

# Heritability of different measures of smooth pursuit eye tracking dysfunction: A study of normal twins

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## Abstract

Research studies have found that smooth pursuit eye movement dysfunction may serve as an index of genetic liability to develop schizophrenia. The heritability of various measures of smooth pursuit eye tracking proficiency and the saccades that occur during smooth pursuit was examined in 64 monozygotic (MZ) and 48 dizygotic (DZ) twin pairs. Two age cohorts were assessed (11–12 and 17–18 years of age). Intraclass correlations indicated significant similarity in the MZ twins for almost all measures in both age cohorts, whereas few of the DZ twin correlations attained significance. Biometrical modeling indicated that genetic mechanisms influence performance on both global and specific eye tracking measures, accounting for about 40% to 60% of the variance. These findings suggest that the underlying brain systems responsible for smooth pursuit and saccade generation during pursuit are under partial genetic control.

**Descriptors:** Smooth pursuit, Saccades, Schizophrenia, Twin studies

Recent advances in molecular genetics have helped researchers identify the mode of genetic transmission for debilitating hereditary illnesses such as Huntington's disease (Morrel, 1993). Despite these advances, research efforts to identify schizophrenia-related genes have met with limited success, in part because linkage studies rely on symptom presentation to identify individuals as affected. However, the use of symptoms to identify the boundaries of the schizophrenia spectrum is a major source of variance in these studies, because of the lack of agreement regarding what constitutes schizophrenia "spectrum" symptoms. This problem can lead to the misidentification of family members as gene carriers when they are not. In addition, reliance on symptom assessment fails to identify individuals who, although symptom free, are carriers of a predisposing gene or genes. These factors add error variance in studies that attempt to identify genetic material linked to schizophrenia. The use of biological markers that reflect genetic susceptibility to schizophrenia holds great promise as a case identification aid because it would enable the identification of genetic risk in asymptomatic and symptomatic relatives who possess the abnormal gene(s).

Numerous research investigations conducted across various laboratories have found that smooth pursuit eye movement dysfunction, the inability to visually follow a slowly moving target smoothly,

may serve as an index of genetic liability to develop schizophrenia (for a review see Iacono, 1998; Levy, Holzman, Matthyse, & Mendell, 1993). Eye tracking impairment has been found to be stable over time in both normal individuals and patients with schizophrenia. Abnormal smooth pursuit has been found in higher proportion among patients with schizophrenia than normal controls. In addition, family studies of schizophrenia have found that asymptomatic first-degree relatives of patients have significantly more impaired eye tracking than normal control subjects. With one exception (Litman et al., 1997; see also Holzman, Levy, & Abel, 1997), the several twin studies of pursuit eye tracking that have been conducted have been interpreted as consistent with the notion that pursuit tracking performance is a heritable trait. Holzman et al. (1988) and Matthyse, Holzman, and Lang (1986) analyzed their data using latent structure modeling. Their data were fit by a model suggesting that impaired eye tracking and schizophrenia are expressions of a single underlying trait that is transmitted as an autosomal dominant gene. Grove, Clementz, Iacono, and Katsanis (1992), examining the eye tracking of patients with schizophrenia and their first-degree relatives, found that eye tracking impairment fit a mixed model with a large single gene effect. The use of deviant pursuit in genetic modeling may facilitate research seeking to identify the genetic transmission of the disorder. For instance, several studies have found linkage of schizophrenia to chromosome 6 (Moises et al., 1995; Schwab et al., 1995). Arolt et al. (1996), following the lead of these investigators, tested for linkage between DNA markers on chromosome 6 and eye tracking dysfunction. They found linkage between chromosome 6 and impaired tracking (but not to schizophrenia), providing further suggestive evidence that eye tracking dysfunction can serve as a susceptibility marker for schizophrenia.

