Investigating the Genetic and Environmental Structure of Cloninger's Personality Dimensions in Adolescence

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In this study we examined the genetic and environmental structure of four dimensions from Cloninger's personality system: novelty-seeking (NS), harm-avoidance (HA), reward-dependence (RD), and persistence (PS). Although adult twin studies suggest that these personality dimensions are moderately heritable, this is the first twin study of Cloninger's personality dimensions in adolescence — a period marked by significant physiological and social changes. Study participants included 1851 adolescent twins between the ages of 11 and 18 years; 878 complete twin pairs and 95 singleton-responding twins. Subjects were participants in two community-based samples of twins residing in the state of Colorado. Results indicated that cross-sectional mean levels for NS, HA and RD tended to show modest increases across the adolescent years, while PS showed modest mean decreases. Consistent sex differences in means were found only for RD. Univariate biometrical twin models were used to decompose trait variance into genetic and environmental sources. Results indicated that for NS, HA and RD additive genetic influences and unique environmental effects were sufficient to explain the data. PS, however, could be explained by unique and common environmental effects only, with different patterns of common environmental effects for males and females. We found moderate heritability estimates for NS, HA and RD ranging from .28 to .36 — with no evidence for sex-limitation in those influences.

Several personality theories propose that individual differences in personality may be reduced to three basic factors or dimensions. These are generally known as the 'Big Three' theories of personality (Cloninger, 1986; Eysenck & Eysenck, 1975; Gray, 1990; Tellegen, 1985). All of these Big Three models share similar conceptualizations. They are all neurobiological-based models that assume orthogonal independent brain systems underlying individual differences in personality. Despite nonagreement in the causal pathways and theoretical conceptualizations of their behavioral constructs, all three models approach the study of personality using a similar neurobiological scheme.

Cloninger's (1986) personality model proposes three genetically homogenous and independent temperament dimensions: novelty-seeking, harm-avoidance, and reward-dependence. Each of Cloninger's personality constructs is hypothesized to involve a distinct brain mechanism. Novelty-seeking is hypothesized to be involved in activation (in response to novel stimuli), harm-avoidance with inhibition (in response to punishment or nonreward), and reward-dependence with maintenance (of behaviors that were previously rewarded). In addition, each of these behaviors is hypothesized to be primarily involved with a different neurotransmitter system (novelty-seeking with dopamine, harm-avoidance with serotonin, and reward-dependence with norepinephrine). Cloninger's Tridimensional Personality Questionnaire (TPQ; Cloninger, 1987c) was later shown to measure four temperament dimensions rather than three, as persistence was found to be a genetically independent fourth dimension, rather than a subscale of reward-dependence (Cloninger et al., 1993; Stallings et al., 1996).

Cloninger's model is appealing in the context of behavioral genetics in that it asserts to measure the underlying genetic influences of the phenotypic structure of personality. In addition, Cloninger's personality traits have also shown to serve as moderators for other phenotypes such as psychopathology and substance use. In a study by Mulder et al. (1999) DSM-III-R cluster A personality disorders were correlated with low reward-dependence and high harm-avoidance, whereas DSM-III-R cluster B personality disorders were correlated with high novelty-seeking, and DSM-III-R cluster C personality disorders were correlated with high harm-avoidance and low novelty-seeking. These results suggest that

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personality disorders and psychopathology may be conceptualized as the extreme ends of normal personality dimensions, and in order to better understand the complex etiology behind personality disorders and psychopathology the etiology of normal personality should be examined as well.

Individual differences in personality have also been shown to be predictors of substance abuse. For example, there has been some suggestion that certain personality profiles in adolescence, based on Cloninger's constructs, may predict problem substance-use later in life. Cloninger suggests two distinct pathways to problem alcohol-use (Cloninger, 1987a; Cloninger et al., 1988). Type I is characterized by high reward-dependence, high harm-avoidance, and low novelty-seeking which leads to such problem behavior as loss-of-control drinking, difficulty in terminating binges, guilt feelings, and later onset. In contrast, type II is characterized by high novelty-seeking, low harm-avoidance, and low reward-dependence, which leads to such behaviors as antisocial personality, persistent seeking of substances for their euphoric effects, and early onset of inability to abstain. Evidence supporting Cloninger's theory has been found in a sample of 431 Swedish males, which indicated that the three personality dimensions, measured at age 11, predicted type II alcoholism at age 27 (Cloninger et al., 1988). Although Cloninger's theory originally focused on alcoholism, it has been shown to predict other types of problem substance use in adolescents and adults, such as cigarette smoking and marijuana use (Masse & Tremblay, 1997; Pomerleau et al., 1992; Wills et al., 1994). However, a recent review of the literature by Howard et al. (1997) found mixed results regarding the strength of Cloninger's theory to predict substance-related behaviors. They found that novelty-seeking does indeed predict early-onset alcohol abuse and criminality, as well as discriminate between alcoholics exhibiting antisocial behavior and their nonantisocial counterparts, smokers versus nonsmokers, and substance abusers versus nonabusers. However, findings for harm-avoidance and reward-dependence were much less consistent.

