Age-Related Changes in Heritability of Behavioral Phenotypes Over Adolescence and Young Adulthood: A Meta-Analysis

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The relative proportions of genetic and environmental variance in behavioral measures have been studied extensively. A growing body of literature has examined changes in heritability measures over time, but we are unaware of any prior efforts to assess developmental heritability changes for multiple behavioral phenotypes using multiple data sources. We have chosen to explore the proportional genetic influences on a variety of behaviors during the genetically and environmentally labile adolescent and young adult years. This meta-analysis examined 8 behavioral domains and incorporated only primary research articles reporting two or more heritability time points in order to minimize the age-to-age error variability. Linear regression analyses revealed significant cross-time heritability increases for externalizing behaviors, anxiety symptoms, depressive symptoms, IQ, and social attitudes and nonsignificant increases for alcohol consumption, and nicotine initiation, but no evidence of heritability changes for attention-deficit/hyperactivity disorder. A variety of mechanisms may underlie these findings including the rising importance of active genotype-environment correlation, an increase in gene expression, or proportional reductions in environmental variance. Additional longitudinal studies and the inclusion of measures of total variance in primary research reports will aid in distinguishing between these possibilities. Further studies exploring heritability changes beyond young adulthood would also benefit our understanding of factors influencing heritability of behavioral traits over the lifespan.

Since it is now widely acknowledged that both nature and nurture provide substantial contributions to most behaviors, attention has shifted to the characteristics of these influences.

Many articles have focused on quantifying the magnitude of genetic and environmental influences for a behavioral trait at a single time point, but a growing number of articles have begun to explore differences across the lifespan. Heritability measures, assessing the portion of total phenotypic variance attributed to additive genetic factors, are entirely reliant on the genetic and environmental variance in a population at the time of sampling. Therefore, studies undertaken at different times when varying environmental factors are at play, or under a diversity of genetic influences, will quite likely yield distinct heritability measures. This is readily apparent for assessing various population level differences, but it is also relevant to changes across the lifespan.

Given that our genetic endowment is unchanging throughout the lifespan, it can be difficult to conceive of how genetic influences may grow or diminish over time, but several factors may work in concert to achieve this. Gene expression changes underlie some of this dynamic process, as genes turn 'on' and 'off' over time and in response to environmental or developmental cues (e.g., puberty; Whitelaw & Whitelaw, 2006). Additionally, the environmental context of gene expression can in turn affect the influence of a gene product. But genetic influences only represent part of the total picture. A key issue in developmental behavior genetics is the extent to which environmental exposures have a persistent effect on behavior. Transient effects of the environment will have little impact on heritability estimates over time, but enduring environmental impacts would be expected to drive heritability estimates lower as experiential effects accumulate (Eaves et al., 1986).

As genetic and environmental influences act to create a phenotype, they can also interact in ways that are not strictly additive. For instance, given the same environmental conditions, people with differing genes may respond in divergent ways by a mechanism known as genotype-environment interaction. Additionally, genetic mediation of exposure to the environment is termed genotype-environment correlation (*r*GE) and

Received 8 March, 2007; accepted 3 April, 2007.

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can take three forms: passive, active, and evocative/reactive. Passive *r*GE occurs because parents provide both genes and early environments to their children, while active *r*GE is the tendency for people to actively seek out environments that reinforce their genotypic dispositions. Evocative/reactive *r*GE results from the elicitation of environmental responses by genetically influenced behaviors (Plomin et al., 1977; Scarr & McCartney, 1983).

Adolescence and young adulthood are developmental periods of substantial flux in gene expression and emerging environmental opportunities. Whereas early childhood environments are largely determined by parents (conferring passive *r*GE), adolescents and young adults have an increasing capacity to select into an ever-expanding range of environments (active *r*GE). If the growing active *r*GE influences exceed the declining rate of passive *r*GE influence in adolescence and young adulthood, as we hypothesize, heritability estimates should increase over this timeframe. However, if the genetic influences on behavior increase at the same rate as total environmental variance, the heritability measures will remain the same over time. Heritability measures may even decrease if the environmental variance increases to a greater extent than genetic influences.

