Behav Genet (2016) 46:100–113
DOI 10.1007/s10519-015-9733-7

ORIGINAL RESEARCH

Widowhood and the Stability of Late Life Depressive Symptomatology in the Swedish Adoption Twin Study of Aging

Christopher R. Beam[1,3] · Robert E. Emery[1] · Chandra A. Reynolds[2] · Margaret Gatz[3,4] · Eric Turkheimer[1] · Nancy L. Pedersen[3,4]

Received: 30 March 2015 / Accepted: 1 August 2015 / Published online: 25 August 2015

Abstract Although the Swedish Adoption Twin of Aging (SATSA) has been used to investigate phenotypic stability of late life depressive symptoms, the biometric processes underlying this stability have not been studied. Under a reciprocal effects modeling framework, we used SATSA twins' Center for Epidemiologic Studies Depression (CES-D) Scale data across 5 waves (from 1987–2007) to test whether the reciprocal exchange between twins within a family and their nonshared environments (P<=>E) promote the accumulation of gene-environment correlation (*r*GE) over time. The model generates increasing *r*GE that produces subsequent stable environmental differences between twins within a family—a process hypothesized to explain stability in chronic late life depressive symptoms. Widowhood is included as a stressful life experience that may introduce an additional nonshared source of variability in CES-D scores. Genetic effects and nonshared environmental effects are primary sources of stability of late life depressive symptoms *without* evidence of underlying *r*GE processes. Additionally, widowhood explained stable differences in CES-D scores between twins within a family up to 3 years after spousal loss.

Keywords Depressive symptomatology · Late life depression · Reciprocal effects model · Gene-environment correlation · Nonshared environment

Introduction

Depressive symptoms have been found to increase across late adulthood (Alexopoulos 2005). The developmental processes leading to these increases, however, are not well understood. Published longitudinal twin studies of late life depressive symptomatology (hereafter, the term "depression" is used for simplicity) have shown that genetic factors primarily account for phenotypic stability over time, while nonshared environmental factors account for change (Carmelli et al. 2000; McGue and Christensen 2003; Neiss and Almeida 2004). These results are at odds with the hypothesis that depression results from the tendency for elderly people to increasingly select into environments that support their depression, a negative feedback loop thought to give rise to chronic depressive symptoms (Fiske et al. 2009). The aims of this paper are to propose and test a developmental model that shows how elderly people shape their environments to support increasing stability of depression (Beekman et al. 2002; Wetherell et al. 2001).

Dickens and Flynn's (2001) reciprocal effects model (REM) provides the theoretical framework for our study of the development of late life depression. The model is as follows: Based on genetically and environmentally influenced differences in phenotype (e.g., depressive traits or symptoms), people nonrandomly select environments that support the development of the phenotype. These environments, in turn, subsequently evoke further changes in phenotype, thereby supporting increased development. Over time, the mean effect of environmental factors

Electronic supplementary material The online version of this article (doi:10.1007/s10519-015-9733-7) contains supplementary material, which is available to authorized users.

✉ Christopher R. Beam
beamc@usc.edu

[1] University of Virginia, Charlottesville, VA, USA

[2] University of California, Riverside, CA, USA

[3] Department of Psychology, University of Southern California, 3620 South McClintock Avenue, Los Angeles, CA, USA

[4] Karolinska Institute, Stockholm, Sweden

increases, becoming a stable cause of phenotypic differences. One of the statistical consequences of their REM is the accumulation of gene-environment correlation, which is the nonrandom match between people and environments. Gene-environment correlation (*r*GE) is the hypothesized mechanism underlying environmental stabilization of late life depression (Dickens et al. 2011). The accumulation of *r*GE in the REM, thus, has an additional statistical consequence: increasing correlations across nonshared environmental factors over time.

The REM framework emphasizes that the conditions under which late life depression develop are not random. Thus, it is important to consider precipitant factors of depression that may contribute to the reciprocal exchange between the elderly and their environments. One of the most studied causes of late life depression is conjugal loss (Bruce 1980, 2002), as the widowed are thought to socially isolate themselves more so than the still-married (Blazer 2003). Becoming widowed, then, may be a negative life experience that may also maintain depression. We propose that some widowed elderly choose isolative environments after conjugal loss, causing the genetic and environmental factors underlying depression to become increasingly correlated with subsequent environmental factors. The overall process maintains depression across late life. Thus, an additional aim of the current paper is to test whether becoming widowed contributes to the reciprocal feedback loop that maintains depression.

Traditional longitudinal twin models

Traditional twin studies make the assumption that genetic and environmental factors operate independently of one another (Neale and Cardon 1992). Elsewhere, simulation studies have been used to show that ignoring *r*GE can misattribute environmental stability of development to genetic influences (Beam and Turkheimer 2013; Dickens and Flynn 2001). In one study of cognitive ability (de Kort et al. 2014), for example, more than half the genetic variance in stability of twins' cognitive scores was accounted for by underlying *r*GE processes.

The Dickens and Flynn (2001) version of the REM we propose to explain within-family environmental stabilization of depression is an extension of the basic genetic simplex model (Boomsma and Molenaar 1987; De Kort et al. 2014; Eaves et al. 1986). At the phenotypic level, depression develops via the transmission of a weighted (autoregressive) effect of the trait score at previous measurement occasions plus novel variation at the current measurement. Genetic simplex models allocate stability and change processes of phenotypic depression scores to genetic, common environmental, and nonshared environmental factors.

The genetic simplex model is represented in multilevel notation in Fig. 1. Between-family factors are those that are shared within a family but differ between families. Within-family factors are those that differ between twins in a family. The phenotype at the first occasion, D_{i1}, is a linear combination of the total between- and within-family genetic factors (the sum of effects shared by twins, A^b, and effects not shared by twins, A^w), a between-family (or common) environmental factor (E^b), and a within-family (nonshared) environmental factor (E^w). At occasions after the first occasion, $D_{it>1}$, twins' scores are an additive function of the weighted effects of previous genetic and environmental scores and unique genetic and environmental variation introduced at the current occasion (e.g., the equation for between-family environmental effects: $E^b_{ft} = c_{ARt,t-1}E^b_{ft-1} + uE^b_{ft}$). The genetic and environmental components reproduce the phenotypic developmental process under the assumption that genetic and environmental factors—and their transmission over time—operate independently of one another.

The P<=>E longitudinal twin model

The reciprocal effects (P<=>E) longitudinal twin model that we propose accommodates *r*GE, but violates the assumption that transmission of genetic and environmental effects occur independently of one another. In our model (Fig. 2), the nonshared environmental effect at time *t*, E^w_t, is regressed on the phenotype, D_{it-1}, at time $t-1$ (labeled as b_{PE}). Conceptually, individual twins influence their own environments, a feature that better represents actual development than the genetic simplex model. Twins with higher phenotypic depression scores, for example, are predicted to select more depressive environments, whereas twins with lower depression scores select less depressive environments. By tracing paths, E^w_t is correlated with previous within-family genetic and environmental variables at *t*-1 (A^w_{t-1} and E^w_{t-1}, respectively). Thus, if P<=>E effects were significant, *r*GE and nonshared environmental correlations would increase over time via phenotypic transmission (b_{PE}) of within-family differences.

The P<=>E parameter represents real developmental processes that induce within-family *r*GE to explain stable twin differences in depression. Within-family genetic and environmental variation underlying depression makes it more (or less) likely for individual twins to seek out depressive environments (known as active *r*GE) and for depressive environments to evoke depressive behaviors (known as evocative *r*GE) from them (Jaffee et al. 2013). Grouchier twins, thus, may develop late life depression more often than even-tempered twins because they are, in part, less desirable to be around and therefore less likely to

