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## Genetic influences on the stability of low self-control: Results from a longitudinal sample of twins

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### ARTICLE INFO

### ABSTRACT

Gottfredson and Hirschi's theory contained two propositions that have been the source of an emerging line of empirical scrutiny. First, according to the general theory of crime, levels of self-control are largely determined by parental management techniques and not by biogenic factors. Second, Gottfredson and Hirschi argued that low self-control should remain relatively stable over the life course. Data from twins drawn from the National Longitudinal Study of Adolescent Health were used to test these two hypotheses. The results of univariate model-fitting techniques revealed that genetic factors accounted for between 52 and 64 percent of the variance in low self-control, with the remaining variance attributable to the nonshared environment. Further, low self-control was stable over a two-year time period ( $r = .64$ ). Bivariate Cholesky decomposition models indicated that the stability of self-control was determined almost exclusively by genetic factors, and that genetic factors also explained a moderate amount of change in self-control.

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### Introduction

The stability of antisocial behaviors over long periods of human development is one of the most consistently replicated findings within criminology. Studies using diverse samples, collected at different time periods in different countries, and analyzed using a variety of statistical techniques have all pointed to the same conclusion—namely, that the best predictor of future criminal behavior is a history of childhood and adolescent misconduct (Cairns & Cairns, 1994; Caspi & Moffitt, 1995; Gottfredson & Hirschi, 1990; Loeber, 1982; Moffitt, 1993; Nagin & Farrington, 1992; Nagin & Paternoster, 2000; Olweus, 1979; Sampson & Laub, 1993). While behavioral stability is a generally accepted “fact” among criminologists, the factors that promote or ultimately lead to stability remain clouded in disagreement (DeLisi, 2005; Gottfredson & Hirschi, 1990; Moffitt, 1993; Nagin & Paternoster, 1991; Sampson & Laub, 1993).

One explanation that has gained prominence in recent years is Gottfredson and Hirschi's (1990) general theory of crime also known as self-control theory. Self-control theory is often distilled down into one proposition: that crime, delinquency, and analogous behaviors are all caused by low self-control. Indeed, this part of their theory has undergone numerous empirical testing resulting in wide support across

studies (Pratt & Cullen, 2000). Gottfredson and Hirschi also argued, however, that their theory could explain why behavior is stable over the life course. According to the general theory, antisocial behavior is stable because its underlying cause (i.e., low self-control) is also stable. Since individual differences in self-control persist over time, and since antisocial behavior is a manifestation of low self-control, then individual differences in delinquent involvement will also persist over time.

In contrast to other hypotheses derived from self-control theory, comparatively less research has evaluated the theory's stability postulate. Importantly, the studies that have examined the stability of low self-control have, in general, failed to identify the underlying mechanisms that ultimately lead to stability. The current study addressed this gap in the literature by examining two intertwined issues. First, and in line with prior research, the stability of low self-control during adolescence was estimated (Arneklev, Cochran, & Gainey, 1998; Beaver & Wright, 2007; Burt, Simons, & Simons, 2006; Hay & Forrest, 2006; Mitchell & MacKenzie, 2006; Turner & Piquero, 2002; Winfree, Taylor, He, & Esbensen, 2006). Second, the factors that explain the stability of low self-control were also examined. Specifically, models were estimated to determine the proportion of variance in the stability of low self-control that was accounted for by environmental effects and the proportion of variance in the stability of low self-control that was accounted for by genetic influences. To do so, a sample of twins from the National Longitudinal Study of Adolescent Health (Add Health) was analyzed.

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## Literature review

### *The stability of low self-control*

Gottfredson and Hirschi (1990) claimed that levels of self-control are largely determined by the way in which parents socialize their children (Hay, 2001; Hirschi & Gottfredson, 2001; Unnever, Cullen, & Pratt, 2003; cf. Wright & Beaver, 2005). Parents who are attached to their child, who monitor their child closely, who recognize when their child is misbehaving, and who correct their child's transgressions will instill high levels of self-control in their child. In contrast, parents who fail to engage in these childrearing practices will tend to raise children with much lower levels of self-control. Up until around the ages of eight to ten, parents have the ability to alter their child's level of self-control. After this time, however, levels of self-control become relatively stable throughout the remainder of the life course (Gottfredson & Hirschi, 1990; Turner & Piquero, 2002).

A handful of studies have tested Gottfredson and Hirschi's (1990) hypothesis regarding the stability of low self-control. Arneklev et al. (1998) first examined the stability of self-control using a convenience sample of college students. Self-control was measured at the beginning of the academic semester and at the end of the semester using Grasmick, Tittle, Bursik, and Arneklev's (1993) battery of questions. They calculated *t*-tests, zero-order correlations, and hierarchical linear models to determine whether levels of self-control remained relatively constant over the course of a semester. The strongest support for the stability of self-control was found when a bivariate correlation was calculated between scores on the self-control scale at Time 1 and at Time 2. The correlation coefficient was large and statistically significant ( $r=.82$ ), indicating relatively little change in the rank-ordering of college students on low self-control over one academic semester.

