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Marriage and Genetic Variation across the Lifespan: Not a Steady Relationship?

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Abstract The prevalence of marriage varies across the lifespan, as does its importance to reproduction and the nurturance of children. We examined genetic and environmental influences on self-reported marriage at each decade from 20 through 70 years of age, using data collected for the Duke Dementia Study, a followed-up subset of the World War II Veteran Twin Registry. Genetic influences best fit a common factor model, supplemented by another, age-specific, genetic factor at age 30. Broad heritability increased from age 20 through 40, and then decreased to zero by ages 60 and 70. A longitudinal Cholesky model best described environmental influences on marriage across the lifespan. Shared environmental factors showed their greatest influence at age 20, no influence at 30 or 40 years, and then, reappeared with influence at 60 and 70. Variance due to error and unique environmental influences increased steadily to age 50 years and then declined slightly.

Keywords Marriage · Divorce · Individual differences · Lifespan · Genetics · Heritability · Twins · Endophenotypes · Natural selection · World War II veteran · Adult development · Pair bonding

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Introduction

She: Love, says the poet, has no reasons.
He: Not even after fifty years?
She: Particularly after fifty years.
–Archibald MacLeish, *The Old Gray Couple*

Marriage and divorce necessarily depend on the mutual contributions of a couple through their dyadic interaction and behavior. We typically think of marriage and divorce as social outcomes that involve at least two individuals' phenotypes. Even so, individuals differ in their propensity to form and maintain heterosexual pair bonds. Genetic factors account, in part, for phenotypic individual differences in the propensity to marry and to divorce (D'Onofrio et al. 2005; Jockin et al. 1996; Johnson et al. 2004; McGue and Lykken 1992; Trumbetta 2001; Trumbetta and Gottesman 1997, 2000; Trumbetta et al. 1995; Turkheimer et al. 1992).

Most genetically informative studies of marital status phenotypes have been cross-sectional, with only one occasion of measurement (Jockin et al. 1996; Johnson et al. 2004; McGue and Lykken 1992) or two (Turkheimer et al. 1992; Trumbetta et al. 1995; Trumbetta and Gottesman 1997, 2000). Despite evidence of important changes in marital quality and stability across the lifespan (Karney and Bradbury 1995; Levenson et al. 1993), no prior studies have investigated patterns of consistency and change across the adult lifespan in the relative influences of genetic and environmental factors on marriage.

Most previous twin studies of genetic influences on marital status examined individual differences in either the propensity ever to marry or, if married, ever to

divorce. Heritability estimates for marriage propensity have varied. In the World War II Veteran Twin Registry, estimates of genetic influence on males' ever marrying ranged from .57, based on responses to a 1972 questionnaire, downward to .44, based on a later, 1985 questionnaire (Trumbetta et al. 1995; Trumbetta and Gottesman 1997, 2000), whereas the Minnesota Twin registry produced higher heritability estimates for marriage in a mixed-sex sample, at 0.72 in the univariate case and at 0.83 in a bivariate model with personality traits (Johnson et al. 2004). Similarly, divorce risk was less heritable in the World War II sample than in the Minnesota sample, with genes explaining only 27% and 15% of variance in divorce risk among the World War II veteran twins in 1972 and 1985, respectively (Trumbetta et al. 1995; Trumbetta and Gottesman 1997, 2000) but 52% of divorce risk variance among the Minnesota pairs (McGue and Lykken 1992).

The notable discrepancies in heritability estimates for marriage and divorce between these Minnesota and WWII twin registries probably reflect both error variance and demographic differences between the samples. While random error may contribute to the between-sample differences, the World War II sample also may contain more within-sample error. Compared to the one-state Minnesota sample, this national sample's cross-jurisdictional heterogeneity of marital law could reduce the phenotypic ''signal to noise'' ratio for marriage, thereby increasing error and related estimates of nonshared environmental effects while decreasing heritability estimates.

Demographic differences also probably contributed to the two samples' discrepant heritability estimates for marriage and divorce. These include the Minnesota sample's mixed-sex composition, later historical period and cohort, and younger age at marital status ascertainment. Heritability estimates are higher for women than for men for life history measures and fitness traits, such as lifetime reproductive success (Pettay et al. 2005), so we might expect to see the same for marriage. However, in the Minnesota sample, neither the heritability of marriage (Johnson et al. 2004) nor that of divorce (Jockin et al. 1996) showed sex differences, so its higher heritability estimates cannot be attributed to the inclusion of women.

Heritability of fertility-related phenotypes also can vary by historical period. Greater latitude of individual choice in fertility-related behaviors leads to increasing levels of biological (relative to environmental) influences on them (Udry 1996). Therefore, the higher heritability of marriage and divorce in the Minnesota sample may reflect increasing personal choice for marriage, divorce, and cohabitation during the latter half of the 20th century. Demographic studies of human fertility show a similar effect, with the heritability of fertility increasing historically with widening availability of contraception (Kohler et al. 1999). Compared to their World War II predecessors who were born between 1917 and 1927, the Minnesota sample's generation, born between 1936 and 1955, likely had more freedom to express phenotypically any relevant genetic predispositions toward remaining unmarried, toward cohabitation without marriage, or, if ever married, toward divorce.

Discrepancies between the Minnesota and WWII samples may also stem from age differences. The mean age of ascertainment for marriage of Minnesota twin pairs was between 39 and 40 years, and for divorce (among those ever married), between 40 and 42 years (McGue and Lykken 1992). These Minnesota twins were approximately one to two decades younger than the World War II pairs, who averaged 50 years of age in 1972 and 63 years of age in 1985 when assessed for marital status. Also, within-sample evidence from these twin registries suggests that the relative strength of genetic and environmental influences on marital status varies with age. In the Minnesota sample, twins over age 40 showed a higher heritability for divorce than younger twins (McGue and Lykken 1992), suggesting that the influence of genetic factors on divorce may increase from early to mid-adulthood. In the World War II sample, surveyed in 1972 and 1985, a period that roughly corresponds to that from early-middle to late-middle age, the heritability of both ever marrying and ever divorcing declined (Trumbetta et al. 1995). Although differential attrition of divorcés may have accounted, in part, for these observations (Trumbetta et al. 1995), evidence both within and between samples remains sufficient to expect that, for men, genetic influences on marriage and divorce may increase from early to mid-adulthood, and then, decline.