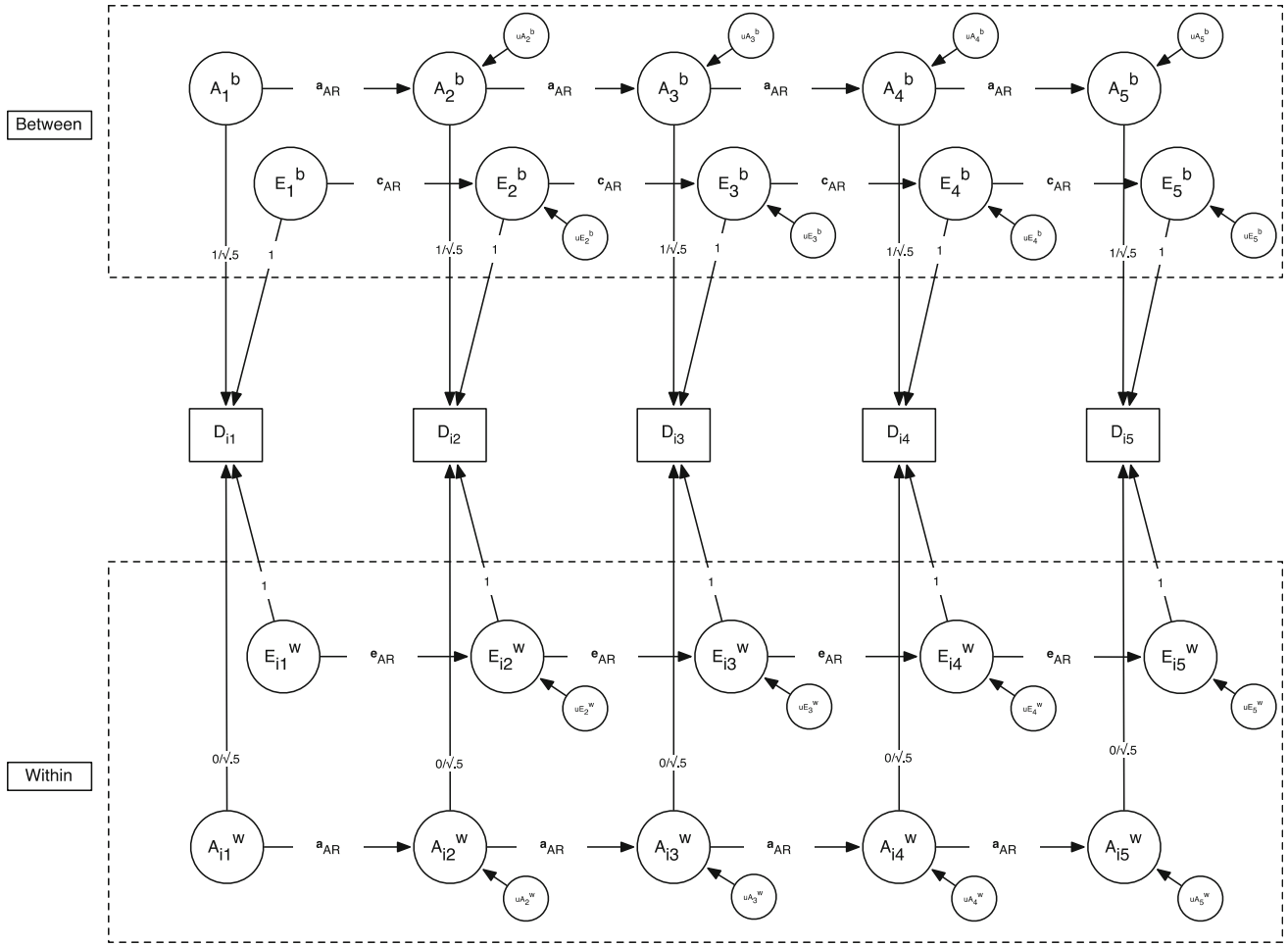

Fig. 1 The baseline longitudinal twin model. Biometric components of D_{it}, phenotypic CES-D scores for twin i at time t, are estimated between- and within-families; A_t^b = between-family genetic effect at time t; E_t^b = between-family (common) environmental effect at time t; A_t^w = within-family genetic effect at time t; E_t^w = within-family (nonshared) environmental effect at time t; uA_t^b = unique between-family genetic effect at time t; uE_t^b = unique between-family environmental effect at time t; uA_t^w = unique within-family genetic effect at time t; uE_t^w = unique within-family environmental effect at time t; a_{ar} = auto-regression coefficient between adjacent genetic components; c_{ar} = auto-regression coefficient between adjacent common environmental components; e_{ar} = auto-regression coefficient between adjacent nonshared environmental components; the between-family and within-family genetic loadings for the MZ twins are 1 and 0, respectively, to meet the assumption that MZ twins share 100 % of their genes; the between-family and within-family genetic loadings for the DZ twins are both $\sqrt{.5}$ to meet the assumption that DZ twins share 50 %, on average, of their segregating genes

be invited to social events. The initial genetic and environmental differences between them—transmitted via their phenotypes—leads to the expectation that grouchier twins are matched to progressively more isolated environments and consequently become more depressed.

Becoming widowed

As noted above, becoming widowed is an ideal precipitant factor to include in the REM of late life depression. First, twin and longitudinal studies have been used to increase knowledge on the causal influences of widowhood on depression (Bonanno et al. 2002; Lichtenstein et al. 1996; Osler et al. 2008). This body of work has shown that widowed twins are more likely to experience greater depression at follow-up than their still married co-twins. Second, a subset of widowed adults, moreover, remains at risk for chronic depression after conjugal loss (Galatzer-Levy and Bonanno 2012; Lee and DeMaris 2007)—symptoms that may not fully diminish over longer periods of time (Lucas et al. 2003). In the context of the reciprocal exchange between twins' depression scores and their nonshared environmental factors, we considered whether becoming widowed contributes to the accumulation of *r*GE and the maintenance of late life depression.

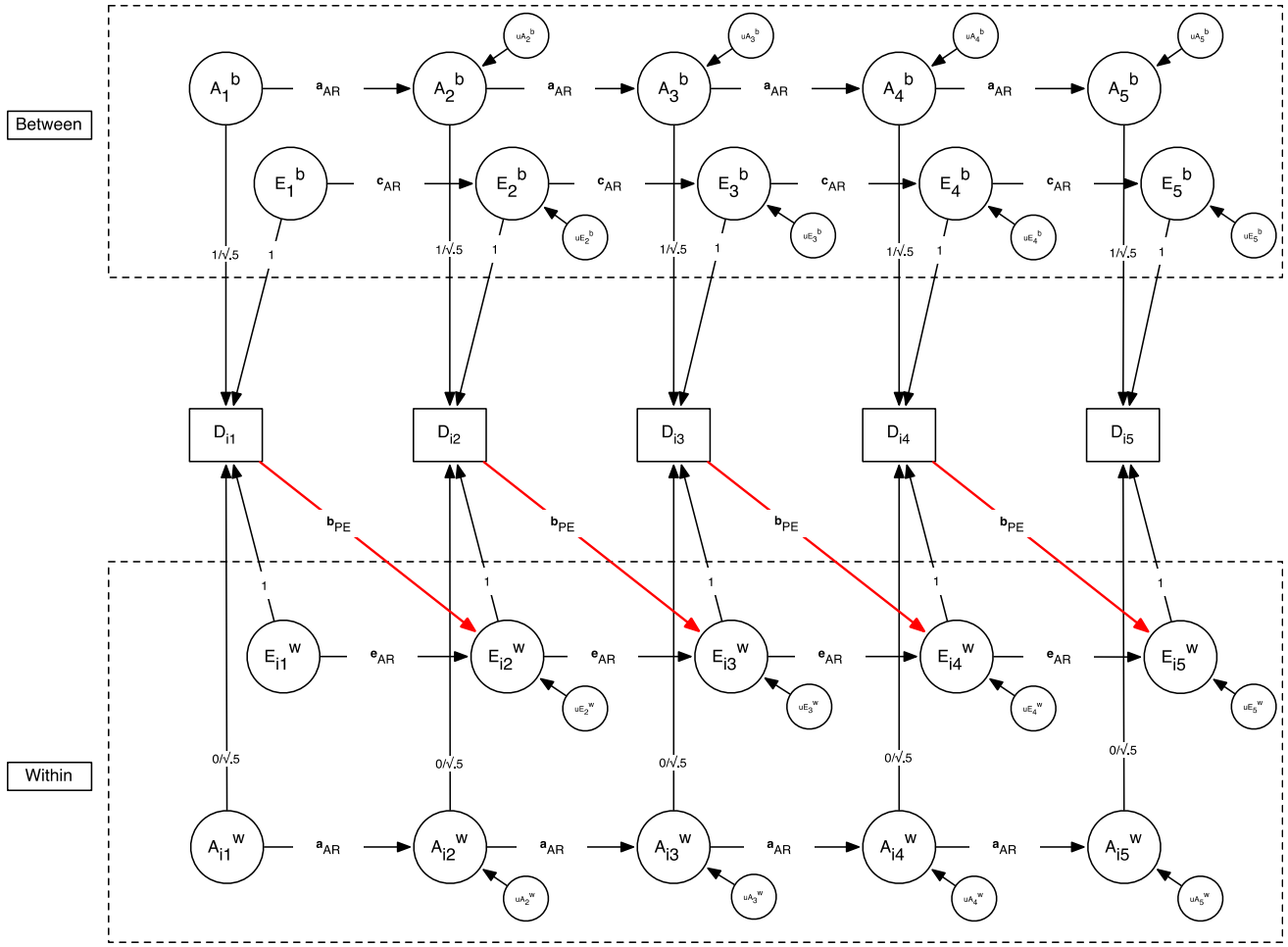

Fig. 2 The P<=>E longitudinal twin model. Biometric components of D_{it}, phenotypic CES-D scores for twin i at time t, are estimated between- and within-families; A_t^b = between-family genetic effect at time t; E_t^b = between-family (common) environmental effect at time t; A_t^w = within-family genetic effect at time t; E_t^w = within-family (nonshared) environmental effect at time t; uA_t^b = unique between-family genetic effect at time t; uE_t^b = unique between-family environmental effect at time t; uA_t^w = unique within-family genetic effect at time t; uE_t^w = unique within-family environmental effect at time t; a_{ar} = auto-regression coefficient between adjacent genetic components; c_{ar} = auto-regression coefficient between adjacent common environmental components; e_{ar} = auto-regression coefficient between adjacent nonshared environmental components. The thick *red line* represents that the P<=>E parameter, b_{PE}, was only estimated in the DZ group. The between-family and within-family genetic loadings for the MZ twins are 1 and 0, respectively, to meet the assumption that MZ twins share 100 % of their genes; the between-family and within-family genetic loadings for the DZ twins are both $\sqrt{.5}$ to meet the assumption that DZ twins share 50 %, on average, of their segregating genes (Color figure online)

