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Genetic and Environmental Influences on Negative Life Events From Late Childhood to Adolescence

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Abstract

This multi-wave longitudinal study tested two quantitative genetic developmental models to examine genetic and environmental influences on exposure to negative dependent and independent life events. Participants ($N = 457$ twin pairs) completed measures of life events annually from ages 9 to 16. The same genetic factors influenced exposure to dependent events across time, and increased in magnitude during the transition to adolescence. Independent events were less genetically influenced than dependent events in boys, but not girls. Shared environmental influences decreased in magnitude as youth transitioned into adolescence. Nonshared environmental influences were mostly age-specific and contributed significantly to both types of events at all ages. Results provide theoretical implications for developmental risk pathways to stress exposure and stress-related psychopathology.

Keywords

Life events; adolescence; development; genetic and environmental influences; twin study

Exposure to stressful life events (SLEs) is a risk factor for many forms of psychopathology, including internalizing disorders (e.g., Hammen et al., 1991; Hammen, 2005; Kim et al., 2003; Kessler, 1997; Rudolph et al., 2000), externalizing disorders (e.g., Ge et al., 2002; Robinson et al., 1995; Timmermans et al., 2010), and other negative psychosocial outcomes in adults and children (e.g., Grant et al., 2004). Understanding the etiology and developmental course of exposure to SLEs may further elucidate risk pathways for psychopathology and other negative outcomes.

Although stress is often considered an environmental risk factor for psychopathology (e.g., Kessler, 1997; Rutter, 2000), there is increasing evidence that SLE exposure is genetically influenced (Kendler & Baker, 2007; Kendler et al., 1993; Kendler & Karkowski-Shuman, 1997; Plomin et al., 1990; Silberg et al., 1999); that is, an individual's genes may predispose him or her to experience SLEs. To date, most of the genetically informative studies examining the etiology of life events have been limited to adult samples. Furthermore, little is known about how the effects of genetic and environmental influences on life events change across the transition from childhood to adolescence, a developmental period associated with increased and unique stressors. In the present study, we conducted developmental behavior genetic modeling to examine genetic and environmental influences on the stability and change in stressful life events from age 9 to 16.

Heritability of Life Events

In a seminal publication, Plomin et al. (1990) examined the genetic and environmental influences on exposure to life events in an adult sample of twins. Results indicated that 40% of the variance in life events could be explained by genetic factors, suggesting that life events were not simply “environmental” experiences. Following this report, the behavioral genetic literature has provided substantial evidence that exposure to SLEs is genetically influenced. The heritability of SLEs has been replicated consistently in adult and adolescent samples (e.g., Bemmels et al., 2008; Bolinskey et al., 2004; Kendler et al., 1993), and a systematic review of 12 studies investigating the heritability of life events found that heritability estimates ranged from 27% to 47% (Kendler & Baker, 2007). That is, between one quarter and one half of the variance in exposure to stressful events can be explained by genetic factors.

How is it that exposure to a putatively environmental variable such as life events is heritable? The distinction between types of life events, specifically independent and dependent events (or uncontrollable and controllable, respectively), has been helpful in explaining how genes influence SLEs (Hammen, 2005; Kendler & Prescott, 1999). Dependent life events are those that are due to the behavior or characteristics of the individual, such as the end of a romantic relationship or being fired from a job. On the other hand, independent or “fateful” life events are not related to the individual’s behavior or characteristics (e.g., the death of a family member due to illness; Brown & Harris, 1978; Hammen, 1991). In adults and adolescents, genetic factors significantly influence exposure to dependent (or controllable) life events, whereas they have less or no influence on independent (or uncontrollable) life events (e.g., Bemmels et al., 2008; Billig et al., 1996; Kendler et al., 1993; Plomin et al., 1990; Silberg, et al., 2001).

The evidence that genes influence dependent but not independent life events implies that an individual’s genes may lead him or her to either evoke or select certain environments and life events. This phenomenon has been described as evocative or active gene-environment correlation (i.e., rGE; Plomin, DeFries & Loehlin, 1977; Scarr & McCartney, 1983), and suggests that individuals evoke responses from their environment that complement their genetic makeup or actively seek out environments or experiences that are consistent with their genes. For example, a child with a genetic propensity for depression may withdraw and reject others’ attempts to engage socially, thereby generating interpersonal stress (i.e., negative reactions from peers). Using this theoretical framework, the heritability of life events that are dependent on one’s behaviors or characteristics provides valuable evidence for the link between genetic propensity and SLEs.

