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White Matter Integrity Predicts Delay Discounting Behavior in 9- to 23-Year-Olds: A Diffusion Tensor Imaging Study

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Abstract

Healthy participants (n = 79), ages 9–23, completed a delay discounting task assessing the extent to which the value of a monetary reward declines as the delay to its receipt increases. Diffusion tensor imaging (DTI) was used to evaluate how individual differences in delay discounting relate to variation in fractional anisotropy (FA) and mean diffusivity (MD) within whole-brain white matter using voxel-based regressions. Given that rapid prefrontal lobe development is occurring during this age range and that functional imaging studies have implicated the prefrontal cortex in discounting behavior, we hypothesized that differences in FA and MD would be associated with alterations in the discounting rate. The analyses revealed a number of clusters where less impulsive performance on the delay discounting task was associated with higher FA and lower MD. The clusters were located primarily in bilateral frontal and temporal lobes and were localized within white matter tracts, including portions of the inferior and superior longitudinal fasciculi, anterior thalamic radiation, uncinate fasciculus, inferior fronto-occipital fasciculus, corticospinal tract, and splenium of the corpus callosum. FA increased and MD decreased with age in the majority of these regions. Some, but not all, of the discounting/ DTI associations remained significant after controlling for age. Findings are discussed in terms of both developmental and age-independent effects of white matter organization on discounting behavior.

INTRODUCTION

Delay discounting occurs when individuals prefer to receive smaller immediate rewards rather than larger delayed rewards; the subjective value of the reward is “discounted” as the delay to reward delivery increases. The rate at which delayed rewards decline in subjective value is an individual difference variable that is hypothesized to reflect a type of impulsivity characterized by the inability to consider future events when making decisions (Ainslie, 1975). In humans, discounting rates are typically assessed by presenting individuals with a series of hypothetical forced-choice questions, such as “Would you rather have $2 now or $10 in 365 days?” The subjective value of the delayed reward is assessed by finding the “indifference point” at which the individual has no preference for one reward over the other.

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Indifference points are plotted against time to determine discounting rates (e.g., Madden et al., 2004; Richards, Zhang, Mitchell, & de Wit, 1999).

Functional neuroimaging studies suggest that activity in the prefrontal cortex (PFC) affects choice behavior on discounting tasks. In one fMRI study of delay discounting in adults (McClure, Laibson, Loewenstein, & Cohen, 2004), BOLD activity in two sets of neural regions was compared. When participants chose the delayed reward instead of the immediate reward, there was greater activity in a set of prefrontal and parietal areas (including the dorsolateral PFC) than in a set of regions that were preferentially activated for choices in which money was available immediately, including the ventral striatum, medial orbito-frontal cortex (OFC), medial PFC, posterior cingulate, and left posterior hippocampus. The authors suggest that activity in the first set of regions may help to override the drive from the second set of regions to accept the immediate reward. McClure, Ericson, Laibson, Loewenstein, and Cohen (2007) conducted an fMRI study of delay discounting of primary rewards, identifying a set of regions that were preferentially activated for immediate rewards, including the nucleus accumbens, subgenual cingulate, medial OFC, posterior cingulate, precuneus, and dorsal anterior cingulate. Activation in these regions predicted selection of smaller sooner rewards. Wittman, Leland, and Paulus (2007) compared BOLD responses to short versus long delays in healthy adult controls. They found that when individuals chose delayed rewards, there was strong activation in the bilateral posterior insular cortex, as well as in the left posterior cingulate, superior temporal gyrus, angular gyrus, inferior parietal lobule, and cuneus; there were no regions that were more strongly activated when individuals chose immediate rewards. Hariri et al. (2006) used an fMRI paradigm to examine relationships between BOLD activity during a different reward-related task and behavioral responses on a delay discounting task. They found that individuals who discounted delayed rewards more steeply had greater activation in the ventral striatum during the reward task, especially when receiving feedback regarding monetary gains (as opposed to losses). In addition, steep discounters had high gain-related (vs. loss-related) activity in the medial PFC and low gain-related activity in the lateral OFC and dorsal PFC. In general, these findings are consistent with the assertion that, in adults, limbic areas such as the ventral striatum may be involved in selecting small immediate rewards, whereas other regions such as the DLPFC are involved in selecting larger delayed rewards. However, not all findings are entirely consistent with this limbic versus frontal dichotomy. In an fMRI study comparing adult methamphetamine-dependent participants and controls, Monterosso et al. (2007) found that, when making more difficult choices in the discounting paradigm (i.e., choices involving values close to their indifference points), both groups showed activation in the ventrolateral PFC and DLPFC bilaterally, in the anterior cingulate cortex and supplementary motor area, and in areas around the right and left intraparietal sulcus. In addition, group differences were seen in left DLPFC and right intraparietal sulcus activation: Control subjects showed less activation in these regions when making easy choices than when making difficult choices, whereas for methamphetamine-dependent subjects, the activation did not attenuate for easy choices. Together, these studies suggest that the PFC is a particularly salient node in the network that supports delay discounting behavior.

Whether these regions are involved in discounting behaviors in adolescents as they are in adults is unknown. It is broadly asserted that adolescents, as a group, are more impulsive and less future-oriented than adults. Moreover, imaging studies and studies of cognitive development indicate that many brain regions, including the PFC, are still developing into young adulthood (Giedd, 2004; Gogtay et al., 2004; Giedd et al., 1996, 1999; Reiss, Abrams, Singer, Ross, & Denckla, 1996; Pfefferbaum et al., 1994). Processes that affect cortical development during adolescence include increasing myelination of white matter, which follows a posterior to anterior pattern of development (Sampaio & Truwit, 2001; Sowell, Thompson, Holmes, Jernigan, & Toga, 1999; Reiss et al., 1996; Pfefferbaum et al., 1994), along with an early increase in gray matter volume followed by subsequent loss of gray matter due to pruning in late adolescence (Giedd et al., 2006; Giedd, 2004; Gogtay et al., 2004). There may also be more subtle ways in which the brain's capacity for structural integration is increasing during adolescence. For instance, there is recent interest in how white matter becomes directionally organized to enhance information-processing capacities.

Diffusion tensor imaging (DTI) measures the rates of diffusion of water molecules and can therefore be used to assess not merely white matter volume, but white matter organization (Basser, 1995). One major outcome variable from DTI studies is *fractional anisotropy* (*FA*), which reflects the extent to which diffusion of water molecules is anisotropic (not equal in all directions). Higher FA values are believed to reflect increasing organization of white matter tracts, which forces water molecules to travel in parallel with them. An additional outcome variable from DTI studies is *mean diffusivity (MD)*, a measure of the total amount of water molecule diffusion in a region. Higher MD values are believed to reflect increasing tissue bulk (regardless of fiber orientation). Some white matter changes (such as increasing myelination) may affect the measurements of both FA and white matter density, whereas other changes (such as changes in fiber orientation) affect FA only (Barnea-Goraly et al., 2005). Higher values of frontal lobe FA have been observed in older versus younger adolescents (Barnea-Goraly et al., 2005; Klingberg, Vaidya, Gabrieli, Moseley, & Hedehus, 1999), and developmental changes in white matter organization are associated with cognitive skills such as general intelligence and cognitive control (Liston et al., 2006; Schmithorst, Wilke, Dardzinski, & Holland, 2005).

Concordant with findings from the imaging literature, behavioral findings on executive tasks also suggest continuing maturation throughout adolescence, and several of these findings pertain to how adolescents make reward-relevant decisions (Conklin, Luciana, Hooper, & Yarger, 2007; Luciana, Conklin, Hooper, & Yarger, 2005; Crone & van der Molen, 2004; Hooper, Luciana, Conklin, & Yarger, 2004; Luna, Garver, Urban, Lazar, & Sweeney, 2004; De Luca et al., 2003). Given this late maturation of complex decision-making behavior, a continuing developmental emergence of discounting behavior throughout adolescence is expected.