All of these previously observed, age-related differences in heritability were for ''lifetime ever'' phenotypes of marriage and divorce. Studies of consistency and change across the lifespan, however, must narrow the marital phenotype from previous lifetime measures to a more specific, time-constrained measure, such as *current marriage.* Analyses of *current marriage* in the 1972 and 1985 World War II data demonstrate even more pronounced changes in heritability than those observed for the phenotype of *lifetime ever* marriage or divorce. For the 1972 data, genetic effects accounted for the majority of variation in current marriage, with estimates of $h^2 = 0.51$ and $e^2 = 0.49$, and zero effect of

shared environments, but for the 1985 data, variation in current marriage was entirely environmental, with estimates of shared and non-shared environmental contributions of $c^2 = 0.24$ and $e^2 = 0.76$, respectively, and zero genetic influence (Trumbetta 2001, 2004).

This shift from strong genetic influences in 1972 to purely environmental influences on current marriage in 1985 is more striking in its historical context. As previously noted, when environmental constraints decrease and individual freedom of choice increases, the ratio of genetic to environmental influences on voluntary behaviors increases. Legislative and social changes during the 1970's and early 1980's, including the adoption of no-fault divorce and increasing acceptance of non-marital cohabitation, reflected increasing choice for domestic arrangements. No-fault divorce itself seems to have increased the prevalence of divorce in some states (Rodgers et al. 1997, 1999b). Therefore, holding all else constant, one would expect an increase, rather than a decrease, in the heritability of both marriage and divorce through the historical changes of the 1970's and 1980's in the U.S. This contrary evidence from the World War II Twin Registry suggests that, for any given age, lifespan development can eclipse secular changes in determining the relative importance of genetic and environmental factors to marriage propensity.

Whereas previous studies, including our own, have examined the general propensity *ever* to marry, and if married, *ever* to divorce, this study examines the time-constrained phenotype of *current marriage* to uncover sources of consistency and change in the biometric/genetic architecture of marriage over the lifespan. We consider current marriage, assessed at each decade point across the adult lifespan from age 20 to 70 in an attempt to answer two primary questions:

1. Do the relative contributions of genetic and environmental factors to phenotypic variance in current marriage change over time?
2. If so, how can we best characterize consistency and change in the specific sources of genetic and environmental variance as well as in their relative levels of influence on marriage propensity across the lifespan?

Method

Sample

The NAS-NRC World War II Veteran Twin Registry was initially ascertained through birth records from 40 states of live, Caucasian male multiple births during the years 1917–1927. These records were matched to names in Master Index of the Veterans Administration, 1958–1959, yielding a total of 15,924 pairs of twins in which both had served in the U.S. armed forces. In response to the initial contact in 1965, 8,747 complete twin pairs responded to the first of numerous questionnaires. By 1988, a total of 9,213 complete pairs from the initial 15,924 pairs were believed to have survived and to be living in the continental United States (Breitner et al. 1995). An initial follow-up in 1990–1991 revealed 1,235 additional deaths across 1,207 pairs and an additional 39 pairs with a twin or cotwin residing out of the U.S., leaving 7,967 pairs eligible for study (Breitner et al. 1995). Among those pairs, 1,575 individuals were not located and 1,650 individuals refused participation, so 12,709 participants or proxies representing 5,699 full pairs completed the Telephone Interview for Cognitive Status in 1990–1991 by which they were enrolled in the Duke Dementia Study (Breitner et al. 1995).

The current study uses follow-up data from this sample, collected from 1997 – 1999 through telephone interviews. At initial enrollment in the Duke Dementia Study, 1,311 of the 12,709 individuals had already lost their twin, with another 52 shortly thereafter losing a twin either to death or dementia. Between initial enrollment and the 1997–1999 follow-up interviews, another 2,649 individuals or their co-twins were deceased, 1,316 individuals refused participation, and 331 individuals or their proxies either could not be located or otherwise became unavailable for interview (Brenda Plassman, personal communication). The remaining participants in the 1997–1999 telephone interviews of the Duke Dementia Study numbered 7,050 individuals and/or their proxies ($n = 501$). Complete pairs numbered 2,976, of whom 1,424 pairs were monozygotic (MZ) and 1,367 pairs were dizygotic (DZ), with 185 pairs of unknown zygosity (UZ). The participants' twin status and age provide an unusual opportunity to examine genetically informative data about marriage patterns across several decades of the adult lifespan.

Measures

Telephone interviewers obtained complete marital and cohabitation history from participants' self-report and/or proxy report for septuagenarian and octogenarian participants. We ascertained all current and previous marriages and non-marital cohabitations, their beginning and end dates, and whether relationships terminated as a result of separation, divorce, or death, or, in the case of cohabitation, as a result of subsequent

marriage. In order to obtain the *current marriage* phenotype, we coded each individual's marital status as either married or not married at each decade point from 20 through 70 years of age. For any given age, varying numbers of pairs provided sufficient information to encode their marital status, but 1,266 MZ and 1,189 DZ full pairs provided sufficient information to encode their marital status as either married or not married at each of the six decade marks from age 20 to 70 years.