The Swedish adoption twin study of aging

The Swedish Adoption Twin Study of Aging (SATSA) is a unique data source for studying developmental and psychosocial processes contributing to stability of late life depression (Finkel and Pedersen 2004; Fiske et al. 2003). First, the sample mainly compromises twins in mid- to late-adulthood followed (almost) every 3 years from 1984 to 2007, making SATSA the most intensive longitudinal twin study of the elderly. Second, the measurement window makes it possible to test whether P<=>E processes contribute to stable within-family environmental differences between twins (Dickens et al. 2011). Third, marital status was collected at each wave, so that the effects of becoming widowed on twins' subsequent depression scores can be tested.

Finally, widowhood is an ideal within-family difference variable to include as a predictor in the P<=>E longitudinal models, for the reason that the causal effects of becoming widowed on depression scores have already been supported in SATSA (Lichtenstein et al. 1996). In a co-twin-control analysis of twins discordant for widowhood status, widowed twins were found to have higher subsequent depression scores at 3-year follow-up than their still

married co-twins. The present study extends Lichtenstein et al.'s finding by including additional twin data collected since the publication of their results (data collected in 2004 and 2007), applying a fresh approach to consider environmental dynamics that perpetuate increasing depressive symptoms across time.

The purpose of the present study is to fill a gap in the late life depression literature by testing whether depression is increasingly stable in the elderly, and, if so, whether the reciprocal exchange between the elderly and their environments account for increasing stability of depression. The main hypothesis is that P<=>E effects produce accumulating within-family *r*GE in twins that, in turn, cause stable within-family environmental differences in late life depression (Blazer 2003; Bruce 2002). The longitudinal P<=>E model was compared to traditional longitudinal twin models (Eaves et al. 1986) that assume independence among genetic and environmental factors over time. We also provide an update to Lichtenstein et al.'s (1996) SATSA finding that becoming widowed predicts higher subsequent depression scores in widowed twins using additional data.

Method

Sample

The Swedish Adoption/Twin Study of Aging (Finkel and Pedersen 2004) is a longitudinal gerontological twin study designed to assess physical and psychological health in a representative sample of middle- and late-aged Swedish adults. The questionnaire-based component of SATSA began in 1984, with follow-up measures sent to all still living twins every three years until 1993. Two additional follow-up questionnaires were sent to still living twins in 2004 and 2007. Marital status information was collected at 6 waves (1984, 1987, 1990, 1993, 2004, and 2007) and depressive symptom measures were administered at the last 5 of these waves. The overall number of twin pairs from which the current study sample was drawn was 1,919 twin pairs. The sample size used in current study was based on the number of families who responded to the first questionnaire in 1984 and consisted of 1,277 families (MZMis 190, DZMis 350, MZFis 226, DZFis 511), with a total of 2479 individual twins. The multilevel approach used here permitted inclusion of twin families where only 1 twin had available data for at least 1 wave.

The age range of the sample in 1984 was 26 to 93 years ($M_{1984}$is 61.20 (*SD*is 14.02). The mean age of the twins at each wave used in the current study are provided in Table 1. The discrepancy between the increase in mean age over the measurement window and interval of time between waves reflects the higher retention of younger twins and attrition among older twins. Mortality was a major cause of attrition across the 20-year measurement window. In 1987, 3.10 % (n = 119) of the total sample was deceased, with increases at each follow-up wave: 7.22 % (n = 277) in 1990, 10.34 % (n = 397) in 1993, 26.73 % (n = 1026) in 2004, and 30.41 % (n = 1167) in 2007.

Table 1 Means and standard deviations of CES-D scale scores and percentage of widowed twins at each wave of measurement

Wave	Age		CES-D scale score		Widowed
	Mean	*SD*	Mean	*SD*	%
1987	63.32	13.80	11.38	8.75	3.59
1990	64.63	12.96	11.61	8.46	2.94
1993	66.35	13.03	12.07	8.97	3.07
2004	71.44	11.41	12.46	8.45	3.19
2007	72.98	10.73	12.65	8.83	1.77

Measures

Depressive symptomatology was measured using the Center for Epidemiologic Studies-Depression (CES-D) scale (Radloff, 1977). Twins rated how often they experienced each symptom over the past 7 days on a scale of 0–3, with 0 = *rarely or none of the time (less than 1 day),* 1 = *some or a little of the time (1–2 days),* 2 = *occasionally or a moderate amount of time (3–4 days); and* 3 = *most or all of the time (5–7 days).* The reliability and validity of the CES-D scale has been well-studied, with 4 facets typically found in factor analyses: depressed affect, lack of well-being, somatic and retarded activity, and interpersonal difficulties (Hertzog et al. 1990; Shafer 2006). The log of the sum of all 20-items was used at each wave to normalize the distributions, as the raw scale scores were positively skewed. The test–retest reliability across all five measurements was substantial (McDonald's ω is 0.85; McDonald1999).

Table 1 presents the means and standard deviations of the CES-D scale scores over the 5 waves. Across all twins, CES-D scores appear to increase by about 1.3 units over the 20-year measurement window, with means below the threshold for clinically significant depressive symptoms (<16 units; Radloff 1977). Despite the slight increase in means over time, the longitudinal trajectory plots of the twins' scores (not presented) were consistent with an autoregressive process rather than a linear growth process. At the individual level, twins who reported higher scores at one wave generally reported lower scores at the subsequent wave (and vice versa).

The *widowhood* variable is a binary coded variable that was operationalized as a change from being married at time t (e.g., 1987) to widowhood status at time $t + 1$ (e.g.,

1990). Twins who became widowed between any two waves were coded 1. Twins who reported the same marital status or were missing at either of the adjacent waves were coded 0. All twins were, thus, coded either 0 or 1 at every wave. Twins were only given the "becoming widowed" code once during the study period, regardless of whether they remarried and became widowed a second time during the study. For example, a twin who became widowed between 1987 and 1990, remarried by 1993, and was widowed a second time between 2004 and 2007. As noted above, widowhood was treated as a within-family variable in the longitudinal multilevel analyses. Across all waves, 14.56 % of the individual twins (Nis 2479) became widowed at some point between 1984 and 2007. The percentages of twins who became widowed between adjacent waves (e.g., between 1984 and 1987, between 1987 and 1990, and so on and so forth) are given in the rightmost column of Table 1 and sum to the total percentage across all waves. The widowhood covariates were included in all multivariate analyses.

Data analysis

A mixed-effects variance components modeling approach was used to analyze the SATSA twins' CES-D data, as described by McArdle and Prescott (2005). Genetic, common environmental, and nonshared environmental factors are estimated between-families and within-families, with the traditional three assumptions made in conventional twin designs (Neale and Cardon 1992): First, monozygotic (MZ) twins share 100 % of their genes whereas dizygotic (DZ) twins share 50 % of their genes, on average. In multilevel models, zygosity differences determine the magnitude of genetic variability ascribed to the between-family level (factors shared by twins) and the within-family level (factors not shared by twins). Second, common environmental factors refer to any environment (e.g., parental socioeconomic status, rearing environment) that makes twins from the same family more similar to one another and are modeled at the between-family level. Third, nonshared environment factors refer to any environment that makes twins different from one another, including measurement error, and are modeled at the within-family level.

A note about the structure of SATSA twin data is in order at this point. SATSA contains families with pairs of twins who were reared together as well as reared apart from one another. For this reason, common environmental factors are not assumed in groups of twins reared apart from one another (that is, the factors are set to zero). In the current study, however, there were too few widowed twins to conduct a multigroup model that adjusts for rearing status between twin groups. As a result, twins reared together and reared apart were pooled within their respective zygosity groups. This decision did not appear to impact the final multivariate structural equation models, as all common environmental factors ultimately were removed from the models, which we explain below.