Stressful Life Events in Adolescence

There is considerable evidence suggesting that the social, psychological, and biological changes that occur during the transition from childhood to adolescence are associated with increased stress (for reviews see Arnett, 1999; Compas et al., 2001; Grant et al., 2004). Studies have shown that events occurring during adolescence, such as pubertal transition (Ge et al., 2001, 2003) and a new focus on interpersonal relationships (Rudolph, 2002; Rudolph & Hammen, 1999), contribute to increased stress in youngsters. Furthermore, the experience of stress during adolescence has consistently been associated with increased risk for psychopathology and poor psychosocial outcomes. For example, SLEs are associated with depressive symptoms (Ge et al., 1994; 2001), continuity and co-occurrence of internalizing and externalizing symptoms (Kim et al., 2003; Timmermans et al., 2010) and increased risk for onset of psychosis in youth (Tessner et al., 2011). Consistent with these findings, a review of the literature (Grant et al., 2004) found that of the 60 studies examining

the association between stress and psychopathology prospectively in children and adolescents, 53 studies reported evidence of stressors contributing to psychopathology.

Despite the large literature on stress in adolescence and its consequences, very few studies have measured the contributions of genetic influences to SLE exposure in adolescence. Thus, SLEs have been largely regarded as environmental factors in the developmental literature, despite strong evidence of genetic influences on SLE exposure in adults.

There are few studies that have examined the etiology of SLEs during adolescence in a genetically informative sample. Recently, Bemmels and colleagues (2008) administered a life events interview to a large sample of twins, adopted siblings, and biological siblings to determine the etiology of independent and dependent life events. In this community sample, genetic factors explained 45% of the variance in dependent life events and only 7% of the variance in independent life events. Furthermore, results consistent with higher magnitudes of genetic influences on dependent than independent life events were found across the entire sample, suggesting that the results of the twin sample were not biased by the limitations of the twin method.

There is even less literature regarding the role of genetic and environmental influences in the development of SLEs across time. One exception is a study by Silberg and colleagues (1999), in which the authors assessed the association between dependent life events and depression in a longitudinal sample of twins in preadolescence and again in adolescence. Results of quantitative genetic modeling suggested that life events were heritable in both developmental stages (h^2 wave 1 = .46; h^2 wave 2 = .91), but that shared environmental factors played a larger role in preadolescent stress exposure and genetic influences were more substantial in adolescence. Additionally, a proportion of the genetic variation in life events was shared with depression, indicating that the same genetic influences may contribute to SLE exposure and depression. This study represents one of few to capture the change in magnitude of genetic and environmental influences on life events in adolescence, and provides some evidence that certain genetic vulnerabilities to SLEs may be “turned on” during the transition to adolescence and contribute to depression vulnerability (Silberg et al. 1999).

The Current Study

The etiological influences on exposure to SLEs in adolescence are largely unknown, as are the factors influencing stability and change in SLEs in adolescence. In this study, we used three quantitative genetic models to examine the genetic and environmental influences on exposure to SLEs across time. Briefly, the Cholesky model provides a general description of the contributions of genetic and environmental influences to the covariances of SLEs between twins. The common factor and simplex models are developmental models that describe the pattern of genetic and environmental influences over time. The common factor model assumes that the stability of behavior across ages is explained by the same genetic and environmental influences contributing to the variance in life events across development, whereas developmental changes are influenced by age-specific effects. In the simplex model, stability of behavior across ages is explained by continuation effects that are transmitted from the earlier age to the next, and developmental changes are influenced by new genetic and environmental influences that occur at specific ages. Elucidating the etiological influences on SLE exposure will inform theories of psychopathology risk and resiliency in adolescence.

We assessed both dependent and independent SLEs to determine if etiological influences differed between these constructs. We focused on “daily” stressors rather than severe or

traumatic events in order to capture the most common events that occur in the lives of adolescents. Based on theory and empirical evidence, we hypothesized that dependent SLEs would be influenced by genetic and shared and nonshared environmental factors, whereas independent SLEs would be explained mostly by shared and nonshared environmental factors. We also examined gender differences in the genetic and environmental influences on stability and change in SLEs during adolescence, as there is clear evidence of gender differences in stress and stress-related pathology during adolescence (e.g., Hankin & Abramson, 2001; Mezulis et al., 2010; Rudolph & Hammen, 1999).