Analyses

We assumed that the dichotomous variable of marriage represents a multifactorial threshold trait for an underlying, continuous, normal distribution of liability. Therefore, we calculated polychoric correlations of twin-cotwin similarity for marriage at each decade from 20 through 70 years of age using PRELIS 2.54. We then fitted a standard, univariate, biometric model (Fig. 1) to MZ and DZ twin correlations for marriage at each decade, using LISREL 8.54 weighted least squares analysis and asymptotic weight matrices from PRELIS 2.54. Because MZ twins are genetically identical and DZ twins share, on average, half of their genes, correlations between latent additive genetic factors (A) were set at 1.0 and 0.5 for MZ and DZ pairs, respectively. For latent non-additive genetic influences (D), MZ twins are modeled as correlated at 1.0 and DZ twins at 0.25, the expected values under conditions of either genetic dominance or epistasis involving a single gene-by-gene interaction. By definition, latent shared environments (C) correlate perfectly (1.0), and evidence across a number of phenotypes supports the equal environments assumption, by which shared environmental effects are equivalent across zygosity (Kendler et al. 1993; Xian et al. 2000). All sources of discordance between MZ twins and all sources of non-genetic discordance between DZ twins are modeled in the latent nonshared environmental factor (E), which also includes all uncorrelated measurement error.

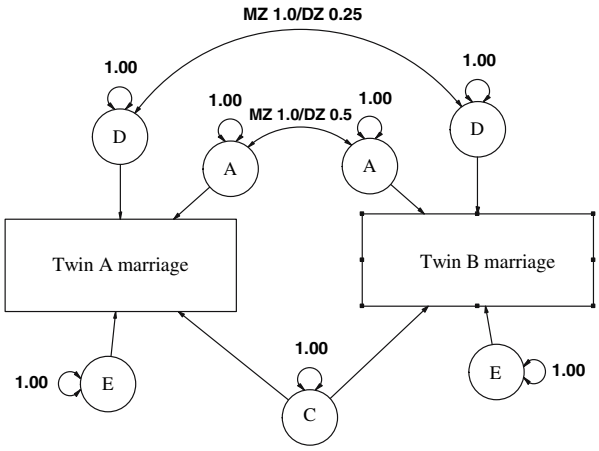

Fig. 1 Univariate biometric model of marriage at one time

When using a classic twin design in this way to partition the variance of a single phenotype, limited degrees of freedom prevent modeling of all four components of variance ($ACDE$) simultaneously. The ACE model is the standard model for twin studies and usually fits the data when the MZ twin correlations exceed DZ correlations. MZ correlations in excess of twice the DZ correlation, however, provide evidence consistent with non-additive genetic variation, and in those cases, the ADE model usually results in better fit than the ACE model. Of course, as ACE and ADE models are not nested, a direct comparison of fit is impossible, but when the ACE model produced an estimate of zero for c^2, the ADE model usually yielded non-zero parameter estimates and an equal or better overall fit. For the sake of parsimony, in all of our univariate and multivariate model fitting, we dropped any parameters LISREL estimated as equal to zero when dropping those parameters either improved or did not significantly worsen the fit of the model. Our goodness-of-fit criteria included change in chi-square ($\Delta\chi^2$) as a function of change in degrees of freedom (Δdf), the root mean square error of approximation (RMSEA), and Akaike's Information Criterion (AIC, Akaike 1987), which balances goodness-of-fit with parsimony.

We first tested for invariance in the genetic architecture of current marriage over time. We selected a standard ACE model and fitted it to the phenotype of current marriage at each of the six-decade points, constraining the parameter estimates for genetic and environmental contributions to marriage to be equal across all six occasions. We then created a model in which sources of genetic and environmental variance and their parameter estimates were free to vary by decade. To that end, we fitted ACE, ADE, AE, CE, and E models to the data for current marriage at each respective decade and created a composite model of the best fitting models for each respective decade point. We compared the fit of this composite model to that of the time invariant model to test whether the relative genetic and environmental contributions to current marriage were generally stable or if they changed significantly across the adult lifespan.

Following this initial test of lifespan invariance in the biometric architecture of current marriage, we fitted competing, longitudinal models of consistency

and change to the data. Our longitudinal models included three primary models and three composite models. The three primary models were a full Cholesky model, a common pathway model, and an independent pathways model. In each of the composite models, an independent pathway for one component (either A, C, or E) modified the general Cholesky model. For simplicity, we confined these longitudinal models to their ACE components, as the non-additive genetic variance term (D) is uninformative about the exact nature of any non-additive effects.

Our first (full Cholesky) model considers variation in current marriage at each time as the sum of additive genetic and of shared and nonshared environmental effects from each prior wave plus any new genetic and environmental influences. As Fig. 2 shows, the variable of current marriage at each decade loads on latent genetic and environmental factors specific to its decade, as well as on age-specific latent genetic and environmental factors from all previous decades. This saturated model suggests that new sources of genetic and environmental influences appear at each occasion of measurement, and that marriage at any given age may be predicted by the incremental accumulation over time of all prior genetic and environmental influences. (Maximum likelihood methods produce biased fit statistics for Cholesky models under certain conditions (Carey 2005), and appropriate corrections are still in development. Applications to weighted least squares methods have yet to be explored.)

Our second model, the common pathway model (Neale and Cardon 1993), tested how well a single endophenotype (Gottesman and Gould 2003) can account for variation in marriage across the lifespan. This model (Fig. 3) resembles a classic single-factor model. In it, current marriage at any age represents an expression of a general marriage factor, *M*, which is decomposed into its genetic and environmental sources of variance. This model also includes age-specific genetic and environmental factors to explain any residual, age-specific phenotypic variance in current marriage.

Our third, independent pathways, model (IP, Neale and Cardon 1993) deconstructs variation in current marriage into both constant and age-specific sources of variance. As seen in Fig. 4, the IP model does not assume a monolithic endophenotype over time but, instead, assumes a single factor across the lifespan for each of the genetic, shared environmental, and nonshared environmental influences on marriage, with different proportional levels of influence at each decade. This model also includes supplemental, age-specific factors to account for any residual, age-specific phenotypic variance attributable to latent genetic or environmental factors.