Twins provide an excellent opportunity to explore these theories. Active *r*GE theory predicts that the genetically identical monozygotic (MZ) twins will be more likely to select similar environments than the less genetically similar dizygotic (DZ) twins. This may be reflected by consistently high (or even increasing) MZ twin correlations and progressively lower DZ twin correlations. While active *r*GE is one possible explanation for heritability increases over time, this could also occur through cumulative effects of one set of consistently expressed genes overshadowing occasion-specific environmental effects. A third possibility is that a new set of genes is expressed during development that boosts the genetic proportion of the variance whether or not environmental effects have a cumulative impact (Eaves et al., 1986). Finally, a reduction in environmental variance would also result in increased heritability estimates. This could be accomplished through a declining role of shared environmental effects or passive *r*GE as well as through reduced error variance due to the greater ability of older subjects to reflect on and report reliably on their behaviors.

In the study of heritability, most research has focused on extracting a single measurement; however, some studies have embraced the dynamic nature of heritability and assessed changes over time. Some meta-analyses have addressed this issue within one or two behavioral domains (McGue et al., 1993; Rhee & Waldman, 2002), but concerted efforts to assess heritability changes across multiple phenotypes simultaneously have been limited. Also, prior meta-analyses have predominantly incorporated research articles reporting heritability at only one timepoint, while the current meta-analysis is restricted to data from articles reporting heritability measures at multiple timepoints, thereby providing better control for differences between populations, analysis methods, and, in the case of longitudinal studies, interpersonal and intergenerational variation.

The purpose of this meta-analysis is to determine whether measures of heritability for a variety of phenotypic domains manifest increases over time. We have chosen to restrict our study to adolescence and young adulthood due to the genetic dynamicism and crescendo of environmental variability during this time of life, and only phenotypes which had been studied repeatedly were included in our analyses. This resulted in one grouping of externalizing behaviors encompassing antisocial behavior, alcohol consumption, and nicotine initiation, another cluster of internalizing behaviors including symptoms of anxiety and depression, and a remaining group of other phenotypes such as IQ, attention-deficit/hyperactivity disorder (ADHD), and social attitudes.

Methods

Search Strategy and Inclusion Criteria

Keyword searches using PubMed and PsychINFO databases were conducted initially to identify twin and adoption studies for each domain of interest which reported heritability estimates or twin correlations at two or more time points to minimize variability in cross-time measures. For inclusion, studies must have reported at least one heritability measure for subjects between the ages of 13 and 25 since the focus of this meta-analysis was to examine heritability changes during the development period of adolescence and young adulthood. Searches were limited to articles published in English, and references from the research articles and review articles identified from these searches were then examined to reveal any additional studies that may have been missed in the database searches. Frequently, the same set of subjects was used to study similar phenotypes multiple times. When this occurred, the most recent study reporting the most time points was selected for inclusion. Both longitudinal and cross-sectional studies were considered. While longitudinal studies examining the same subjects over time are preferred, cross-sectional studies drawing subjects from the same population and using the same analysis methods were also included. Longitudinal studies in which the rater changed over time (i.e., parent-report in wave 1 and self-report in wave 2) were not used since this introduces an additional source of cross-time differences. For the eight behavioral domains of interest, each was required to have at least four samples represented. When males and females are reported separately, they constitute distinct samples. Similarly, studies with multiple cohorts reporting a single heritability measure at each time-point comprise a singular sample. When studies reported twin

https://doi.org/10.1375/twin.10.3.423 Published online by Cambridge University Press