Although Arneklev et al.'s (1998) study provided initial evidence supporting Gottfredson and Hirschi's stability hypothesis, their use of a nonrepresentative sample of college students made it difficult to generalize their findings to other demographic groups. To address this limitation, Turner and Piquero (2002) used a nationally representative sample of respondents from the National Longitudinal Sample of Youth (NLSY) to examine the stability of self-control over a twelve-year time period. Self-control was indexed by using both behavioral and attitudinal measures. The results of their analysis revealed a moderate degree of stability and a moderate degree of change in self-control (correlation coefficients ranged between .33 and .68), providing mixed support for the stability hypothesis.

Burt et al. (2006) analyzed data from the Family and Community Health Study (FACHS) to determine the stability of self-control in a sample of African American children over two measurement waves. At Wave 1, the children were between the ages of ten and twelve years old and at Wave 2 the children were between the ages of twelve and fourteen years old. Burt et al. used a thirty-nine item scale to measure low self-control. To examine the stability in low self-control, they recoded the self-control scale at Wave 1 into quartiles and the self-control scale at Wave 2 into quartiles. Then they examined whether respondents were likely to shift from one quartile to another quartile between the two waves. Contrary to self-control theory, Burt et al. (2006) found a substantial amount of change in self-control. Studies by Mitchell and Mackenzie (2006) and by Winfree et al. (2006) also failed to support the stability thesis.

Recently, Hay and Forrest (2006) used a semi-parametric group-based modeling strategy to test the stability hypothesis. Using five waves of data from the NLSY and a behavioral measure of self-control, they found very high levels of stability for 84 percent of the sample. They did, however, identify roughly 16 percent of the sample that exhibited a significant amount of change after the age of ten. In short, Hay and Forrest's analysis of the NLSY data indicated some evidence in favor of the general theory of crime.

Using data from the Early Childhood Longitudinal Study, Kindergarten Class (ECLS-K), Beaver and Wright (2007) estimated the stability of self-control from kindergarten through first grade in over 17,000 children. They used questions derived from the Social Skills Rating Scale to measure self-control. The results of their structural equation models revealed that self-control was a highly stable trait (stability coefficients ranged from  $\beta=.96$  to  $\beta=.84$ ) during a time period when Gottfredson and Hirschi (1990) would expect to observe at least some change in self-control. Nonetheless, their work revealed that self-control was a time-stable trait during childhood.

### *Why is low self-control stable?*

Taken together, prior investigations detected moderate- to high-levels of stability in self-control over long periods of the life course, providing at least some support in favor of Gottfredson and Hirschi's stability hypothesis (but see Burt et al., 2006). Despite the strong linkages in self-control over time, the factors that are responsible for maintaining levels of self-control remain poorly understood (Caspi & Moffitt, 1995). The current authors present two alternative views—one derived from Gottfredson and Hirschi's theory and one derived from behavioral genetic research—that have the potential to account for the stability in self-control.

### *Stability according to the general theory of crime*

Gottfredson and Hirschi (1990) were very vague in explaining why self-control should remain relatively stable. A close reading of their theory, however, seems to suggest that levels of self-control from childhood through adolescence would remain stable because parental management techniques would also remain stable. Since childrearing tactics are the main incubators of self-control, as long as parenting techniques remain stable, self-control should also remain stable. Gottfredson and Hirschi (1990) were also quick to point out that initial levels of self-control may generate social consequences that contribute to stability. For example, Evans, Cullen, Burton, Dunaway, and Benson (1997) found that individuals with low self-control were likely to have bad social relationships, to associate with criminal peers, to have less educational training, to live in bad neighborhoods, and to have problems obtaining stable employment. All of these experiences embed individuals into lifestyles that promote the use of low self-control and block opportunities for the development of self-control.

Nagin and Paternoster (2000) also interpreted Gottfredson and Hirschi's theory in a similar way when they concluded that:

According to Gottfredson and Hirschi, the window for the development of self-control is fairly short. They suggest that it closes by age 8 or 10. Moreover, they argue that self-control is relatively stable over time because good self-control is difficult to undo and initially poor self-control is difficult to remedy as those low in self-control both miss out on and misplay opportunities to improve their lots in life. One's initial socialization experience, then, is the mechanism controlling the distribution of self-control in the population. Once established early in life, the level of self-control across persons in the population is relatively stable. (p. 121)

To summarize, it appears that Gottfredson and Hirschi (1990) identified two interrelated processes that lead to the stability of self-control. First, they argued that levels of self-control are largely determined by parental socialization techniques. Second, individuals lacking self-control are likely to find themselves in situations that value low self-control (e.g., associating with delinquent peers) and that discourage the use of self-control. As a result, initial levels of self-control—levels that were created by parents—are maintained because of the criminogenic social environments and the negative social consequences that oftentimes covary with levels of self-control. The

important point to bear in mind, however, is that according to the general theory, the development of self-control and the stability of self-control are due solely to social factors and are entirely free from genetic effects (Beaver & Wright, 2005b; Gottfredson & Hirschi, 1990; Wright & Beaver, 2005).