Recently, we reported behavioral findings from a large adolescent sample that completed delay and probability discounting tasks. The delay discounting task was administered to all participants, whereas the probability discounting task was administered only to 11- to 23-year-olds. We reported that delay discounting matures with age but that probability discounting does not (Olson, Hooper, Collins, & Luciana, 2007). Specifically, age was

negatively correlated with the rate of delay discounting in healthy 9- to 23-year-olds. In addition, delay discounting rates were lower in individuals with higher verbal intelligence, independent of the effect of age. We also found that errors of commission on a measure of behavioral inhibition were associated with delay discounting, but not after controlling for the effects of age and verbal intelligence. In contrast, probability discounting was unrelated to age in 11- to 23-year-olds and was not associated with performance on other executive function task measures. Probability discounting was associated with self-reported rates of externalizing behaviors in the sample. There were no sex differences in discounting rates for either task. There also was no significant Sex by Age interaction on discounting rate. Most of the participants who contributed data to the behavioral study also completed an artifact-free DTI scan, and they are the focus of the current report.

Accordingly, the present study aims to examine whether indices of white matter organization (FA and MD) are related to discounting behavior in this adolescent sample and, if so, the extent to which these associations are age-dependent. This approach is similar to that employed by others to examine DTI–behavior relations with respect to both individual differences and overall developmental patterns (Beaulieu et al., 2005; Nagy, Westerberg, & Klingberg, 2004). The fMRI literature on delay discounting has identified a possible network of regions activated when a larger later reward is chosen; these primarily include lateral prefrontal regions and parietal and temporal regions. In contrast, a different set of regions is typically activated when smaller sooner rewards are chosen, primarily including the ventral striatum and medial prefrontal regions. Therefore, we hypothesize that adolescents with more mature patterns of white matter organization (higher FA and lower MD) in regions promoting the selection of delayed rewards (lateral prefrontal and temporal/parietal regions) will exhibit less steep discounting rates. Given that delay discounting is associated with age and verbal IQ in this sample and that these same associations have also been reported by others (see Olson et al., 2007 for a review), an additional study aim is to investigate whether these same aspects of white matter organization are associated with discounting after controlling for the effects of age and verbal IQ. The specificity of observed effects is also addressed by examining DTI correlates of probability discounting behavior. Because pubertal status affects the timing of neurobehavioral development (Giedd et al., 2006), the role of pubertal status in the development of delay discounting behavior was considered.

METHODS

Participants aged 9 to 23 years were recruited. Individuals younger than 18 years were recruited from 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's participation. Individuals ages 18 and up were recruited through posted advertisements at the University. Inclusion criteria included being 9- to 23-year-olds; 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 were right-handed, as assessed by the Edinburgh Handedness Inventory (Oldfield, 1971), and without 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. Intelligence was measured by the four subtests of the Wechsler Abbreviated Scale of Intelligence (WASI; The Psychological Corporation, 1999). Pubertal status was assessed using the questionnaire version of the Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988). Participants and parents provided informed consent or assent according to local IRB requirements. Table 1 summarizes demographic data. Participants' racial/ ethnic backgrounds were Caucasian (68), African American (1), Hispanic (3), Asian/Pacific Islander (2), other (including multiracial) (4), and not reported (1). Participants completed a 3-hr screening session on one day and were tested on another day. Testing included a structural magnetic resonance imaging scan and a neurocognitive battery.

MRI Data Acquisition

MRI image acquisition was performed on a Siemens 3-Tesla Trio scanner (Siemens Medical Systems, Erlangen, Germany) using an eight-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 (MP-RAGE) sequence (TR = 2530 msec, TE = 3.65 msec, TI = 1100 msec, 240 slices, voxel size = 1.0 × 1.0 × 1.0, flip angle = 7°, FOV = 256 mm). An axial hyper-echo turbo spin echo (TSE) sequence was used to collect proton density (PD) images (TR = 8550 msec, TE = 14 msec, 80 slices, voxel size = 1.0 × 1.0 × 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 = 12.5 sec, TE = 98 msec, 64 slices, voxel size = 2.0 × 2.0 × 2.0 mm, 0 mm skip, FOV = 256 mm, 2 averages, *b* value = 1000 sec/mm^2). Thirteen unique volumes were collected to compute the tensor: a *b* = 0 sec/mm^2 image and 12 images with diffusion gradients applied in 12 noncollinear 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].

MRI Image Analysis

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 coregistered using an affine transformation with trilinear interpolation.

The diffusion tensor was computed using the Diffusion Toolbox (FDT) from the FMRIB library. A linear affine transformation was applied to diffusion-weighted images to correct for the distortions caused by eddy currents (Haselgrove & Moore, 1996). Six maps of the apparent diffusion coefficient were computed using the single *b* = 0 image and the 12 eddy current corrected diffusion-weighted images. The diffusion tensor was then derived, and FA and MD maps were created. Prior to normalization, the FA and MD images were corrected for geometric distortion due to magnetic field inhomo-geneity by unwarping PD-aligned *b* = 0 images and applying this transformation to the FA and MD images (Ardekani, 2003).

Normalization of imaging data required several steps. First, because of differences in the size and shape of children's brains compared to adults', as an initial template we used an age-appropriate single-subject T1 image chosen from our own database of normal adolescents, which was aligned to the Montreal Neurological Institute (MNI) template. Second, we used a subsample of data from the larger study to create a study-average template. To do this, T1 images from this subsample were coregistered to their PD images using a six-parameter affine transformation and then aligned to the single-subject template using a nonlinear warp (Ardekani, 2003). A new template was created by averaging the subsample of normalized T1 images. We then returned to the 79 PD coregistered T1 images from the present analysis, and performed the normalization using the study-average template instead of the single-subject template. The distortion-corrected FA and MD images were normalized by applying the nonlinear warps derived during normalization of the T1 images to the study-average template. 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. Voxelwise 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 (SPM2; Wellcome Department of Cognitive Neurology, London, UK). All analyses were thresholded at $p < .001$, uncorrected for multiple comparisons, with an extent threshold of 25 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. Because the rate of brain development in numerous regions varies by sex (Giedd et al., 1996), sex was entered as a covariate in all of the DTI analyses.

Discounting Task (Richards et al., 1999)

The delay discounting task was programmed in E-prime (Psychology Software Tools; www.psnet.com). 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?"). Discounting was assessed at six delays (1, 2, 10, 30, 180, and 365 days later). Participants ages 11 and up also completed a probability discounting task. On each trial, participants chose between an immediate amount of money or $10 available with a given probability (i.e., "Would you rather have $2 for sure or $10 with a 50% chance?"). Discounting was assessed at five probabilities (25%, 50%, 75%, 90%, and 95% chance of winning). The immediate or certain amount was determined by an adjusting-amount procedure (Richards et al., 1999) involving random selection within a fixed interval that depended on previous choices. Task instructions appeared onscreen and were read aloud. Afterward, the computer "randomly" selected one trial, and the participant received a cash payment based on that trial. (Participants were told that all trials had an equal chance of being selected, but 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.

RESULTS

Behavioral data analysis was conducted using SPSS for Windows, version 12.0.0 (SPSS, Chicago, IL). Behavioral data were inspected for normality to ensure that they met the assumptions for parametric statistics.

Discounting data were analyzed as follows. The adjusting-amount procedure (Richards et al., 1999) results in the establishment of one indifference point for each delay interval. Indifference points reflect the subjective value of the delayed amount. For instance, $10 thirty days from now may have the same subjective value as $3 immediately; in that case, $3 is the "indifference point" for a 30-day delay. Indifference points were established for each participant at each interval and were plotted against time (at time = 0, the subjective value of $10 was assumed to be $10 for all subjects). As Myerson, Green, and Warusawitharana (2001) suggest, the areas under these discounting curves (area under the curve, AUC) were calculated by summing the resulting trapezoids. The AUC method of quantifying discounting behavior has been used in a variety of published findings (e.g., Olson et al., 2007; Field, Santarcangelo, Sumnall, Goudie, & Cole, 2006; Ohmura, Takahashi, & Kitamura, 2005; Dixon, Marley, & Jacobs, 2003; Du, Green, & Myerson, 2002) and provides a straightforward measure of discounting behavior that is not linked to any particular theoretical framework (Myerson et al., 2001).