Based on the three twin assumptions above, twins' phenotypic CES-D scores are decomposed into the following random variance components (subscript 1 for twin 1 and subscript 2 for twin 2):

$$CESD_{ft,1} = b_{0t} + w_{ab}A^b_{ft} + E^b_{ft} + w_{aw}A^w_{ft,1} + E^w_{ft,1},$$

and

$$CESD_{ft,2} = b_{0t} + w_{ab}A^b_{ft} + E^b_{ft} + w_{aw}A^w_{ft,2} + E^w_{ft,2}.$$

The CES-D scores of twin 1 and twin 2 in family, f, at wave, t, are decomposed into between-family and within-family genetic and environmental factors. All biometric regressors have a mean of zero so that the expectation of the phenotypic mean $E[CESD]_{ft,i}$ equals the intercept, b_{0t}. At the between-family level, a genetic factor (A^b) and a common environmental factor (E^b) shared by both twins are estimated. At the within-family level, a genetic factor unique to twins raised in the same family (A^w) and a nonshared environmental factor unique to twins raised in the same family (E^w) are estimated. The total genetic effect, A, for each twin is equal to:

$$A_{ft,1} = w_{ab}A^b_{ft} + w_{aw}A^w_{ft,1},$$

and

$$A_{ft,2} = w_{ab}A^b_{ft} + w_{aw}A^w_{ft,2}.$$

The variances of the A^b and A^w factors are constrained to be equal. The weights, w, are fixed values that indicate the proportion of genetic information shared by each twin pair. In MZ pairs, w_{ab} equals 1 and w_{aw} equals 0 to satisfy the assumption that identical twins share all of their genes. In DZ pairs, w_{ab} equals 0.5 and w_{aw} equals 0.5 to satisfy the assumption that fraternal pairs of twins share only half of their segregating genes, on average; the other half varies between them. The weights of the genetic factors between- and within-families were scaled so that $w^2_{ab} + w^2_{aw} = 1$, with the expectation that A^b and A^w are uncorrelated, $E\left[A^b_{ft}, A^w_{ft,i}\right] = 0$.

The *baseline longitudinal twin model* was initially fit to the twins' data and is a standard genetic simplex model (de Kort et al. 2014; Dolan et al. 2014; Eaves et al. 1986) that fits autoregressive pathways between adjacent waves for the genetic and environmental factors (as depicted in Fig. 1). The between- and within-family genetic and

environmental factors are correlated via first-order autoregressions ($t > 1$):

$$A^b_{ft} = a_{ARt,t-1}A^b_{ft-1} + uA^b_{ft}$$

and

$$E^b_{ft} = c_{ARt,t-1}E^b_{ft-1} + uE^b_{ft}$$

and

$$A^w_{ft,i} = a_{ARt,t-1}A^w_{ft-1,i} + uA^w_{ft,i}$$

and

$$E^w_{ft,i} = e_{ARt,t-1}E^w_{ft-1,i} + uE^w_{ft,i}.$$

The autoregressive coefficients ($a_{ARt,t-1}$, $c_{ARt,t-1}$, and $e_{ARt,t-1}$) and the disturbances (uA^b_{ft}, uE^b_{ft}, $uA^w_{ft,i}$, and $uE^w_{ft,i}$) were freely estimated in the baseline model. The first-order autoregressive processes between the genetic and environmental factors function independently and are believed to represent the phenotypic (i.e., observable) autoregressive process. The variances also were freely estimated at t is 1. The A^w autoregressive coefficients were not estimated in the MZ group, as there is no within-family genetic

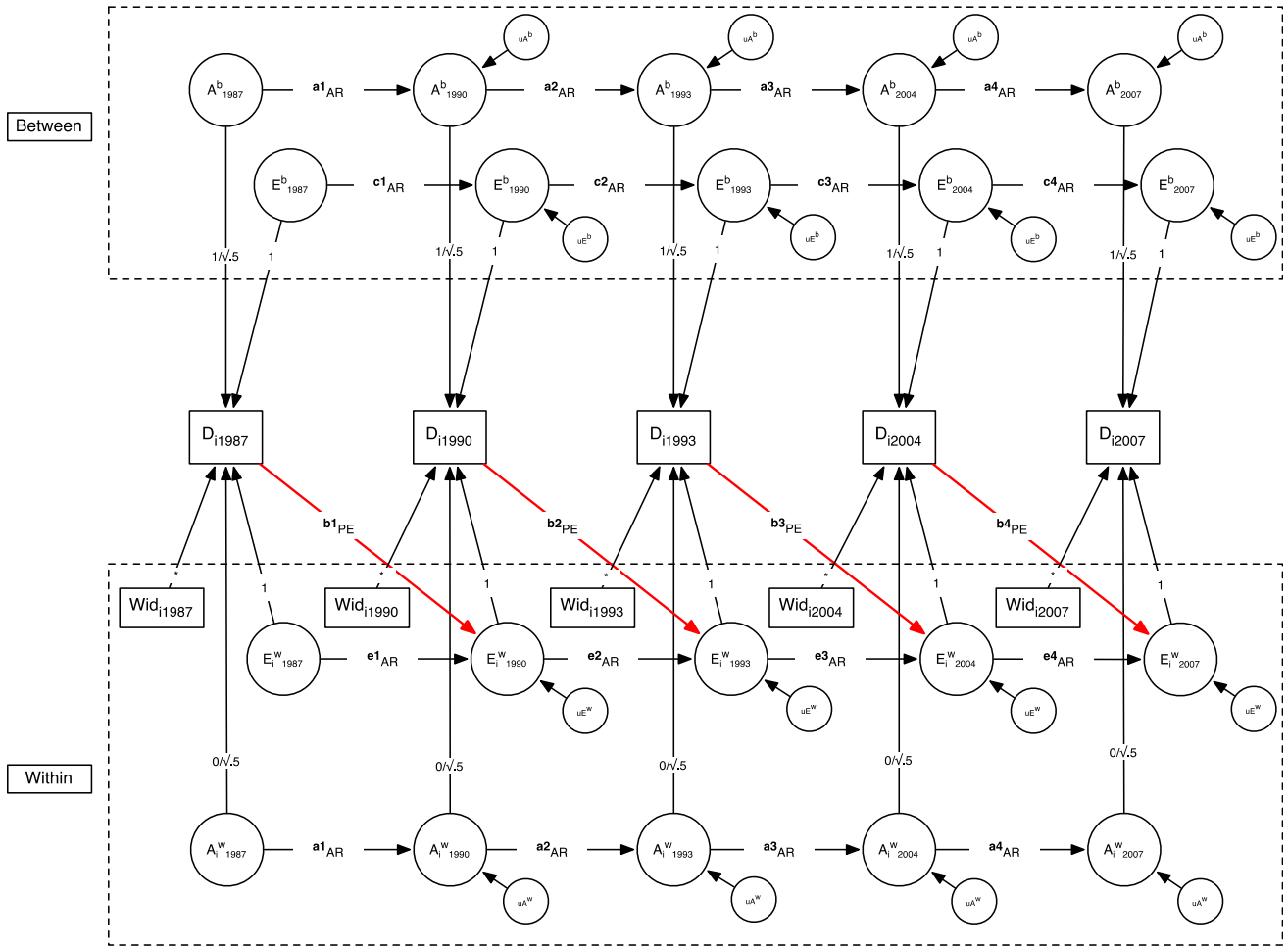

Fig. 3 SATSA P<=>E longitudinal twin model with widowhood covariates. Biometric components of D_{it}, phenotypic CES-D scores for twin i at wave t, are estimated between- and within-families; A^b_t = between-family genetic effect at time t; E^b_t = between-family (common) environmental effect at wave t; A^w_t = within-family genetic effect at wave t; E^w_t = within-family (nonshared) environmental effect at wave t; uA^b_t = unique between-family genetic effect at wave t; uE^b_t = unique between-family environmental effect at wave t; uA^w_t = unique within-family genetic effect at wave t; uE^w_t = unique within-family environmental effect at wave t; a_{ar} = auto-regression coefficient between adjacent genetic components; c_{ar} = auto-regression coefficient between adjacent common environmental components; e_{ar} = auto-regression coefficient between adjacent nonshared environmental components; Wid_{it} = the within-family effect of twin i's widowhood status on CES-D score at wave t. The *red lines* represent the P<=>E parameters, b_{PE}, and were estimated separately between adjacent waves only in the DZ group. The between-family and within-family genetic loadings for the MZ twins are 1 and 0, respectively, to meet the assumption that MZ twins share 100 % of their genes; the between-family and within-family genetic loadings for the DZ twins are both $\sqrt{.5}$ to meet the assumption that DZ twins share 50 %, on average, of their segregating genes (Color figure online)

variation. The five widowhood covariates were included in the baseline model (as portrayed in Fig. 3).

