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Abstract

Previous research has demonstrated stability of cognitive ability and marked heritability during adulthood, but questions remain about the extent to which genetic factors account for this stability. We conducted a 35-year longitudinal assessment of general cognitive ability using the Armed Forces Qualification Test administered to 7,232 male twins in early adulthood and readministered to a subset of 1,237 twins during late middle age. The proportion of variance in cognitive functioning explained by genetic factors was .49 in young adulthood and .57 in late middle age. The correlation between the two administrations was .74 with a genetic correlation of 1.0, indicating that the same genetic influences operated at both times. Genetic factors were primarily responsible for stability, and nonshared environmental factors were primarily responsible for change. The genetic factors influencing cognition may change across other eras, but the same genetic influences are operating from early adulthood to late middle age.

Genes Determine Stability and the Environment Determines Change in Cognitive Ability During 35 Years of Adulthood

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Abstract

Previous research has demonstrated stability of cognitive ability and marked heritability during adulthood, but questions remain about the extent to which genetic factors account for this stability. We conducted a 35-year longitudinal assessment of general cognitive ability using the Armed Forces Qualification Test administered to 7232 male twins in early adulthood and re-administered to a subset comprising 1237 twins during late middle age. The proportion of variance in cognitive functioning explained by genetic factors in young adulthood was .49 and it was .57 in late middle age. The correlation between the two administrations was 0.74 with a genetic correlation of 1.0, indicating that the same genetic influences operated at both times. Genetic factors were primarily responsible for stability and non-shared environmental factors for change. The genetic factors influencing cognition may change across other eras, but from early adulthood to late middle-age the same genetic influences are operating.

Introduction

Spearman described a general factor common to many mental abilities that he called "*g*" (Neisser, Boodoo, Bouchard, Boykin, Brody, Ceci, Halpern, Loehlin, Perloff, Sternberg, & Urbina, 1996) which accounts for at least 50% of the variance across a range of mental tests and is one of the most replicated findings in psychology (Neisser et al., 1996). General cognitive ability correlates with numerous other characteristics, such as academic and occupational success (Neisser et al., 1996). There has been very little longitudinal, genetically informative research that addresses this trait during the period from early adulthood to late-middle adulthood, a period that has been quite understudied in many domains. Three important questions about general cognitive ability during this period are: a) What are the magnitudes of genetic and environmental influences during this period?; b) Do the same or different genetic and environmental influences operate at different ages?; and c) What are the genetic and environmental influences on change and stability over time? In order to answer these questions unambiguously a study must have: a) longitudinal data; b) a long time interval; c) the same measure at each assessment; and d) a genetically informative sample. To our knowledge, no study to date has met all of these criteria. Midlife remains a sorely understudied period, with the bulk of developmental research investigating childhood, adolescence or old age. Most of the extant genetically informative studies are cross-sectional, and the few longitudinal studies cover relatively short time intervals. In many studies different measures of *g* are used at different ages and each measure will have its own unique method variance.

What are the magnitudes of genetic and environmental influences during this period? Bouchard and McGue (2003) reviewed the behavior genetic studies of cognitive ability and suggested that the relative strength of genetic and environmental influences on intelligence varies with age. A number of investigators have concluded that the influence of genetic factors increases, while the influence of shared environmental factors decreases with age, at least until middle age (McGue, Bouchard, Iacono & Lykken, 1993; Plomin & Spinath, 2004; McCartney, Harris, and Bernieri, 1990;). Vogler (2006) suggested that the heritability of cognitive functioning appears to be relatively stable over time with some decline in heritability in older cohorts and the results of several studies support this (Pedersen et al..1992; Finkel et al., 1995, 1998; Posthuma et al., 2001; McGue & Christensen, 2002; Plomin, 1994). Reynolds et al. (2005) in a study with assessments at ages 50, 60, 70, and 80 years found an inverted U-shaped pattern for genetic variance, i.e., genetic variance increased somewhat from age 50 to 60 years and then decreased. Among studies of adults, the limited number that utilized a true longitudinal design, the relatively brief time intervals utilized, and the preponderance of subjects over the age of 65 years essentially precludes a definitive answer to the question of whether the magnitude of genetic and environmental influences changes from early adulthood to late middle age.