In light of these studies, it is quite surprising that there are no twin studies investigating the genetic and environmental structure of Cloninger's personality constructs during adolescence. Extensive data from twin and adoption studies confirm the importance of genetic factors in explaining individual differences in personality (Eaves et al., 1989; Eaves et al., 1998; Eaves et al., 1999; Finkel & McGue, 1997; Goldsmith, 1983; Jang et al., 1996; Loehlin, 1992; Loehlin & Martin, 2001; McCartney et al., 1990; Viken et al., 1994). Although there has been some variability in the interpretation of the findings due to differential sample selection and the range of personality traits addressed, the evidence is largely consistent. In general, there are genetic influences on essentially all personality traits; the genetic effect is substantial, accounting for between 40% and 60% of the variance; the influence of shared environmental effects (including shared family influences) is negligible. The important environmental influences appear to be unique environmental experiences specific to individuals, or nonshared environmental factors that contribute to within-family differences in personality. However, most of the available data has come from studies of adult populations and investigations of early childhood and infant temperament. There is very limited data from adolescent populations.

The purpose of this study is to better understand the genetic and environmental structure of Cloninger's personality constructs during adolescence — a period marked by significant biological and environmental changes. For the present study we used a sample of 1851 same-sex and opposite-sex adolescent twins from a community-based sample of twins residing in Colorado. This is the first twin study of Cloninger's personality model in adolescence. Twin studies are able to tease apart biological influences from environmental ones by investigating the differential patterns of correlations and covariances between monozygotic (MZ) and dizygotic (DZ) twins. The use of same-sex and opposite-sex twins further allows us to examine whether there are different genetic and environmental influences for males and females.

The aims of this study are threefold: 1) to investigate age differences on Cloninger's personality dimensions during adolescence, as indexed by age-cohort mean levels and variability; 2) to estimate heritability for Cloninger's personality constructs in adolescence; and 3) to examine whether there are differential heritability patterns between males and females in Cloninger's dimensions in adolescence.

Materials and Methods

Participants

The sample included 1851 adolescents 11 to 18 years of age (878 complete twin pairs and 95 singleton-responding twins). Complete pairs included 714 same-sex twin pairs (199 MZ male twin pairs; 220 MZ female twin pairs; 158 DZ male twin pairs; 137 DZ female twin pairs), and 164 opposite-sex twin pairs. The adolescent twin participants were drawn from two sources: the Colorado Longitudinal Twin Sample (LTS) and the Colorado Twin Registry (CTR; P.I.: J.K. Hewitt), both community-based samples of adolescent twins residing in Colorado. Twins were assessed in their homes separately by different interviewers. Informed parental consent and participant assent were obtained and twins were reimbursed $30 for participation. All consent forms and assessment protocols were approved by the Institutional Review Board (IRB) of the University of Colorado.

The average age of the sample was 14.5 years (SD = 2.2). Consistent with the demographics of the state of Colorado, the twins in our sample were largely Caucasian. The ethnicity distribution of the sample is

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as follows: 85.5% Non-Hispanic Caucasian, 7% Hispanic, 2% African American, 3% Asian, 2% Native American, and 0.5% other.

Zygosity Determination

Zygosity for all same-sex twin pairs was determined by two methods (opposite-sex twins were immediately assigned as dizygotic). During interview sessions, interviewers rated each twin pair on a nine-item assessment of physical characteristics (Nichols & Bilbro, 1966); ratings were used to make a judgment of zygosity. A second zygosity rating was based on genotyping each individual for 11 highly informative short tandem repeat polymorphisms (STRPs) using standard polymerase chain-reaction (PCR) methods and ABI 377 genotyping technology. Marker discordance for members of a twin pair indicates their dizygotic origin, while marker concordance across all genotyped markers indicates their monozygotic origin. The average heterozygosity of the markers exceeds 0.75, and gives a posterior probability of MZ misdiagnosis of less than 0.0001. Only twins whose tester ratings and genotypic data agree on zygosity determination were used in the current analyses.