#### *A behavioral genetics view of stability*

Whereas Gottfredson and Hirschi (1990) dismissed the possibility that the stability of self-control is influenced by genetics, findings from an emerging line of behavioral genetic research have pointed to a very different conclusion (Caspi & Moffitt, 1995; Goldsmith, 1983; Haberstick, Schmitz, Young, & Hewitt, 2005; Johnson, McGue, & Krueger, 2005; Larsson, Larsson, & Lichtenstein, 2004; McGue, Bacon, & Lykken, 1993; Reiss, Neiderhiser, Hetherington, & Plomin, 2000). Researchers working from the behavioral genetic tradition typically employ samples of twins to estimate the proportion of variance in a particular trait or behavior that is accounted for by genetic factors and the proportion of variance that is explained by environmental influences (Walsh, 2002). Historically behavioral genetic model-fitting techniques have been used to estimate the heritability of traits and behaviors. Recently, however, more elaborate models have been calculated to determine genetic and environmental influences on behavioral stability (Caspi & Moffitt, 1995). Findings from these studies have unequivocally revealed that genetic factors are salient contributors to the stability of behaviors and personality traits, such as low self-control (Haberstick et al., 2005; Johnson et al., 2005; Larsson et al., 2004; Reiss et al., 2000; Saudino & Cherny, 2001; van Beijsterveldt, Bartels, Hudziak, & Boomsma, 2003; van der Valk, van den Oord, Verhulst, & Boomsma, 2003).

To illustrate, van Beijsterveldt et al. (2003) examined the genetic and environmental influences on the stability of aggression in a large sample of twins. In line with previous research (Haberstick et al., 2005; Johnson et al., 2005; van der Valk et al., 2003), they found that aggression was relatively stable from childhood through adolescence ( $\beta$  ranged from .41 to .77). Next, they estimated the relative contributions of genetic and environmental influences on the stability of aggression. The results of their biometric statistical analyses revealed that nearly 65 percent of the variance in the stability of aggression was accounted for by genetic effects.

In a similar vein, Larsson et al. (2004) examined the genetic and environmental contributors to the stability of ADHD in a sample of 1,480 twin pairs. They found that ADHD was relatively stable over a five-year period and that genetic effects were the overriding cause of this stability. Genetic factors have also been found to be important sources of stability in other personality traits, such as negative emotionality and constraint (McGue et al., 1993). In summary, research has demonstrated substantial genetic effects on the stability of antisocial behaviors and on the stability of personality traits, hinting at the possibility that the stability of self-control may be partially accounted for by genetic effects.

## Methods

### *Data*

Data for this study came from the National Longitudinal Study of Adolescent Health (Add Health). The Add Health used multistage stratified sampling techniques to obtain a nationally representative sample of American adolescents in grades seven through twelve (K. M. Harris et al., 2003; Udry, 2003). In 1994, over 90,000 adolescents attending 132 middle and high schools were administered a self-report at school. To gain more detailed information about some of the respondents, follow-up interviews were conducted with a random sample of 20,745 adolescents and 17,700 of their primary caregivers (typically the mother) in their home. During these interviews, youths were asked about their involvement in delinquency, about their personality traits, about

their social relationships, and about other issues pertinent to adolescence. Approximately one to two years after the first wave of data were collected, a second round of interviews was conducted. The questions asked at the Wave 2 interview were very similar to those asked at Wave 1. The third and final wave of data was collected in 2001–2002 when the respondents were eighteen to twenty-six years old ( $N=15,197$ ). Given that most of the respondents were young adults at Wave 3, the survey instruments were redesigned to include questions relevant to people in their twenties. For example, questions about employment, educational achievements, and marriage were asked at Wave 3.

The Add Health data also contained a subsample of siblings (K. M. Harris et al., 2003). At Wave 1, respondents were asked whether they currently resided with a co-twin, with a half-sibling, with an unrelated sibling (e.g., a stepsibling), or with a cousin. If they responded affirmatively and if their sibling was between the ages of eleven and twenty years old, then they were also added to the sample. Finally, a probability sample of full siblings was chosen for inclusion in the Add Health data (Jacobson & Rowe, 1999). In total, 3,139 sibling pairs were retained in the sample. An extensive amount of research has used the sibling-pairs sample to estimate genetic influences on a range of different behaviors and personality traits (e.g., Cleveland, Wiebe, & Rowe, 2005; Haynie & McHugh, 2003; Jacobson & Rowe, 1999; Rowe, Jacobson, & Van den Oord, 1999). Importantly, a recent examination of the demographic characteristics of the sibling-pairs data revealed that they closely approximated those of the larger Add Health sample (Jacobson & Rowe, 1998).