These three models all assumed that genetic and environmental influences on phenotypic variance would follow similar longitudinal patterns. However, genetic and environmental influences may behave differently over time, with some influences reflecting a more constant source of variation (as in an IP model) and others, the accrual of many time-specific sources of variation (as in a Cholesky model). Therefore, after testing the three general longitudinal models in which genetic and environmental factors were constrained to behave similarly, we modified the initial Cholesky

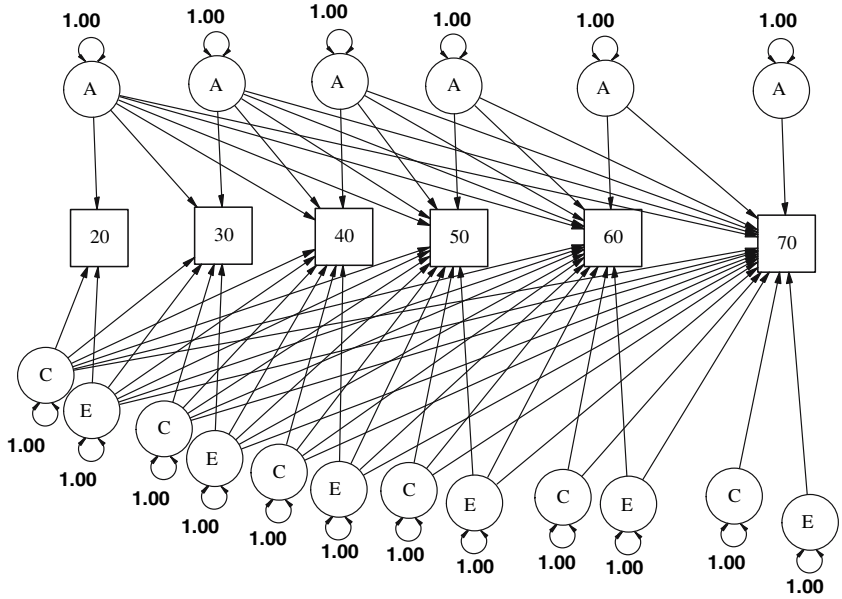

Fig. 2 Saturated Cholesky model of current marriage across the lifespan

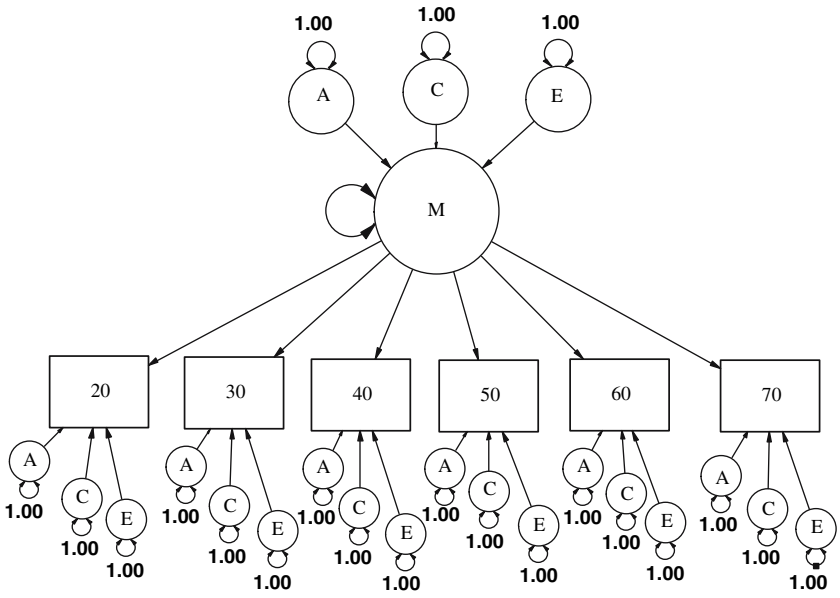

Fig. 3 Common pathway: Single factor model of current marriage endophenotype across the lifespan

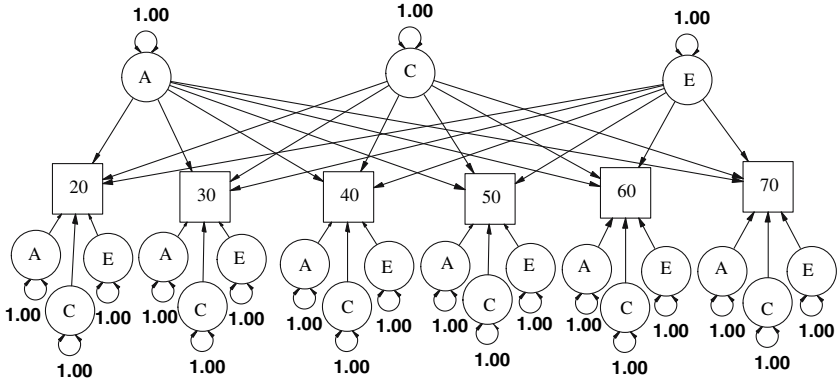

Fig. 4 Independent pathways model of current marriage across the lifespan

model by including one independent pathway submodel at a time for each one of the three (*ACE*) sources of variance (van Beijstervelt et al. 2003). This tested whether these three general sources of phenotypic variance showed different longitudinal patterns of influence.