Measures

Tridimensional Personality Questionnaire. The Tridimensional Personality Questionnaire (TPQ) was administered to adolescents 16 to 18 years of age. The version used in the present study was a shortened 54-item self-administered true–false instrument (Heath et al., 1994). The TPQ was originally designed to assess three higher order personality dimensions: novely-seeking (NS), harm-avoidance (HA), and reward-dependence (RD) (Cloninger, 1986, 1987b). However, consistent with Cloninger's revised model (Cloninger & Svrakic, 1994; Cloninger et al., 1993), we scored persistence (PS) as a separate higher-order dimension. We should also point out that although Cloninger's TPQ has been superseded by the Temperament and Character Inventory (TCI; Cloninger et al., 1994), the four temperament dimensions of the TPQ and TCI are the same. As is typical with self-report personality questionnaires, respondents occasionally skip items (or both 'true' and 'false' answers are circled). For this reason, the mean of the items endorsed was computed for each dimension, rather than the sum of endorsements. If more than two items were skipped (or could not be scored), the dimension score was coded as missing. Internal consistencies (Cronbach's alpha) for the TPQ dimensions were .82, .66, .66, and .53 for HA, NS, RD, and PS, respectively.

Junior Temperament and Character Inventory (J-TCI). The J-TCI was administered to adolescents 11 to 15 years of age. The J-TCI was developed to assess the same four temperament dimensions as the TPQ (NS, HA, RD and PS), as well as three additional character dimensions (self-directedness, cooperativeness, and self-transcendence) in children and adolescents (Cloninger et al., 1994). The J-TCI is appropriate in content and reading level for children as young as 7 years of age. The J-TCI is a revision of the adult TCI in which each item is rewritten in short concrete statements and more abstract items are omitted. In this study only the four temperament dimensions of the J-TCI were available, administered as a 55-item self-report true–false questionnaire. Internal consistencies (Cronbach's alpha) for the four J-TCI temperament dimensions were .78, .68, .58, and .58 for HA, NS, RD, and PS, respectively.

Data Transformation and Analysis. Because the TPQ and J-TCI were designed to measure the same temperament dimensions (but utilize somewhat different items), analyses were conducted on standardized scores (the standardization was conducted within each age cohort/instrument, and the standardized scores for each cohort were then appended together for analysis of the full sample). To further assure that the younger and older participants can be classified on the same distribution of liability for each dimension, correlations between the TPQ and J-TCI dimensions and Eysenck's personality questionnaire (EPQ; Eysenck & Eysenck, 1975) dimensions, which were administered to all participants in the sample, were examined. Since NS and HA have been shown to correlate with extraversion and neuroticism in adults (Heath et al., 1994; Stallings et al., 1996), the correlation patterns between these two personality systems were examined across the two measures. Except for the correlation between NS and extraversion, the correlation patterns were extremely similar across the two measures. These data suggest that it is unlikely that the TPQ and J-TCI dimensions are assessing substantially different constructs in the 11 to 15 years and 16 to 18 years age cohorts. For all descriptive analyses comparing age and sex differences in means and variances we used the total sample of 1851 individuals, including twin pairs and single-responding twins (singletons).

For biometrical model-fitting analyses we used only the subset of 878 complete twin pairs (1756 individuals). In addition, since each of the personality dimensions was significantly correlated with age (age correlations from the full sample were .12, .25, .17, and –.13; for HA, NS, RD, and PS, respectively), we age-corrected (i.e., obtained residual scores using standard regression procedures) and rank-normalized scores within the J-TCI and TPQ groups prior to performing model-fitting analyses. For each of the four personality dimensions univariate twin models were fit to 10 twin covariance matrices (MZ-male, MZ-female, DZ-male, DZ-female, and DZ-opposite-sex twins in the 11 to 15 years age cohort; and the same five zygosity groups in the 16 to 18 years age cohort). Age corrections were conducted within each measure/cohort. However, age effects across measures and/or cohorts may still exist and can be analyzed. Note that age cohort is also confounded with assess-