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Many studies have demonstrated that the eye tracking of schizophrenia patients is abnormal. However, these family, twin, and genetic modeling studies have provided little insight into what types of tracking deviations are most heritable, or whether there is any difference in the heritability of different aspects of pursuit performance. This lack of insight derives from the fact that these studies have relied on global measures of pursuit dysfunction. These measures provide for the quantification of individual differences in pursuit tracking ability in a general fashion, without taking into account the specific nature of the tracking deficit. No study has examined the genetic influences on eye tracking by differentiating between smooth pursuit eye movements and the saccadic defects that occur during smooth pursuit. Saccades are high-velocity eye movements that, along with low-velocity smooth pursuit, enable individuals to keep their eyes fixated on the target. Because the high- and low-velocity eye movements are controlled by different parts of the central nervous system, Abel and Ziegler (1988) have argued that in order to gain a precise understanding of the nature of the eye tracking deficit in schizophrenia and its neuroanatomical locus, assessment of smooth pursuit should include classification of the saccades that occur during pursuit.

The present study is the first to examine the heritability of various components of smooth pursuit tracking, including the differential heritability of the saccades that occur during smooth pursuit, in a large sample of normal monozygotic (MZ) and dizygotic (DZ) twins. Two age cohorts were examined, one 11–12 years of age and the second 17–18 years of age. We have found (Katsanis, Iacono, & Harris, 1998) that during preadolescence the oculomotor system is still developing and has not reached adult levels of functioning. Given the developmental differences and the fact that all the twin studies to date have been conducted on adults, it is of interest to determine how the heritability of smooth pursuit performance varies with age. Examination of these two samples made it possible to determine the presence of genetic influences on eye tracking during both preadolescence and late adolescence.

## Method

### Subjects

The subjects were MZ and DZ reared-together male twin pairs who were participants in an ongoing longitudinal investigation, the Minnesota Twin Family Study (MTFS). Twenty-nine MZ and 20 DZ twin pairs were between the ages of 17 and 18 and 35 MZ and 28 DZ twin pairs were 11–12 years old. The MTFS was designed to examine the genetic and environmental influences on the development of substance use and related behavioral disorders. Potential participants were identified from birth certificates and were recruited, along with their parents, by telephone and mail. An overview of the research design, including a thorough characterization of the twin families and the extent to which they are representative of twin families in Minnesota can be found elsewhere (Iacono, Carlson, Taylor, Elkins, & McGue, 1999).

Zygoty was determined using the following three methods: (a) parents completed a zygoty questionnaire that included questions regarding the twins' physical similarity and frequency with which family members and others confused them; (b) research staff determined zygoty after rating twins on physical similarity (e.g. eye color, ear shape, etc.); and (c) zygoty was determined using an algorithm based on ponderal index, cephalic index, and fingerprint ridge count. In the event that the above three methods disagreed, a serological examination was carried out on 12 blood group antigens and protein polymorphisms. To assess the efficacy

of our twin identification procedure, we assessed 50 twin pairs with all four methods. We found that when the three methods involving behavioral and physical measures agreed, the serological analysis always confirmed this agreement.

Study participants were administered structured interviews to assess the presence of various psychiatric conditions, including externalizing childhood disorders (conduct, oppositional defiant, attention deficit hyperactivity, and drug/alcohol use disorders), major depression, and substance abuse/dependence. Only subjects who did not satisfy the diagnostic criteria for any of the assessed psychiatric disorders were included in the present investigation.

### Psychophysiological Assessment

The data presented in this report were collected as part of a 3.5-hr battery of psychophysiological tests that began with an oculomotor assessment that has been described in detail elsewhere (Katsanis et al., 1998). The target, a luminescent dot subtending  $0.4^\circ$  of visual arc, oscillated at 0.4 Hz with an excursion of  $\pm 10^\circ$  of visual angle for 30 s. Subjects were asked to follow the target with their eyes, to try not to blink, and to hold their head still. Head movement was kept at a minimum with the use of chin, forehead, and occipital supports.

Horizontal eye movements were recorded with an Eye Trac Model 210 eye movement monitor and infrared (IR) spectacles (Applied Science Laboratories, Inc., Waltham, MA). The lower bound of accuracy with this instrument is  $0.25^\circ$  of visual angle and it has a time constant of 4 ms. Vertical eye movements were recorded from electrooculogram electrodes placed on the outer canthus and above one eye and were digitized on line at 256 Hz. Data were recorded on a Grass Model 12A Neurodata acquisition system with 1/2 amplitude low frequency filter settings at 0.01 Hz (18 dB/octave roll-off). The 50% bandwidth for high frequency filtering was set to 30 Hz (18 dB/octave roll-off).

### Eye Movement Analysis

A comprehensive assessment of eye tracking performance was carried out using both global and specific measures. A detailed description of the scoring of these measures has been reported in Katsanis et al. (1998).