Do the same or different genetic and environmental influences operate at different ages? The extent to which genetic influences change at different ages is not well understood for most traits, but it has important implications for gene association studies. If the sample being studied is heterogeneous with regard to age and different genes are influencing the trait at different ages, this heterogeneity would increase the difficulty of

detecting an association. Because an individual's genotype is fixed at conception, it is tempting to think of the influence of genetic factors as a stable, static phenomenon. McClearn (1993) quotes Francis Galton's observation, made in 1875: "It must be borne in mind that the divergence of development, when it occurs, need not be ascribed to the effect of different nurtures, but it is quite possible that it may be due to the appearance of qualities inherited at birth, though dormant (Galton, 1875)." While the individual's genotype does not change during the lifespan, different genes may be expressed at different developmental periods (Vogler, 2006). As Plomin et al. (1993) pointed out, because a trait is equally heritable at two ages does not mean that the trait is stable with regard to its genetic determinants and different heritabilities at two ages do not necessarily reflect instability of genetic influences.

Previous longitudinal studies addressing the question of whether different genetic factors were operating at different ages included children only. A number of studies have found evidence for new genetic influences arising over the course of childhood and adolescence (Wilson & Matheny, 1983; Eaves, Long, & Heath, 1986; Petrill, Lipton, Hewitt, & Plomin, 2004; Cardon, Fulker, Defries, & Plomin, 1992; Defries, Plomin, & Labuda, 1987) and some have not (Bartels, Rietveld, Van Baal, & Boomsma, 2002; Polderman, Gosso, Posthuma, Van Beijsterveldt, Heutink, Verhulst, & Boomsma, 2006).

What are the genetic and environmental influences on change and stability over time? McGue and Christensen (2002), using a cohort-sequential design, examined a sample of twins over age 70 on a battery of cognitive measures and found a heritability of 0.06 for the linear change over four occasions spanning six years. Plomin et al. (1994) in a longitudinal study of subjects in the second half of life found that genetic factors

accounted for nearly 90% of the stability. Reynolds et al (2005) in a study of latent growth parameters at age 65 found that the heritability of linear change was 0.01, while the nonshared environment explained 0.99 of the variance. The quadratic trend (acceleration of cognitive change over time or "change in the change") had a heritability of 0.43 and a contribution from the nonshared environment of 0.57. DeFries et al (1987) found that the genetic contribution to the cognitive stability observed between age 4 and adulthood was 0.28.

We are unaware of any genetically informative studies that span the period from early adulthood/adolescence to late middle age that utilize a true longitudinal design and repeated administration of the same cognitive measure. The Vietnam Era Twin Study of Aging (VETSA) (Kremen, Thompson-Brenner, Leung, Grant, Franz, Eisen, Jacobson, Boake & Lyons, 2006) provides a unique opportunity to investigate the determinants of cognitive development from adolescence/early adulthood to late middle age, an important but understudied period in developmental aging research.

Materials and Methods

Subjects

Age 20 Armed Forces Qualification Test (AFQT) scores were obtained from military records for 7232 (1669 identical pairs, 1303 fraternal pairs, and 1288 unpaired twins) men from the Vietnam Era Twin Registry (VETR). Age 55 AFQT scores were obtained from the 1237 men who participated in VETSA. VETSA participants were assessed at one of the two testing sites (Boston University {BU} and the University of California, San Diego {UCSD}) or in rare circumstances they elected to have a research assistant travel to them. Informed consent was obtained after the nature and possible

consequences of the studies were explained. The study was approved by the BU and UCSD Institutional Review Boards. Zygosity for VETR members was initially determined by a combination of questionnaire and blood group type. Eisen et al. (1989) reported that zygosity determined utilizing this protocol was 95% accurate. VETSA participants underwent additional zygosity testing via analysis of 25 microsatellite markers.

VETR members are representative of all twins who served in the military during the Vietnam War on a variety of socio-demographic variables (Eisen, True, Goldberg, Herderson, & Robinette, 1987; Goldberg, True, Eisen, Henderson, & Robinette, 1987). In VETSA 48.5% of subjects that we contacted agreed to participate. Given that participation entailed a two or three day trip to Boston or San Diego, we believe that the participation rate is reasonable. Moreover, we have carefully compared participants to non-participants.

Studies have shown that differences in socioeconomic status between the military veterans and non-veterans, when observed, are modest in size, contrary to a popular assumption that military men came from lower socioeconomic strata (Boulanger, 1981; Cooper, 1977). The demographic characteristics of VETSA participants is very similar to that of 2003 U.S. Census data for men in their 50s in education, median self-income, ethnicity, marital status, and employment. VETSA participants do not differ significantly from the overall subject pool in age at induction, race, marital status, education at induction, branch of military, Vietnam service, combat experience, or lifetime prevalence of the more common types of psychopathology. Although the VETSA sample was drawn

from individuals who were members of the military, two-thirds were not stationed in a war zone.