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ment: TPQ versus J-TCI. In addition, the sample is cross-sectional and therefore may also be confounded with birth cohort. Twin models decompose phenotypic variance into four etiological sources: additive genetic effects (A), nonadditive, or dominant genetic effects (D), shared or common environmental effects (C), and unique environmental effects (E). The full ACDE model cannot be fit to data from twins raised together because C and D are confounded, so ACE and ADE models were evaluated separately. Fitting models to the 10 data groups allowed us to test for sex and age cohort/instrument heterogeneity in the genetic and environmental sources of variance. Model-fitting analyses were performed using the structural equation modeling statistical program Mx (Neale, 1999). Standard chi-square difference tests were used to compare the fit of alternative nested models, and Akaike's Information Criterion (AIC; Akaike, 1974) was used to compare the fit of nonnested models (see Neale & Cardon, 1992 for details).

Three types of models were fit to each personality dimension: 1) Sex and age/instrument-limitation models allowed for differential magnitudes in parameter estimates for males and females, for age/instrument cohorts, and for both sex and age/instrument (corresponding homogeneity models constrain parameter estimates across sex and/or age/instrument to be equal). 2) Scalar effects models constrained parameter estimates across sex or age/instrument cohorts (or both) to be equal, but allowed the total variances to differ by sex and/or age/instrument cohorts. 3) General sex-limitation models allowed for testing whether different genes (or environmental factors) influence personality in males and females, by allowing the genetic correlation (or shared environmental correlation) for opposite-sex twins to be freely estimated. Note, since twins are the same age (and both were assessed with the same instrument) an analogous general age/instrument-limitation model is not possible. We also did not fit an ADE sex-limitation model since there was no evidence for differential additive genetic effects between males and females, and therefore it would generally not be plausible to find differential nonadditive genetic effects.

Results

Descriptive Analyses

We first examined raw means (Table 1) and variances (Figure 1) for each of the four personality dimensions by age. We found modest, but significant age-to-age differences in means for all four scales. These differences show a general tendency for older adolescents (i.e., ages 16 to 18 years) to score modestly higher on the NS, HA, and RD scales than younger adolescents (i.e., 11 to 15 years). For PS an opposite trend was found. These means are very similar to the TPQ means reported by Schmitz et al. (2004). Their sample participants were measured at ages 17, 19, and 20 years-of-age, so it overlaps to some extent with the age distribution in our adolescent sample. Although there were some significant age-to-age differences in variances for the four dimensions, there were no general trends for increasing or decreasing variance with age.

Table 1 also breaks down raw means by sex for the four personality dimensions at each age. RD shows consistent sex differences in means, with females scoring higher than males throughout adolescence. HA shows significant sex differences in means in later adolescence, with females scoring higher than males. Males scored significantly higher than females on NS for some ages, but there was no general trend for sex differences. PS generally did not show sex differences in means throughout adolescence (although there was a significant difference at age 11, it should be interpreted with caution due to the small sample size at that age).

Twin correlations for males and females in each cohort are shown in Table 2. NS correlations for MZ twins are moderate and twice the magnitude of the

Table 1

Raw Means Across Age and Gender

Age:		11	12	13	14	15	16	17	18
NS	Males	0.44	**0.41**[2]	0.45	0.46	**0.50**[1]	0.53	0.52	**0.52**[1]
	Females	0.37	**0.35**	0.41	0.43	**0.45**	0.54	0.53	**0.47**
HA	Males	0.26	0.26	0.26	0.23	**0.25**[1]	**0.28**[1]	**0.29**[2]	**0.29**[2]
	Females	0.32	0.29	0.29	0.26	**0.30**	**0.36**	**0.38**	**0.39**
RD	Males	0.54	**0.46**[2]	**0.49**[2]	**0.52**[2]	**0.51**[2]	0.60	**0.58**[2]	**0.56**[2]
	Females	0.56	**0.59**	**0.62**	**0.67**	**0.62**	0.65	**0.68**	**0.69**
PS	Males	**0.62**[1]	0.70	0.66	0.68	0.64	0.63	0.61	0.60
	Females	**0.77**	0.72	0.65	0.74	0.63	0.65	0.63	0.62
Sample size	Males	28	241	81	144	103	111	104	100
	Females	36	248	83	117	127	113	94	121

Note: Significant gender differences are shown in bold.