Method

Participants

A sample of 457 same sex twin pairs (245 monozygotic pairs, 212 dizygotic pairs) from the Longitudinal Twin Study (LTS) - a sample of same-sex twin pairs recruited through the Colorado Department of Health born between 1986 and 1990 in Colorado -were included in the analyses. Parents were invited to participate if they lived within 2–3 hours of Boulder and 50% of those who were invited did so. Individuals were included in the analysis if they had data at one or more time points. We found no evidence of attrition effects in our sample, as there was no significant difference in rates of SLEs reported between individuals who completed the study and those who discontinued the study prematurely (all tests $p > .05$). Sample size at each time point is presented in Appendix 2. Across the eight time points, approximately 20% of all possible data points were missing.

The race and ethnic distribution of the sample was 86.6% Caucasian, 8.5% Hispanic, 0.7% African-American, 1.2% Asian, and 2.9% other, which corresponds well to that reported for Boulder County, Colorado in the 1990 United States Census (89.5% Caucasian, 3.8% Hispanic, 0.9% African-American, 2.4% Asian, and 3.4% other; U. S. Census Bureau, 1990). The mean number of years of education was 14.29 years for mothers and 14.42 years for fathers. Of all parents, 5% did not complete high school, 29% completed high school without post-secondary education, 49% had some post-secondary education, and 17% had some graduate-level education. Additional details regarding the sample and recruitment procedures can be found in Rhea et al. (2006).

Measures

Life Events—To measure SLEs, we used the Life Events Scale for Adolescence (LESA; Graber, Brooks-Gunn & Warren, 1995). This scale asks 54 questions about life events in the domains of family, school, and peer events and was administered to the twins annually from ages 9 to 16. Participants were asked to indicate which life events had happened to them in the past year, and rate each event that had occurred on a 7-point scale (“impact rating”) ranging from 1 (very upset) to 7 (very pleased).

In this study, dependency of events was assessed by a panel of raters using a Likert like scale to determine the status of an event: a rating of 1 or 2 indicated that the event’s occurrence was certainly or almost certainly independent of a person’s behavior, 3 indicated that the event was possibly dependent or at least partly dependent on the person’s behavior, and 4 or 5 indicated that the event’s occurrence was almost certainly or certainly due to the person’s behavior (Hammen, 1991). Items with a score of three or higher were given a rating of dependent. With good interrater reliability ($\kappa = .74$), 34 items were designated as dependent and 20 items as independent.

Impact ratings of all 54 items were assessed across participants to identify events that were generally seen as negative by adolescents. Using a method developed by Trombello et al.

(2011), 95% confidence intervals were calculated for each item based on the impact ratings made by the entire sample of adolescents, and items that had a confidence interval below 4 (“neutral”) at every time point were deemed negative. This method resulted in 19 life events that were both dependent and negative and 10 life events that were both independent and negative (see items listed in Appendix 1).

The distribution of frequency counts of life events at each time point was highly skewed; thus, both categories of life events were binned into ordinal variables, with the assumption that a continuous, normal liability distribution underlies the ordinal variables. Derks et al. (2004) reported that when skewed data were analyzed, the parameter estimates were biased (i.e. shared environmental effects were underestimated and the nonshared environmental effects were overestimated). They also demonstrated that although transformations (e.g., square root transformation) did not remove this bias, the correct parameter estimates were recovered when analyses were conducted on ordinal variables. Appendix 2 shows how life events scores were binned into ordinal variables and the percentages of the sample in each category. The number of categories in the ordinal variables was chosen to avoid small cell sizes.

Statistical Analyses

Mplus (Muthen & Muthen, 2010) was used to conduct the data analyses. Given that several ordinal variables were examined, the weighted least square mean and variance (WLSMV) estimation method was used. When WLSMV is used, Mplus uses pairwise deletion. Statistical significance of the parameters was determined by their p -values, which are based on the ratio of each parameter to its standard error, which yields a z -statistic. Given that the χ^2 is sensitive to sample size, additional fit indices, including the comparative fit index (CFI; Bentler, 1990), the Tucker-Lewis index (TLI; Bentler, 1990), and the root mean square error of approximation (RMSEA; Browne & Cudeck, 1987) were assessed. A CFI and TLI greater than .95 and RMSEA less than .06 indicate good model fit (Hu & Bentler, 1998). In Mplus, a parameter approaching its boundary can cause the model to fail. Therefore, parameter estimates that were equal to or very close to zero or one had to be fixed to zero or one.