Measures

Armed Forces Qualification Test (AFQT)

The AFQT is a 50-minute paper-and-pencil test consisting of 100 multiple-choice items administered just prior to military induction (Bayroff & Anderson, 1963). It is highly correlated with measures of general cognitive ability (Uhlaner & Bolanovich, 1952). For example, McGrevy and Knouse (1974) found a correlation of 0.84 (after correcting for restriction of range) between the AFQT and Wechsler Adult Intelligence Scale (1955) scores. Items equally represent the four domains of vocabulary, arithmetic word problems, knowledge and reasoning about tools and mechanical relations, and visual-spatial organization (1952). In VETSA, we administered the same AFQT version given to participants approximately 35 years earlier.

Analysis of Twin Data

AFQT scores were recorded as percentiles based on military norms. For the purposes of genetic model-fitting the raw percentiles were transformed to their normal deviates. We conducted biometrical modeling to quantify genetic and environmental determinants of the traits being studied. In biometric analysis, we assume that observed variation in the trait is due to a mixture of latent additive genetic (A), shared environmental (C), and nonshared environmental (E) factors. The additive genetic component of variance, usually called "heritability," is due to genes with influences that combine additively. The common environmental variance includes all environmental influences shared by members of a twin pair that serve to make the twins similar to one another (e.g., the

family environment). The unique environment entails non-shared environmental factors that make members of a twin pair different from one another, plus measurement error. Our analyses utilized the simplest and most frequently applied multivariate model, the *correlated factors model*. In the *correlated factors model*, in addition to the A, C and E parameters of the univariate model, we also add correlations between latent variables, r_A, r_C, and r_E. These symbols represent the genetic correlation, the shared environmental correlation and the nonshared environmental correlation, respectively.

Results

The mean age of participants at the baseline AFQT administration was 19.8 years (±1.5; range = 16-31 years). Mean age for re-administration during VETSA was 55.4 years (±2.48; range 51-60 years). The correlation of AFQT at age 20 with AFQT at age 55 was 0.74 ($p<.001$). Mean baseline AFQT score (n=7,232) was 54.6 (±23.5). Mean AFQT score at age 55 (n=1,237) was 64.1 (±20.9). Change could only be computed for the 1,237 subjects who participated in VETSA. Although the magnitude of the change was small (3 percentile points; 0.16 SDs), the mean at age 55 was significantly higher than at age 20 (paired samples t-test = -6.26, $p<.001$). At age 55 the correlation between the AFQT and the Wechsler Abbreviated Scale of Intelligence IQ was 0.84 (after correcting for restriction of range).

Maximum likelihood estimates of correlations for MZ and DZ twins are given in Table 1, indicating the principal trends in the data. The cross-pair correlations for MZ twins are all greater than those for DZs, suggesting that genetic effects play some role in individual differences. However, cross–twin correlations for DZ twins exceed one half of

their respective MZ correlations which may reflect environmental effects shared by twins, genetic consequences of assortative mating, or both (Eaves, 1982). The cross-twin cross-age correlations for MZ twins are similar to the cross-twin within-age correlations, indicating a very high level of long-term stability in the effects of familial (genetic and/or shared environmental) influences.

Table 1 about here

Model Fitting Analyses

Maximum-likelihood estimates of the contributions of additive genetic effects (A), environmental effects shared by twins (C), and non-shared (individual-specific) environmental effects unique to individual twins (E) were obtained using the structural equation modeling program Mx (Neale, Boker, Xie, & Maes, 2003). Several structural models were fitted to the data and likelihood-ratio tests were conducted for differences between means and covariance structures (see Table 2). Model 1 allows each variance, covariance, and mean to take its own value in MZ and DZ twins. Subsequently, reduced models with fewer parameters are tested to determine if they fit the data as well as the full model (Model 1). Models 2 through 6 presented in Table 2 are compared to Model 1. These tests indicated that there is no mean difference in AFQT scores between twins randomly designated as 'A' versus 'B' or between MZ and DZ twins. These tests also indicated a significant mean difference between AFQT scores at baseline and follow-up and a significant difference in covariances for MZ versus DZ pairs, as would be expected if there were genetic effects. We see that the full bivariate saturated "ACE" model

(model 6), which allows means to differ only between baseline and follow-up, gives a fit that is comparable to the most general model (#1) that imposes no constraints. Although model 6 can be reduced by omitting the genetic ($p=.32$) and shared environmental ($p=.26$) effects unique to follow-up, we take the conservative approach of including these parameters to preclude any bias in the estimate of heritability that may occur at follow-up.