[1] $p < .05$

[2] $p < .01$

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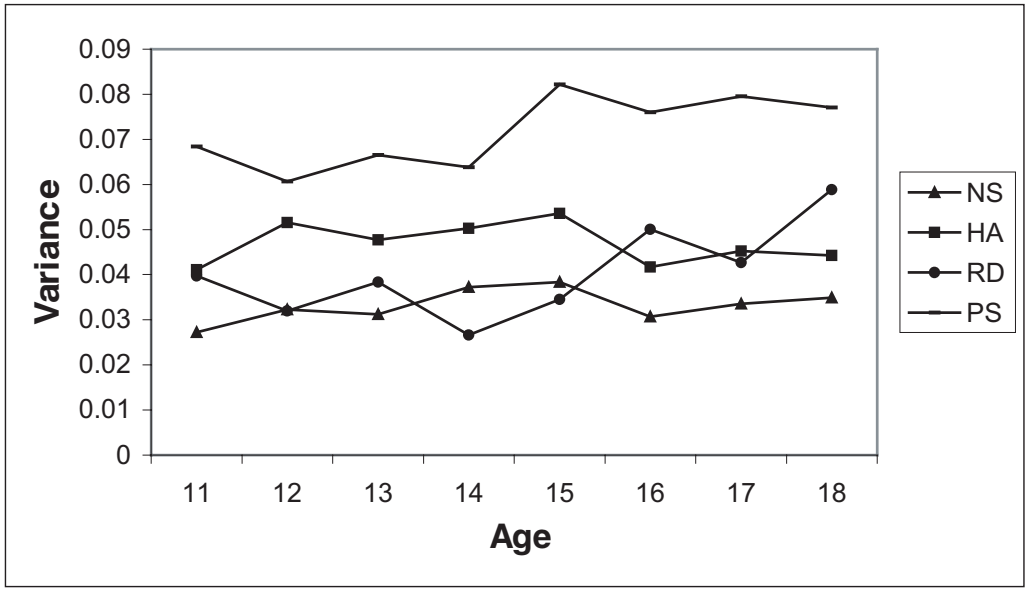

Figure 1
Variances by age.

correlations for both same-sex and opposite-sex DZ twin correlations for both measures, which suggests substantial heritability for NS in both measures. For HA and RD MZ twin correlations are generally moderate and higher than DZ same-sex and opposite-sex twin correlations, which suggests substantial heritability for the J-TCI as well as the TPQ. However, TPQ twin correlations for DZ females are relatively moderate which can also suggest some shared environmental influences for females in the TPQ measure. For J-TCI PS twin correlations across all five zygosity groups are moderate and similar. This correlational pattern suggests substantial shared environmental influences. For TPQ PS twin correlations range from low to moderate with no consistent pattern across zygosity groups (i.e., MZ twins do not necessarily have higher correlations than DZ twins) which again, suggests substantial shared environmental influences. The low correlation for opposite-sex twin pairs as opposed to same-sex DZ twin pairs for HA in both measures, for NS in the J-TCI measure, and for PS in the TPQ measure, suggests the potential for differential genetic and environmental influences for males and females for these traits.

Model-Fitting Analyses

For NS, HA and RD, homogeneity models constraining parameter estimates across sex and age cohort/instrument provided acceptable fits to the data. ADE homogeneity models provided very good fits as indicated by the Akaike information criterion (–31.83, –33.85, –24.34 for NS, HA, and RD respectively). However, standard χ^2 difference tests to evaluate the fit of alternative nested models indicated that dominance effects (D) could be dropped without a significant decrement in fit, so that the more parsimonious AE model was considered the best-fitting model for HA, NS, and RD ($\Delta\chi^2_{(1)} = 3.82$, $\Delta\chi^2_{(1)} = 1.45$, $\Delta\chi^2_{(1)} = 1.47$, for NS, HA, and RD respectively; $p > 0.05$ for all). Although we did not find significant differential heritability patterns between males and females by χ^2 difference test, a sex limitation model also provided a good fit to the data for HA ($\chi^2_{(25)} = 15.90$, $p = 0.918$, AIC = –34.09).