Figure 3 depicts the *P<=>E longitudinal twin model* with the five widowhood covariates. The red paths in the model represent the reciprocal exchange between twins and their nonshared environments and induce within-family *r*GE. Whereas in other P<=>E models (De Kort et al. 2014), the b_{PE} regression coefficients represent the relation between twins' total phenotypic score and their subsequent nonshared environments, this model assumes that environmental selection at wave t based on past behavior at wave t-1 only occurs within-families:

$$E^{w}_{ft,i} = b_{PEt,t-1} CESD^{w}_{t-1} + uE^{w}_{ft,i}$$

The autoregressive coefficients ($b_{PEt,t-1}$) and disturbances ($uE^{w}_{ft,i}$) were freely estimated in the baseline model. The effect was only estimated in the DZ group (indicated by the thick red colored pathways). In the MZ group, the P<=>E autoregressive process is the same as the nonshared environmental autoregressive process depicted in Fig. 1 for the reason that there is no within-family genetic variation. Thus, the P<=>E autoregressive coefficients $b_{PEt,t-1}$ are redundant with the nonshared environmental auto-regressive coefficients $e_{ARt,t-1}$. Nonshared environmental stability, therefore, is captured by constraining the nonshared environmental factors and autoregressions to be the same between the MZ and DZ groups, but the P<=>E process is *only* estimated in the DZ group.

In the DZ group, the nonshared environmental variables, $E^{w}_{ft,i}$ at wave $t > 1$, are regressed on the previous nonshared environmental factor and phenotype at wave $t - 1$:

$$E^{w}_{ft,i} = e_{ARt,t-1} E^{w}_{ft-1,i} + b_{PEt,t-1} CESD^{w}_{t-1} + uE^{w}_{ft,i}$$

The nonshared environmental auto-regressive coefficients, $e_{ARt,t-1}$, are the same as in the MZ group, as are the time specific nonshared environmental disturbances (residual), $uE^{w}_{ft,i}$. The P<=>E parameter, $b_{PEt,t-1}$, represents the transmission of DZ twin i's previous within-family phenotypic score to DZ twin i's subsequent nonshared environmental score. By path tracing rules, the P<=>E parameter necessarily transmits effects of the within-family genetic factor ($A^{w}_{ft-1,i} * b_{PEt,t-1}$) and the nonshared environmental factor ($E^{w}_{ft-1,i} * b_{PEt,t-1}$) at wave $t - 1$ via the phenotype at wave $t - 1$.

The P<=>E parameter has three consequences in the DZ group. First, it necessarily induces accumulating within-family *r*GE. Second, it changes the meaning of the nonshared environmental effects when $t > 1$ for the reason that the parameter correlates $E^{w}_{ft,i}$ with $A^{w}_{ft-1,i}$ (Dolan et al. 2014). Third, the P<=>E parameter increases the stability of nonshared environment over time as twins are matched to environments well-suited to their phenotypic depressive symptoms.

Finally, the becoming widowed covariates also were included in the model to test the psychosocial hypothesis that conjugal loss contributes to stability of late life depression, above and beyond the P<=>E process. Widowhood effects were tested based on the interval of time between measurement occasions. In SATSA, short-term effects were defined as becoming widowed within a 3-year period prior to follow-up (e.g., between 1987 and 1990) whereas long-term effects were defined as the 11-year period prior to fellow-up (i.e., between 1993 and 2004). The effects were operationalized in this way because precise dates of bereavement were not recorded in SATSA. Additionally, in all multivariate models, twins' CES-D scores at all 5 waves were regressed on their age at recruitment into the study (1984) to take into account age heterogeneity at each wave of measurement.

Missingness due to attrition and dropout is an issue in all longitudinal studies. For our purposes, missing data were assumed to be missing at random (MAR) and full-information maximum likelihood estimation with robust standard errors (MLR) was used to handle missingness (Muthén and Kaplan 1985; Raykov 2005). The CES-D data here were not analyzed thoroughly for missingness patterns, as the primary objective in this study was to demonstrate how P<=>E effects can be fit into longitudinal twin models to test whether *r*GE processes contribute to twin differentiation in late adulthood.

Exploratory data analyses were conducted in R 3.1.2 (R Core Team 2014). Multivariate mixed effects structural equation models were estimated in M*plus* 7.3 (Muthén and Muthén 2014). The Satorra–Bentler scaled Chi-square difference test was used to calculate a Chi-square distributed test statistic to compare nested models (Satorra and Bentler 2001). Additionally, the root mean square error of approximation (RMSEA) was used to evaluate model fit (Browne and Cudeck 1992). Models with RMSEA values below 0.05 are considered to fit the data well, and models with values below 0.08 are considered to fit the data adequately. The Akaike Information Criterion (AIC) and Bayesian information criterion (BIC) also were used to evaluate model fit (Burnham and Anderson 2004). Both the AIC and BIC indexes are computed to balance model parsimony and model complexity (Kline 2005). Lower values indicate better model fit to the data.

Results

Table 2 presents the longitudinal correlations among the CES-D and widowhood variables. The lag-1 correlations among the CES-D scores suggest high stability across

Table 2 Longitudinal correlations among CES-D and widowhood variables

	$CES\text{-}D_{1987}$	$CES\text{-}D_{1990}$	$CES\text{-}D_{1993}$	$CES\text{-}D_{2004}$	$CES\text{-}D_{2007}$	Wid_{1987}	Wid_{1990}	Wid_{1993}	Wid_{2004}	Wid_{2007}
$CES\text{-}D_{1987}$	1.00									
$CES\text{-}D_{1990}$	0.64	1.00								
$CES\text{-}D_{1993}$	0.60	0.63	1.00							
$CES\text{-}D_{2004}$	0.41	0.47	0.50	1.00						
$CES\text{-}D_{2007}$	0.44	0.47	0.48	0.63	1.00					
Wid_{1987}	0.15	0.11	0.09	0.04	0.01	1.00				
Wid_{1990}	0.05	0.17	0.03	0.16	−0.03	−0.05	1.00			
Wid_{1993}	0.03	0.12	0.12	0.02	0.02	−0.06	−0.05	1.00		
Wid_{2004}	0.07	0.10	0.18	0.19	0.19	−0.07	−0.05	−0.08	1.00	
Wid_{2007}	−0.05	−0.04	0.00	0.13	0.11	−0.05	−0.02	−0.06	−0.10	1.00

Table 3 MZ and DZ intraclass correlation coefficients

	$CES\text{-}D_{1987}$	$CES\text{-}D_{1990}$	$CES\text{-}D_{1993}$	$CES\text{-}D_{2004}$	$CES\text{-}D_{2007}$
ICC_{MZ}	0.26	0.36	0.25	0.36	0.43
ICC_{DZ}	0.15	0.14	0.13	0.27	0.26

Coefficients estimated using M*plus* 7.3 (Muthen and Muthen 1998–2012) sample statistics output function

waves. Unsurprisingly, the correlation is lower at the 11-year interval between waves 3 and 4. At higher order lags (i.e., 2, 3, and 4), the correlations tend to decrease, but do remain stable, consistent with an auto-regressive process (Guttman 1954). The correlations of interest between CES-D scores and widowhood are the wave concurrent correlations (e.g., time 1). These correlations ranged between 0.1–0.2, with the largest correlation at wave 4, that is, the interval in which the largest proportion of twins experienced conjugal loss.

Table 3 presents the MZ and DZ intraclass (twin) correlation coefficients. Across all waves, the MZ correlations were greater than the DZ correlations. This pattern of difference indicates that underlying genetic factors partly explain individual variation in CES-D scores at each wave. The MZ twin correlations, however, were not twice as great as the DZ twin correlations at all waves of measurement, which suggests that common environmental effects also may account for variation in CES-D scores at some waves. Common environmental factors may be an artifact of the SATSA subsample analyzed in this study, as twins of this age likely have lived independently of one another for quite awhile (McGue and Christensen 2013). Alternatively, the twins may have adopted a particular set of control beliefs taught to them during childhood, for example, that were well-developed by adulthood and that similarly influenced their likelihood of becoming depressed. The modest MZ twin correlations suggest that