#### Global Measures

Before eye movement scoring, all records were reviewed and epochs with blink and other artifacts were identified and removed.

*Root-mean-square error (RMS error).* RMS error, representing the deviation between the target and the IR signal, was computed, as has been traditional in our laboratory, after the two signals were aligned for phase differences (Iacono & Lykken, 1979).

*Saccade rate.* The saccade rate was calculated as the number of saccades that occurred during the task divided by the time in seconds.

*Amplitude and velocity of saccades.* In addition to the frequency, saccades have two other features: amplitude and velocity. To obtain a "global" index of these properties for the purpose of this study we also computed the average saccade amplitude in degrees and average velocity in degrees per second.

#### Smooth Pursuit Measures

*Amount of pursuit.* The amount of pursuit is the amount of time in seconds that the eyes engaged in smooth pursuit. This measure

was derived after epochs containing saccades and fixations were removed from the eye movement signal. This task was carried out using a computer program written with ASYST software (Version 3.1; Asyst Software Technologies, 1990).<sup>1</sup>

**Pursuit gain.** Gain reflects the ability of the smooth pursuit system to match eye velocity to target velocity. When performance is optimal, gain is equal to 1.00. Gains of less than 1.00 reflect an inability of the smooth pursuit system to lock the fovea onto the target. We computed frequency- and time-domain gain using intervals of smooth pursuit free of saccades and fixations. Time-domain gain reflects the extent that eye velocity matches target velocity. Frequency-domain gain was defined as the ratio of the power present at 0.4 Hz in the eye and target signal.

#### Measures of Saccade Types

Saccades that were more than 0.5° of visual angle were identified and classified as corrective or intrusive saccades. For each saccade type, we assessed its frequency, amplitude, and velocity.

**Corrective saccade measures.** Corrective saccades appear to compensate for a mismatch between the position of the target and the eye. They are subclassified as catch-up saccades, back-up saccades, and initiation saccades. *Catch-up saccades* are less than 5° in amplitude. They are generated when the pursuit eye velocity is less than the target velocity. *Back-up saccades* are generated when the eyes are ahead of the target and function to move the eyes from a position ahead of the target to the target. *Initiation saccades* occur primarily during the target turnaround point when the eyes stop to reverse direction. They consist of a brief fixation followed by a saccade in the direction of target motion.

**Intrusive saccade measures.** Intrusive saccades interrupt smooth pursuit by taking the eyes away from the target. They can be subclassified into anticipatory saccades and square wave jerks. *Anticipatory saccades* are at least 5° in amplitude. They move the eyes in the direction of the target to a point ahead of the target. *Square wave jerks* are pairs of back-to-back saccades roughly equivalent in size that have an intersaccadic interval of 80–450 ms.

#### Data Analyses

The distributions of most of the eye tracking variables were skewed; therefore, all measures were normalized using the van der Waerden transformation. With this transformation the data are ranked and the data values are replaced with the expected value for a score of that rank from the normal distribution (Guilford & Fruchter, 1979). Because we expected twins to be similar on the dependent measures, all intraclass correlations were assessed using one-tailed significance tests. Similarly, because MZ twin pairs were expected to be more similar on the measures than the DZ twins, all comparisons between MZ and DZ twins were assessed using one-tailed  $z$  tests.

Similarity between twins was measured by computing intraclass correlations. Intraclass correlations can be used to infer the presence of three broad classes of etiological influences. Additive genetic influences (which increase phenotypic similarity among biological relatives) are inferred when the MZ correlation is roughly

double the DZ correlation for a trait. Shared environmental influences (which also serve to increase phenotypic similarity among family members) are inferred when the MZ and DZ correlations are of similar magnitude. Nonshared environmental influences (unique environmental experiences that serve to decrease phenotypic similarity) are typically calculated as a residual to additive genetic and shared environmental influences. The nonshared environment estimate is always expected to exceed zero because it includes nonetiological influences (e.g., measurement error) that contribute to decreased phenotypic similarity among family members.