Phenotypic Stability—Correlations were calculated for each category of life events separately. Cross-trait phenotypic correlations (e.g., age 9 life events score with age 10 life events score for twin 1), within-trait cross-twin correlations (e.g., age 9 life events score for twin 1 with age 9 life events score for twin 2), and cross-trait cross-twin correlations (e.g., age 9 life events score in twin 1 with age 10 life events score in twin 2) were calculated to assess phenotypic stability over time and provide initial estimates of genetic and environmental influences on life events.

Based on the assumption that, on average, monozygotic twins (MZ) share 100% of their genes and dizygotic twins (DZ) share 50% of their genes by descent, comparing the magnitude of cross-trait cross-twin correlations can provide information about genetic and environmental influences on stability of SLEs. When correlations are greater in MZ twin pairs than DZ twin pairs ($r_{MZ} > r_{DZ}$), there is evidence of genetic influences on the stability of life events over time. If r_{MZ} is greater than twice r_{DZ} , this suggests the influence of dominant genetic effects, whereas if r_{MZ} is less than twice r_{DZ} , there is evidence of shared environmental effects on the stability of SLEs.

To examine these effects, phenotypic correlations were fixed to be equal between twin 1 and twin 2 and between MZ and DZ twin pairs; within-trait cross-twin correlations and cross-trait cross-twin correlations varied between MZ and DZ twin pairs; and equivalent cross-trait cross-twin correlations between twin 1 and twin 2 were fixed to be equal. To examine

gender differences, a model in which parameters were restricted to be equal in boys and girls was tested and was compared (using a chi-square difference test) to the model where parameters were free to vary across boys and girls.

Model Fitting—We conducted genetic modeling in three steps. In the first step, we fit the Cholesky model to the data, which provides an atheoretical assessment of genetic and environmental influences on developmental stability. This model decomposes the covariance of life events across ages into genetic (a^2), shared environmental (c^2), and nonshared environmental (e^2) influences, and assumes that these factors also influence subsequent ages. For example, genetic influences at age 9 influence all life events at all ages, genetic influences at age 10 influence life events at age 10 to 16, genetics influences at age 11 influence life events at age 11 to 16, and so on. Appendix 3 shows an example of this model for genetic influences only for clarity, although the Cholesky model also includes shared (C) and nonshared (E) environmental influences.

Next, we used the Cholesky model as a reference to examine the fit of two developmental models: the simplex model and the common factor model. The simplex model (see Panel B of Figure 1) explains phenotypic stability by assuming that past influences on life events are transmitted over time (continuation effects), via autoregressive effects among the latent variables. The simplex model also includes new influences (innovation effects) that are not impacted by previous time points that emerge at each time point. As the latent factor at the first time point cannot be due to continuation effects, it is assumed to be an innovation effect. The simplex model is likely to fit better to data in which correlations decrease as time points become more temporally distant (e.g., correlations between age 9 and age 10 are higher than correlations between age 9 and age 13). In contrast, the common factor model (see Panel A of Figure 1) assumes that there is one consistent underlying factor explaining influences on life events at each age, and also includes age-specific influences.

As with the correlation models, potential gender differences in the parameter estimates of each genetic model were tested by comparing the fit of a model where parameter estimates were allowed to vary across gender and a model where parameter estimates were constrained to be equal across gender. The difference in the fit of these two models was tested by conducting a chi-square difference test.

Results

Correlations

A model estimating cross-trait phenotypic correlations, within-trait cross-twin correlations, and cross-trait cross-twin correlations was fit to the data for each life events category. For negative independent events, a model fixing parameters estimate to be equal across gender did lead to a decrement in fit, $\chi^2(97) = 121.13$, $p = .05$, suggesting gender differences. There was no evidence of gender differences in negative dependent events, $\chi^2(97) = 96.57$, $p = .49$. Therefore, results for independent negative events are presented separately for boys and girls, and results for dependent negative events are presented for the entire sample.