Table 1

Research Articles Included in Analyses

First author	Year	Instrument	Methods	Informant	Sex	Number of subjects	Age	Type	Cohort Origin
Externalizing behaviour									
Burt	2005	Diagnostic Interview for Children and Adolescents-R, adapted	SEM	Parent, self, rater	M,F	1397–1506	10–15	Longitudinal	US, MN
Jacobson	2002	ASB symptoms — Questionnaire adapted from DSM-III-R Conduct Disorder, ASPD criteria	SEM	Self	M F	3960–3974 2775–2811	20–58 23–63	Retrospective	Virginia Twin Registry
Lyons	1995	ASPD symptoms — from DSM-III-R	SEM	Self	M	6452	36–55	Retrospective	Veterans, US
O'Connor	1998	Behavior Problem Index — adapted from CBCL	SEM	Parent, self, rater	M,F	790	10–21	Longitudinal	NEAD, US
Tuvblad	2006	31–34 item questionnaire*	SEM	Self	M	849–1184	13–20	Longitudinal	TCHAD, Sweden
					F	849–1184	13–20		
Grove	1990	Diagnostic Interview Schedule — Antisocial Behavior	Max likelihood	Rater	M,F	65	16–68	Adoption, Retrospective	Minnesota Study of Twins Reared Apart
McGue	1993	MPQ — aggression factor scale	Correlations	Self	M,F	254	17–37	Longitudinal	US
McGue	2006	Youth problem behavior, adult psychopathology	SEM	Self	M	472–481	17.5, 20.7	Retrospective, Longitudinal	MTFS, US
					F	608–628	17.5, 20.7		
Alcohol consumption									
Rose	2001	Frequency of drinking	SEM	Self	M,F	2206	16–18.5	Longitudinal	Finland, Central Population Registry
Kaprio	1992	Quantity consumed	SEM	Self	M	1796	18–29	Longitudinal	Finnish Twin Cohort
					F	1974	18–29		
Koopmans	1997	Alcohol use	SEM	Self	M F	573–1154 727–1378	12–25 12–25	Cross-sectional	Dutch Twin-Family Study on Health-Related Behavior
Malone	2004	Dependence — Composite International Diagnostic Interview	SEM	Self	M	502–578	17–24	Longitudinal	MTFS, US
Hopfer	2005	Average drinks consumed per episode in past year	SEM	Self	M,F	3634–4432	16–22.5	Longitudinal	Add Health, US
Smoking initiation									
Madden	1999	Initiation	SEM	Self	F	14,110	18–46	Cross-sectional	Australia, Sweden, Finland †
					M	1041	18–46	Cross-sectional	Australia
					M	13,395	18–46	Cross-sectional	Sweden, Finland
White	2003	Smoking involvement	SEM	Self	M,F	1843–2892	13–25	Longitudinal	Australia

Table 1 (CONTINUED)

Research Articles Included in Analyses

First author	Year	Instrument	Methods	Informant	Sex	Number of subjects	Age	Type	Cohort Origin
Smoking initiation (CONTINUED)									
Koopmans	1997	Initiation	SEM	Self	M F	573–1154 727–1378	13–21 13–21	Longitudinal	Dutch Twin-Family Study on Health-Related Behavior
Miles	—	Initiation	SEM	Self	M	938	12.5–15	Longitudinal	VTSABD, US
					F	1139	12.5–15		
Slomkowski	2005	High contact — past 30 d. smoking	SEM	Self	M,F	1421	15.5–16.5	Longitudinal	Add Health, US
		Low contact — past 30 d. smoking	SEM	Self	M,F	1421	15.5–16.5		
Depression symptoms									
Rice	2002	Mood and feelings questionnaire	SEM	Parent	M,F	128–330	8–17	Cross-sectional	South Wales, UK
O'Connor	1998	CDI	SEM	Parent, Self, rater	M,F	395	10–21	Longitudinal	NEAD, US
Silberg	2001	CAPA — Depression	SEM	Self	F	667–844	8–17	Cross-sectional	VTSABD, US
Eley	1999	CDI	SEM	Self	M	230–255	8–16	Cross-sectional	Register for Child Twins, UK
					F	249–288	8–16		
Pogue-Geile	1985	MMPI-D_{30}	Correlations	Self	M	266	20–25	Longitudinal	Indiana U. Twin Panel
Dworkin	1976	MMPI-D	Correlations	Self	M,F	84	15.9–27.9	Longitudinal	Boston, US
Anxiety symptoms									
Silberg	2001	Overanxious disorder symptoms	SEM	Self	F	667–844	8–17	Cross-sectional	VTSABD, US
Eley	1999	State-Trait Anxiety Inventory for Children (trait scale)	SEM	Self	M	230–255	8–16	Cross-sectional	Register for Child Twins, UK
				Self	F	249–288	8–16		
Dworkin	1976	MMPI-A	Correlations	Self	M,F	84	15.9–27.9	Longitudinal	Boston, US
Gjone	1996	CBCL — Internalizing behaviors	Regression	Parent	M,F	382–596	5–15	Cross-sectional	Norwegian Medical Birth Register
Social attitudes									
Koenig	2005	9-item Religiousness scale	SEM	Self	M	546	33	Retrospective	Minnesota Twin Registry
Abrahamson	2002	Conservatism scale‡	SEM	Rater	M,F	654	12–15	Adoption, Longitudinal	CAP, US
Eaves	1997	Conservatism scale‡	Correlations	Self	M,F	100–723	9.5–75+	Cross-sectional	MCV Cardiovascular Twin Study, Virginia 30,000
Pogue-Geile	1985	Wiggins's religiousity	Correlations	Self	M	266	20–25	Longitudinal	Indiana U. Twin Panel