To estimate the proportion of variance in each oculomotor measure associated with genetic and environmental factors, we used biometrical models, which can be fit to the variance and covariance data from the MZ and DZ twins pairs. Our method for these analyses is outlined fully in Katsanis, Iacono, McGue, and Carlson (1997) and will be briefly reviewed. As already indicated, the variance in a phenotype ( $V_P$ ) can be decomposed linearly into that proportion associated with additive genetic effects ( $V_A$ ), shared environmental effects ( $V_C$ ), and nonshared environmental effects ( $V_E$ ; Eaves, Eysenck, & Martin, 1989). This standard biometrical model is thus defined as:

$$V_P = V_A + V_C + V_E.$$

Under this standard model (referred to as the ACE model), the expected phenotypic covariances for MZ and DZ twins are given by:

$$\text{COV}_{\text{MZ}} = V_A + V_C$$

$$\text{COV}_{\text{DZ}} = \frac{1}{2} V_A + V_C.$$

Variance components (A, C, and E) were estimated by maximum likelihood by fitting the biometrical model given by the above equations to the observed MZ and DZ variances and covariances using the Mx software program (Neale, 1994).

## Results

### Global Oculomotor Functioning

Table 1 presents the medians and percentile ranks of all variables as a function of zygosity and age. Table 2 presents the intraclass correlations for the oculomotor measures. The correlations for the global oculomotor variables were significant for both the 11- and 17-year-old MZ twins, suggesting moderate to high similarity on RMS error and overall saccade frequency, amplitude, and velocity. The only variables that the DZ twins were found to be similar on at a statistically significant level were saccade amplitude and velocity for the 11-year-old group. To determine whether the MZ twins' intraclass correlations were significantly larger than those of the DZ twins, we computed standard  $z$  transformation tests in each age cohort. Eleven- and 17-year-old MZ twins had significantly greater saccade frequency similarity than did the DZ twins. In addition, 17-year-old MZ twins had significantly more similar saccade velocity than the DZ pairs. These results suggest significant genetic influence on the general oculomotor measures.

To examine the extent to which twin similarity in smooth pursuit eye tracking increases with age, we contrasted the correlations between each age cohort separately for the MZ and DZ twins. Only oculomotor measures that were found to be significantly similar in at least one of the two age cohorts were examined. These

<sup>1</sup>The computer program used for data quantification and processing, called CASPER, was written by Brett Clementz and modified by Tom Zielund.

**Table 1.** Medians and Percentile Ranges for All Measures as a Function of Age and Zygosity

Measures	11-year-old male twins				17-year-old male twins			
	MZ twins (n = 70)		DZ twins (n = 56)		MZ twins (n = 58)		DZ twins (n = 40)	
	Median	25th–75th percentile	Median	25th–75th percentile	Median	25th–75th percentile	Median	25th–75th percentile
Global								
RMS error	107.50	77.50–149.50	126.00	81.75–226.25	69.00	58.75–84.00	84.50	66.00–113.50
Saccade rate	1.55	1.29–1.81	1.55	1.27–1.79	1.51	1.22–1.69	1.45	1.18–1.74
Saccade amplitude (deg)	2.80	2.05–3.83	3.25	2.23–4.05	1.73	1.11–3.23	2.39	1.27–3.03
Saccade velocity (deg/s)	0.10	0.08–0.12	0.10	0.08–0.12	0.07	0.06–0.11	0.08	0.07–0.10
Smooth pursuit								
Time domain gain	0.91	0.82–0.95	0.84	0.72–0.93	0.96	0.93–0.99	0.94	0.87–0.99
Frequency domain gain	0.89	0.82–0.94	0.84	0.70–0.92	0.95	0.91–0.98	0.94	0.87–0.97
Smooth pursuit (s)	27.63	26.89–28.14	27.78	27.04–28.20	28.29	27.60–28.77	27.81	27.34–28.45
Corrective saccade								
Catch-up frequency	1.15	0.81–1.30	1.18	0.85–1.38	1.13	0.85–1.42	1.15	0.83–1.45
Back-up frequency	0.09	0.05–0.19	0.18	0.05–0.16	0.14	0.07–0.24	0.11	0.05–0.16
Initiation frequency	0.12	0.05–0.16	0.09	0.02–0.16	0.05	0.02–0.09	0.06	0.02–0.09
Intrusive saccade								
Anticipatory frequency	0.05	0.00–0.12	0.09	0.02–0.2	0.00	0.00–0.05	0.02	0.00–0.07
Square-wave-jerk frequency	0.07	0.02–0.12	0.05	0.02–0.07	0.05	0.00–0.10	0.05	0.00–0.07

Note: MZ = monozygotic; DZ = dizygotic; RMS = root-mean-square.

analyses indicated that 17-year-old MZ twins were significantly more similar for amount of smooth pursuit ( $p < .05$ ) than 11-year-old MZ twins. For the DZ twins a significant decrease in similarity was noted for overall saccade amplitude ( $p < .05$ ) from early to late adolescence.

