.179	-.008

Table 25. Partial correlations between subcortical volumes in Freesurfer regions of interest at time 1 and probability discounting at time 2, controlling for probability discounting at time 1, age at time 1, sex, IQ, and total brain volume, n = 90.

	Left	Right
Nucleus accumbens	-.040	-.163
Amygdala	-.130	-.110
Caudate	-.208	-.167
Cerebellum (cortex)	-.078	-.086
Cerebellum (white matter)	.154	.027
Hippocampus	-.076	-.115
Pallidum	-.031	.083
Putamen	-.130	-.079
Thalamus	-.111	-.033
Ventral diencephalon	.143	.161

Table 26: White matter tracts where MD at time 1 is associated with delay AUC at time 2, controlling for time 1 delay AUC.

Region	**Cluster size (voxels)**	**Max Partial r**	**x**	**y**	**z**
1. Fornix (cres)/ stria terminalis/ anterior thalamic radiation in posterior left thalamus/ hippocampus	46	- .3725	-24	-32	4

Table 27: White matter tracts where FA at time 1 is associated with delay AUC at time 2, controlling for time 1 delay AUC, in participants age 9-15.

Region	**Cluster size (voxels)**	**Max Partial r**	**x**	**y**	**z**
1. Left inferior fronto-occipial fasciculus/ anterior thalamic radiation/ uncinate fasciculus/ superior longitudinal fasciculus in inferior frontal gyrus (pars opercularis and triangularis)/ middle frontal gyrus	23	+ .5169	-34	22	18

Table 28: White matter tracts where FA at time 1 is associated with delay AUC at time 2, controlling for time 1 delay AUC, age, sex, and IQ (whole sample).

Region	**Cluster size (voxels)**	**Max Partial r**	**x**	**y**	**z**
Right anterior thalamic radiation/ corticospinal tract in amygdala/ pallidum/ hippocampus	21	- .3951	12	-6	-12

Table 29: White matter tracts where FA at time 1 is associated with delay AUC at time 2, controlling for time 1 delay AUC, age, sex, and IQ, for participants age 9 to 15 at time 1.

Region	**Cluster size (voxels)**	**Max Partial r**	**x**	**y**	**z**
Right superior longitudinal fasciculus/ inferior longitudinal fasciculus/ inferior fronto-occipital fasciculus/ anterior thalamic radiation in lateral occipital cortex/ angular gyrus/ precuneus/ superior parietal lobule	38	- .5138	28	-56	34

Table 30: White matter tracts where high MD at time 1 is associated with high probability AUC at time 2, controlling for time 1 probability AUC, age, sex, and IQ (whole sample).

Region	**Cluster size (voxels)**	**Max Partial r**	**x**	**y**	**z**
Left inferior longitudinal fasciculus/ inferior fronto-occipital fasciculus/ forceps major in lateral occipital cortex/ cuneus/ precuneus	20	+ .3640	-26	-68	30

Figure 1. Mean delay and probability discounting AUCs at time 1 (left) and at time 2 (right).