Table 1 presents correlations for dependent and independent life events. In the diagonal cells, the first number represents the phenotypic correlation within a time point ($r = 1.0$) and the second and third numbers represent the within-trait cross-twin correlations for MZ and DZ twins, respectively. In the off-diagonal cells, the first number represents phenotypic correlations across time points, followed by the MZ and DZ cross-trait cross-twin correlations.

In both categories of life events, positive phenotypic correlations across time points of modest to moderate magnitude suggest some stability in life events during the transition from childhood to adolescence. For negative dependent life events, the correlations among MZ twins was larger than among DZ twins both within and across time points, suggesting genetic influences on the exposure to life events as well as their developmental stability. General trends also suggest a possible increase of genetic effects during adolescence, as the difference between MZ and DZ correlations became greater over time.

As expected, there was a different pattern in correlations of negative independent life events. For both boys and girls, there was evidence of phenotypic stability over time. In boys, the evidence of genetic influences on independent life events was not consistent, as MZ correlations were often very close to or less than DZ correlations. In girls, evidence of genetic influences on independent events was more consistent, although to a lesser extent than with dependent events.

Model Fitting Results

First, the fully saturated Cholesky model was tested. Next, the common factor and simplex models were tested for each variance component separately (A, C, and E) as well as jointly, to determine the pattern of genetic and environmental influences on the developmental stability of life events. For example, the first model included a common factor model for A and a Cholesky model for C and E (i.e., the “Common Factor A” model in Table 2), the second model included a common factor model for C and a Cholesky model for A and E (i.e., the “Common Factor C” model in Table 2), and so on. Table 2 provides model fit statistics of all models. Model fit indices indicated that the full Cholesky model fit the data well, as did the more parsimonious developmental models (see Table 2 for results).

Figure 1 (Panel A) shows genetic influences from the common factor model for dependent life events, including standardized parameter estimates. These estimates can be used to calculate the contribution of genetic influences on life events, as well as the proportions of each variance component that are explained by common and age specific influences. For example, at age 9, the contribution of common genetic factors (those that influence all ages) can be calculated by squaring the common factor genetic path ($.37^2 = .14$), and the contribution of genetic influences specific to age 9 can be calculated by squaring the age-specific genetic path ($.40^2 = .16$). The overall genetic variance in dependent life events at age 9 is the sum of these two numbers $[(.14) + (.16) = .30]$. The same calculations can be used to calculate the magnitude of shared environmental and nonshared environmental influences (see Tables 3–4).

Figure 1 (Panel B) shows the genetic influences from the simplex model for dependent life events, including standardized parameter estimates. To calculate the genetic variance explained by continuation effects at age 10 (genetic effects that were also present at age 9), the continuation effect coefficient is squared and multiplied by the total genetic variance at age 10 $[(.89^2) \times (.42^2) = .14]$. To calculate the genetic variance explained by innovation effects at age 10 (genetic effects that were not present at age 9), the innovation effect coefficient is squared and multiplied by the total genetic variance at age 10 $[(.45^2) \times (.42^2) = .03]$. The overall genetic variance at age 10 is calculated by summing these numbers $(.14 + .03 = .17)$. The same calculations can be used to calculate the magnitude of shared environmental and nonshared environmental influences (see Tables 3–4).

Due to space limitations, figures presenting the full common factor ACE and simplex ACE models (including parameter estimates) for dependent and independent events are presented in the online supplement. Online Supplements 1 and 2 present results from the common factor ACE and simplex ACE model for dependent events. Online Supplements 3 and 4

present the common factor ACE model for independent events for boys and girls and Online Supplements 5 and 6 present results from the simplex ACE models for boys and girls.

Table 3 provides the proportions of variance explained by genetic and environmental influences on dependent life events at each age. The common factor ACE model suggests genetic factors, the majority of which were common to all ages, contributed marginally to life events up to age 12 (5% –14% of the variance), after which genetic influences increased and remained higher throughout adolescence (35% – 48%). Conversely, shared environmental influences, which were mostly due to common influences, were greater in magnitude before age 12 (12% – 43%), after which they decreased and remained minimal throughout adolescence (1% – 16%). Finally, the model indicated that nonshared environmental effects were generally age specific and consistent across ages (18%–47%). To test whether these developmental patterns were statistically significant, we tested three models in which the magnitude of common genetic, shared environmental, and nonshared environmental influences at each age was fixed to be equal at all time points. Chi-square difference tests indicated that only the magnitude of common nonshared environmental influences could be fixed to be equal across time, $\chi^2(8) = 11.44, p = .18$. Fixing the magnitude of common genetic, $\chi^2(8) = 21.59, p < .01$, and shared environmental influences, $\chi^2(9) = 52.70, p < .01$, across time led to a significant decrement in fit. These results suggest that the overall increase in genetic influences and decrease in shared environmental influences were significant.