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Table 2 about here

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Effects of A, C, and E at baseline and at follow-up derived from the full bivariate model are presented in Figure 1 as both path coefficients and proportions of variance; correlations between the latent factors (e.g., shared environmental influences) are also presented. There was not a statistically significant difference in the percentage of variance in AFQT scores explained by genetic effects in early adulthood (49%) *versus* that at the 35-year follow-up (57%). However, before concluding that these two values are the same, it is prudent to consider the statistical power of the comparison. Therefore, we calculated the power of the test for differences in heritability assuming that the heritability during early adulthood is 0.5 and heritability at follow-up is 0.6, and the genetic correlation between occasions is assumed to be 1.00. Sample sizes approximated those of the current study, i.e. 200 MZ and 170 DZ pairs measured on both occasions and a further 1800 MZ and 1500 DZ pairs measured only at induction. The power of the test for heterogeneity of heritability over waves ($\alpha=0.05$) is 43%. This indicates that even if

the observed values are truly different from one another, 57% of the time with our sample size, we would fail to conclude that the difference is significant.

The correlation between genetic influences at baseline and late middle age does not differ significantly from unity. The correlation between shared environmental effects in early adulthood and late middle age is 0.77. The shared environmental and genetic influences that affected twins' performance as young adults have persisted into later life. As described above, the phenotypic correlation between AFQT at age 20 and at age 55 is 0.74; modeling results indicate that 71.3% of this phenotypic correlation is attributable to genetic influences operating at both occasions, 22.4% is due to shared environmental influences on both occasions, and 6.3% due to nonshared environmental influences. Although the average change in AFQT scores from age 20 to age 55 was significant, but rather small, we conducted analyses to determine the extent to which genetic and shared environmental factors contributed to change. These two factors had very modest effects on change during this interval (1.7% and 15.2% for genetic and shared environmental influences, respectively). By far the greatest influence on change in AFQT was the nonshared environment, which accounted for 83.1% of the variance in change.

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Figure 1 about here

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Discussion

Our findings with regard to the stability of an individual's general cognitive ability over three and one-half decades of adulthood indicate that there is a slight, but significant increase in the level of performance and there is a substantial coefficient of

stability (i.e., correlation from baseline to follow-up). The pattern of correlations within individuals, across twin pairs, and across-twins/across-times provides for some interesting additional conclusions. If one wanted to predict how a given individual would score on the AFQT when he is 55 years old, one would do approximately equally well by predicting from the individual's own score at age 20 or from his MZ co-twin's score at age 55. MZ co-twins are as similar to one another when measured at the same time as an individual is similar to himself over the 35 year interval. The observed genetic and shared environmental correlations indicate no new genetic or shared environmental influences that manifest themselves over the extended interval of adulthood. This genetic correlation is different from several studies of children and adolescent twins and adoptees assessed at different ages (described above), suggesting that during some developmental periods there are changes in the genes that are influencing cognitive ability. However, it is also possible that the use of different measures at different occasions in those studies of younger subjects resulted in the lower genetic correlations. Although we found that the same genes were operating over the 35 year period from age 20 to age 55, it is quite possible that some different genetic influences could be operating 35 years later, when subjects are 90 years old. Indeed it is unknown if the same genes would be operating 10 or even 5 years later. Although the estimates of heritability during early adulthood and late middle age were 49% and 57%, respectively, our data do not provide the statistical power necessary to determine whether heritability increases significantly.

The cross-temporal correlation for non-shared environmental effects, though significant, is only moderate. This result is consistent with the view that non-shared environmental effects are highly age-specific, i.e., much of the variation within MZ pairs

reflects short-term environmental influences contributing to differences in test performance across time. Our data confirm this view because the average correlation between early adult and follow-up scores (0.74) is similar to the correlation between MZ twins measured at the same time (0.71). From the genetic and shared environmental correlations, it is possible to infer that the observed phenotypic stability primarily reflects genetic and shared environmental influences; 71.3 % of the correlation between age 20 and age 55 scores is due to genetic factors and 22.4% and 6.3% is due to the shared and non-shared environments, respectively.

Although there was considerable stability over time, there was still a substantial amount of variability in the entire sample, with the greatest increase in AFQT score being 57 points and the greatest decrease being 55 points. There was a change of at least 10 points (½ SD unit) among 44.6% of the subjects and 17.6% of the subjects had a change of 20 points (1.0 SD unit) or greater. Changes during this time were overwhelmingly (83.1%) due to aspects of the environment that were not shared by twins. That is, the same type of influences that make identical twins different from each other are the ones that make an individual perform differently at age 55 from how he performed at age 20.