Validity of Discounting Data

Typically, not all participants produce behavior consistent with discounting when completing discounting tasks. Because of inattention, poor motivation, and/or desire to complete the task quickly, some participants complete the task quickly and erratically. Indifference points should decrease as the time until reward delivery increases. If this is not the case, discounting behavior has not occurred. Discounting curves from participants producing erratic data are typically excluded from further analysis (Reynolds, Richards, & de Wit, 2006; Dixon et al., 2003).

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 increased (Dixon et al., 2003). Because the subjective value at the first point (time = 0 or odds against = 0) is assumed to be $10, a value lower than $10 at the first point assessed (time = 1 day or odds against = .0526) was counted as a decrease. Only participants who consistently discounted were included in the remaining analyses. Younger participants were not more likely than older participants to produce inconsistent discounting data. The final analyses were conducted on a subset of those cases who had complete data on the behavioral measures and usable imaging data (n = 79). This sample substantially overlaps our report (Olson et al., 2007) that focused exclusively on behavioral findings. One individual is excluded here because of poor imaging data. We reran all analyses presented in the behavioral paper minus this participant, and all reported behavioral associations (summarized in the Introduction above) remained significant.

Discounting Behavior in Relation to Pubertal Status

The distribution of PDS stages was as follows: prepubertal, $n = 4$ (0 male); early pubertal, $n = 8$ (5 males); mid-pubertal, $n = 14$ (9 males); late pubertal, $n = 26$ (14 males); postpubertal, $n = 22$ (5 males). Age was highly correlated with PDS total score [$r(72) = .873$, $p < .001$]; age also was highly correlated with PDS stage [r(72) = .860, $p < .001$]. Delay AUC was slightly more highly correlated with age [r(72) = .347, $p < .01$] than with PDS total score [$r(72) = .328$, $p < .01$]. After controlling for age, PDS total scores were not significantly correlated with delay AUC, indicating that pubertal status did not contribute to the prediction of delay discounting above and beyond the contribution made by age. Given these results and the undersampling of pre- and early pubertal stages, we emphasized age rather than PDS scores in the remaining analyses.

Relationships between Delay Discounting and Fractional Anisotropy

There were a number of areas where high FA was correlated with high delay AUC (Table 2; Figure 1), indicating that individuals who discounted less steeply had better white matter organization in these regions. In contrast, there were no areas where there was a significant association in the other direction (i.e., high FA with low delay AUC). As noted above, sex was entered as a covariate in all analyses. Clusters are numbered within Table 2 and will be referenced here by these numbers. There were several medium-sized right frontal clusters; these included clusters (1 and 2) with fibers from the anterior thalamic radiation (ATR), inferior fronto-occipital fasciculus, uncinate fasciculus (UF), forceps minor, and superior longitudinal fasciculus (SLF) in the region of the inferior frontal gyrus and the frontal pole, as well as a cluster (3) with fibers from the SLF near the precentral and middle frontal gyri. There was a medium-sized cluster (4) including fibers from the corticospinal tract near the right amygdala and the hippocampus, and a medium-sized cluster (7) including fibers from the ATR near the thalamus. There also was a medium-sized left frontal cluster (5) including fibers from the UF, inferior longitudinal fasciculus (ILF), and inferior fronto-orbital fasciculus, extending from the temporal pole and the parahippocampal gyrus to insular and frontal orbital cortices. There was a large left-hemisphere cluster (6) including fibers from the inferior fronto-occipital fasciculus, ILF, ATR, and UF, extending through the inferior and superior temporal gyri, the parahippocampal gyrus, and the temporal fusiform cortex, as well as a similarly placed but smaller cluster in the right hemisphere (8). Finally, there was a left-sided cluster (9) including fibers from the splenium of the corpus callosum and forceps major. Partial *rs* reflecting the relation between FA and delay discounting behavior controlling for participant sex but without controlling for age or VIQ were moderate in magnitude and ranged from .37 to .49. The mean FA value for each voxel cluster was extracted and plotted against delay AUC (Figure 2).

Age Influences on Relations between Delay AUC and FA

Given that delay discounting rates decrease with age, the possibility that the observed associations between delay AUC and FA were attributable to age was investigated. First, relationships between age and FA were examined for each cluster from Table 2. The mean FA value for all voxels within the cluster was extracted and correlated with age using Pearson correlations (Table 4; Figure 3). As indicated in Table 4, correlations with age

ranged from .13 to .52. Given the possibility of nonlinear relationships between age and changes in neural development (e.g., Giedd et al., 1999), linear, quadratic, and cubic models were fit to the age–FA associations and were compared using Akaike's Information Criterion (AIC) (Motulsky & Christopoulos, 2004; Akaike, 1974) to assess model fit. In three of the FA clusters (Clusters 3, 4, and 9), the relationship between age and mean FA in the voxel was nonlinear (cubic). In Clusters 1, 2, 5, 6, 7, and 8, the relationship was linear.

Thus, in the majority of the identified clusters where there was a significant association between delay AUC and FA, there also was a significant linear relationship between age and FA. In order to address whether the age–FA relationship accounted for the delay AUC–FA relationship, age was entered into the regression equation in SPM2 to assess whether there were significant associations between FA and delay AUC over and above the effects of age. Sex also was entered as a covariate. A right frontal region (Cluster 1) remained significant after controlling for age, as did the large region (6) in the left temporal lobe (Table 2). These two regions reflect areas where there was an age-independent relationship between FA and delay AUC, whereas there was an age-dependent relationship between FA and delay AUC in the remaining regions.

The delay AUC–FA analysis was repeated using total scores on the PDS rather than age as a covariate in order to explore whether controlling for pubertal status produced different results. The same voxel clusters remained after controlling for PDS total scores as remained after controlling for age.

As noted above, although the majority of clusters showed linear associations between age and FA, in three clusters the relationship was nonlinear. To further explore the nature of these nonlinear effects, the sample was divided at the median for age (16.5 years), and the associations between FA and delay AUC were examined within each age group. For these analyses, the *t* threshold was set at 3.0 and the minimum cluster size was set at 10 voxels. Within the younger sample, four clusters indicated areas where high FA was associated with high delay AUC. These clusters overlapped Clusters 1, 3, 6, and 8 (as represented in Table 2), although they were smaller in volume. Within older participants, there were two very small clusters where FA was significantly associated with delay AUC, and these clusters overlapped Clusters 2 and 8 (from Table 2). Three notable trends from this pattern are that (i) overall, the age effects appeared to be stronger in younger participants; (ii) FA in posterior regions, including the temporal lobe, was more strongly associated with delay AUC in younger versus older participants; and (iii) frontal FA values were associated with delay AUC across the full age range studied here.

Relationships between Delay Discounting and Mean Diffusivity

There were a number of areas where low MD was correlated with high delay AUC (Table 3; Figure 1), indicating that individuals who discounted less steeply had greater white matter density in these regions. In contrast, there were no areas where there was a significant association in the other direction, that is, low MD with low delay AUC. There was a large right (Cluster 1) and a medium left (Cluster 9) frontal cluster, including fibers primarily from the SLF, as well as the ATR and corticospinal tract (in the right hemisphere cluster), near the precentral gyrus and middle frontal gyrus. There were bilateral clusters (2 and 3) in

the cerebral peduncles, including fibers from the ATR and corticospinal tract, near the amygdala and the globus pallidus. There was a medium right (4) and large left (5) temporal/parietal cluster, including fibers from the inferior and superior longitudinal fasciculi, inferior fronto-occipital fasciculus, and ATR (on the left only); the cluster was located near the supramarginal gyrus, superior temporal gyrus, parietal operculum cortex, and planum temporale on the left, and near the inferior, middle, and superior temporal gyri, parahippocampal gyrus, and temporal fusiform cortex on the right. There was a right parietal cluster (Cluster 6) including fibers from the inferior and superior longitudinal fasciculi, inferior fronto-occipital fasciculus, and ATR in the region of the angular gyrus, supramarginal gyrus, and lateral occipital cortex. An additional right frontal cluster (7) included fibers from the inferior fronto-orbital fasciculus, ATR, SLF, UF, and forceps minor, and was located near the frontal orbital cortex, insula, inferior frontal gyrus, frontal operculum cortex, and pars triangularis. Finally, there was a left-sided cluster (8), including fibers from the cingulum, forceps minor, ATR, SLF, and UF near the anterior cingulate gyrus and the paracingulate gyrus in the frontal lobe. Partial *r*s reflecting the associations between mean diffusivity and delay discounting behavior, controlling for participant sex, were somewhat stronger in magnitude than those observed for FA and ranged from .40 to .52. The MD value for each voxel cluster was extracted and plotted against delay AUC (Figure 4).