Results

Univariate models

Relative levels of genetic and environmental influence on current marriage varied significantly across the decades. Compared to the baseline, time-constrained model (χ^2 = 3481.21, 153 df), the model in which components of variance and their loadings were free to vary by age and in which parameters estimated as zero were dropped fit the data significantly better (χ^2 = 196.48, 143 df; $\Delta\chi^2$ = 3284.73, Δ df = 10, $P < 0.001$). Table 1 presents parameter estimates and 95% confidence intervals for the best fitting univariate models of current marriage at each age. Broad heritability (h^2) of marital status increased from age 20 to age 30 and remained steady through age 40, with increasing frequency of marriage in the sample (from 8.3% to 87.6% to 92.3%). Genetic influences included non-additive as well as additive effects at ages 30 and 40 years. Then, at age 50, the heritability of marriage declined to near zero, despite a change of less than 1% in the frequency of marriage (92.0%) from the previous decade. This net shift of only 9 individuals from marriage to non-marriage among the complete pairs, however, represents an overall change in marital status in 295 individuals between the ages of 40 and 50 years. After age 50, variance in marital status attributable to genetic sources fell to zero, even though marriage rates remained fairly constant at age 60 (92.7%), and declined significantly only by age 70 (85.8%). Common environmental factors influenced the likelihood of

Table 1 MZ and DZ correlations for marriage, with best-fitting univariate biometric models of current marriage at each decade.

Married at age	Phenotypic correlations				Percentage of phenotypic variance attributable to:				
	MZ	*n* pairs	DZ	*n* pairs	a^2	d^2	c^2	e^2	h^2
20	0.569	1302	0.439	1217	0.26 (0.22, 0.30)	–	0.31 (0.28, 0.34)	0.43 (0.40, 0.45)	0.26
30	0.386	1301	0.142	1214	0.18 (0.13, 0.24)	0.20 (0.15, 0.31)	–	0.61 (0.58, 0.64)	0.38
40	0.383	1299	0.166	1209	0.27 (0.23, 0.30)	0.11 (0.08, 0.15)	–	0.62 (0.59, 0.65)	0.38
50	0.034	1298	0.021	1206	0.04 (0.03, 0.04)	–	–	0.96 (0.94, 0.99)	0.04
60	0.074	1298	0.239	1206	–	–	0.14 (0.13, 0.15)	0.86 (0.83, 0.89)	–
70	0.126	1270	0.218	1192	–	–	0.18 (0.17, 0.18)	0.82 (0.79, 0.85)	–

a^2 – additive genetic factors
d^2 – non-additive genetic factors
h^2 – broad heritability (total genetic factors)
c^2 – shared environmental factors
e^2 – non-shared environmental factors plus error variance

current marriage most strongly at age 20, but their effects declined to zero at ages 30 through 40 and emerged again at lower levels at ages 60 and 70. The proportion of variance in current marriage attributable to error and nonshared environment increased across the lifespan to age 50, and then declined slightly.

Multivariate models

Table 2 presents goodness-of-fit measures for the multivariate models.

Only one model fit the data better than the full Cholesky model. This model included a single independent pathway that accounted for genetic influences on current marriage across the full lifespan. In the best-fitting version of this model, only at age 30 were there any additional, age-specific genetic influences. All other original age-specific genetic pathways were estimated as equal to zero and dropped without any significant decrement in fit. This composite model and its parameter estimates appear in Fig. 5 and Table 3. It suggests that the same genetic factors contribute throughout the lifespan to variation in current marriage, but with different levels of effect relative to environmental factors. It also suggests influences of an additional genetic factor at age 30. The slight discrepancies between this model's estimates for parameters beyond age 50 and those of the univariate models probably reflects limited statistical power to differentiate genetic and common environmental effects when the phenotype is dichotomous and e^2 is high (Neale et al. 1994).

While sources of genetic influence remained relatively constant, sources of environmental influence on current marriage varied across the lifespan, as the full independent pathways model and the two Cholesky models with their respective independent environmental pathways did not fit the data as well as either the full Cholesky or the Cholesky modified with a single independent pathway for genetic influences. It also can be inferred, from the relatively poor fit of the common pathways model, that the propensity for marriage cannot be reduced to a single phenotype or endophenotype across the lifespan.

Discussion

Marriage across the lifespan does not appear to reflect a uniform phenotype but a heterogeneous one. Different individuals' varying patterns of marriage history are influenced by genetic and environmental factors that are, themselves, characterized by both continuity and change. Although the proportional influences of genetic and environmental factors on marriage varied, the source factor of genetic influences remained constant throughout adulthood with some additional genetic influence at age 30. Both the sources and levels

Table 2 Fit indices for multivariate models

Primary models	$\chi 2$	df	RMSEA	$\Delta\chi 2$ (from model 1)	Δdf	*P*	AIC
1. CH	115.58	108	0.007				–98.42
2. CP	468.73	137	0.044	353.15	30	<0.001	194.73
3. IP	236.59	127	0.027	121.01	19	<0.001	–5.43
4. IP A, CH CE	116.65	110	0.007	1.07	2	0.586	–103.35
5. IP C, CH AE	148.81	110	0.017	33.23	2	<0.001	–71.19
6. IP E, CH AC	193.10	114	0.023	77.52	12	<0.001	–34.90

CH – Cholesky
IP – Independent pathways

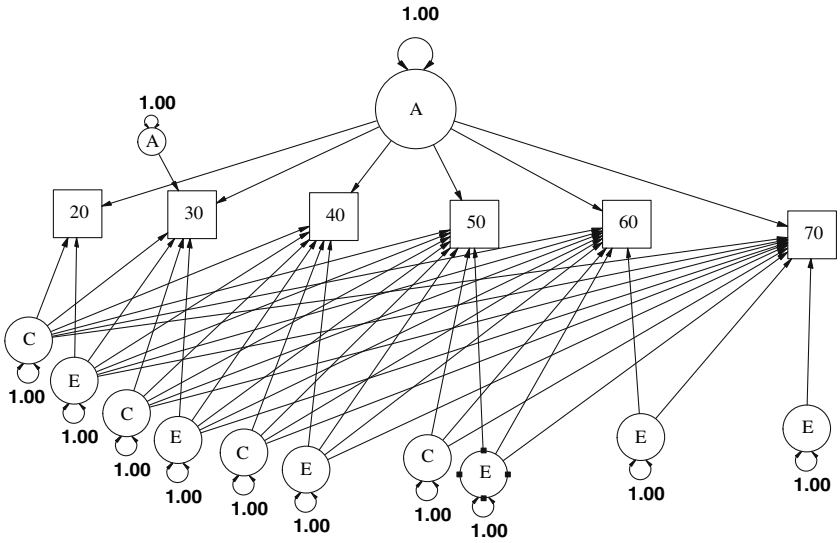

Fig. 5 Final, composite model of current marriage across the lifespan

Table 3 Parameter estimates for composite model (standard errors in parentheses)