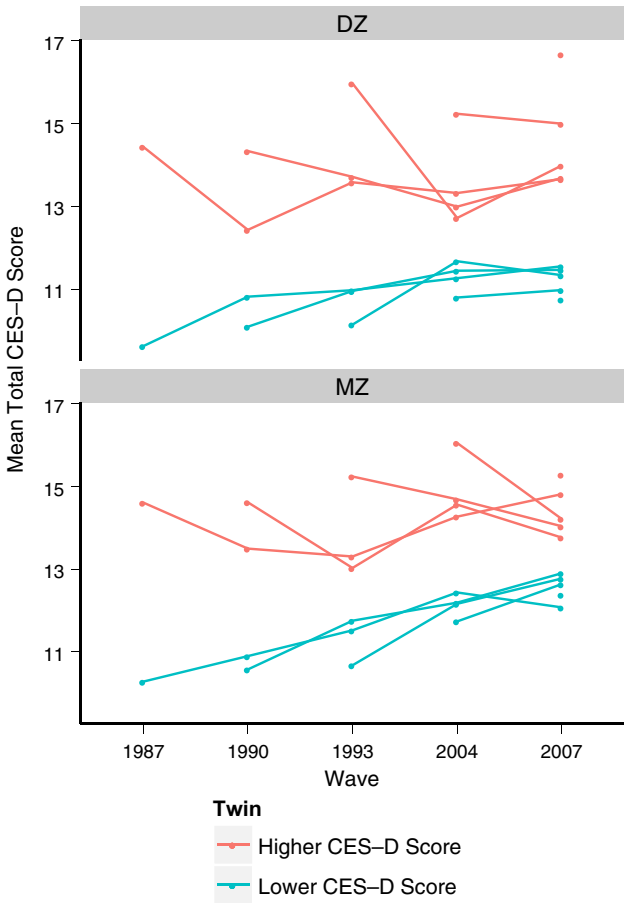

Fig. 4 Stability of within-family differences in DZ and MZ twins' total CES-D scores. At each wave, *t*, twins were rank ordered from low to high CES-D scores within their families. Means at each wave $\geq t$ were calculated for twins with lower and higher scores, and then plotted. The procedure was repeated until all *t* were exhausted (Color figure online)

nonshared environmental factors primarily explain twin differences in CES-D scores across all waves.

Next we examined the stability of within-family differences in MZ and DZ twins' CES-D scores over the five waves. At each wave, *t*, twins were rank ordered by their

Table 4 Model fit of longitudinal twin models

Model	χ^2	df	Model comparison	LR	Δdf	S–B LR test	RMSEA	AIC	BIC
1. Baseline AE simplex	296.18	129	–	–		–	0.03	21,751.06	22,012.77
2. Common A and unique A components	290.55	132	1	−6.83	3	–	0.03	21,738.93	21,983.18
3. Common A component only	302.53	137	2	11.83	5	0.037	0.03	21,742.95	21,958.13
4. Common E and unique E components (no simplex E)	286.21	135	2	0.74	3	0.863	0.03	21,734.33	21,961.14
5. Common E and simplex E	228.51	131	4	42.01	4	0.000	0.03	21,681.40	21,931.47
6. Common E and constrained simplex	232.75	133	5	4.11	2	0.128	0.03	21,682.31	21,920.75
7. P<=>E effects	231.71	129	7	2.79	4	0.594	0.03	21,686.43	21,948.14
8. Short-term widowhood effects constrained	233.59	136	7	0.16	3	0.984	0.02	21,676.43	21,897.42

All models adjust for the effects of age on CES-D scores at all 5 waves at the between-family level

A genetic component; *E* nonshared environmental component; *P<=>E* represents the phenotype-environment transmission coefficient; *S–B LR* Satorra–Bentler likelihood ratio difference test for nested models; *RMSEA* root mean square error of approximation; *AIC* akaike information criterion test; *BIC* Bayesian information criterion test

CES-D scores within their families so that means for twins who reported *higher* and *lower* scores at waves $\geq t$ could be calculated. For example, MZ and DZ twins were rank ordered according to their within-family CES-D scores at Wave 1. Means across all waves (Waves 1–5) for twins higher and lower on CES-D scores were calculated and plotted (Fig. 4). The process was subsequently repeated across Waves 2-5 and plotted. In both MZ and DZ twins, Fig. 4 shows that the twin with the higher CES-D score at wave *t* remained the more depressed twin, on average, at all subsequent waves. As expected, the within-family differences in the mean longitudinal trajectories were larger in the DZ group than the MZ group. All twins showed initial convergence (e.g., between 1987 and 1990), but the MZ twins demonstrated stronger convergence toward one another over time whereas the DZ twins' trajectories did not demonstrate as strong convergence. Rather, after initial convergence, the DZ twins' trajectories slightly diverge from one another (e.g., between 1990 and 1993) or remain relatively parallel (e.g., between 1993 and 2004). One interpretation, thus, is that within-family *r*GE may explain DZ twins tendency to diverge from one another over the long term rather than converge toward one another.

Model fitting results are presented in Table 4. Full longitudinal ACE simplex models were initially fit to the data. Preliminary analyses revealed that the common environmental variance estimates were either essentially zero (and nonsignificant) or negative. Only genetic and nonshared environmental components were estimated in the multivariate analyses. The baseline longitudinal AE model fit the data well (RMSEA is 0.03). The improvement in model fit for Model 2 suggested that a time-constant genetic factor fit the data better than a genetic simplex structure. Model 3 further suggested the wave specific genetic factors could not be set to zero without significant loss in model fit, suggesting that novel sources of genetic variance are introduced over time. In addition to the nonshared environmental simplex structure, adding a time-constant nonshared environmental factor (Model 4) significantly fit the data better than Model 2. The simplex structure could not be dropped from the model, as indicated by the significant decrement in model fit of Model 5. The auto-regressive coefficients between waves 3 years apart, however, could be constrained to be equal without loss of fit (Model 6 compared to Model 4).

The addition of the P<=>E effects in Model 7 did not provide a significant improvement in model fit. The uncorrelated genetic and nonshared environmental stability and change processes in Model 6 provided the most adequate fit to the data among the models tested here. The nonsignificant P<=>E effects in Model 7 were small (about 0.02).

The final model, Model 8, tested the short-term and long-term effects of widowhood on twins' subsequent CES-D scores. The constraint placed on the short-term effects did not result in significant loss of model fit when compared to Model 6. Model 8 was accepted as the final best-fitting model. Parameter estimates and their 95 % confidence intervals from this model are presented in Fig. 5. Based on these estimates, we calculated the proportions of variance in CES-D scores accounted for by each of the genetic and environmental factors (Table 5). The time-constant genetic factor (h^2_{TC}) accounted for approximately 22-26 % of the variance in CES-D scores over time while time-specific genetic factors (h^2_{TS}) accounted for 2–16 % of the variance in CES-D scores. All