The VETSA sample includes only men, so our findings may not generalize to women. Our likelihood computations assume that data are missing at random or missing completely at random, however we know that is not the case. Individuals who scored below the 10^{th} percentile did not qualify for induction. VETSA participants also had slightly higher AFQT scores at age 20 than VETR members who did not participate in VETSA. This difference may be due to factors such as people of higher cognitive ability being more inclined to participate in scientific research and being easier to locate. In

principle, correction for biases of ascertainment and follow-up are possible as long as we can specify the selection criteria at both stages. However, it has been shown that the effects of far more extreme selection on twin resemblance, typically, do not lead to substantial biases in the patterns of twin correlation (Martin & Wilson, 1982).

The findings reported here regarding cognitive ability have some interesting similarities and differences with results we obtained for another widely studied human characteristic – body mass index (BMI). In a sample of twins from the VET Registry that overlaps somewhat with the VETSA sample reported in this paper, the average BMI increased dramatically from age 20 to age 48, from a mean of 22.7 ($\pm$3.0) to 27.8 ($\pm$4.2) (Franz, Grant, Jacobson, Kremen, Eisen, Xian, Romeis, Thompson-Brenner & Lyons, 2007). Genetic factors were the primary source of variation in BMI at age 20 and age 48, but unlike our findings for cognitive ability, the heritability of BMI decreased significantly from age 20 to age 48 (heritability 76% and 59%, respectively). Shared environmental influences accounted for a negligible (7 percent, ns) amount of variance in BMI at each time point. The phenotypic correlation for BMI across the 28 year interval was 0.52. The genetic correlation of the overlap between age 20 and age 48 BMI was estimated at 0.60, indicating that some, but not all of the genetic influence on BMI is accounted for by the same genes over time. Some of the genes that influence BMI during middle age were not operating in early adulthood. This result is quite different from that for general cognitive ability.

Our results support the remarkable conclusion that virtually all of the genetic influences on cognitive performance expressed in young adulthood are still manifest 35 years later. We found little support for the possibility that "new" genes exert their

influence over the course of adult development. A practical implication of this finding is that studies, such as genome wide association studies, seeking to identify specific alleles that influence general cognitive ability, may safely combine subjects from age 20 to 60 years. However, it is quite possible that in older age, genetic (and environmental) influences that were not operating earlier may begin to do so. The relative influence of the non-shared environment remained stable over time, but there was only very modest overlap of the features of the nonshared environment influencing cognition in early adulthood versus late middle age. These findings provide what we believe are the first long-term longitudinal results to challenge suggestions that decades of exposure to environmental influences over the course of the lifespan might attenuate the influence of genetic factors.

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Table 1: Maximum likelihood estimates of correlations for AFQT scores at baseline ("Age 20") and during VETSA ("Age 55")

	Twin 1 Age 20	*Twin 2 Age 20*	*Twin 1 Age 55*
	Monozygotic		
Twin 2 Age 20	.75		
Twin 1 Age 55	.74	.70	
Twin 2 Age 55	.69	.75	.75
	Dizygotic		
Twin 2 Age 20	.52		
Twin 1 Age 55	.70	.42	
Twin 2 Age 55	.45	.79	.47

For all correlations, $p<.001$

Table 2: Bivariate longitudinal ACE Model: Model comparison statistics

Model		-2LL	k	LRT	Δdf	p	AIC
1	Saturated Model	15097.344	28	-	-	-	-
2	Means equal for A and B twins	15101.134	24	3.790	4	.435	-4.210
3	Means equal for MZ and DZ twins	15099.980	24	2.636	4	.620	-5.364
4	Means equal for induction and follow-up	15171.044	24	73.700	4	<.001	65.700
5	Variances and covariances equal for MZ and DZ twins	15340.484	18	243.141	10	<.001	223.141
6	Bivariate ACE model	15122.444	11	25.100	17	.092	-8.900

-2LL: -2 times the log-likelihood
k: Number of free parameters in model
LRT: Likelihood ratio test. Models 2-6 are compared against Model 1. The LRT is distributed as chi-square statistic
Δdf: Change in degrees of freedom of saturated model and submodel
p: Significance level of chi-square test
AIC: Akaike's Information Criterion. An index of the balance between goodness of model fit and parsimony

Figure 1: Bivariate correlated factors model for AFQT scores at baseline and during VETSA from the bivariate ACE model. A=additive genetic; C=shared family environment; E = non-shared environment; R_G=Genetic correlation; R_C=Shared environment correlation; R_E=Non-shared environment correlation. Values adjacent to lines are path coefficients; values in parentheses are the 95% CIs; values in brackets are standardized variance components (h^2=genetic influences; c^2=shared environment; e^2=non-shared environment).