The final analytical sample was restricted to dizygotic (DZ) twins and monozygotic (MZ) twins. The strategy of only analyzing twin pairs to estimate genetic influences on behavioral stability has been used frequently by past researchers (Haberstick et al., 2005; Hewitt, Emde, & Plomin, 2001; Larsson et al., 2004; Saudino & Cherny, 2001). Once twins whose zygosity was unknown were removed from the data, the sample size included  $N=741$  twin pairs,  $n=452$  DZ twin pairs ( $n=248$  same-sex pairs,  $n=204$  opposite-sex pairs), and  $n=289$  MZ twin pairs.

### *Measurement*

*Low self-control.* When detailing their general theory of crime, Gottfredson and Hirschi (1990) identified six different dimensions to the underlying construct of low self-control. Specifically, they argued that people with low self-control are self-centered, are impulsive, are risk seekers, prefer simple tasks, prefer physical activities over mental activities, and have a temper. Although there is disagreement over the best way to measure low self-control (DeLisi, Hochstetler, & Murphy, 2003; Longshore, Stein, & Turner, 1998; Longshore, Turner, & Stein, 1996; Marcus, 2004; Piquero & Rosay, 1998), the most widely-used measurement strategy has been the battery of questions developed by Grasmick et al. (1993). Unfortunately the Grasmick et al. (1993) scale was not available in the Add Health data; however, a recent meta-analysis by Pratt and Cullen (2000) revealed that the relationship between low self-control and criminal outcomes is not contingent on the use of the Grasmick et al. (1993) scale.

Prior researchers analyzing the Add Health data have identified items that approximated the different components to self-control (Perrone, Sullivan, Pratt, & Margaryan, 2004). First, adolescents were asked whether they had trouble getting along with their teachers. Perrone et al. (2004) conceptualized this item as tapping the bad temper component of self-control. Another question indexing a bad temper was also identified: whether the adolescent had trouble getting along with other students. In addition, respondents were asked whether they had problems keeping their mind focused, whether they had trouble getting their homework finished, and whether they had problems paying attention in school. According to Perrone et al. (2004, p. 302), “these questions tap into the simple tasks, physical activities, and impulsivity components of self-control.” Finally, respondents were asked whether they felt they did everything just right. This last question captured

variation in the self-centeredness dimension of self-control. Items were recoded so that higher scores reflected lower levels of self-control. The exact same questions were available at Wave 1 and Wave 2, and thus the measurement of low self-control was identical between waves.<sup>1</sup>

It is important to note that Hirschi (2004, p. 543) has recently redefined self-control and argued that self-control is “the tendency to consider the full range of potential costs of a particular act.” He then developed a nine-item self-control scale that was based on this new definition. Three of these items pertained to school: (1) Do you like or dislike school, (2) How important is getting good grades to you, and (3) Do you finish your homework? The items used by Hirschi paralleled some of the items available in the Add Health data. Therefore the low self-control scales used in the current analyses measured both the older definition of self-control (Gottfredson & Hirschi, 1990) and Hirschi’s (2004) newer definition of self-control (Piquero & Bouffard, 2007).

A series of additional statistical specifications were calculated to assess the psychometric properties of the low self-control scales. First, all of the items were factor analyzed and the results of the analyses and an inspection of the scree plots revealed that the six items loaded on one latent construct, which is consistent with Gottfredson and Hirschi’s argument that self-control is a unitary factor. Second, reliability analyses were calculated for the low self-control scales by calculating Cronbach’s alphas. The results indicated that the low self-control scales had moderate internal consistency (Wave 1  $\alpha = .67$ ; Wave 2  $\alpha = .67$ ). Third, the predictive validity of the low self-control scales was examined. To do so, two delinquency scales were created: a Wave 1 delinquency scale and a Wave 2 delinquency scale. These scales have been used by prior researchers (e.g., Beaver, 2008; Beaver & Wright, 2005a) and measured a wide array of different delinquent activities, including fighting, stealing, and vandalism, among others. The bivariate correlations between the Wave 1 low self-control scale and the delinquency scales were statistically significant ( $r = .28, p < .01$  for the Wave 1 delinquency scale;  $r = .20, p < .01$  for the Wave 2 delinquency scale), as was the association between the Wave 2 low self-control scale and the Wave 2 delinquency scale ( $r = .30, p < .01$ ). Importantly, these effect sizes approximated those reported in the meta-analysis by Pratt and Cullen (2000). Taken together, it appeared as if the low self-control scales in the Add Health data were reliable and valid indicators of self-control.