Table 1 (CONTINUED)

Research Articles Included in Analyses

First author	Year	Instrument	Methods	Informant	Sex	Number of subjects	Age	Type	Cohort Origin
IQ									
Boomsma	2002	RAKIT, WISC, RAVEN, WAIS, WAIS-3R	SEM	Rater	M,F	380–674	5–50	Cross-sectional	Netherlands Twin Register (NTR)
Wilson	1986	WISC-R	Correlations	Rater	M,F	526	5–15	Longitudinal	Louisville Twin Study
Akerman	1992	Verbal Ability Test	Correlations	Self	M	39	13–18	Longitudinal	Project Metropolitan, Sweden
Fischbein	1979	Differential Ability Test — Verbal, Military IQ Test — Verbal	Correlations	Self	M	95–110	12–18	Longitudinal	Swedish Longitudinal TwinStudy (SLU)
Osborne	1973	verbal IQ	Correlations	Rater	M,F	31–63	12–20	Cross-sectional	Kentucky, Georgia twins
Loehlin	1989	WISC, WAIS, + Revised scales (2nd wave)	SEM	Rater	M,F	258	3–24	Adoption, Longitudinal	Texas Adoption Project
ADHD									
Gjone	1996	CBCL — inattention	SEM	Parent	M	380–490	5–15	Longitudinal	Norwegian Medical Birth Register
					F	400–562	5–15		
Larsson	2006	Inattention, derived from DSM	SEM	Parent	M	986–1035	8–17	Longitudinal	TCHAD, Sweden
					F	990–1010	8–17		
Hay	2004	Inattention	SEM	Parent	M,F	1160	7–15	Longitudinal, Cross-sectional	Australian Twin Registry
Nadder	2002	ADHD-CAPA	SEM	Teacher	M	988	8–16	Longitudinal	VTSABD, US
					F	1206	8–16		

Note: SEM = structural equation modeling; ASPD = antisocial personality disorder; NEAD = Nonshared Environment and Adolescent Development; TCHAD = Twin Study of Child and Adolescent Behavior; MPQ = Multidimensional Personality Questionnaire; MTFS = Minnesota Twin Family Study; Add Health = US National Longitudinal Study of Adolescent Health; VTSABD = Virginia Twin Study of Adolescent Behavioral Development; CAP = Colorado Adoption Project; CDI = Child Depression Inventory; CAPA = Child and Adolescent Psychiatric Assessment; CBCL = Child Behavior Checklist; * Developed by the Dept of Criminology, Stockholm University (Ring, 1999) using items derived from the project 'Delinquent Behavior among Young People in the Western World' (Junger-Tas et al., 1994); †Australian National Health and Medical Research Council, Finnish Twin Cohort, New Swedish Twin Registry; ‡ Wilson and Patterson conservatism scale (1970)

correlations, but not heritability estimates, the Falconer formula $h^2 = 2(r\text{MZ}–r\text{DZ})$ was employed to estimate heritability (Falconer & Mackay, 1996).