Age	FAC A	A20	A30	A40	A50	A60	A70
20	0.58 (0.14)	–					
30	0.49 (0.16)		0.34 (0.30)				
40	0.50 (0.14)			–			
50	0.27 (0.19)				–		
60	0.31 (0.15)					–	
70	0.09 (0.14)						–
Age		C20	C30	C40	C50	C60	C70
20		0.50 (0.14)					
30		–0.04 (0.18)	0.19 (0.21)				
40		–0.12 (0.20)	–0.11 (0.45)	0.30 (0.32)			
50		–0.06 (0.20)	–0.24 (0.34)	0.16 (0.53)	0.27 (0.23)		
60		–0.04 (0.17)	–0.04 (0.44)	0.31 (0.29)	0.20 (0.22)	–	
70		–0.02 (0.15)	0.19 (0.38)	0.20 (0.40)	0.28 (0.39)	–	–
Age		E20	E30	E40	E50	E60	E70
20		0.63 (0.05)					
30		0.05 (0.09)	0.78 (0.04)				
40		–0.28 (0.12)	0.53 (0.09)	0.52 (0.12)			
50		–0.14 (0.12)	0.32 (0.09)	0.58 (0.12)	0.55 (0.10)		
60		–0.13 (0.08)	0.24 (0.07)	0.22 (0.12)	0.68 (0.12)	0.42 (0.18)	
70		–0.07 (0.07)	0.13 (0.06)	0.42 (0.12)	0.47 (0.12)	0.60 (0.20)	0.26 (0.45)

of environmental influence on current marriage changed over time. Environmental influences tended to accrue: sources of variance from earlier decades continued to contribute significantly to variation in marriage propensity at later ages.

Unfortunately, in this and in most samples, age and historical circumstance remain confounded. As we consider how best to interpret our results, we will consider historical context and demographics before exploring the implications of our findings for normative lifespan development of pair bonds, evolutionary adaptations of fertility-related behaviors, and individual differences in marriage-related phenotypes. We also will revisit the equal environments assumption of twin studies in the specific case of marriage-related phenotypes.

Historical context

Any observed changes or consistencies in relative influences of genetic and environmental factors on marital status over the lifespan are, by definition, embedded in their historical context. The World War II Veteran Twins, born from 1917 to 1927, varied greatly in the historical circumstances they faced at age 20. Some men reached age 20 during the Great

Depression; others, during WWII; and still others, after their return to post-war life. Twins within each pair faced the same historical incentives or disincentives to marry, and this may account for some shared environmental influences on marriage at age 20. However, by the time any men in the sample reached age 30, WWII had ended. The increased genetic variance in marriage at age 30 probably reflects, in part, the greater stability of the relevant post-war environment. The feasibility of marriage for this cohort probably varied less from 1947 to 1957 than from 1937 to 1947. In addition, the end of the war probably provided a first opportunity for marriage for much of this veteran sample and with increased opportunities to marry, the heritability of marriage increased (as Udry 1996, would predict).

From 1960 to 1985, however, as the World War II generation entered midlife, the percentage of non-institutionalized, married women participating in the civilian paid labor force expanded from 27.6 to 60.8% (Bureau of the Census 1999). This may have increased divorce risk, particularly for women in workplaces where men outnumbered women (Trent and South 2003). These workforce changes coincided with secular increases in divorce rates and, ultimately, with the institution of no-fault divorce. Although some of the initial increase in divorce rates with the passage of such legislation may have represented a backlog of individuals who would otherwise have divorced previously, evidence suggests an even greater increase in divorce rates following no-fault legislation (Rodgers et al. 1997, 1999b). The precipitous drop in observed genetic variance in marriage around age 50 may reflect, in part, the heterogeneity of participants' ages relative to the relaxation of restrictions on divorce in their respective home states.

Over this same historical period, divorce laws began to reflect greater gender equity. Early-life divorces in the World War II generation may have reflected husbands' phenotypes more strongly, with their wives' phenotypes more strongly reflected in mid- to late-life divorces. More recent questionnaire data show that wives, more often than husbands, initiate divorce proceedings (Amato and Previti 2003), so the decline in genetic influences on men's likelihood of marriage later in life might be an historical (and/or lifespan-related) shift in which spouse more often initiated divorce. Although this hypothesis is consistent with our results, an empirical test of it would require spousal information beyond the scope of our veteran-only data.

Demographics

The findings of other twin registries illustrate the importance of demographic as well as historical factors. Participants in the Vietnam Twin Registry (VET) averaged 38 years of age in 1987 when their marital status was assessed, very close to the mean age of the Minnesota sample. By doubling the difference between the MZ and DZ correlations, we obtain from the VET data heritability estimates forever having married of 0.32 and for having ever divorced of 0.42 (Beth Jerskey and Michael Lyons 2005, pers. comm.). The divorce estimate compares with that of the World War II sample but is lower than that of the largely civilian, Minnesota sample of similar age and cohort, probably reflecting a combination of important demographic and life experience differences between the samples. Forever marrying, the VET heritability estimates are lower than either the World War II or the Minnesota sample, also probably reflecting a combination of initial demographic selection into Vietnam era military service and the accrual of life experiences associated with Vietnam veteran status. Economically disadvantaged individuals were disproportionately represented among Vietnam veterans and, as economic disadvantage appears to depress the full expression of genetically associated cognitive abilities (Turkheimer et al. 2003), so too, poverty may prevent full expression of relevant marital phenotypes (Cherlin 2004). Consistent with Udry's (1996) view, economic disadvantage in this sample and the limitations it likely imposed on marriage were associated with lower heritability estimates for marriage.