Age Influences on Relations between Delay AUC and MD

Given that delay discounting rates decrease with age, the possibility that the observed associations between delay AUC and MD were attributable to age was investigated. First, relationships between age and MD were examined for each cluster from Table 3. The MD value across all voxels within each cluster was extracted and correlated with age using Pearson correlations (Figure 5; Table 4). As indicated in Table 4, correlations with age ranged from −.30 to −.65. As with the FA analyses, linear, quadratic, and cubic models were fit to the age–MD associations, and the models were compared using AIC to assess model fit. All relationships were linear, indicating that linear models provided the best description of the age–MD relations within these clusters.

Thus, in all of the identified clusters where there was a significant association between delay AUC and MD, there also was a significant linear relationship between age and MD. In order to address whether the age–MD relationship accounted for the delay AUC–MD relationship, age was entered into the regression equation in SPM2. There were two regions that remained significant after controlling for age (Table 3). These regions were near the right frontal lobe (Cluster 1, involving the SLF) and right amygdala/globus pallidus (Cluster 2, involving the corticospinal tract and ATR). In these regions, there is an age-independent relationship between delay AUC and MD; in the remaining regions, the relationship is age-dependent.

The delay AUC–MD analysis was repeated using total scores on the PDS rather than age as a covariate in order to explore whether controlling for pubertal status produced different results. Controlling for pubertal status rather than age resulted in the addition of one small cluster (28 voxels).

Evidence of Specificity

To address the question of whether the observed associations reflect a specific relationship between delay discounting and white matter organization or whether they reflect more general processes, we computed Pearson correlations between several other behavioral measures and the mean voxel values in the FA and MD clusters from the voxelwise analysis of delay discounting AUC (Table 4).

Verbal IQ was selected as a correlate because delay discounting behavior is known to be correlated with verbal IQ in this sample (Olson et al., 2007); if some general cognitive process involved in completion of both verbal intelligence subtests and the delay discounting task accounted for the findings from the voxelwise analysis, then verbal IQ would also be correlated with the mean FA and MD values in these clusters. A *p* value of .01 was used to assess significance due to the large number of comparisons in the table. There were no significant associations between verbal IQ and mean FA or MD cluster values (Table 4).

To extend the specificity analysis to the voxel level, verbal IQ was entered as a covariate in the SPM analyses. For FA, seven of nine clusters (1, 2, 3, 5, 6, 7) continued to show significant associations between FA and delay AUC after controlling for the effect of verbal IQ (Table 2). For MD, the relations between delay AUC and MD remained significant even after controlling for verbal IQ in Regions 1, 2, 3, 4, 5, 6, and 8 (Table 3). One cluster (Cluster 7) was no longer significant after controlling for verbal IQ. This analysis confirms that the associations between delay AUC and the DTI variables are not attributable to the effect of verbal IQ and illustrates that the observed associations between delay AUC and the imaging variables are not attributable to general cognitive processes common to the discounting task and the verbal intelligence measures.

Further evidence of specificity comes from an analysis of the probability discounting data. In addition to the delay discounting task, a probability discounting task was administered to participants ages 11 and up; 62 participants had valid data on both tasks. This task involves many of the same behavioral elements as the delay discounting task, including reading items and choosing between two numeric options. There was a single cluster (FA Cluster 4) with a significant correlation between probability discounting and mean FA; there were no significant correlations for the remaining FA clusters or for any of the MD clusters (Table 4).

DISCUSSION

Overall, our results confirmed the hypothesis that higher FA and lower MD values in white matter pathways that interconnect the lateral prefrontal and temporal–parietal cortices with other brain regions are associated with lower rates of delay discounting in a healthy 9-to-23-year-old sample. We found several white matter regions where increased white matter organization, indicated by higher FA values, was associated with an increased preference for larger delayed rewards. Primarily, these regions included pathways in the region of the frontal cortex bilaterally, including areas near the insula and the OFC as well as the dorsolateral region, as well as a large extent of a pathway, the ILF, in the left temporal lobe.

Individuals with relatively high levels of white matter organization in these areas discounted less steeply, independently of the effects of age and verbal IQ. In addition, we identified pathways where relatively low levels of mean diffusion were associated with an increased preference for larger delayed rewards. These included tracts coursing bilaterally through frontal, temporal–parietal, limbic and striatal regions. Clusters of lower MD in the right frontal lobe, left temporal lobe, and right amygdala/basal ganglia were significantly associated with less steep discounting, independent of the effects of age and verbal IQ. Overall, our results indicate that white matter fiber organization contributes to individual differences in discounting behavior. Our findings suggest that effects in some, but not all, regions are attributable to developmental changes.

Although functional imaging studies have tended to emphasize the role of lateral frontal regions in promoting selection of larger later rewards, in the present study, the largest cluster was in the left temporal lobe involving the ILF. This tract connects the occipital lobe with anterior portions of the temporal lobe (Catani, Jones, Donato, & ffytche, 2003), and its function is debated (Mandonnet, Nouet, Gatignol, Capelle, & Duffau, 2007). It has been implicated in emotional processing, in visual memory, and in semantic processing (Mandonnet et al., 2007; Catani et al., 2003; Ross, 1980). The finding of significant temporal involvement in delay discounting through the involvement of this pathway is consistent with the animal literature. In rodents, hippocampal lesions produce preferences for smaller sooner rewards on discounting tasks (for a review, see Cardinal, 2006). The left temporal lobe is involved in processing information regarding time and/or duration. A left-hemisphere advantage has been documented for temporal processes including ordering stimuli, perceiving simultaneity, perceiving temporal order, duration discrimination, and gap detection (for a review, see Elias, Bulman-Fleming, & McManus, 1999). Increasing left temporal lobe white matter organization may lead to an improved ability to tolerate delays by facilitating the comparison between the two durations involved in each question (immediate vs. delayed rewards). Interestingly, the PFC has also been implicated in the accurate processing of time delays (Koch, Oliveri, Carlesimo, & Caltagirone, 2002; Mangels, Ivry, & Shimizu, 1998). Perhaps better accuracy in processing delays leads to improved preference for delayed rewards because there is a “default mode” of choosing immediate rewards in the absence of accurate delay interval processing.

Within the left hemisphere, increased FA in the region of the UF was also associated with a greater tolerance for delayed rewards. The UF connects the anterior temporal lobe with the OFC. Low FA in this region has been associated with relatively deficient levels of general intelligence, memory, and executive function in schizophrenia-spectrum disorders (Nakamura et al., 2005; Nestor et al., 2004; Kubicki et al, 2002), with dementia of the Alzheimer’s type (Taoka et al., 2006), as well as with socioemotional difficulties in postinstitu-tionalized children (Eluvathingal et al., 2006). Together with other pathways in the left hemisphere, such as the ATR that connects anterior and dorsomedial regions of the thalamus with dorsolateral regions of the frontal lobe, the ILF and the UF may facilitate the language and emotion-based evaluative processes as well as behavioral control functions that facilitate the ability to delay gratification. In the right hemisphere, the ILF and the UF again were identified as pathways where increased white matter organization was associated with lower discounting rates, as were the SLF and inferior occipito-frontal fasciculus

(IFOF). The SLF is a long association bundle with four branches that contribute, respectively, to language functions (arcuate fasciculus), to spatial attention and working memory via connections between the parietal and frontal cortices, and to language articulation processes (Makris et al., 2005). The IFOF is also a long association pathway that interconnects the occipital lobe with the frontal cortex. Given that the portions of these pathways that were implicated in the current study were largely located in temporal and frontal regions, they are situated to integrate the emotional and cognitive demands inherent to the process of delayed gratification.