Plan of analysis

The analysis for this study proceeded in two steps. First, basic univariate variance components models were estimated. These variance component models are employed frequently in behavioral genetic research and the results reveal the percentage of variance in low self-control that is attributable to genetic factors (A), shared

environmental factors (C), and nonshared environmental factors (E). Fig. 1 provides a schematic diagram of how these models were estimated. The circles represent the latent factors of A, C, and E. The square boxes, which are labeled Twin 1 and Twin 2, represent scores on the low self-control scale for one twin (i.e., Twin 1) and for their co-twin (i.e., Twin 2), respectively. The double-headed arrows indicate the covariation between the latent factors. For MZ twins, the correlation between genetic factors (A) was fixed to 1.00 because MZ twins share 100 percent of their DNA. For DZ twins, the correlation between genetic factors (A) was fixed to .50 because DZ twins share, on average, 50 percent of their segregating DNA. The correlation between shared environmental factors (C) was fixed to 1.00 for both MZ and DZ twins because their shared environments are, by definition, equal. The correlation between nonshared environmental factors (E) was free to vary because nonshared environments between twins are, by definition, orthogonal to each other. The model in Fig. 1 was estimated separately for the Wave 1 low self-control scale and for the Wave 2 low self-control scale.

The second step in the analysis was to estimate the stability of self-control and to determine the degree to which genetic and environmental factors were implicated in promoting both stability and change in self-control. To do so, a bivariate Cholesky decomposition model was calculated, which partitioned the covariation (i.e., stability) between the two self-control scales into genetic, shared environment, and nonshared environment components (Neale & Cardon, 1992). As Fig. 2 shows, the Cholesky decomposition model built on and extended the univariate model presented in Fig. 1. Two comments about Fig. 2 are warranted. First, A1, C1, and E1 represented genetic, shared environmental, and nonshared environmental effects that were common to both of the low self-control scales. The path estimates of  $a_{11}$ ,  $a_{21}$ ,  $c_{11}$ ,  $c_{21}$ ,  $e_{11}$ , and  $e_{21}$  were used to gain estimates of the genetic and environmental effects on the stability of self-control. For example, the total correlation (i.e., stability estimate) between the Wave 1 and Wave 2 low self-control scales was equal to:

$$r = (a_{11} * a_{21}) + (c_{11} * c_{21}) + (e_{11} * e_{21}) \tag{1}$$

The proportion of covariation attributable to genetic effects, therefore, was equal to the product of ( $a_{11} * a_{21}$ ) divided by the total correlation (i.e.,  $r$  from Eq. (1)). The proportion of the covariation due to shared environmental effects was equal to the product of ( $c_{11} * c_{21}$ ) divided by the total correlation (i.e.,  $r$  from Eq. (1)). Lastly, the proportion of the covariation that was the result of nonshared environmental factors was equal to the product of ( $e_{11} * e_{21}$ ) divided by the total correlation (i.e.,  $r$  from Eq. (1)) (Neale & Cardon, 1992; for an application see Larsson et al., 2004).

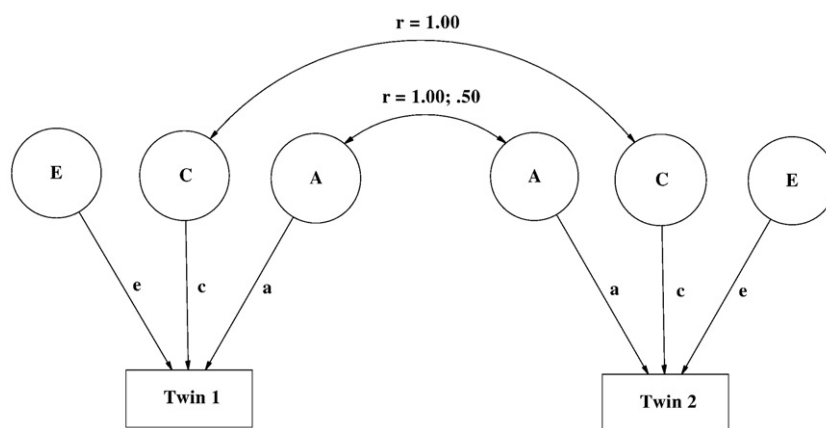


Fig. 1. Basic univariate variance components model for the low self-control scales. Notes: A=genetic factors, C=shared environmental factors, and E=nonshared environmental factors.