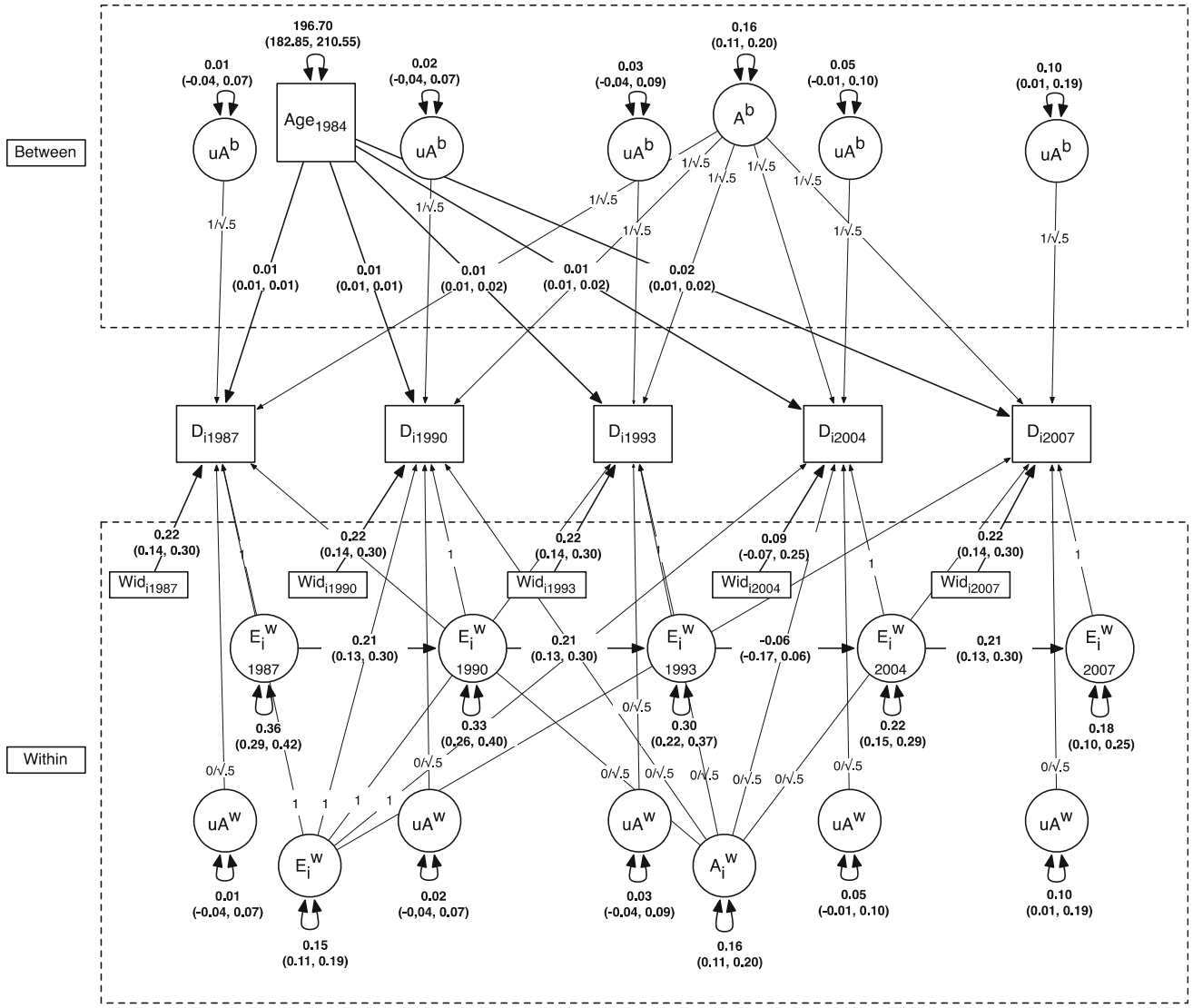

Fig. 5 Best fitting model (Model 8) with unstandardized MLR parameter estimates and 95 % confidence intervals. Upper and lower bounds of the 95 % confidence intervals are presented in parentheses below the parameter estimate. Biometric components of D_{it}, phenotypic CES-D scores for twin i at time t, are estimated between- and within-families; A^b = between-family genetic effect at time t; A_i^w = within-family genetic effect at time t; E_i^w = within-family (nonshared) environmental effect at time t; uA^b = unique between-family genetic effect at time t; uA^w = unique within-family genetic effect at time t; E_{it}^w = unique within-family environmental effect at time t; Wid_{it} = the within-family effect of twin i's widowhood status on CES-D score at wave t; Age = the effects of age at recruitment (1984) into SATSA. Fixed paths and their respective loadings have been *greyed* for clarity of presentation of the parameter estimates

time-specific genetic factors were nonsignificant except for the Wave 5 effect (see Fig. 5). The time-constant nonshared environmental factor (e_{TC}^2) accounted for approximately 21–25 % of the variance in CES-D scores across all waves whereas time-specific nonshared environmental factors (e_{TS}^2) accounted for the largest portion of variance at each wave (29–51 % of the variance in CES-D scores). The estimates do not add up to 1.0 for the reason that the age and widowhood covariates account for the remainder of the variance in CES-D scores.

The significant auto-regression coefficient between time-specific nonshared environmental factors 3 years apart also accounted for stable twin differences over time (Fig. 5). The time-constant nonshared environmental factor plus the time-specific nonshared environmental auto-regressive process explain the observed stability of within-family differences in CES-D scores. Additionally, the time-constant within-family genetic factor also explains the larger difference between DZ twins than MZ twins over time.

Table 5 Proportions of variance in CES-D scores explained by genetic factors, nonshared environmental factors, age, and widowhood variables

	Wave				
	1987	1990	1993	2004	2007
Phenotypic mean	2.25	2.30	2.35	2.54	2.61
Phenotypic variance	0.70	0.69	0.67	0.61	0.65
h^2_{TC}	0.22	0.23	0.23	0.26	0.24
h^2_{TS}	0.02	0.02	0.04	0.08	0.16
e^2_{TC}	0.21	0.22	0.22	0.25	0.23
e^2_{TS}	0.51	0.50	0.46	0.36	0.29
Wid_{it}	0.005	0.004	0.005	0.001	0.01
Age_{1984}	0.03	0.02	0.04	0.05	0.08

Phenotypic mean and variances are in log units of CES-D scores. h^2 proportion of variance attributed to genetic factors (heritability); e^2 proportion of variance attributed to nonshared environmental factors; *TC* time-constant factor; *TS* time-specific factor; *Wid* widowhood status; *Age* age of twin at recruitment into SATSA in 1984

Figure 5 also presents the effects of widowhood on subsequent CES-D scores ($b_{wid\text{-}3\text{-}year}$ and $b_{wid\text{-}11\text{-}year}$). Only the short-term ($b_{wid\text{-}3\text{-}year}$) effect of widowhood was significant, a finding that is consistent with the short-term effect of widowhood on twins' CES-D scores found by Lichtenstein et al. (1996). The dichotomous operationalization of the widowhood variable means that twins widowed at any point between waves that were 3 years apart experience a 0.22 increase in log CES-D units at follow-up (equivalent to 1.25 points in raw score units).

Discussion

The aims of the current study were to add to prior longitudinal twin studies on the biometric stability and change processes of late life depression (Carmelli et al. 2000; McGue and Christensen 2003; Neiss and Almeida 2004) by specifying a P<=>E regression effect that allowed genetic and environmental effects to correlate with one another over time (Beam and Turkheimer 2013; Dolan et al. 2014; Eaves et al. 1977; Fiske et al. 2009). The specification of P<=>E effects arguably do a better job of representing real developmental processes, because they model niche-picking behavior not represented in models that assume independence among genetic and environmental components. We did not detect the P<=>E effect in the current study. The findings of significant time constant genetic and nonshared environmental components and the nonshared environmental simplex structure underlying the twins' CES-D scores suggest that both genetic *and* nonshared environmental contribute to stability of depression in the elderly. The results also provide an update to prior work on depressive reactions to widowhood in SATSA (Lichtenstein et al. 1996).

While a time-constant genetic factor accounted for stability of CES-D scores in SATSA, the results also support two different nonshared environmental processes as sources of stability. First, the time-constant nonshared environmental factor is a constant source of difference in CES-D scores between twins and is interpreted as a lower bound reliability estimate of the nonshared environment. Second, the auto-regressive relation among the time-specific nonshared environmental factors further suggests time-limited stability of differences in late life CES-D scores between twins, similar to findings on general cognitive ability scores in the SATSA (Dickens et al. 2011). As is often found in longitudinal twin studies (Carmelli et al. 2000; McGue and Christensen 2003), nonshared environmental effects also contributed to fluctuations in CES-D scores over time in SATSA. Nonshared environmental stability between twins suggests that environmental factors contributing to depression (e.g., social networks, coping skills) may be established prior to late adulthood and remain consistent throughout late adulthood.

There are two reasons P<=>E effects were not observed in the SATSA CES-D data. First, one indicator of underlying *r*GE is decreasing DZ twin correlations with stable (or increasing) MZ twin correlations (Beam and Turkheimer 2013). Assuming elderly select environments based on phenotypic differences, the expectation would be that DZ twins would gravitate toward increasingly different environments over time, because of their genetic and environmental differences. Within-family *r*GE would only drive down DZ similarity. In SATSA, however, differences between the MZ and DZ twin correlations were relatively stable across the 5 waves, which suggests that genetic and nonshared environmental processes were relatively constant across the measurement period observed here or were established earlier in life, as supported by these results.

Second, phenotype-environment longitudinal models require extremely large samples of twin pairs to have the power to detect the effect (Dolan et al. 2014). A post hoc power analysis suggested that a sample of twins four times the size of the SATSA sample used in the current study was needed to have 50 % power to detect the small effects (approximately 0.02) observed in Model 7 in Table 4.

Finally, bereavement processes also were found to predict within-family differences in twins' CES-D scores. The significant effect of widowhood on subsequent CES-D scores between measurements 3 years apart is consistent with findings suggesting that the effects on depressive symptoms of becoming widowed are time limited (Bonanno et al. 2004; Lee and DeMaris 2007; Lichtenstein et al. 1996). Notably, the addition of two more waves of data in SATSA did not change the results found by Lichtenstein et al. (1996). The long-term effect was not significantly different from zero and is a finding consistent with arguments that widowed adults more often are resilient than not (Bonanno et al. 2004; Lucas et al. 2003), possibly because they create meaning from their loss or successfully construct adaptive post-widowhood identities (Martin-Matthews 2011). Future research on widowhood would benefit from knowledge about how risks for depression associated with genetic and environmental factors change in the months and years following conjugal loss.

The present results should be interpreted in light of the following limitations. First, SATSA contains samples of twins reared apart and reared together. There were too few widowed twins, however, to estimate 4-group models that adjust for the different rearing statuses in SATSA. This limitation is overcome in the current study by the fact that rearing status differences would only bias the estimation of common environmental factors. The effects of common environmental factors remains unclear, as effects were observed in univariate analyses of CES-D scores in the 1987 wave of SATSA (Gatz et al. 1992) but not observed in the current multivariate longitudinal analysis and in other twin samples (Kendler and Prescott 1999; McGue and Christensen 2003). Second, sex differences in CES-D scores have been found in SATSA (Gatz et al. 1992), but were not considered here to increase the sample size to detect P<=>E effects. Third, the widowhood variables were only considered to be within-family environmental variables. Marital status has been found to be genetically influenced (Kendler and Baker 2007), for example, which means that common genetic and environmental factors may mediate the correlation between widowhood and late life depression. Fourth, sample attrition due to death may have influenced the results, as mortality has been found to be correlated with higher baseline CES-D scores in SATSA but not overall depressive symptom levels (Fiske et al. 2003). Thus, the SATSA CES-D data probably did not meet the MAR assumption made in this study.