No regions were identified where increased white matter organization or tissue density was associated with smaller AUCs—that is, there were no regions where better white matter organization predicted increased preference for smaller sooner rewards. Functional imaging studies have generally found that increased activity in the ventral striatum and in medial prefrontal areas promotes selection of smaller sooner rewards (Hariri et al., 2006; McClure et al., 2004). It is unclear why high FA and low MD in these areas was not associated with higher rates of delay discounting. One interpretation of the lack of findings in the reverse direction is that the key event in influencing delay discounting behavior in this age range is the maturation of the DLPFC and associated white matter connections. The ventral striatum may be more clearly implicated in behavioral patterns exhibited by clinical groups where impulsive behavior is a maximally salient trait.

Our initial hypothesis was that developmental changes in white matter organization in the PFC would be associated with developmental changes in the delay discounting rate. Our results suggest that there are both age-dependent and age-independent relationships between white matter development and delay discounting. Age-dependent effects on delay AUC were widespread and appeared to be stronger in younger (9- to 16.5-year-old) versus older (17- to 23-year-old) participants. When age was entered as a covariate in the regression analyses involving the total sample, there were several regions where the associations between FA or MD and discounting remained significant. These included portions of the right SLF in the frontal lobe near the precentral and middle frontal gyri for both FA and MD (with additional right frontal regions of the ATR and IFOF for FA only), a cluster near the globus pallidus and the amygdala involving fibers from the corticospinal tract and the ATR (for MD only), and a large region in the left temporal lobe for both FA and MD involving fibers from the ILF and the IFOF (with additional regions of the superior longitudinal fasciculus and ATR for MD only). To summarize, white matter microarchitecture in right frontal and left temporal regions (as well as near the globus pallidus and the amygdala for MD only) was associated with individual differences in delay discounting performance that were not attributable to age in this sample. In contrast, white matter tracts in left frontal and right temporal and parietal regions, as well as pathways near the amygdala, hippocampus, thalamus, anterior cingulate/ paracingulate gyrus, and splenium of the corpus callosum, showed age-dependent associations between white matter organization and delay discounting behavior.

These results suggest the possibility that developmental changes in discounting behavior may be attributable to structural changes in the brain in the latter set of regions, whereas individual differences in discounting (apart from developmental considerations) may be

attributable to differential patterns of white matter organization in left temporal and right frontal regions. Individual differences in delay discounting have been identified on the basis of a variety of correlates, including intelligence (de Wit, Flory, Acheson, McCloskey, & Manuck, 2007; Olson et al., 2007), and behavioral/personality traits, including aggression (Bjork, Hommer, Grant, & Danube, 2004) and impulsivity (Kirby & Petry, 2004). Because several associations between delay discounting and white matter organization remained significant after controlling for the effect of verbal IQ in this sample, it is unlikely that the influence of the right frontal and left temporal regions is attributable to IQ. One possibility is that left temporal and right frontal white matter organization may differ in individuals who are more or less aggressive or impulsive. However, in this sample, there was no association between delay discounting behavior and overall scores on a measure of externalizing behavior (Olson et al., 2007). The nature of the individual difference reflected in the age-independent effect of left temporal and right frontal white matter is therefore somewhat unclear, but further research should address whether more specific aspects of externalizing behavior (such as aggression or impulsivity) may contribute to this association.

We have presented some evidence of specificity of the reported associations to delay discounting behavior as opposed to global cognitive processes: The identified voxel clusters were not associated with verbal IQ. With the exception of FA Cluster 4, they also were not associated with probability discounting rates. The incomplete overlap of the delay and probability discounting samples limits our ability to make direct comparisons with respect to the imaging data. However, the probability discounting findings and the verbal IQ findings together provide some preliminary evidence that the reported associations do not reflect general or global cognitive processes, instead reflecting processes more specific to the delay discounting task.

That said, although relatively little is known about the functional correlates of the brain's white matter structure as it develops, it is most certainly the case that maturational increases in white matter organization will influence functioning in both broad and narrow cognitive domains. We view it as a future goal to determine whether fiber pathways that are associated with specific tasks can be described so that the complete network underlying each task can be better understood and then discriminated from the networks that underlie different tasks.

There are a variety of possible process-related explanations for the age-associated decrease in the delay discounting rate. Nine- and 10-year-olds were not more likely to produce invalid task data, suggesting that they were able to comprehend the task. However, for several reasons, younger children may evaluate the relevant quantities differently from older adolescents and adults. Older children may have more subjective experience of waiting through long delays. Relatedly, younger children may perceive a given delay as longer because it reflects a larger fraction of their lifetimes. It is also the case that younger children may place greater subjective value on small amounts of money than older children (although many discounting studies have used equivalent monetary rewards for younger and older participants; e.g., Lamm, Zelazo, & Lewis, 2006; Scheres et al., 2006; Green, Fry, & Myerson, 1994). These processes likely contribute to differences in the discounting rate; evaluation of the mechanisms of change in delay discounting with maturation is a direction for future research.

Age-related changes in behavior and cognition during adolescence may arise, in part, because the pubertal transition confers a number of hormonal and other neurochemical changes that interact with other aspects of brain development to alter behavior, and these changes may differently affect cognition in the two sexes (Overman, 2004). A stringent analysis of pubertal effects on performance and on brain development would require adequate samples of males and females spanning the full range of pubertal development, because males and females vary in the time course of puberty-related hormonal changes (Giedd et al., 2006). Unfortunately, we had limited ability to analyze possible changes in the association between white matter development and delay discounting during the transitions from prepuberty to puberty to postpuberty because our sample included very few prepubertal participants. In addition, the process would need to be examined separately for each sex, given the differences in the neurobiological underpinnings and timing of puberty in the two sexes. Expansion to a younger sample would permit this type of analysis. In addition, in our sample, pubertal status was highly correlated with chronological age, making it difficult to isolate maturational changes that can be attributed to puberty but not age. For instance, although we found that pubertal development correlated with delay discounting performance, this relationship was no longer significant after controlling for age. Similarly, we conducted separate analyses entering PDS total scores rather than age into the regression equations as a covariate; the same voxel clusters remained significant whether we controlled for age or for pubertal status scores.

At a behavioral level, sex did not affect performance on the discounting task (Olson et al., 2007). In addition, the interaction between sex and age also was not a significant predictor of discounting behavior. There were two voxel clusters in the voxelwise analysis where there was a sex difference in FA, and there were three clusters where there was a sex difference in MD. We controlled for these sex differences in the regression analyses by entering sex as a covariate. The effect of covarying sex on the DTI findings was minimal; the relative strengths of the associations between delay AUC and FA or MD in different voxel clusters was slightly different when sex was covaried, but no clusters were added or deleted. Thus, it appears that the impact of sex differences on the reported brain–behavior relationships is minimal. A more detailed analysis of potential sex differences is an important direction for future research.

The present study is not without limitations. Longitudinal follow-up studies are needed in order to assess whether developmental changes in white matter organization within individuals predict behavioral maturation on discounting tasks. In addition, given the low numbers of pre- and early pubertal participants, our ability to interpret the developmental implications of this study is limited, especially in relation to pubertal status. The ability to assess relationships between cognitive factors, behavioral factors, and demographic factors may be limited by nature of our sample, which is predominantly Caucasian, of relatively high socioeconomic status, and of above-average intelligence. The reported associations should be examined in groups with average or low-average intelligence to determine their generalizability. In addition, we did not use tractography to localize significant clusters in relation to probabilistic maps of white matter pathways derived from our own sample. However, we did set a conservative FA threshold of 0.2 for white matter masking, thereby reducing confounds due to partial volume effects and increasing the likelihood that

significant structure–function relationships involved white matter versus other tissue compartments. Additionally, visual inspection of these clusters indicated that they were located on or near major white matter tracts, versus areas with higher concentrations of gray matter or cerebrospinal fluid.

Because delay discounting rates are disturbed in various forms of psychopathology, these findings may have implications for high-risk samples. There is a large body of literature documenting elevated delay discounting rates in individuals meeting criteria for substance abuse and dependence (for a review, see Reynolds, 2006). Elevated delay discounting rates have also been found in populations with impulse control disorders, including pathological gamblers (Alessi & Petry, 2003). In addition, relative to healthy controls, elevated rates of delay discounting are seen in individuals with schizophrenia and schizoaffective disorder (Heerey, Robinson, McMahon, & Gold, 2007), social anxiety (Rounds, Beck, & Grant, 2007), and depressive symptomatology (Yoon et al., 2007). It is unknown whether disturbance in the discounting rate precedes and predicts the development of associated psychopathology, or whether it occurs as a result. In addition, the relationships between age, verbal IQ, white matter organization, and delay discounting may differ in high-risk versus low-risk samples; an extension of these findings to high-risk adolescent samples is an important goal for future research.