A study based on an Australian twin sample, born from 1893 to 1965 and heterogeneous for age, cohort, and historical period, examined marital instability, defined broadly to include any dissolved non-marital cohabitations of six months or greater duration (D'Onofrio et al. 2005). The heterogeneous sample and phenotype, as well as the secular changes in norms for marriage, divorce, and cohabitation they represented, likely accounted for marital instability's low heritability ($h^2 = 0.15$; 95% confidence interval = 0.05–0.19) compared with estimates for marriage or divorce, more narrowly defined, from less heterogeneous samples.

Even though the WWII follow-up sample produced higher heritability estimates than the Australian sample for marriage-related phenotypes, demographic features of the veteran participants in the 1997–1999 telephone interviews including marital history suggest that the WWII data nevertheless produced underestimates of population-level genetic influences on

marriage over the lifespan. The veteran twins who survived and participated in telephone interviews were doubly selected. Military selection removed individuals who were most likely, through impairments in mental or physical health, either never to marry or, if married, to divorce. Participants in the WWII Twin Registry reported significantly higher rates of marriage and widowhood and significantly lower rates of never marrying or divorcing at midlife (1972 and 1985) than their counterparts in the general population (Trumbetta 1997). From that initial group, twins who survived with their cotwin until the late 1990's and participated in telephone interviews probably represent, on average, the healthiest members of this already healthy registry. Heritability estimates from these long time survivors are probably somewhat lower than would be obtained had detailed marital history data for the full, original registry been available. With this double selection for military service and for longevity, our estimates of lifetime genetic contributions to current marriage are almost certainly underestimates of genetic variance in the general population for marriage across the lifespan.

Lifespan development

The declining importance of common environmental factors relative to genetic factors from age 20 through 40 years is consistent with previous research on the sociology of marriage. To the degree that local and familial culture influence norms for age at first marriage (including attitudes toward premarital sex, out-of-wedlock births, and the independent residence of unmarried adult offspring), shared environmental factors would tend to influence variation in marriage at ages below the median for a first marriage, which, in this sample, was approximately 25 years. It also may be expected that, when marriage became normative for this population, the influences of shared environments would decline to near zero.

As the population's female: male sex ratio increases with age, and men's chances of marriage may become less dependent on genetically-influenced traits for which women might select at earlier ages, we would expect the heritability of marriage to peak around age 40 for men, with environmental relative to genetic influences on marriage increasing for the rest of the lifespan. This pattern of declining genetic influence is also consistent with the small, but increasing numbers of men who experienced the death of a spouse after age 40. The increasing mixture of widowhood with divorce in the aging sample contributes increasing heterogeneity to the current marriage phenotype. Although one might expect this to result in continuously increasing estimates for e^2 across the lifespan, the data instead show a leveling off of the combined effects of error and unique environments after age 50, which may represent a ceiling effect.

Evolutionary adaptations of fertility-related behaviors

Insofar as marriage or marriage-like alliances fulfill the evolutionary function of reproduction and safe rearing of offspring to maturity, the finding of genetic variation at the ages of peak family formation raises questions for evolutionary psychology. Fisher's Fundamental Theorem of Natural Selection suggests that as organisms, over the course of evolution, reach an asymptote of optimal adaptation, the heritability of fitness traits approaches zero (Fisher 1930). Several mechanisms, however, may continue to introduce genetic variance for fertility-related traits back into the population, including mutation, frequency-dependent selection, heterozygote advantage, and sexual antagonism (Rodgers et al. 2003, 2001). Perturbing forces also can increase the proportion of phenotypic variation attributable to genetic factors, including secular changes in norms for sexual attraction, marriage, cohabitation, contraception, and family size (Rodgers et al. 2001, 2003).

Insofar as a trait is adaptive, we expect it to show little variation due to genetic factors under these conditions of "purifying selection," but with conditions of "balancing selection," in which optimal adaptation requires genetic variety, more genetic variation for fertility related phenotypes may be observed (Hughes and Burleson 2000). For humans and for *Drosophila*, the heritability of most fitness traits averages around 20% (Hughes and Burleson 2000). Our findings of higher heritability for marriage at 30 and 40 years of age suggest that marriage may be more closely aligned with balancing selection and also may reflect more than a simple index of fitness. Even if purifying selection had more effect on marriage than hypothesized, marriage also may be one of those fertility-related traits under such strict societal regulation in the past that it precluded person-driven variety of expression, so that the loosening of that regulation has allowed the emergence of phenotypic variation (Rodgers et al. 2001). In addition, natural selection may act on different traits now than it did in our evolutionary history (Kirk et al. 2001).

In our current lifespan data, heritability for marriage seems to peak at the ages when marriage is most normative. Genetic influences on the propensity to

form pair bonds seem to be greatest at ages when reproduction and the rearing of young children occur, and it may be, that as a person ages, the ''drive'' to have children recedes, which may allow environmental influences to eclipse genetic ones (Trumbetta and Gottesman 2000). Highest genetic variation at times of peak family formation also may reflect variation in sexual behavior and/or mating strategies (Trumbetta and Gottesman 2000).

The WWII cohort came of age in an era of heterosexual marriage, so some of the genetic factors involved in sexual orientation may be implicated in our data. Although the evolutionary implications of sexual orientation variation in the population continue to be studied, recent evidence that relatives, and especially female maternal relatives, of male homosexuals show higher birth rates (Camperio-Ciani et al. 2004; King et al. 2005) suggests that somewhat different selective advantages for sexual orientation may operate at familial and individual levels.