Alcohol consumption: The quantity or frequency of drinking was the selected operationalization for this domain.

Nicotine initiation: When multiple measures were assessed, only 'initiation' was included.

ADHD: Frequently, ADHD was assessed using measures of inattention, hyperactivity, and/or impulsivity. 'Inattention' was deemed the most representative metric for this domain in the absence of a composite measure.

Externalizing behavior: This domain encompassed a variety of related measures including: antisocial behavior, aggression, conduct disorder, oppositional defiant disorder, and early problem behavior.

Social attitudes: Studies reporting the heritability of 'religiousness' or 'conservatism' were considered for inclusion in this domain. Where both were reported, 'conservatism' was included.

IQ: In studies which only reported IQ subscore heritabilities, 'verbal IQ' was retained as it is more stable and representative of global intellectual functioning. However, total IQ heritability measures were preferred.

Anxiety symptoms: Any study reporting heritability of anxiety symptoms measured using any of a range of instruments was included.

Depressive symptoms: Assessment of depressive symptoms by a variety of instruments was considered acceptable for inclusion here.

Statistical Analyses

Each phenotypic domain was analyzed separately. Since the number of subjects per study varied considerably, weighted averages of the heritability measures (and ages) were used. For studies that reported one heritability measure for a range of ages, the midpoint age was selected for analysis. For retrospective studies asking participants to reflect on childhood behaviors, 'childhood' was assigned an age of 12. Linear regression analyses were performed with study as a random effect using Proc Mixed in SAS (SAS Institute, 2005). Some studies reported results separately for males and females; therefore, additional analyses were performed in which sex was included as a covariate. For studies reporting total subject numbers but sex-specific heritability measures, analyses assumed equal proportions of males and females.

Weighted averages of the shared environmental estimates for three behavioral domains, nicotine initiation, IQ, and social attitudes, were also analyzed using linear regression in the same manner as heritability estimates. The remaining behavioral domains lacked estimates of shared environment from some studies or contained numerous estimates of zero which impaired meaningful analyses of this measure.

Results

Externalizing Behaviors

Externalizing behavior assessed independently demonstrated a moderate and statistically significant increase in heritability per year (+.008, $t = 3.21$, $p = .004$). The effect size of alcohol consumption was the greatest of all the externalizing behaviors (+.014), but fell short of statistical significance ($t = 1.36$, $p = .191$). Initiation of nicotine use demonstrated an effect size of only +.004 which was not significant ($t = 1.44$, $p = .164$).

Internalizing Behavior

The internalizing behaviors, symptoms of anxiety and depression, demonstrated differing patterns of change despite correlated genetic influences (Hettema et al., 2006; Kendler et al., 2006; Middeldorp et al., 2005).