The ability to tolerate increasing delays as individuals anticipate future rewards is a critical aspect of motivated behavior. The findings from this study contribute to the definition of salient neural pathways in cortical and subcortical regions that contribute to this complex behavior. Overall, our data indicate that higher FA and lower MD values in white matter pathways that interconnect the lateral prefrontal and temporal/parietal cortices with other brain regions are associated with lower rates of delay discounting in a healthy adolescent sample. Some of these associations are not independent of the effect of age and, therefore, likely reflect developmental processes, whereas others, particularly in the left temporal and right frontal lobes, are age-independent; these age-independent effects may reflect individual differences in temporal information processing ability and/or in aspects of personality and behavioral functioning such as impulsivity or aggression.

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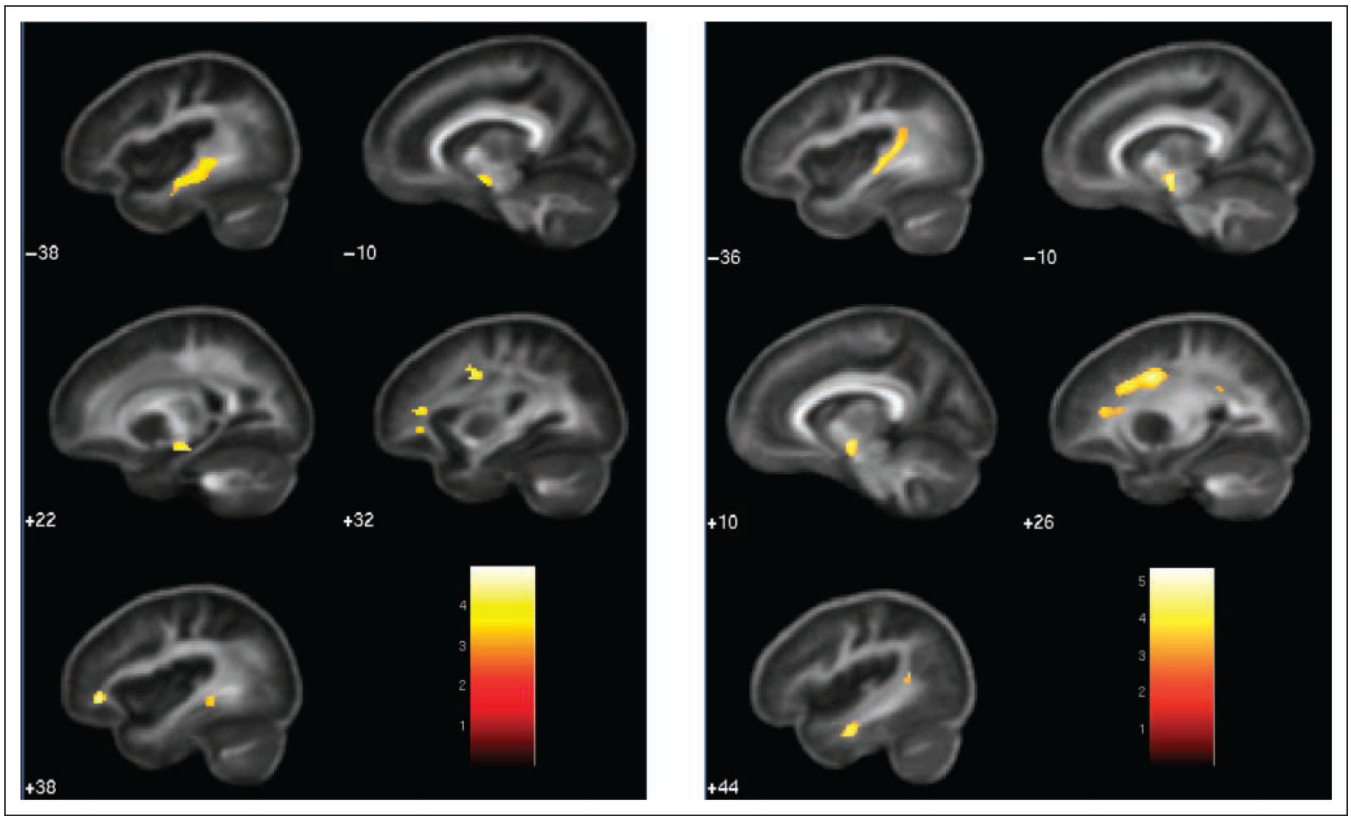

Figure 1.

Left: Selected sagittal slices where high FA is associated with high delay AUC. Right: Selected sagittal slices where low MD is associated with high delay AUC. Signed numbers are *x*-coordinates in standard MNI space.

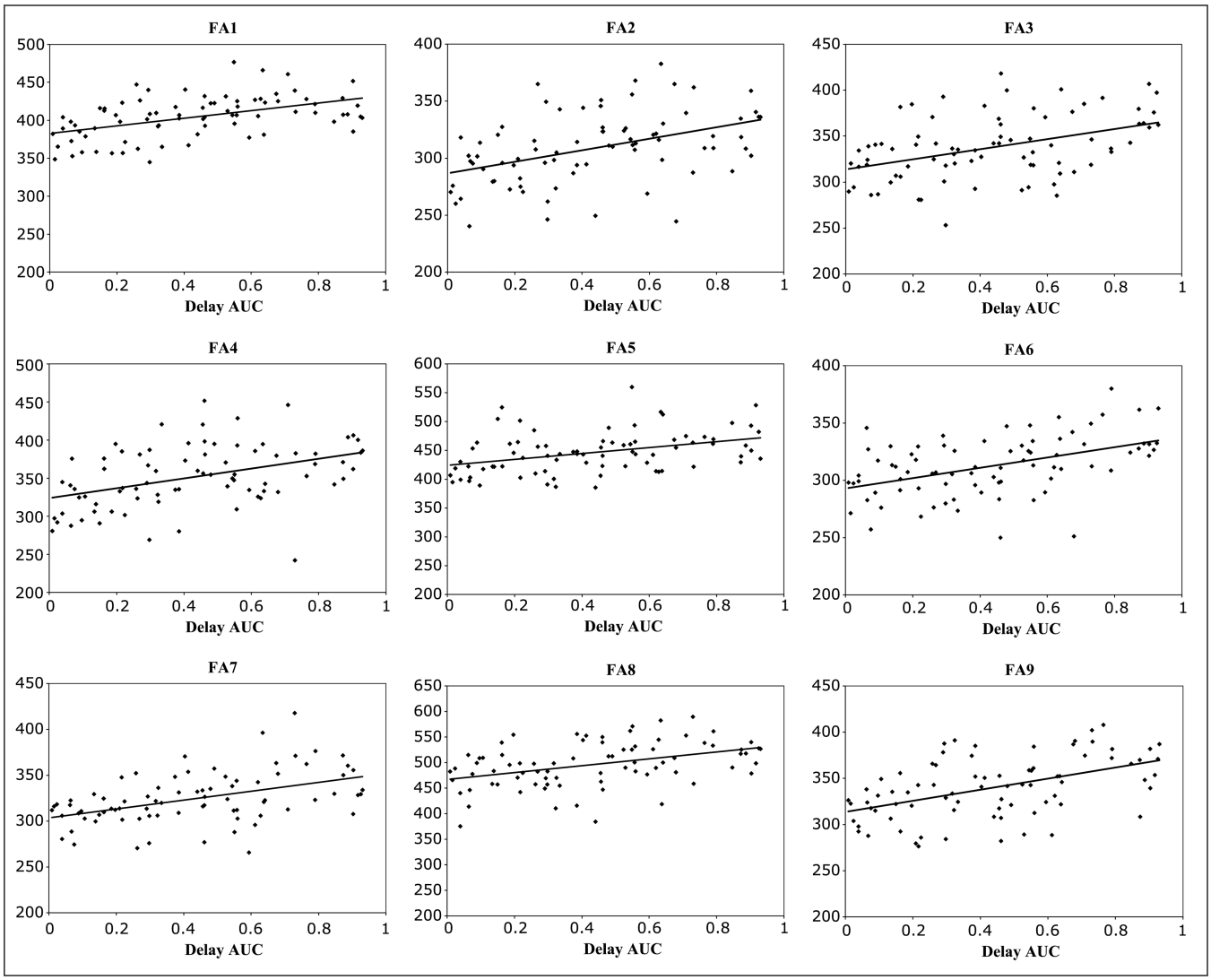

Figure 2.
Scatterplots of delay AUC-mean FA correlations for Table 2 voxel clusters.