Overall, the intraclass correlations for both age groups suggest a genetic influence on the global measures of oculomotor func-

tioning. Univariate biometrical models were fit to the data on the global oculomotor measures. Figure 1 shows the standardized estimate of the proportion of total phenotypic variance owing to additive genetic ( $h^2$ ;  $V_A / V_P$ ), shared environmental ( $c^2$ ;  $V_C / V_P$ ), and nonshared environmental factors ( $e^2$ ;  $V_E / V_P$ ) when the ACE model ( $V_P = V_A + V_C + V_E$ ) was fit to the twin data from both age groups for each oculomotor measure. As indicated earlier, given

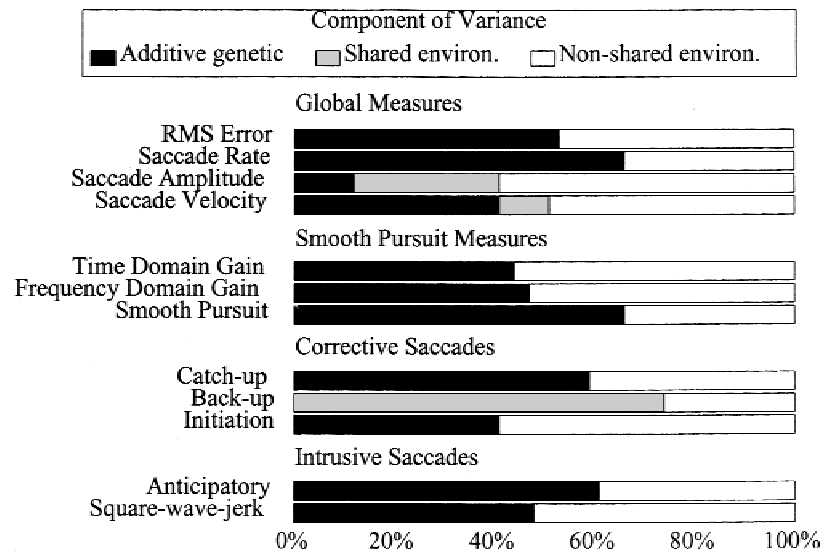
**Table 2.** Intraclass Correlations of the Oculomotor Measures for 11- and 17-year-old Monozygotic and Dizygotic Twin Pairs

Measures	11 year olds			17 year olds		
	$R_{MZ}$	$R_{DZ}$	$z^\dagger$	$R_{MZ}$	$R_{DZ}$	$z^\dagger$
No. of pairs	35	28		29	20	
Global measures						
RMS error	.48**	.14	1.43	.61***	.26	1.42
Saccade rate	.69***	-.11	3.18***	.68***	.19	2.04*
Saccade amplitude (deg)	.46**	.46**	0.00	.40*	.06	1.17
Saccade velocity (deg/s)	.54***	.38*	0.77	.53**	.08	1.64*
Smooth pursuit						
Time domain gain	.44**	.05	1.58	.47**	.28	0.71
Frequency domain gain	.49**	.01	1.99*	.48**	.28	0.72
Smooth pursuit (s)	.59***	.12	2.09*	.81***	.07	3.39***
Corrective saccade						
Catch-up frequency	.63***	-.15	2.78**	.64***	.17	1.88*
Back-up frequency	.22	.38*	-0.66	.28	.13	0.43
Initiation frequency	.40**	.07	1.32	.44**	.15	1.03
Intrusive saccade						
Anticipatory frequency	.52***	.23	1.28	.66***	.18	1.96*
Square-wave-jerk frequency	.53***	.28	1.13	.54**	-.32	1.94*

Note:  $R_{MZ}$  = intraclass correlation for monozygotic twins;  $R_{DZ}$  = intraclass correlation for dizygotic twins.