Individual differences

Mediators of genetic influences on fitness-related behaviors may include attachment mechanisms, which have shown genetically influenced variation (Finkel et al. 1998). Although the specific biological mediators of genetic effects on attachment remain, in large part, unknown, animal models of pair bonding suggest that neuropeptide levels play an important role. Evidence of the effects of oxytocin and vasopressin (Young et al. 1998) and of dopamine (Edwards and Self 2006) on pair bonding in voles suggests consideration of the hypothesis that heritable variation in neuropeptides and neurotransmitters may account, in part, for individual differences in marriage propensity and, if neuropeptide levels and actions vary with development, they may contribute to age-related variability in the levels of genetic influence on marital status. Other potential mediators may be found in the system of mirror neurons that are required for the development of empathy and interpersonal relationships (Gallese 2003).

Fisher (1930) acknowledged that reproduction in humans follows different patterns than those of other organisms, depending less wholly on chance and varying with individual differences in temperament and disposition. Personality traits mediate some of the genetic influence on marital status (Jockin et al. 1996) and results from twin studies of personality are consistent with our findings. In a sample of 15,000 Finnish twins aged 18–53 years, Viken et al. (1994) found that the sources of genetic variation for Extraversion and Neuroticism remained consistent after ages 29 and 35, respectively, and that heritability estimates generally declined with age. These findings are consistent with our findings of the relatively stable sources of genetic variation and of the age-related decline in heritability of current marriage. Pedersen and Reynolds (1998) found similar levels of stability in the sources of genetic influences on personality traits in the Swedish Adoption/Twin Study of Aging. On the other hand, Loehlin and Martin (2001) followed twins who averaged 48, 56, and 62 years in a longitudinal study of the Australian Twin Registry and found no age-related changes in the genetic architecture of Eysenck's Psychoticism, Extraversion, or Neuroticism. Still, comparisons of cross-sectional studies of different age cohorts are consistent with a slight decline in the heritability of personality traits. Agreeableness and conscientiousness, two traits associated with marital adjustment (Bouchard et al. 1999), may show decreasing heritability, as genetic influences on agreeableness and conscientiousness accounted for .41 and .44 of the variance, respectively, for a sample with mean age of 31 years (Jang et al. 1996) and for only .12 and .29 of the variance in a sample with mean age of 58.6 years (Bergeman et al. 1993). Other traits associated with risk for early marriage, such as age at first intercourse (Dunne et al. 1997; Miller et al. 1999; Rodgers et al. 1999a) or sensation seeking (Zuckerman 2002) are also genetically influenced, and genetic factors associated with them may be expected to influence marriage before age 25, particularly in a sample that predated modern options for contraception.

Some genetic effects on marriage may be detectable in twin similarity of marital quality, usually considered protective against divorce. Intriguing, recent evidence showed that the degree of genetic relatedness of Swedish twin sisters accounted for the degree of similarity between their respective husbands' reports of marital satisfaction (Spotts et al. 2005). This study indicates the importance of spousal phenotype and underlying genotype to marital satisfaction and suggests another possible mediator of genetic influences on marriage and divorce.

The equal environments assumption in studies of marriage

For studies of marriage, the equal environments assumption, by which within-pair DZ twin environments are as similar to each other as within-pair MZ twin environments, must also apply to spouses, such that, within pairs, MZ spouses are no more similar than DZ spouses. This assumption seems warranted in light

of evidence across numerous traits (Lykken and Tellegen 1993). A recent study reported slightly higher resemblance of closest associates for MZ than for DZ twins (r = 0.22 and .14, respectively; Rushton and Bons 2005), but this very slight difference in the similarity of close associates emerged only when spouse and best friend measures were summed together, reinforcing the likelihood that spousal choice alone does not significantly violate the equal environments assumption. Furthermore, Rushton and Bons (2005) examined among their variables education, occupation, and income, all of which define the social stratum by which one's ''field of eligibles'' are generally constrained (Lykken and Tellegen 1993), so that respective spouses of any two unrelated individuals from the same social stratum also will bear a greater than chance resemblance to each other on these dimensions.

Conclusion

Although, as a dyadic phenotype, divorce initially might be thought to have a maximum heritability of 50% for either partner, the upper limit of heritability for divorce may be higher to the extent that partners share traits related to marital stability. Evidence of marital assortment for psychiatric disorders (Maes et al. 1998), and specifically, substance abuse and antisocial personality (Sakai et al. 2004; Vanyukov et al. 1994), as well as underlying tendencies toward psychopathology, as seen in the psychopathic deviate scale on the MMPI-2 (Trumbetta and Phillips 1998), suggest that individuals at high risk for divorce are more likely to select mates who have similar risk profiles. Even so, the estimate of 38% broad heritability for current marriage at ages 30 and 40, suggests that genetic influences on individual differences in marriage are substantial. The peak in heritability at the ages of peak family formation for this veteran twin sample suggests that marriage follows a pattern of balancing selection, and may reflect an adaptive mixture of evolutionary fitness strategies.

Our data indicate that marriage represents more than a single, complex phenotype, and yet, the heritability of marriage suggests a meaningful phenotypic construct. The changing state of marriage has led to explorations of broader phenotypes (D'Onofrio et al. 2005), which have shown less genetic influence. A strategy of more focused phenotyping may help to explore individual differences in the formation and course of pair-bonded relationships. In this and other samples, the effects of age, cohort, and historical period remain confounded, and future studies of multiple cohorts and cultures may help to disentangle some of these confounds.

Advances in quantitative methods and software will likely aid in future examinations of how genetic and environmental influences on marriage-related phenotypes may change over time. Survival analysis with large, genetically informative samples, which once required almost prohibitive amounts of computing time, has become increasingly feasible with faster processing technologies. New software development also will aid investigators in the increasingly sophisticated examination of the time-dependent expression of genetic and environmental influences on complex behavioral phenotypes.

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