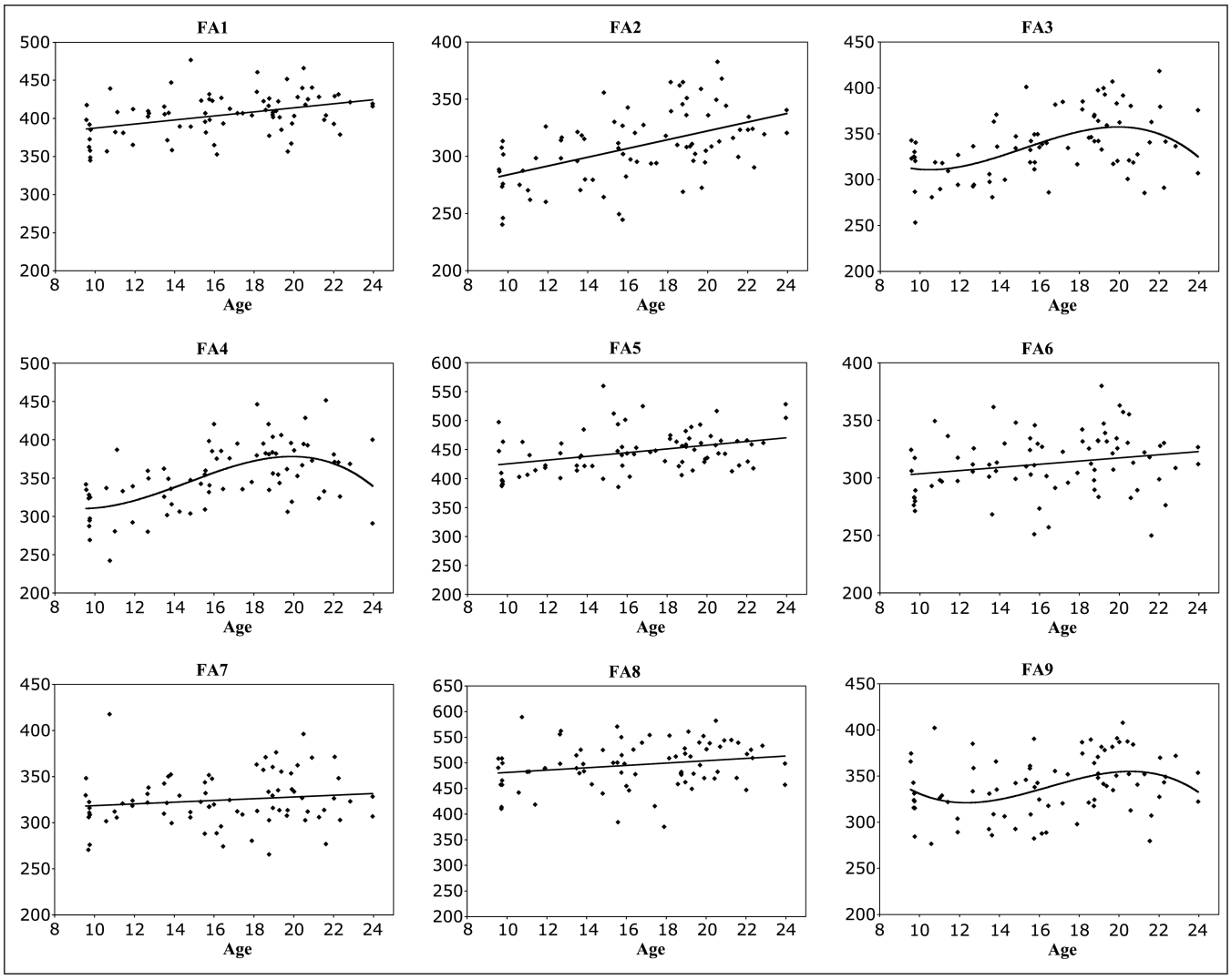

Figure 3.
Plots of age-mean FA relationships for Table 2 voxel clusters.

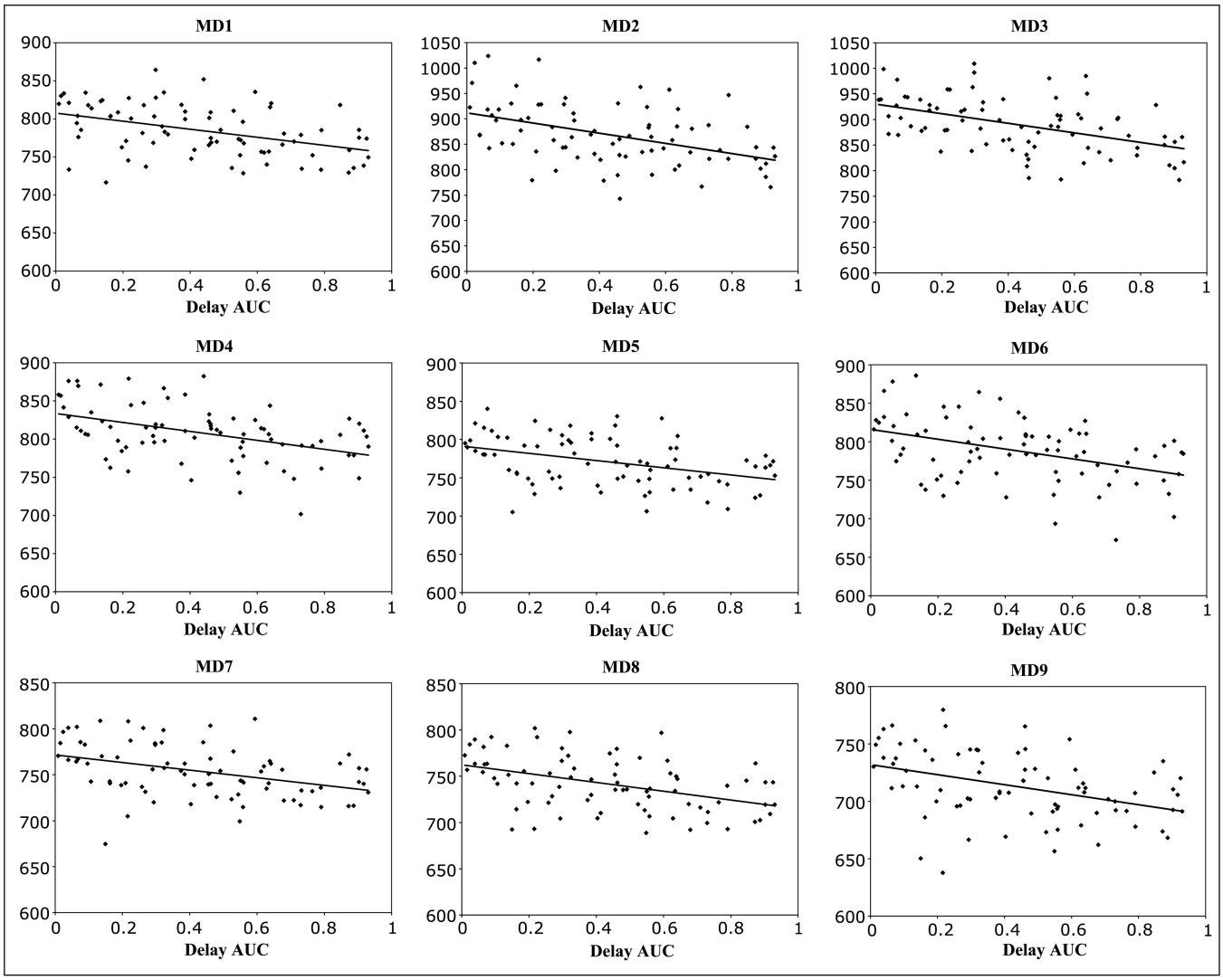

Figure 4.
Scatterplots of delay AUC–mean MD correlations for Table 3 voxel clusters.