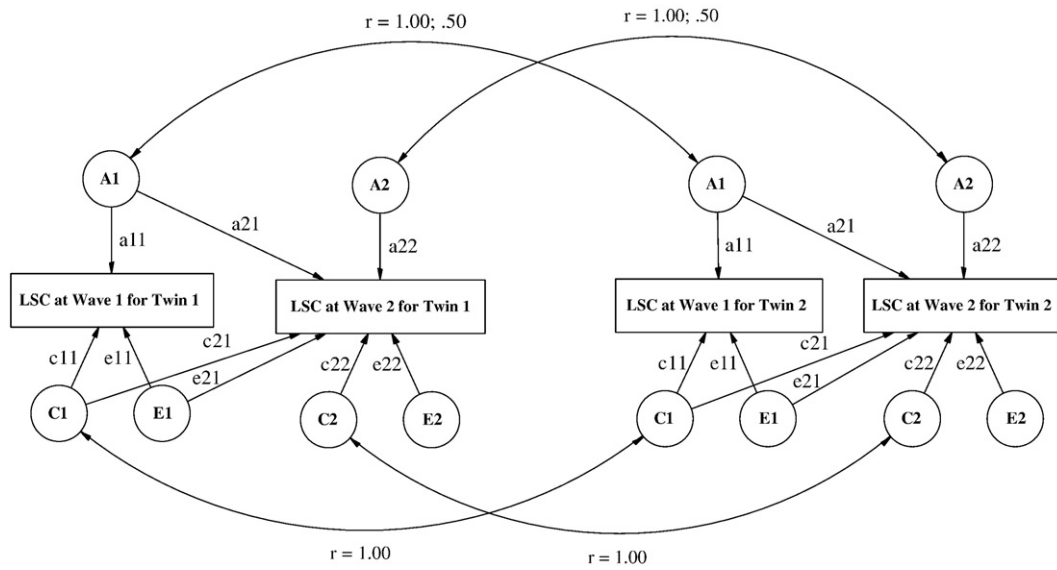


Fig. 2. Bivariate Cholesky decomposition model for the stability of low self-control. Notes: LSC=low self-control; A1, C1, and E1 =genetic, shared, and nonshared environmental effects common to low self-control at Wave 1 and Wave 2. A2, C2, and E2=genetic, shared, and nonshared environmental effects unique to low self-control at Wave 2.

Second, Cholesky decomposition models were also employed to calculate the proportion of change in low self-control that was due to genetic and environmental factors. To arrive at these estimates, the path coefficients  $a_{22}$ ,  $c_{22}$ , and  $e_{22}$  needed to be squared and summed together. The resulting product was then placed in the denominator in three separate equations: one each where the squared  $a_{22}$ ,  $c_{22}$ , and  $e_{22}$  terms were entered into the numerator. The resulting values provided an estimate of the proportion of change accounted for by genetic, shared environmental, and nonshared environmental factors. All of the models were estimated using the statistical software package, Mx, which is a structural equation modeling program particularly useful for analyzing twin data (Neale, Boker, Xie, & Maes, 2002). Mx uses maximum likelihood estimates, and all of the statistical models were fitted to variance-covariance matrices that had been corrected for measurement error.

**Results**

The analysis began by estimating the univariate variance components models for the low self-control scales. A series of iterative steps were taken before arriving at the final models. Mx allows for the comparison of models by constraining the variance estimates for “A” and “C” to zero.

Four different models were estimated: (1) the full ACE model, (2) the AE model (where C is constrained to zero), (3) the CE model (where A is constrained to zero), and (4) the E model (where C and A are constrained to zero). To determine which of the models best fit the data, Akaike's information criteria (AIC) statistics were compared across models (Akaike, 1987). AIC assesses the overall model's goodness-of-fit and the model's parsimony. The model with the smallest AIC was selected as the model that fit the data the best and this model is presented in Fig. 3. The left-hand side of Fig. 3 reveals the results for the Wave 1 low self-control scale and the right-hand side depicts the results for the Wave 2 low self-control scale. Genetic factors accounted for 64 percent of the variance in the Wave 1 low self-control scale, and the remaining 36 percent was due to the nonshared environment. The shared environment had no effect on low self-control at Wave 1. Very similar results were garnered for the Wave 2 low self-control scale, where genetic factors accounted for 53 percent of the variance, shared environmental factors accounted for 0 percent of the variance, and nonshared environmental factors accounted for 47 percent of the variance.

The results thus far have indicated that genetic and nonshared environmental factors accounted for variability in low self-control during adolescence. These univariate models, however, did not provide any information about the stability of low self-control nor did they

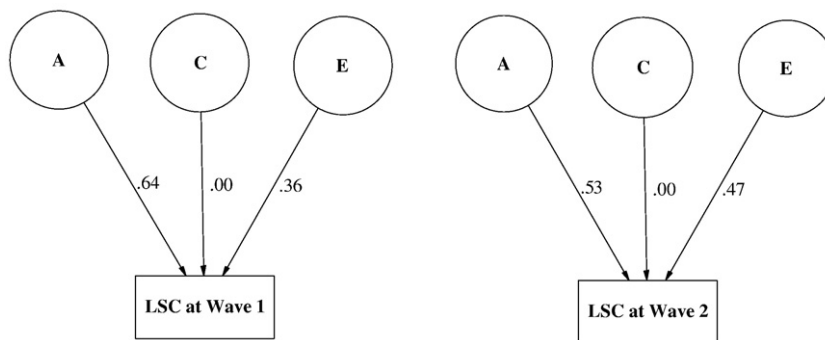
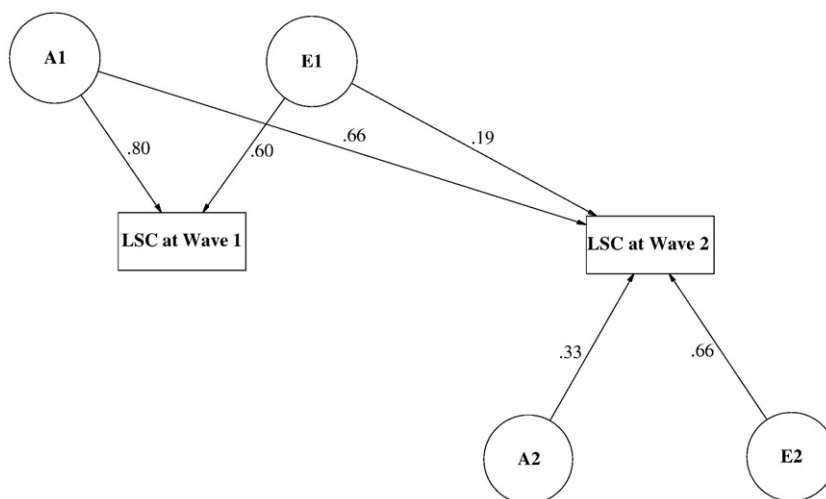