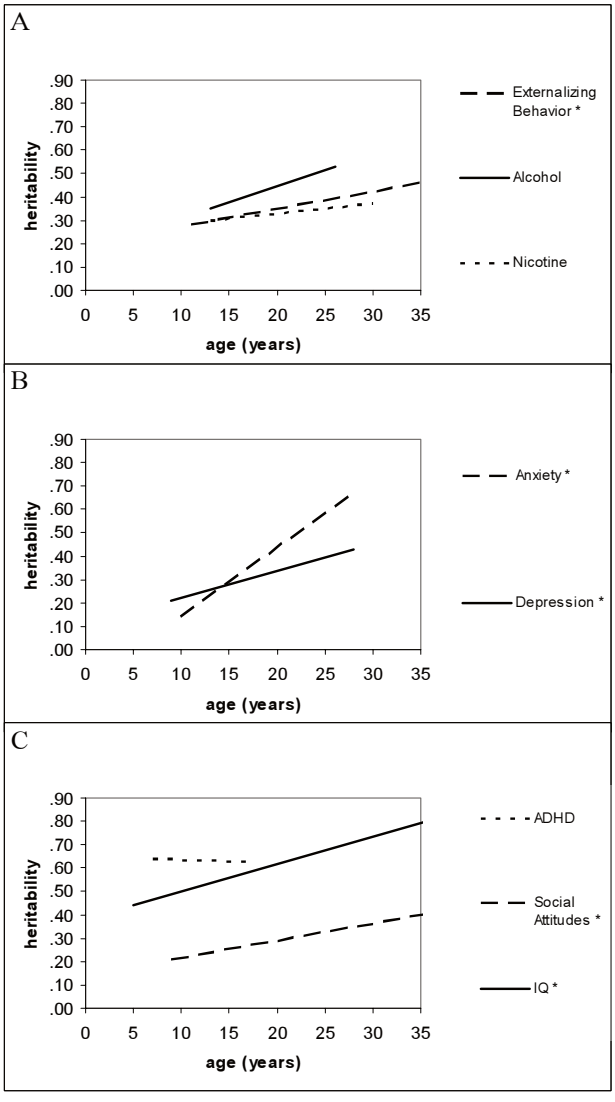

Figure 1

Regression lines for externalizing behaviors (A), internalizing behaviors (B) and other behaviors (C). Asterisks denote a significant difference from 0 for the slope (effect size) at a $p \leq .05$ level. Fit lines were truncated at age 35 to preserve focus on adolescent and young adult years.

https://doi.org/10.1375/twin.10.3.423 Published online by Cambridge University Press

Anxiety symptoms manifest the highest effect size of any domain (+.030, $t = 4.22$, $p = .0056$) while depressive symptoms demonstrated a moderate but significant increase in heritability per year (+.012, $t = 2.2$, $p = .048$). Differences in respondents (parent and self-report) for studies in the depressive symptoms domain may have increased heterogeneity in the data, while anxiety symptoms were exclusively self-reported.

Other Domains

The ADHD domain was conspicuous in its almost complete absence of change over adolescent development (effect size = –.001, $t = –.11$, $p = .918$). It was also unique in that all behavioral reports were by parents or teachers, not the individuals themselves, which is likely related to the fact that ADHD had the youngest average age for any domain. ADHD did have the highest heritability at the mean age for any domain, .635.

The genetic impact on social attitudes, though modest, grows throughout adolescence and young adulthood (+.007, $t = 4.8$, $p < .0001$). Any heterogeneity introduced by including both conservatism and religiousness in this domain does not appear to have impacted the significant increase in heritability seen here, although this may have been aided by homogeneity in the sampled populations which were exclusively drawn from US populations.

Similarly, IQ displayed a large and significant heritability increase per year (+.012, $t = .487$, $p < .0001$). Our results showing increases in the heritability of IQ over time are supported by previously documented reports using data from multiple sources (McCartney et al., 1990; McGue et al., 1993; Plomin, 1986).

Sex Differences

Significant heritability differences between males and females were detected for externalizing behaviors ($F = 11.95$, $p = .0003$) with females demonstrating higher heritability ($t = 3.77$, $p = .001$). No other domains showed significant sex differences in heritability: alcohol consumption ($F = .38$, $p = .691$), nicotine initiation ($F = .98$, $p = .293$), anxiety symptoms ($F = 4.66$, $p = .090$), depressive symptoms ($F = 1.78$, $p = .218$), ADHD ($F = .60$, $p = .566$), social attitudes ($F = .31$, $p = .580$), and IQ ($F = .68$, $p = .418$).

Discussion

A general pattern of increasing heritability from adolescence through young adulthood is evident for the majority of assessed phenotypes. To summarize, we found significant cross-time heritability increases for externalizing behavior, anxiety symptoms, depressive symptoms, IQ, and social attitudes and nonsignificant increases for alcohol consumption and nicotine initiation, but no evidence of heritability changes for ADHD. Considering the already high ADHD heritability measures at the youngest ages included here, it is conceivable that age-related changes were antecedent to the ages studied here. Alternatively, this phenotype may be a temperament variable largely independent of environmental modification. In the cases of alcohol consumption and nicotine initiation, low availability of these substances at the younger ages may have attenuated heritability estimates since expression of these behaviors must follow exposure to the substance. Additionally, for alcohol consumption, quantitation differences and the diversity of populations sampled likely contributed to the lack of statistical significance; however, Maes et al. (1999) also found no evidence of heterogeneity for the genetic effects on alcohol use in male and female twins aged 13 to 16. In other instances, studies modeling age in a continuous fashion may lend support to the significant changes found here such as the study of separation anxiety disorder symptoms by Feigon et al. (2001) in which heritability measures dramatically increased in boys and girls aged 3 to 18.