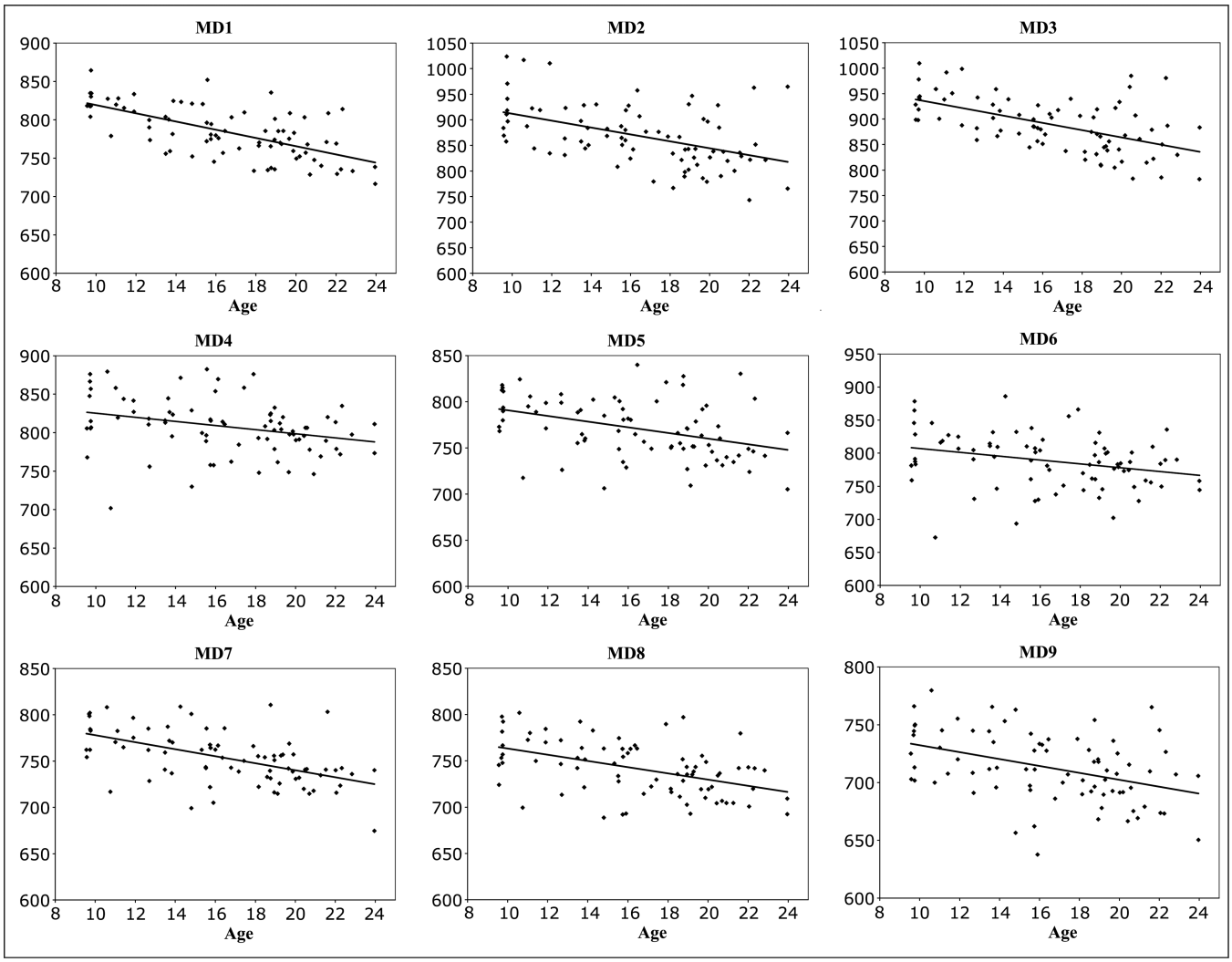

Figure 5.
Plots of age–mean MD relationships for Table 3 voxel clusters.

Table 1

Sample Characteristics

n	**79**
% Female	53.2
% Caucasian	87.2
Age (years)	16.44 (4.10)
Mother's education (years)	15.41 (2.69)
Father's education (years)	15.47 (3.22)
Income	91,493 (71,292)
WASI Verbal IQ	116.13 (9.22)
WASI Performance IQ	112.46 (11.44)
WASI Full Scale IQ	116.18 (9.85)
Delay AUC	.4339 (.27)

Values represent percentages or means (and standard deviations).

Table 2

White Matter Tracts where High FA is Associated with High Delay AUC

Region	Cluster Size (Voxels)	Max Partial r	x	y	z
1. Right: ATR, IFOF	58*,†	.4968	30	38	10
2. Right: IFOF, ATR	39†	.4769	38	40	−4
3. Right: Superior longitudinal fasciculus	61†	.4714	30	4	36
4. Right: corticospinal tract	37	.4514	22	−18	−16
5. Left: Uncinate fasciculus	78†	.4473	−32	6	−14
6. Left: IFOF, inferior longitudinal fasciculus	243*,†	.4432	−36	−34	0
7. Left: ATR	48†	.4273	−6	−6	−10
8. Right: IFOF, inferior longitudinal fasciculus	38	.3889	40	−36	−4
9. Left: Forceps major/splenium of corpus callosum	40	.3790	−18	−44	22

ATR = anterior thalamic radiation; IFOF = inferior fronto-occipital fasciculus.

Clusters remain significant when *age or

†VIQ is entered into the regression equation.

Table 3

White Matter Tracts where Low MD is Associated with High Delay AUC

Region	Cluster Size (Voxels)	Max Partial r	x	y	z
1. Right: Superior longitudinal fasciculus	489*,†	.5245	28	2	36
2. Right: Corticospinal tract, ATR	153*,†	.4983	14	−10	−10
3. Left: ATR, corticospinal tract	57†	.4877	−12	−10	−10
4. Right: Inferior longitudinal fasciculus, superior longitudinal fasciculus	101†	.4514	44	−8	−26
5. Left: Inferior longitudinal fasciculus, superior longitudinal fasciculus, IFOF, ATR	355†	.4264	−38	−40	8
6. Right: Inferior longitudinal fasciculus, superior longitudinal fasciculus, IFOF, ATR	166†	.4239	36	−54	24
7. Right: IFOF, ATR, superior longitudinal fasciculus	146	.4084	28	34	12
8. Left: Cingulum, forceps minor, ATR	104†	.4013	−16	24	28
9. Left: Superior longitudinal fasciculus	60	.4005	−26	−4	38

Clusters remain significant when *age or

†VIQ is entered into the regression equation.

Table 4

Partial Correlations between Mean DTI Values and Variables of Interest, Controlling for Gender

	Delay AUC	Age	VIQ	Probability AUC[a]
FA1	.478*	.406*	.231	.105
FA2	.438*	.510*	.266	.059
FA3	.436*	.425*	.194	.196
FA4	.421*	.522*	.033	.371*
FA5	.394*	.359*	.194	.162
FA6	.461*	.224	.032	.011
FA7	.474*	.133	.130	.103
FA8	.403*	.253	.108	.086
FA9	.485*	.305*	.225	.235
MD1	−.432*	−.653*	.063	−.208
MD2	−.466*	−.461*	−.018	−.224
MD3	−.496*	−.546*	−.078	−.139
MD4	−.430*	−.331*	−.128	−.226
MD5	−.409*	−.389*	.002	−.129
MD6	−.411*	−.304*	−.099	−.132
MD7	−.399*	−.540*	−.046	−.142
MD8	−.445*	−.470*	−.061	−.228
MD9	−.394*	−.412*	−.133	−.196

[a] $n = 62$ (individuals with good delay discounting, probability discounting, and imaging data).

* $p < .01$.