Fig. 3. Results of the univariate variance components model. Notes: LSC=low self-control, A=proportion of variance accounted for by genetic factors, C=proportion of variance accounted for by shared environmental factors, and E=proportion of variance accounted for by nonshared environmental factors.



**Fig. 4.** Results of the bivariate Cholesky decomposition model for the stability of low self-control. Notes: LSC=low self-control, A1=genetic factors common to low self-control at Wave 1 and Wave 2, E1 =nonshared environmental factors common to low self-control at Wave 1 and Wave 2, A2=genetic factors unique to low self-control at Wave 2, E2=nonshared environmental factors unique to low self-control at Wave 2. Path coefficients presented.

provide any information about the genetic and environmental factors that may promote the stability of low self-control. Instead, the Cholesky decomposition model needed to be employed to examine the stability in low self-control. This model can also be used to estimate genetic, shared environmental, and nonshared environmental effects on the covariation (i.e., stability) between the two low self-control scales. Fig. 4 portrays the results of the bivariate Cholesky decomposition model for the stability of low self-control. For ease of interpretation, the coefficients presented are standardized parameter estimates. Note that the shared environmental factor has been removed from the models because it did not account for any variance in the low self-control scales.

Using path tracing rules, it is possible to ascertain the total correlation (i.e., the stability coefficient) between the two self-control scales. The wave-to-wave stability in low self-control was  $r = .64$   $(.80 * .66) + [.59 * .19]$ . This estimate was in line with prior research revealing that low self-control was relatively stable over time (e.g., Beaver & Wright, 2007; Turner & Piquero, 2002). Moreover, the path coefficients in Fig. 4 can also be used to determine the proportion of the stability that was due to genetic factors. The results indicated that 82 percent of the stability in low self-control was accounted for by genetic factors  $(.80 * .66) / .64$  and the remaining 18 percent was due to nonshared environmental factors  $(.60 * .19) / .64$ .

Lastly, the path coefficients in Fig. 4 can be used to estimate genetic and nonshared environmental effects on changes in low self-control. To arrive at these estimates, the path coefficient for the A2 latent construct (.33) was squared (.11), the path coefficient for the E2 latent construct (.66) was squared (.44), and the resulting values were added together (.55). Using these values it was possible to estimate that genetic factors accounted for 20 percent of the change in low self-control  $(.11 / .55)$ , while nonshared environmental factors accounted for 80 percent of the change in low self-control  $(.44 / .55)$ . The results of the bivariate Cholesky decomposition model are summarized in Table 1.

**Table 1**  
Genetic, shared environmental, and nonshared environmental effects on stability and change in low self-control

Model	A	C	E
Factors accounting for stability	.82	.00	.18
Factors accounting for change	.20	.00	.80

Notes: A=genetic factors, C=shared environmental factors, and E=nonshared environmental factors.

### Conclusion

Gottfredson and Hirschi's (1990) theory has become one of the most tested and empirically supported criminological theories (Pratt & Cullen, 2000). Even so, much remains unknown about the causes of self-control and the stability of self-control. The purpose of this study was to shed some light on these issues. The current authors first estimated the proportion of variance in self-control that was attributable to genetic factors, shared environmental factors, and nonshared environmental factors. The results of the model-fitting statistics revealed that genetic effects accounted for 64 percent of the variance in the Wave 1 low self-control scale and for 53 percent of the variance in the Wave 2 low self-control scale. The remaining variance was attributable to nonshared environmental influences. Importantly, the shared environment did not explain any variance in any of the two self-control scales. This finding is revisited momentarily.

The stability of self-control during adolescence was also examined. The results revealed some support in favor of Gottfredson and Hirschi's (1990) stability postulate. For example, the bivariate correlation between the Wave 1 and Wave 2 low self-control scales was  $r = .64$ . This finding added to a growing body of research suggesting that levels of self-control remain relatively stable over the life course (Arneklef et al., 1998; Beaver & Wright, 2007; Hay & Forrest, 2006; Turner & Piquero, 2002). The analysis was extended by estimating the genetic effects and the environmental effects on the stability of self-control. To do so, a bivariate Cholesky decomposition model was estimated. The results of this model revealed that 82 percent of the covariation between the two self-control scales was due to genetic factors and the remaining 18 percent was attributable to the nonshared environment. Moreover, 20 percent of the change in low self-control was due to genetic factors and the remaining 80 percent was due to the nonshared environment. The shared environment did not account for either stability or change in low self-control.