\* $p < .05$ , \*\* $p < .01$ , \*\*\* $p < .001$  (one-tailed).

†The  $z$  statistic is used to test the significance of the difference between  $R_{MZ}$  and  $R_{DZ}$  and is distributed as a standard normal under the null hypothesis of no difference.



**Figure 1.** Percentage of variance in eye tracking measures accounted for by genetic and environmental factors. (RMS = root-mean-square.)

the similarity in the intraclass correlations across age groups, the univariate biometrical models computed separately for each age group yielded similar heritability estimates. Thus, we were able to fit the models to the data from the 11- and 17-year-old cohorts combined (64 MZ pairs and 48 DZ pairs). The increase in power gained from combining the data across ages allows for a more robust estimate of genetic and environmental influences than would be possible from fitting models to data from each age group separately.

The global saccade amplitude and velocity measures were derived by simply averaging across the specific saccade measures. As indicated in the upper portion of Figure 1, variance in each of the global oculomotor measures was associated with additive genetic factors; the average  $h^2$  for the global oculomotor measures was .43. Individual differences in each global measure were associated largely with nonshared environmental factors, whereas shared environmental factors were associated only with variance in global measures of saccade amplitude and velocity.

### Smooth Pursuit Tracking

Examination of the intraclass correlations for the smooth pursuit tracking measures indicated a similar pattern of results as those involving the global oculomotor variables. The correlations were significant for both the 11- and 17-year-old MZ twins, suggesting moderate to high similarity on time- and frequency-domain gain and amount of smooth pursuit. The DZ twins were not found to be significantly similar for any of the variables.

Eleven- and 17-year-old MZ twins had significantly greater smooth pursuit similarity than did the DZ twins. In addition, 11-year-old MZ twins were found to be significantly more similar than the DZ pairs in frequency domain gain. These results also suggest significant genetic influence on the smooth pursuit eye tracking measures. Figure 1 illustrates that individual differences in smooth pursuit eye movements are associated with roughly equal shares of additive genetic and nonshared environmental influence. Furthermore, examination of the data using biometrical analysis indicated that, consistent with the correlational analyses,

the biometrical models computed separately for each age group yielded similar heritability estimates, revealing no heritability differences as a function of age.

### Corrective and Intrusive Saccades

The intraclass correlations involving the anticipatory, catch-up, and initiation saccades and square wave jerks were found to be significant for the MZ twins of both age cohorts. With the exception of the back-up saccades in the 11-year-old cohort, none of the correlations for the DZ twins attained significance. The frequency of back-up saccades was not found to be significantly similar in either the MZ or DZ twins. Anticipatory saccade and square-wave-jerk frequency in the 17-year-old group and catch-up frequency in the 11 and 17 year olds were found to be significantly more similar in the MZ than in the DZ twins. No other comparisons between the MZ and DZ twin pairs attained significance. The biometrical modeling results confirmed what would be expected based on the twin correlations: the frequency of most saccadic movements are influenced by genetic factors. With the exception of the frequency of back-up saccades, individual differences in most saccadic frequencies were associated with roughly similar magnitudes of genetic and nonshared environmental influences (see Figure 1). As with the rest of the eye tracking measures, examination of the heritability of corrective and intrusive saccades as a function of age did not yield significant results. This finding suggests that the heritability was similar for these measures at each age.

### Discussion

The primary objective of the current study was to examine genetic influences on smooth pursuit eye tracking and the saccadic defects that occur during smooth pursuit. We found that MZ twins in both the 11- and 17-year-old cohorts showed significant similarity for all the measures of eye tracking performance. Examination of the intraclass correlations pertaining to the global and smooth pursuit eye tracking measures of the DZ twins revealed a lack of significance. The only exceptions were for saccade amplitude and ve-

locity, which attained significance in the 11- but not the 17-year-old DZ cohort. For the individual saccadic measures, again the MZ twins were found to be significantly more similar than the DZ twins on the overwhelming majority of the measures. This outcome suggests that genetic mechanisms influence performance on the corrective and intrusive saccade measures to an equivalent degree.