The simplex ACE model yielded a pattern of results similar to that of the common factor ACE model. Continuation effects accounted for the majority of the genetic influences over time, and there was little evidence of innovation effects, suggesting that the same genetic factors were influencing all time points. Furthermore, there was a significant increase of genetic effects beginning at age 12 and continuing through adolescence. Both continuation and innovation effects contributed to shared environmental influences, which were higher at ages 9–12, then decreased and remained relatively low throughout adolescence. Nonshared environmental influences were consistent and significant across time, and were mostly explained by innovation effects.

Table 4 provides proportions of variance in independent life events explained by genetic and environmental influences at each age in both the common factor ACE and simplex ACE models. Both models yielded similar results. For boys, genetic influences were modest, increased slightly over time, and were a result of both common and age-specific effects in the common factor model and both continuation and innovation effects in the simplex model. Shared environmental influences contributed significantly to the variance in life events in the middle time points (ages 12–14) but minimally in early and later time points. In general, innovation or age specific effects explained most of the nonshared environmental influences on independent life events in boys. Estimates of nonshared environmental influences were significant and stable across time points.

For girls, genetic influences on independent life events were significant at almost every age and generally increased over time. These influences were explained by both common and age-specific effects in the common factor model and both continuation and innovation effects in the simplex model. Shared environmental influences were higher in late childhood and decreased in early adolescence. Innovation or age specific effects played a significant role in shared environmental influences until age 12, after which continuation or common effects explained most of the shared environmental influences. Finally, nonshared environmental influences were due mostly to innovation or age specific effects in both models and were significant and relatively stable across ages, but of a lesser magnitude than seen in boys.

The gender differences in genetic influences on negative independent life event exposure may be due to two items rated independent (*got picked on by any of your friends; your group of friends got picked on by other groups*) that could be construed as more “dependent” for girls than boys, thus confounding results (we thank two reviewers for this suggestion). In analyses using an independent event composite omitting these two items, gender differences for the common factor and simplex ACE models were marginally significant or nonsignificant [$\Delta\chi^2(40) = 54.83, p = .06$ and $\Delta\chi^2(40) = 47.57, p = .19$], respectively, suggesting the inclusion of these items may have contributed to gender differences in independent event exposure. However, all parameter estimates of genetic and environmental influences on independent events were very similar in the original and new analyses (e.g., the mean heritability of independent events across age in the two models was .20 vs. .18 for boys, .34 vs. .31 for girls, and .27 vs. .23 in the combined sample). Estimates of shared and nonshared environment were also very similar. More detailed results are available upon request.

Discussion

The present multi-wave, longitudinal study examined the genetic and environmental influences on exposure to negative life events and the stability and change in these events from late childhood to adolescence. We focused on this developmental transition because exposure to negative events during this period is a significant risk factor for many negative psychosocial outcomes. Results were compared and contrasted for dependent vs. independent life events, given previous evidence of differential etiology. We fit two developmental quantitative genetic models to the data, each with different theoretical assumptions. In the common factor model, stability of behavior is explained by the same genetic and environmental influences, whereas developmental changes are influenced by age-specific effects. In the simplex model, stability of behavior is explained by continuation effects transmitted from the earlier age to the next, and developmental changes are influenced by new influences “turned on” at each age.

Our results indicated that exposure to dependent and independent life events was stable over time, as evidenced by significant age-to-age correlations from age 9 to 16; however, the magnitude of etiological influences on the stability of life events changed considerably throughout this developmental period. These findings have important implications for etiological models of stress exposure and stress-related psychopathology during the transition from childhood to adolescence.

Etiology of Stability and Change in Negative Dependent Life Events

We hypothesized that genetic and nonshared environmental influences would explain the majority of the variance in dependent events. Both the common factor ACE model and simplex ACE model fit the data well and suggested similar conclusions regarding the pattern of etiological influences on dependent events.