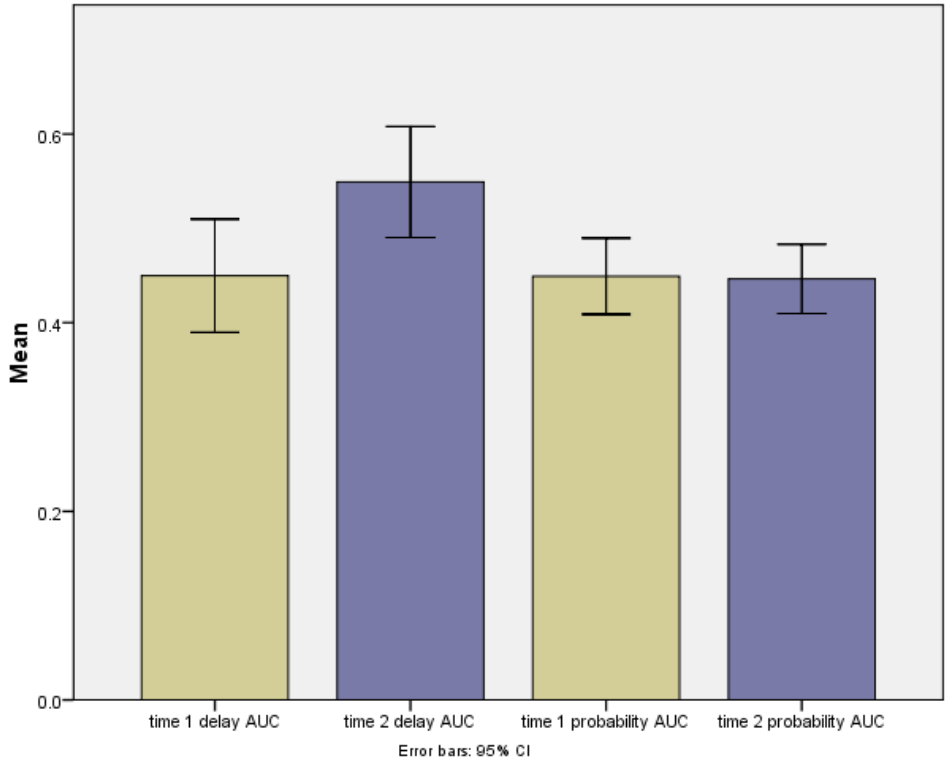


Figure 2. Partial correlation of age with time 2 delay discounting AUC after controlling for time 1 delay discounting AUC.