In conclusion, in a longitudinal twin sample, we demonstrated a substantial stability of depressive symptomatology among older adults. While we applied a reciprocal effects framework to capture gene-environmental processes typically overlooked in longitudinal twin studies, we were not able to show that the observed stability was the result of reciprocal effects. For the most part, genetic contributions were constant over time while environmental effects included continued effects and new effects at each wave of measurement. We found that widowhood was a specific environmental effect that affected depressive symptoms the next wave after bereavement, although not subsequent waves, providing an example of the manner in which environmental dynamics can perpetuate increasing depressive symptoms across time.

Acknowledgments This work was supported by the National Institute of Child Health and Human Development (1R01HD056354-01) and the National Institute on Aging (1F31AG044047-01A1 and T32AG020500).

Conflict of Interest None.

Human and Animal Rights and Informed Consent This report does not contain any studies with animals performed by any of the authors. Informed consent was obtained from all individual participants included in the study.

References

Alexopoulos GS (2005) Depression in the elderly. Lancet 365(9475):1961–1970

Beam CR, Turkheimer E (2013) Phenotype-environment correlations in longitudinal twin models. Dev Psychopathol 25(1):7–16

Beekman ATF, Geerlings SW, Deeg DJ, Smit JH, Schoevers RS, de Beurs E et al (2002) The natural history of late-life depression. Arch Gen Psychiatry 59(2002):605–611

Blazer DG (2003) Depression in late life: review and commentary. J Gerontol A Biol Sci Med Sci 58(3):249–265

Bonanno GA, Wortman CB, Lehman DR, Tweed RG, Haring M, Sonnega J et al (2002) Resilience to loss and chronic grief: A prospective study from preloss to 18-months postloss. J Pers Soc Psychol 83(5):1150–1164

Bonanno GA, Wortman CB, Nesse RM (2004) Prospective patterns of resilience and maladjustment during widowhood. Psychol Aging 19(2):260–271

Boomsma DI, Molenaar PCM (1987) The genetic analysis of repeated measures. I simplex models. Behav Genet 17(2):111–123

Browne MW, Cudeck R (1992) Alternative ways of assessing model fit. Soc Methods Res 21(2):230–258

Bruce ML (1980) Depression and disability in late life: directions for future research. Adv Pathobiol 7:247–248

Bruce ML (2002) Psychosocial risk factors for depressive disorders in late life. Biol Psychiatry 52:175–184

Burnham KP, Anderson DR (2004) Multimodel inference: understanding AIC and BIC in model selection. Soc Methods Res 33(2):261–304

Carmelli D, Swan GE, Kelly-Hayes M, Wolf PA, Reed T, Miller B (2000) Longitudinal changes in the contribution of genetic and environmental influences to symptoms of depression in older male twins. Psychol Aging 15(3):505–510

Core Team R (2014) R: a language and environment for statistical computing. R Foundation for Statistical Computing, Vienna

De Kort J, Dolan C, Kan KJ, van Beijsterveldt C, Bartels M, Boomsma D (2014) Can GE-covariance originating in phenotype to environment transmission account for the Flynn Effect? J Intell 2(3):82–105

Dickens WT, Flynn JR (2001) Heritability estimates versus large environmental effects: the IQ paradox resolved. Psychol Rev 108(2):346–369

Dickens WT, Turkheimer E, Beam CR (2011) The social dynamics of the expression of genes for cognitive ability. In: Kendler KS, Jaffee S, Romer D (eds) The dynamic genome and mental health: The role of genes and environments in youth development. Oxford, New York, pp 103–127

Dolan CV, de Kort JM, van Beijsterveldt TCEM, Bartels M, Boomsma DI (2014) GE Covariance through phenotype to environment transmission: an assessment in longitudinal twin data and application to childhood anxiety. Behav Genet 44(3):240–253

Eaves LJ, Krystyna L, Martin NG, Jinks JL (1977) A progressive approach to non-additivity and genotype-environmental covariance in the analysis of human differences. Brit J Math Stat Psy 30:1–42

Eaves LJ, Long J, Heath AC (1986) A theory of developmental change in quantitative phenotypes applied to cognitive development. Behav Genet 16(1):143–162

Finkel D, Pedersen NL (2004) Processing speed and longitudinal trajectories of change for cognitive abilities: the Swedish Adoption/Twin Study of Aging. Aging, Neuropsychol, Cognit 11(2–3):325–345

Fiske A, Gatz M, Pedersen NL (2003) Depressive symptoms and aging: the effects of illness and non-health-related events. J Gerontrol 58(6):320–328

Fiske A, Wetherell JL, Gatz M (2009) Depression in older adults. Annu Rev Clin Psychol 5:363–389

Galatzer-Levy IR, Bonanno GA (2012) Beyond normality in the study of bereavement: heterogeneity in depression outcomes following loss in older adults. Soc Sci Med 74(12):1987–1994

Gatz M, Pedersen NL, Plomin R, Nesselroade JR, McClearn GE (1992) Importance of shared genes and shared environments for symptoms of depression in older adults. J Abnorm Psychol 101(4):701–708

Guttman L (1954) Some necessary conditions for common-factor analysis. Psychometrika 19:149–161

Hertzog C, Alstine JV, Usala PD, Hultsch DF, Dixon R (1990) Measurement properties of the Center for Epidemiological Studies Depression Scale (CES-D) in older populations. Psychol Assess 2(1):64–72

Jaffee SR, Price TS, Reyes TM (2013) Behavior genetics: past, present, future. Dev Psychopathol 25:1225–1242

Kendler KS, Baker JH (2007) Genetic influences on measures of the environment: a systematic review. Psych Med 37:615–626

Kendler KS, Prescott CA (1999) A population-based twin study of lifetime major depression in men and women. Arch Gen Psychiatry 56:39–44

Kline RB (2005) Principles and practice of structural equation modeling, 2nd edn. Guilford Press, New York

Lee GR, DeMaris A (2007) Widowhood, gender, and depression: a longitudinal analysis. Res Aging 29(1):56–72

Lichtenstein P, Gatz M, Pedersen NL, Berg S, McClearn GE (1996) A co-twin-control study of response to widowhood. J Gerontrol 51B(5):P279–P289

Lucas RE, Clark AE, Georgellis Y, Diener E (2003) Reexamining adaptation and the set point model of happiness: reactions to changes in marital status. J Pers Soc Psychol 84(3):527–539

Martin-Matthews A (2011) Revisiting widowhood in later life: changes in patterns and profiles, advances in research and understanding. Can J Aging 30(3):339–354

McArdle JJ, Prescott CA (2005) Mixed-effects variance components models for biometric family analyses. Behav Genet 35(5): 631–652

McDonald RP (1999) Test theory: a unified treatment. Lawrence Erlbaum Associates, Mahwah

McGue M, Christensen K (2003) The heritability of depression symptoms in elderly Danish twins: occasion-specific versus general effects. Behav Genet 33(2):83–93

McGue M, Christensen K (2013) Growing old but not growing apart: twin similarity in the latter half of the lifespan. Behav Genet 43:1–12

Muthén BO, Kaplan D (1985) A comparison of some methodologies for the factor analysis of non-normal Likert variables: a note on the size of the model. Br J Math Stat Psychol 38:171–189

Muthén LK, Muthén BO (1998–2012) Mplus user's guide. Muthén and Muthén, Los Angeles

Neale MC, Cardon LR (1992) Methodology for genetic studies of twins and families. Springer, Netherlands

Neiss M, Almeida DA (2004) Age differences in the heritability of mean and intraindividual variation of psychological distress. Gerontology 50(1):22–27

Osler M, McGue M, Lund R, Christensen K (2008) Marital status and twins' health and behavior: an analysis of middle-aged Danish twins. Psychosom Med 70(4):482–487

Radloff LS (1977) The CES-D scale: a self-report depression scale for research in the general population. Appl Psychol Meas 1(3):385–401

Raykov T (2005) Analysis of longitudinal studies with missing data using covariance structure modeling with full-information maximum likelihood. Struct Equ Model 12(3):493–505

Satorra A, Bentler PM (2001) A scaled difference Chi square test statistic for moment structure analysis. Psychometrika 66(4):507–519

Shafer AB (2006) Meta-analysis of the factor structures of four depression questionnaires: beck, CES-D, Hamilton, and Zung. J Clin Psychol 62(1):123–146

Wetherell JL, Gatz M, Pedersen NL (2001) A longitudinal analysis of anxiety and depressive symptoms. Psychol Aging 16(2):187–195