Due to the pattern of twin correlations for J-TCI PS, in which DZ and MZ twin correlations were quite similar (suggesting shared environmental effects and no genetic effects) CE models were also fit to this scale. For TPQ PS twin correlations suggest no heri-

Table 2
Twin Correlations by Gender and Age/Instrument

		HA	NS	RD	PS
	N		J-TCI		
MZ Males	132	0.43	0.50	0.21	0.33
MZ Females	140	0.37	0.35	0.36	0.34
DZ Males	106	0.20	0.22	0.10	0.33
DZ Females	94	0.21	0.18	–0.07	0.29
Opposite-sex	98	–0.01	0.03	0.09	0.35
	N		TPQ		
MZ Males	67	0.37	0.25	0.45	0.14
MZ Females	80	0.40	0.37	0.16	0.29
DZ Males	52	0.08	0.12	0.07	0.16
DZ Females	43	0.23	–0.27	0.27	0.50
Opposite sex	66	0.00	0.16	0.25	–0.03

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Table 3

Parameter Estimates for Best-Fitting Models

		a^2	c^2	r_c	e^2	χ^2	p	AIC
HA		0.36 (0.29–0.43)	—	—	0.64 (0.56–0.71)	21.59	0.80	–34.40
NS		0.36 (0.28–0.43)	—	—	0.64 (0.57–0.72)	25.99	0.57	–30.00
RD		0.28 (0.20–0.36)	—	—	0.72 (0.64–0.80)	31.13	0.31	–24.86
PS	males	—	0.27 (0.18–0.36)		0.73 (0.64–0.82)			
	females	—	0.35 (0.26–0.44)		0.65 (0.56–0.74)	31.93	0.16	–18.06
	r_c TPQ cohort	—	—	0.00 (0.00–0.71)				

Note: 95% confidence intervals are shown in parentheses.

tability as well, yet shared environmental influences may be different for males and females given the DZ opposite-sex twin correlation appears lower than the DZ same-sex twin correlation. And indeed a sex-limitation model allowing for differential CE parameter estimates for males and females, while further allowing the shared environmental correlation (r_c) between male and female DZ pairs to be free for the TPQ PS scale (i.e., shared environmental effects were allowed to affect males and females in a different way for PS as measured by the TPQ), was found to provide the best fit for the PS dimension.

Table 3 summarizes parameter estimates for the best-fitting model for each scale. We found moderate heritability estimates for NS, HA, and RD ranging from .28 to .36. For all three of these personality dimensions simple AE homogeneity models ('constrained') provided acceptable fits to the data. There was very little evidence for substantial sex- or age/instrument-limitation in the genetic and environmental influences on these dimensions across the adolescent range. PS, on the other hand, could be explained by unique and common environmental effects, where for the older/TPQ cohort, shared environmental effects affected males and females differently.

Discussion

In this study we investigated the underlying genetic and environmental structure of four temperament dimensions from Cloninger's personality system in adolescence. Our sample consisted of 1851 twins between the ages of 11 and 18 years. We examined age and sex differences in means and in the genetic and environmental structure of Cloninger's dimensions across this age range.

The results showed a tendency of older adolescents (i.e., ages 16 to 18 years) to score higher on the NS, RD, and HA scales than younger adolescents (i.e., ages 11 to 15 years). An opposite trend was found for PS. Although we cannot rule out the possibility that the mean differences we observed are an artifact of using two different instruments for assessing Cloninger's dimensions at ages 11 to 15 years (J-TCI) and 16 to 18 years (TPQ), the two measures were designed to capture the same four temperament dimensions, and the scores were standardized within measurement instrument to minimize measurement differences. Previous studies have provided some evidence for age-related changes in Cloninger's dimensions (Luby et al., 1999); the age trends we observed in the current study may be valid. Luby et al. (1999) investigated the psychometric features of the J-TCI and found a positive correlation between NS and age in a sample of 9- to 13-year-old children (i.e., novelty-seeking increased with age in these children as in our sample). They claim that this type of finding is consistent with child development theories that suggest that as a child grows there is a need to individuate from the family, which may be expressed by a greater tendency for exploration and risk-taking. Contrary to our results, however, Luby et al. (1999) found a negative correlation between HA and age in their sample. This discrepancy could be due to the fact that our sample is older and our age range is broader than Luby et al.'s. The older a child gets the more they may be aware and able to understand the implications of the dangers surrounding them, which in turn may lead to higher harm-avoidance reactions.

Gender differences in means were substantial and persistent throughout adolescence for RD, with females generally scoring higher than males. Males scored significantly higher than females on NS intermittently throughout adolescence, but there was no general trend. These findings are remarkably similar to Luby et al.'s findings. HA showed significant gender differences in means in later adolescence, with females scoring higher than males. There was no general trend for sex differences in means for PS.