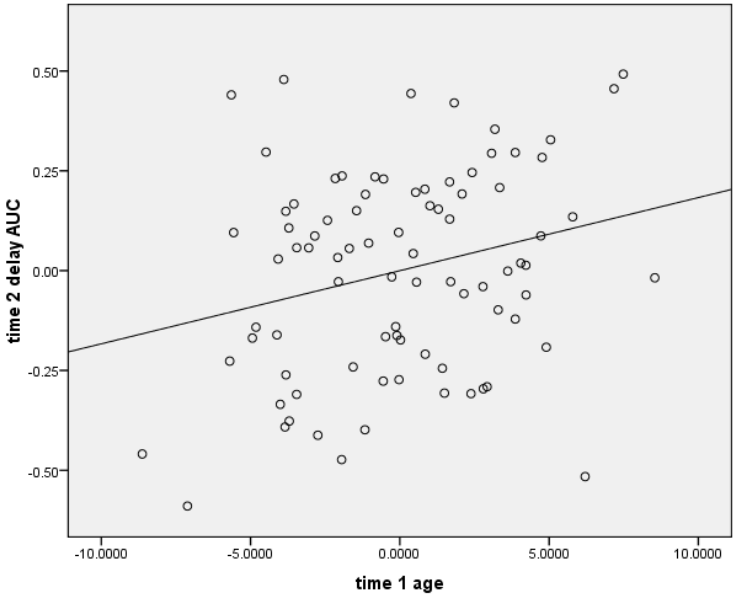
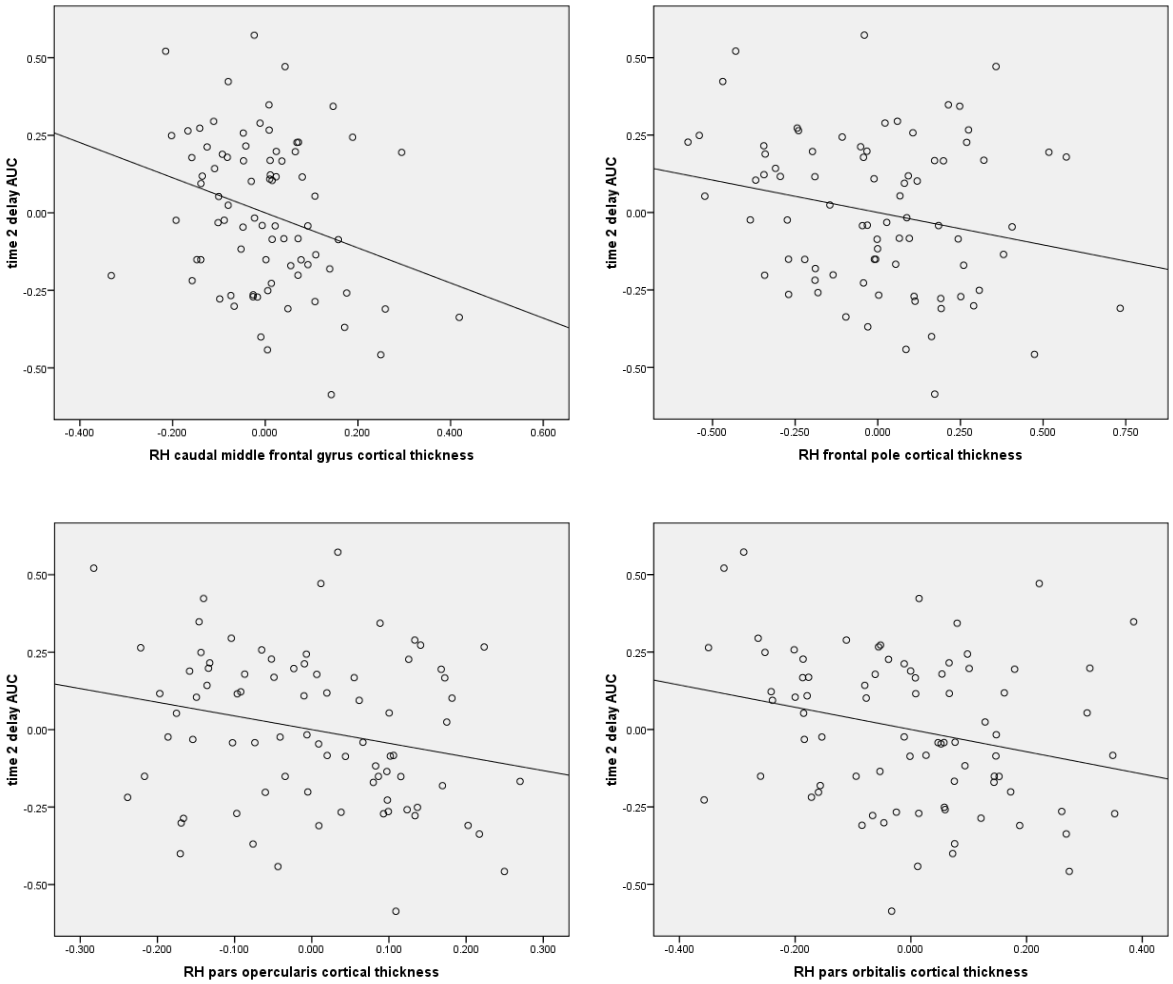
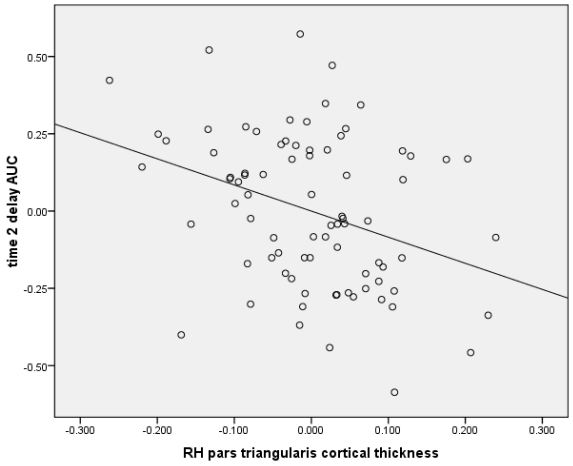