The correlations pertaining to the RMS error measure are in agreement with the correlations reported by Iacono and Lykken (1979) and Iacono (1982), who also examined normal twins. For the smooth pursuit measures, Bell, Abel, Li, Christian, and Yee (1994) reported frequency-domain gain correlations in the magnitude of .91 to .98 for MZ twins. However, our respective intraclass correlations for the two cohorts were substantially lower (.48 to .49) and similar in magnitude to those for the rest of the global eye tracking measures. The reasons for the differences across studies is unclear. Bell et al.'s analyses were based on a relatively small sample of twin pairs ( $n = 8$ ); it is possible that their sample was not representative of twins in the general population. Interestingly, among the smooth pursuit measures, the two with the highest heritabilities were amount of time engaged in smooth pursuit eye tracking ( $h^2 = .66$ ) and saccade frequency ( $h^2 = .66$ ). This outcome suggests that these measures are relatively stable trait indicators of eye tracking performance and our findings suggest they may be promising measures to include in the assessment of eye tracking. To date, these measures have received relatively little attention.

RMS error and amount of smooth pursuit are measures influenced by both the presence of saccades and the quality of smooth pursuit. However, the frequency- and time-domain gain measures are indicative of smooth pursuit movement per se because saccades were eliminated from the calculation of these variables. Yet, all these measures seem to be influenced by hereditary factors to a similar degree. The global saccade measures, which do not reflect smooth pursuit activity, were also found to be heritable. Collectively, these results suggest that these measures have similar heritability; no one measure stands out as obviously more influenced by genetic factors. This result is consistent with the findings of an earlier study in which we found that these measures are highly correlated, suggesting that they tap a similar underlying construct related to the overall quality of pursuit eye tracking (Katsanis et al., 1998). Because ours was not a study of schizophrenia, however, we cannot address directly whether any of these measures would provide a "best index" of genetic risk for schizophrenia.

In a prior study of the developmental differences in smooth pursuit eye tracking proficiency, we found that eye tracking was significantly worse during preadolescence than late adolescence and adulthood. Furthermore, during late adolescence the smooth pursuit system appeared to reach adult levels of functioning (Katsanis et al., 1998). In light of the developmental changes that take

place in the brain during preadolescence and the likelihood that there are individual differences in CNS maturation, we compared heritability across the two cohorts to determine the extent that these differences were genetically influenced. The intraclass correlations of the 11 and 17 year olds revealed few significant differences overall. Only for amount of smooth pursuit was the intraclass correlation significant across age, and then only for the MZ group. In addition, biometrical modeling computed separately for each age cohort yielded similar heritability estimates across groups. Although the sample sizes for each age group were too small to address this issue conclusively using biometrical analyses, the results indicated that heritability was similar for these measures at each age. This finding, coupled with our previous observation that preadolescent's eye tracking is worse than that of the late adolescents (Katsanis et al., 1998), suggests that genetic influences in the rate that the smooth pursuit eye tracking system matures are stable from preadolescence to late adolescence. Thus, once norms are developed to account for the generally poorer performance of younger individuals, it may be possible to use their eye tracking performance to index genetic risk.

Saccades that occur during smooth pursuit have been classified according to their function into compensatory or intrusive saccades. Compensatory saccades are believed to serve a corrective function and attempt to bring the eyes on the target (catch-up, back-up, initiation saccades). On the other hand, intrusive saccades do not serve any apparent function and are believed to be inhibited during smooth pursuit (anticipatory saccades, square-wave jerks). Levy et al. (1993) argued that intrusive saccades such as anticipatory saccades do not reflect the functional integrity of the pursuit system and can be eliminated by simple instruction. The present study suggests that both intrusive and compensatory saccades are not random eye movement activity and that their occurrence may represent relative stable traits that are under partial genetic control. Further research is needed to determine the extent to which these measures can provide meaningful information regarding the integrity of the smooth pursuit system.

A limitation of the present investigation is that a relatively small number of subjects was utilized. Future studies with more subjects, for example, more than 200 pairs of each twin type, would enhance power in the analyses, provide more accurate estimates of heritability, and allow for more sensitive tests of the possibility that some types of tracking irregularity are differentially heritable.

In conclusion, we examined the heritability of various types of eye movement activity using a comprehensive assessment of the smooth pursuit system and the saccades that occur during smooth pursuit. We found that genetic influences exert their effects on global measures of eye tracking dysfunction, on measures of smooth pursuit eye tracking, and on the specific saccadic events that occur during smooth pursuit.

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