Several additional studies have demonstrated changing heritability measures over time but for behaviors too rarely studied to be included in our meta-analysis. It is interesting to note the range of behaviors which show heritability increases such as exercise (Simonen et al., 2004) and sports participation (Stubbe et al., 2005), conflict, involvement with, and regard for parents (Elkins et al., 1997), vocabulary knowledge (Van Den Berg et al., 2004), and eating attitudes and behaviors (Klump et al., 2000).

Cumulatively, a pattern of increasing heritability over adolescence and young adulthood has emerged for a range of behaviors. Several possibilities exist to explain the underlying causes for these observed changes. Active *r*GE theory offers one attractive prospect since an increasing range of environmental options provides individuals with greater opportunities to express their genetic proclivities. Children's gravitation toward particular environments is iteratively reinforced as their genetic propensity toward behaviors is validated by environmental feedback. For example, a predisposition toward disruptive behavior in school may lead to detention with other disruptive children, subsequent friendship with them, and cascading antisocial behaviors fueled by interactions with like-minded peers and a growing sense of identification with this lifestyle. Over time, this process results in growing genetic modification of environmental experiences and thereby increased heritability. It is also possible that the accumulating effects of consistent gene expression from one set of genes gradually accounts for more of the phenotypic variance if environmental effects are only transiently influential. Alternatively, the expression of a novel set of genes would enhance the genetic proportion of the variance. For example, DZ twins may be less correlated due to differences in the developmental timing of their maturation. In one study of genetically influenced eating pathology in 11- and 17-year-old female twins, heritability measures from age 11 twins who had reached puberty matched those of the 17-year-old

https://doi.org/10.1375/twin.10.3.423 Published online by Cambridge University Press

twins (h^2 = .54 for both groups) in sharp contrast to their same-aged prepubertal peers (h^2 = 0; Klump et al., 2003). Clearly, differences in the timing of biological development can underlie behavioral differences, and the time of life studied here is particularly amenable to such occurrences.

A proportional reduction in environmental variance would also result in rising heritability estimates, and declining importance of shared environmental influences is one potential mechanism. To explore the plausibility of this idea we examined the three phenotypes for which we had adequate data to address changes in shared environment over time. Decreases in shared environment were significant for all three domains: nicotine initiation (t = –3.38, p = .0025), IQ (t = –3.63, p = .0014), and social attitudes (t = –3.75, p = .0009). For IQ and social attitudes, these changes likely contributed to the significant increases in heritability demonstrated here. The decreases in shared environmental measures for nicotine initiation, however, did not confer significance to the increase in heritability for this domain. It is also entirely conceivable that measurement error decreases during this timeframe as the subjects become more competent to assess and report their behaviors. Reduced variance due to measurement error would then manifest as an increase in the additive genetic proportion of variance.

Competing theories of gene–environment interaction/correlation may not be mutually exclusive. They may act in concert to yield the patterns of heritability seen for a given phenotype or differing theories may be manifest in different domains. For example, in domains showing limited or nonsignificant heritability increases, it is possible that persisting effects of environmental experiences or declining effects of passive *r*GE may have obscured or counterbalanced increasing genetic influences due to active *r*GE or novel gene expression.

Alternatively, genetic and environmental variation could truly be relatively static. Distinguishing between these possibilities is particularly challenging and beyond the scope of this study since true variance (rather than standardized estimates) would be required from all data sources to accomplish this.

One notable strength of this meta-analysis is the exclusive use of studies which reported heritability measures at two or more timepoints. In doing so, we have reduced the error portion of age-to-age variability inherent in meta-analyses using single-timepoint measures. Use of the same sample populations, measurement instruments, and analytic methods within studies allows for closer comparability for heritability measures across ages.