It is important to point out, however, that the generalizability of the findings may be limited due to two main limitations of the research. First, the Add Health data did not contain all of the measures needed to replicate Grasmick et al.'s (1993) low self-control scale. Still, the low self-control scales used in this study have been employed by prior researchers analyzing the Add Health data (Beaver, 2008; Perrone et al., 2004). The fallibility in the measures of self-control provided a conservative estimate of genetic influences on low self-control. Error in measurement, for example, would attenuate the stability coefficients and it would reduce

the heritability estimates. In addition, measurement error is included in the nonshared environmental (E) component and thus would increase the variance explained by E. Even so, the models revealed a substantial amount of stability in self-control and significant genetic effects on both the development of self-control and the stability of self-control. If the Grasmick et al. (1993) scale was available in the Add Health data, the stability coefficients and the heritability estimates would probably increase. Future research will have to explore this possibility by using different measures of self-control and different samples.

The second main limitation of the current study was that the time interval between measurement waves—approximately a one- to two-year time lag—was relatively short. A more complete test of Gottfredson and Hirschi's (1990) stability postulate would need to examine the stability of self-control over a much longer period of time. Nonetheless, the findings were in line with a voluminous line of literature revealing that certain personality traits are extremely stable over long swaths of the life course (Caspi, 2000). Prospective studies, for instance, have found that personality traits measured early in childhood are significantly predictive of personality traits in early adulthood (Caspi et al., 2003). It would be interesting to examine whether self-control would also remain stable from childhood up through adulthood. Of course, Gottfredson and Hirschi (1990) argued that self-control does not become stable until around the ages of eight or ten, but recent work by Beaver and Wright (2007) has indicated that self-control is relatively stable well before the age of eight. Much more research is needed in order to draw firm conclusions about the development of self-control and the stability of self-control.

#### Implications for Gottfredson and Hirschi's theory

Gottfredson and Hirschi (1990, see especially chap. 3, pp. 60, 96) openly rejected the notion that self-control could be influenced by genetic and biological factors and instead took the position that self-control was created only through social factors. In taking such a position, Gottfredson and Hirschi (1990) failed to leave open the possibility that certain genetic influences, under certain conditions, could predispose an individual to developing low self-control. There is now ample evidence from a diverse range of disciplines, including psychology, psychiatry, neuroscience, behavioral genetics, child development, and even criminology, clearly showing that self-control is guided by genetics and nonshared environmental factors, not shared environmental influences as Gottfredson and Hirschi suggested (Beaver & Wright, 2005b; Cohen, 1999; Dunn & Plomin, 1990; J. R. Harris, 1995, 1998; Pinker, 2002; Rowe, 1994; Wright & Beaver, 2005).

In direct contradiction to the general theory, there was no evidence indicating that the two self-control scales were influenced by shared environmental effects. Unfortunately, most criminological research examining familial effects has focused on shared environmental influences (Beaver, 2008). The current findings and those of many other studies, however, indicate the shared environment has relatively little effect on personality development (Cohen, 1999; J. R. Harris, 1995, 1998, 2006; Pinker, 2002; Rowe, 1994; Wright & Beaver, 2005). The current authors suggest that Gottfredson and Hirschi (1990) revise their theory on the causes of self-control to reflect more accurately the research results garnered from these various lines of inquiry.

It should be noted that shared environmental factors were relatively inconsequential in accounting for the stability of self-control. Genetic effects, however, explained 82 percent of the variance in stability. Clearly, analysis of twins from the Add Health data strongly suggested that the causes of stability are not to be found in the social environment, but rather are more likely to be located within an individual's genotype. If this is the case, then the stability of criminal behavior may also be explained by genetic influences. The findings reported here definitely hinted at this possibility.

This study was not the first to suggest that the stability in antisocial phenotypes may be accounted for by genetic factors. In a large review

of the literature on behavioral continuity, Caspi and Moffitt (1995) concluded that:

Although antisocial behavior per se is not inherited, a variety of biological traits may underlie the heritability coefficients for antisocial behavior by predisposing persons to develop an antisocial phenotype. These traits may be scrutinized empirically to determine if any are stable across development and if the process by which they initiate antisocial behavior might also serve to maintain such behavior. (p. 474)

Self-control may be one trait—a trait largely influenced by genetic factors—that may begin to explain why antisocial behavior is stable.

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#### Note

1. Unfortunately, the same items measuring self-control at Wave 1 and Wave 2 were not collected at Wave 3. As a result, the analysis was confined to the first two waves of data.

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