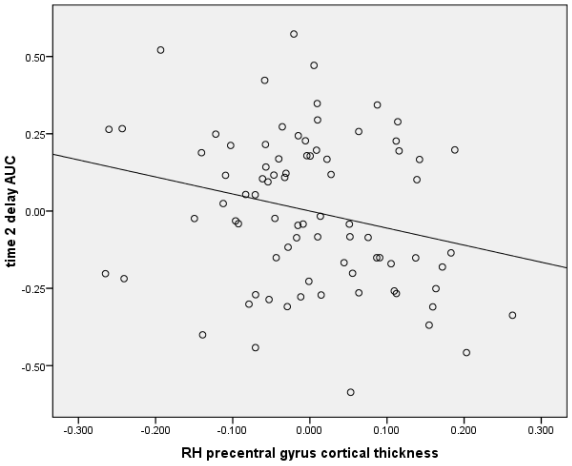
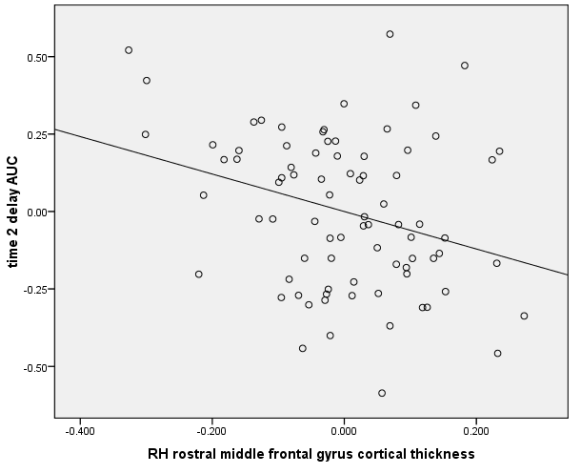