In addition to investigating age differences on the Cloninger's personality dimensions during adoles-

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cence, as indexed by age-cohort mean levels and variability, we were also interested in exploring the genetic and environmental etiology of these scales. For NS, HA, and RD a simple AE homogeneity model, which constrained the male and female additive genetic and unique environmental estimates to be the same, provided an adequate fit to the data. Models constraining parameter estimates across measures/age cohorts provided a good fit as well, indicating that the genetic and environmental influences on these temperament dimensions do not differ by measurement instrument and/or age. Consistent with the personality literature, we found moderate heritability estimates for these dimensions ranging from .28 to .36. These heritability estimates are markedly higher than those found for the TPQ in adolescent adoptive and biological siblings in a study by Schmitz et al. (2004). Schmitz et al. suggest that their heritability estimates are lower than those reported from twin studies of personality due to the high resemblance between MZ twins that is likely due to nonadditive genetic factors. Although we were able to drop nonadditive genetic effects from our models for all four temperament dimensions without a significant decrement in model fit, it is noteworthy that models with nonadditive genetic effects also yielded acceptable fits to the data. Twin correlations for NS, HA and RD suggest some evidence for nonadditive genetic effects as well, but considering our sample sizes, it is likely that we had limited power to detect it. Several studies have found evidence for nonadditive genetic effects for Eysenck's personality system (Eaves et al., 1999; Eaves et al., 1998; Heath et al., 1994), the Multidimensional Personality Questionnaire (MPQ; Finkel & McGue, 1997), and Zuckerman's Sensation Seeking Scale (Koopmans et al., 1995). Heath et al. (1994) also found substantial nonadditive genetic effects in adult twins for NS and HA. Future investigations into the subscales with larger adolescent samples will perhaps reveal a more comprehensive picture of the dynamics behind the genetic structure of Cloninger's temperament dimensions in adolescence.

For PS an environmental (CE) sex-limitation model fit best, where shared environmental effects differed for males and females in the older/TPQ cohort. These findings should be interpreted with some caution however, due to the small number of items that compose the PS dimensions. As the PS scale was later derived as an independent dimension (it was formerly a subscale of RD), it is only composed of six items in the J-TCI measure and five items in the TPQ measure. This dimension may have questionable measurement properties, especially in light of the fact that most studies on personality show moderate heritability and no shared environmental effects. Thus, our findings to the contrary for PS may be the result of tenuous correlation patterns. Furthermore, PS has higher variances than the rest of the scales in our sample, which again leads us to suspect that it may not have enough items to reliably measure the persistence phenotype. However, the internal consistency for PS was the same as for RD (.58), so that we should not rule out the possibility that PS is simply more heritable in adulthood than in adolescence.

Except for PS, Cloninger's personality dimensions did not show substantial shared environmental effects in adolescence across gender or measure/age. Genetic studies on the TPQ in adult twins are consistent in showing no shared environmental effects and moderate heritability (Heath et al., 1994; Heiman et al., 2003; Stallings et al., 1996). However, two studies found gender differences in the genetic structure of the TPQ in adult twins. In Stallings et al.'s (1996) study of twins between ages 50 and 96 years, they report that sex-limitation models showed a significant improvement in fit than models constraining the underlying genetic and environmental structure of the TPQ to be the same for men and women. Similarly, in a study on Australian twins between ages 25 and 89 years, Heath et al. (1994) found much higher additive genetic variance for women and much higher nonadditive genetic variance for men in HA. For RD, a model allowing for a sex-by-genotype interaction, showing differential genes in the two sexes (the genetic correlation in opposite-sex twin pairs was estimated at .34) provided a better fit to the data. Although we did not find significantly different heritability patterns between males and females, for HA and NS, a genetic sex-limitation model also provided a good fit.

In summary, while we found phenotypic mean differences across age and sex, Cloninger's personality dimensions show quite consistent genetic and environmental influences during adolescence for NS, HA, and RD. For PS, shared environmental influences seem to be heterogeneous across sex and age/instrument. Gender differences in the genetic and environmental structure of Cloninger's personality system that have been reported in some adult samples appear to be minimal in adolescence. However, there remains a need for more studies of Cloninger's dimensions in this important age range.

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