Several limitations should be considered in the interpretation of these results. For the present study, a limited number of research articles using unique subject cohorts were available for some domains of interest. Additional studies would be desirable to bolster the number of subjects and enhance confidence in the results. Also, a few studies have modeled age in a continuous fashion. While this approach can provide a more sensitive measure of the impact of age on heritability measures, we were unable to incorporate these models into our analysis. When possible, results from full ACE models were used, but occasionally only best fitting AE model estimates were reported. If undetected shared environment was present in these samples, it would be largely confounded with additive genetic effects, thereby slightly inflating heritability estimates for these studies. However, since this occurs in only three primary articles (Hopfer et al., 2005; Kaprio et al., 1992; and Larsson et al., 2006), it seems unlikely to have greatly influenced our results. Another consideration is that the operationalization of each behavior of interest often varied a little from study to study. The measurement of slightly different behaviors within domains likely contributed to the overall variance of the data and may have impacted the results in some instances.

Cross-cultural differences in reporting could further influence studies of some phenotypes such as alcohol consumption, nicotine initiation, or anxiety or depressive symptoms. The social attitudes domain was a conspicuous exception to this, however, since all studies of this phenotype drew samples from US populations, limiting the cultural variation. A related concern is that within-culture differences from the inclusion of cross-sectional studies may have clouded the results. Different age strata within a population may have experienced different social environments which impacted their heritability measures. However, a test of this idea in three American cohorts born between 1934 and 1974 found that, although prevalence rates for illicit drugs and tobacco varied markedly, heritability estimates could be constrained across groups (Kendler et al., 2005).

It is worth noting that nearly all available research in these areas sampled from Caucasian populations in Europe, Australia, and America. Additional research from culturally and genetically disparate populations would be useful in gleaning a better understanding of human heritability changes over this developmental timeframe.

Despite these potential concerns, evidence for increases in heritability throughout the adolescent and early adult period has been presented for some behavioral domains. These results caution against the common practice of fitting a single heritability statistic to data from participants with a wide range of ages since the proportions of genetic and environmental variability may differ. Concern over acquiring enough subjects should be tempered by the knowledge that increased age heterogeneity could also inflate confidence bounds, thus negating any benefits of a larger sample size for determining a single heritability measure. A cross-sectional approach or inclusion of age as a moderator in analyses may be more appropriate. Additionally, direct comparison with other studies with subjects of different ages may

https://doi.org/10.1375/twin.10.3.423 Published online by Cambridge University Press

not be entirely feasible. It is important to remember that heritability statistics only apply to a specific population at a given time and age-related differences may be at least as important as cohort effects and genetic differences in a population. As repeatedly demonstrated here, marked increases in heritability measures may be manifest over adolescence and early adulthood.

Whether or not heritability continues to increase throughout adulthood and senescence remains to be seen. Heath et al. (1999) found that genetic influences for smoking initiation were high in men and women under 30 (.62), but lower in the over 30 population (.51). Even the high heritability of IQ appears to diminish with advancing age. In a sample of adult twins aged 27 to 59, the verbal and performance IQ heritability measures were .70 and .73 respectively. An older cohort aged 60 to 94 demonstrated corresponding measures of only .56 and .60 (Finkel & McGue, 1998). Heritability measures of personality variables such as neuroticism have also evinced decreases with age in adults (Floderus-Myrhed et al., 1980; Viken et al., 1994). The proportional genetic influence on extraversion may also decline with age (Viken et al., 1994), although this was not universally observed (Floderus-Myrhed et al., 1980).

This suggests that beyond the tumultuous formative years, environmental effects may begin to accumulate and form a larger proportion of the total phenotypic variance. As of now, too little research has been done to deduce the overall pattern of heritability changes in later adulthood. This and other questions will likely be resolved as additional research emerges elucidating the relative genetic and environmental influences that shape behavioral development over the lifespan.

Acknowledgments

This research was supported in part by NIH grants DA011287, MH49492, and MH20030.

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