Figure 3. Partial correlations between cortical thickness and delay discounting at time 2, after controlling for time 1 delay discounting, age, sex, and IQ.




time 2 delay AUC
0.50
0.25
0.00
-0.25
-0.50
-0.300
-0.200
-0.100
0.000
0.100
0.200
0.300
RH pars triangularis cortical thickness


time 2 delay AUC
0.50
0.25
0.00
-0.25
-0.50
-0.300
-0.200
-0.100
0.000
0.100
0.200
0.300
RH precentral gyrus cortical thickness


time 2 delay AUC
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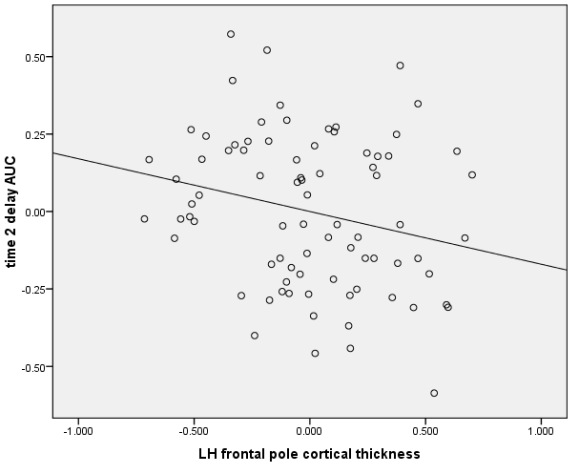
0.00
-0.25
-0.50
-0.400
-0.200
0.000
0.200
RH rostral middle frontal gyrus cortical thickness


time 2 delay AUC
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-0.50
-1.000
-0.500
0.000
0.500
1.000
LH frontal pole cortical thickness

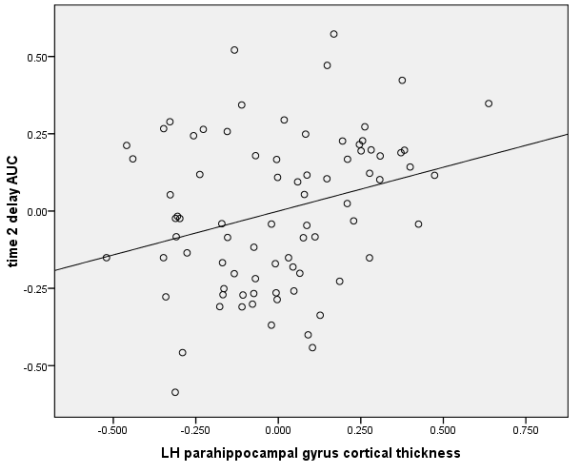
time 2 delay AUC
0.50
0.25
0.00
-0.25
-0.50
-0.500
-0.250
0.000
0.250
0.500
0.750
LH parahippocampal gyrus cortical thickness