The genetic influences on dependent events in our sample were mostly common or continued across ages. That is, the same genetic factors influenced exposure to these events in childhood and adolescence in our sample, rather than “new” genes being triggered in adolescence. Also, the magnitude of these common genetic influences increased during the transition to adolescence and remained elevated until age 16. These results are consistent with those from Silberg et al. (1999), who reported that the magnitude of genetic influences increased from childhood to adolescence. They are also consistent with the hypothesis that gene-environment correlation influences the etiology of dependent life events and results of studies suggesting that genetically-influenced traits—such as pubertal transition (Ge, 1994; 2001), negative emotionality and neuroticism (Lakdawalla & Hankin, 2008; Masten et al.,

1999), and vulnerability to depression (Lakdawalla et al., 2007; Mezulis et al., 2010; Silberg et al., 1999) may influence exposure to SLEs in adolescence and the ability to cope with these events. They also suggest that the genetically influenced mechanisms that contribute to dependent event exposure are influential during childhood, but play a larger role in stress exposure during the developmental transition to adolescence. Indeed, phenotypic studies assessing the developmental changes in stress exposure and reactivity indicate that vulnerability factors associated with stress and psychopathology, such as negative cognitive style, are observed in children but interact with stress more strongly in adolescence (for a review see Lakdawalla et al., 2007).

Results from the current study buttress results from phenotypic studies, suggesting that vulnerability factors for psychopathology, such as rumination, worry, or oppositional behavior, which are presumed to be heritable, may guide children and adolescents to seek out or create dependent life stressors or lead to ineffective coping responses, compounding the effects of SLEs.

Shared environmental factors decreased over time, suggesting they have less influence on exposure to dependent events as a child transitions into adolescence. The influence of family environment and dynamics on dependent events may decrease as youth spend more time away from their family and have more experiences independently of their co-twins and focus more on their peers. This pattern is consistent with studies that have found children to be more likely to report family stressors and adolescents to report interpersonal and peer stressors (Gest et al., 1999; Rudolph & Hammen, 1999; Wagner & Compas, 1990). Furthermore, several studies have found decreasing effects of shared environment on other phenotypes during adolescence (e.g., Bergen et al., 2007).

Shared environmental influences on child dependent life event exposure may include parental characteristics, such as parenting style and personality. For example, an over-controlling parenting style may serve to generate conflict in the household that affects both twins, or a parent who regularly expresses negative emotionality may model reactive, worry-prone behaviors that influence both twins' exposure to stressors, as well as their coping strategies for such events.

Nonshared environmental factors contributed to dependent events consistently from childhood to adolescence through "new", developmentally specific influences at each age. This result should be interpreted with the caveat that nonshared environmental influences may include measurement error.

Etiology of Stability and Change in Negative Independent Life Events

Our results indicated that the simplex ACE model and common factor ACE models fit the independent life events data as well as more saturated models; however, separate parameter estimates for boys and girls were necessary. We hypothesized that life events that were independent of an individual's behavior or characteristics would be minimally influenced by genetic factors, and explained mostly by nonshared environmental influences and, to a lesser extent, shared environmental influences. The simplex ACE model for boys in our sample supported our hypotheses and was consistent with the literature on the etiology of independent events in adults. Genetic factors explained 18% of the variance in independent events on average, which is generally consistent with heritability estimates in the literature for independent life events (17% in a review by Kendler and Baker, 2007). The results suggest that independent life events are more truly environmental events that are less influenced by gene-environment correlation than dependent life events.

The magnitude of shared environmental effects varied considerably across ages (mean $c^2 = .20$) and was accounted for by a combination of common or continuation and age-specific or innovation effects. The relatively higher estimate for shared environmental influences compared to those in the literature (e.g., Bemmels et al., 2008; Silberg et al., 1999) is likely due to the item content in our scale, as several of the independent event items were life events that impacted both twins (e.g., death of a family pet, older sibling moving out).

Finally, nonshared environmental influences were substantial and significant across all time points, and generally represented age specific or innovation effects. These results are consistent with the definition of independent life events as representing “true” environmental effects. As youths’ environment, and related stressors, change from childhood to adolescence (Arnett, 1999; Compas et al., 2001; Grant et al., 2004), there are “new”, age-specific environmental influences on these events across time. Again, these results may also represent a systematic change in measurement error over time.