Figure 4. Partial correlations between white matter volumes and delay discounting at time 2, after controlling for time 1 delay discounting, age, sex, IQ, and total brain volume.

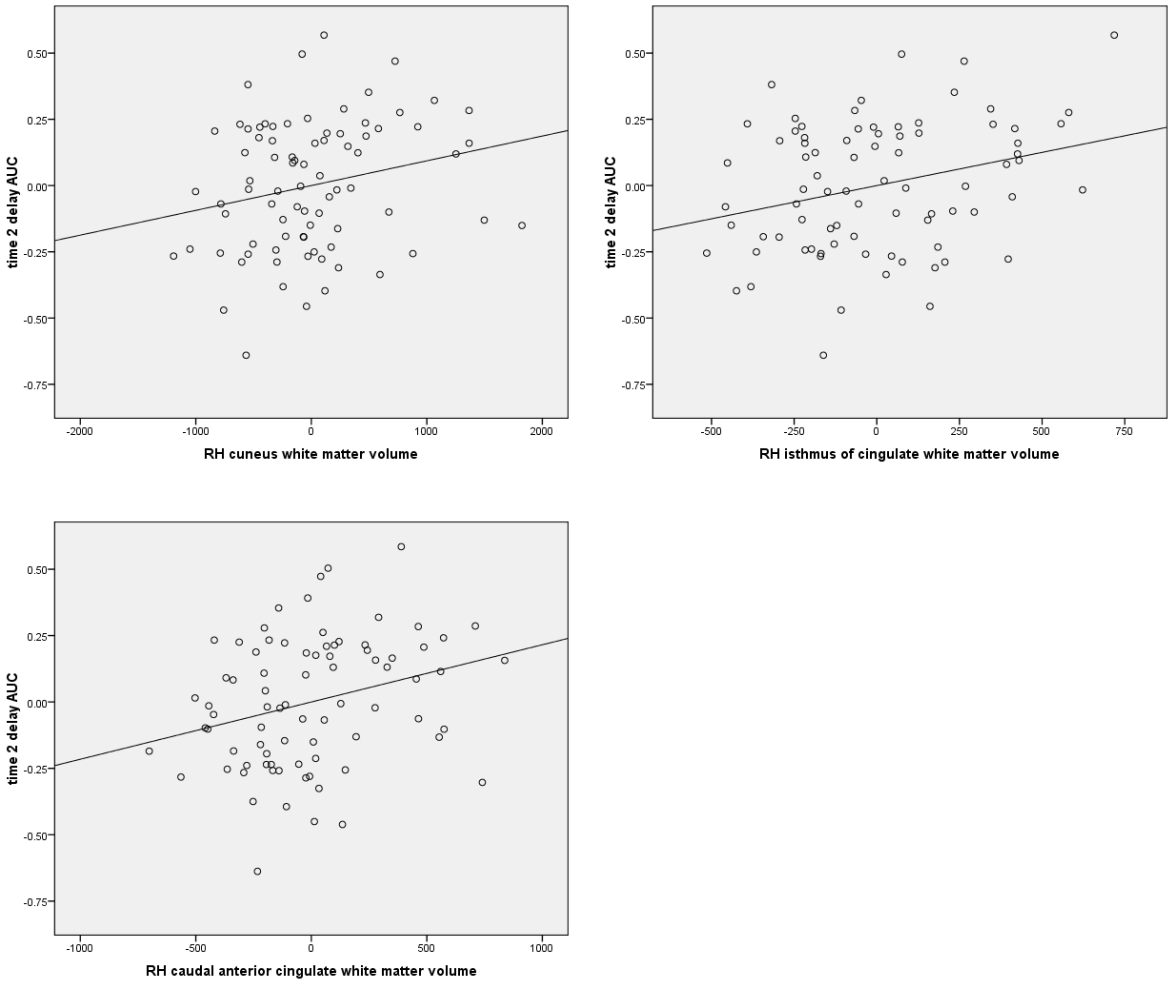

Figure 5. Partial correlations between subcortical volumes and delay/probability discounting at time 2, after controlling for time 1 discounting, age, sex, IQ, and total brain volume.

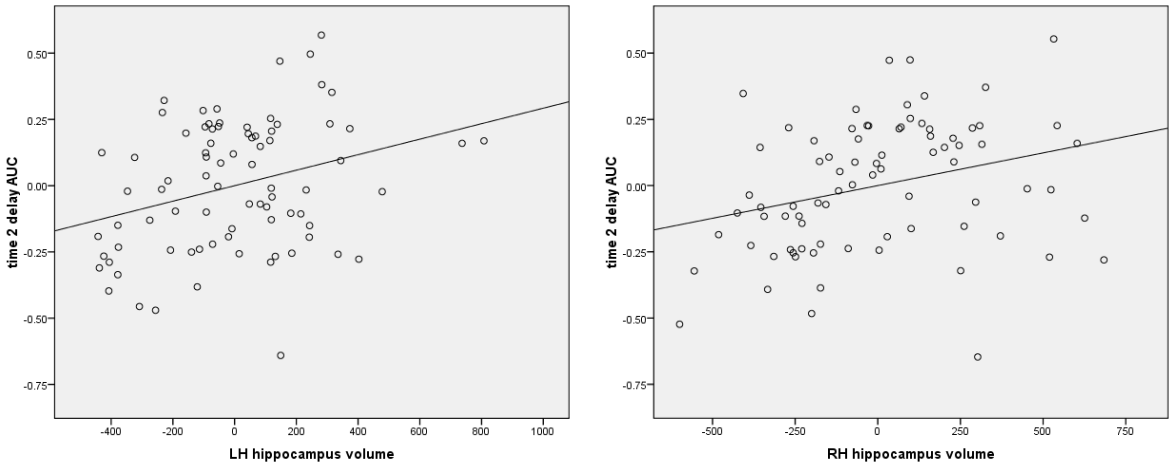

Figure 6. Partial correlation between white matter volumes and probability discounting at time 2, after controlling for time 1 probability discounting, age, sex, IQ, and total brain volume.

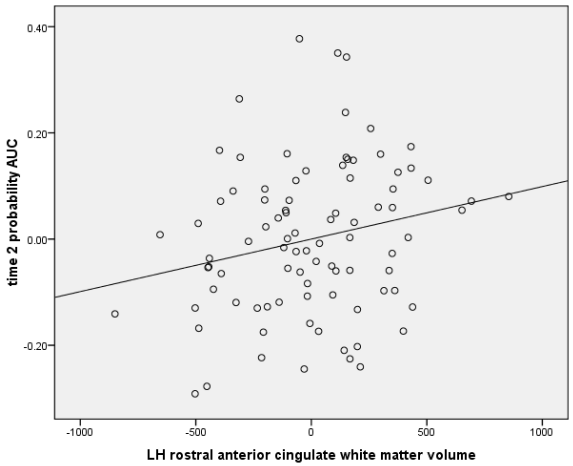

Figure 7. Partial correlation between DTI measures and delay discounting at time 2, after controlling for time 1 delay discounting (whole sample).

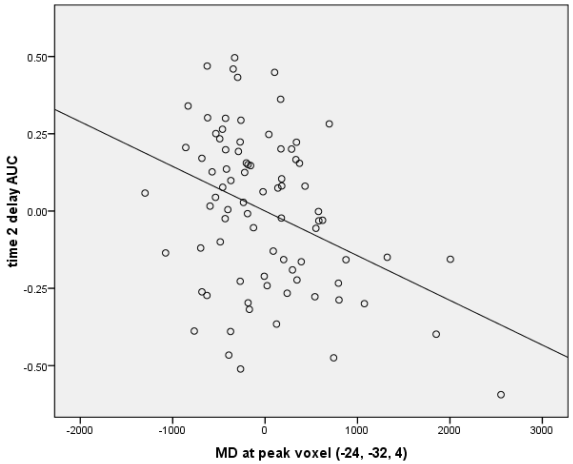

Figure 8. Partial correlation between DTI measures and delay discounting at time 2, after controlling for time 1 delay discounting (9- to 15-year-olds).

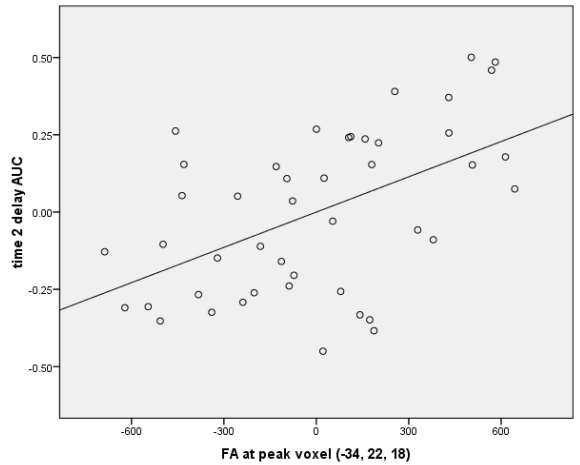

Figure 9. Partial correlation between DTI measures and delay discounting at time 2, after controlling for time 1 delay discounting, age, sex, and IQ (whole sample).

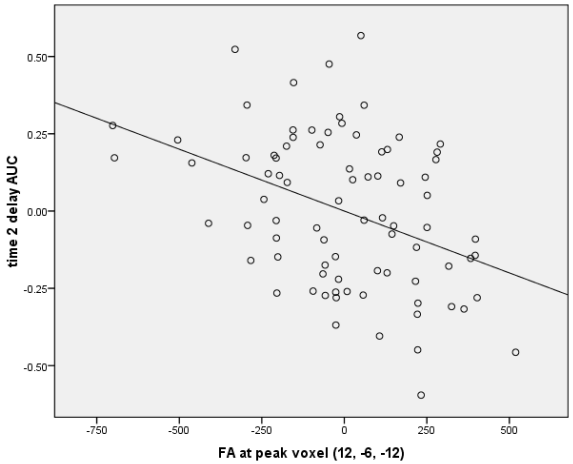

Figure 10. Partial correlation between DTI measures and delay discounting at time 2, after controlling for time 1 delay discounting, age, sex, and IQ (9- to 15-year-olds).

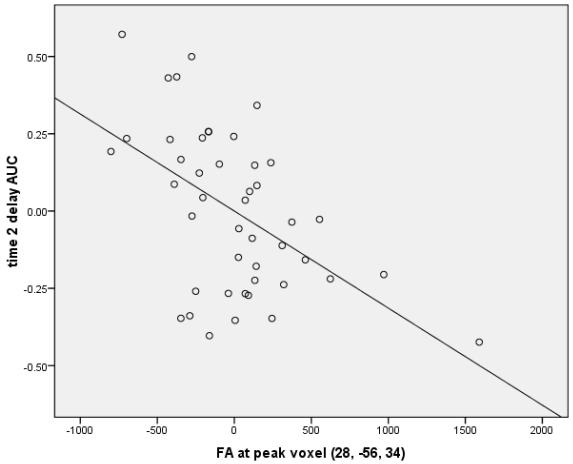

Figure 11. Partial correlation between DTI measures and probability discounting at time 2, after controlling for time 1 probability discounting, age, sex, and IQ (whole sample).