The results for girls differed from those for boys in that genetic influences contributed significantly to the variance in independent events at almost all time points and generally increased from late childhood to adolescence (ages 9–12, mean $h^2 = .22$; ages 13–16, mean $h^2 = .45$). Further, the average heritability estimate across ages (34%) was similar to that for dependent life events (36%). This result was not consistent with our hypothesis, nor with the literature suggesting that genetic factors have little influence on exposure to independent life events (e.g., Bemmels et al., 2008). However, other studies have also found gender differences in the etiology of and exposure to independent life events. For example, Saudino and colleagues (1997) found significant gender differences in parameter estimates for “uncontrollable” life events (i.e., independent events), with higher heritability in females (22%) than males (0%). Additionally, in a large community sample of adolescents, Hankin and colleagues (2007) found that girls were more likely than boys to experience independent life events. Furthermore, the authors reported that this difference was unlikely to be a result of reporting bias, as girls did not report more overall life events than boys, and there were no gender differences in the proportion of threatening to non-threatening events reported.

One possible explanation for the significant genetic variance of exposure to independent events in girls is that a genetically influenced trait, such as personality, may influence how life events are reported in girls more so than in boys. A plausible candidate is neuroticism, given evidence linking it to increased exposure and reactivity to life stressors (Bolger & Zuckerman, 1995; Kendler et al., 2004; Magnus et al. 1993), particularly in females (Costa et al., 2001; Kercher & Rapee, 2009; Roberts & Kendler, 1999). The few genetically-informed studies assessing the association between personality and life events provide some support for this hypothesis, indicating that their association may be genetically mediated, such that genetic influences on life events can be explained- in totality (Saudino et al., 1997) or in part (Billig et al., 1996)- by genetic influences shared in common with personality constructs such as neuroticism. An important consideration for future research is the examination of etiological influences that are common and unique to personality and life event exposure, and possible gender differences in these influences.

Strengths and Limitations

Strengths of the current study include its assessment of life events at multiple time points and longitudinal design, which allowed a robust examination of a critical period in psychosocial development. In contrast, previous life events research has often focused on discrete developmental periods (e.g., adolescence, young adulthood), rather than the period of transition from childhood into adolescence. Given the importance of this transition in many developmental theories of psychopathology (e.g., Hankin & Abramson, 2001), it is important to shed light on the etiology of risk factors associated with this transition. Our

results contribute significantly to this process by estimating the magnitude and developmental patterns of genetic and environmental influences on SLEs. Furthermore, previous studies of life events have relied heavily on retrospective reports of lifetime exposure to SLEs, whereas we used annual reports of SLE exposure. This strength may serve to reduce the methodological limitations and biases associated with long-term retrospective recall of life events.

Another strength of the current study is the type of life events examined. First, we separated dependent and independent life events, as they are differentially related to the behavior and characteristics of an individual (e.g., Hammen, 1991). By comparing and contrasting the etiological pathways to both types of events, we strengthened the evidence of the impact of gene-environment correlation in exposure to dependent events and highlighted gender differences in exposure to independent events. A related strength is our focus on exposure to “daily” events or “hassles”, rather than severe or traumatic events. Much of the behavior genetic literature has focused on traumatic and severe events (e.g., sexual assault, death of a loved one, loss of employment), and daily events or hassles have often been neglected in investigations of etiological pathways to stress. Although the impact of severe events on psychopathology risk is evident (e.g., Kendler, Kuhn & Prescott, 2004), there is evidence suggesting that daily events also have significant impact on psychopathology (e.g., Pettit et al. 2010; Schneiders et al., 2006). This focus on daily events allowed us to examine events that occur regularly in the lives of youth and may increase their risk for stress-related psychopathology.

Our results should be interpreted with some caveats in mind. First, some evidence suggests that self-report measures of life events can lead to several methodological problems, including disentangling subjective versus objective threat and difficulty measuring gender differences in perceived stress (e.g., Hammen & Shih, 2008). In this vein, the use of more objective interview-based assessments has been recommended, in which ratings of events are made by judges based on contextual information (i.e., “contextual threat”), rather than based on the individual’s subjective experience. However, checklists continue to dominate the stress literature (for reviews, see Grant et al., 2004; Monroe, 2008). In the current study, our assessment of the valence of each event was determined by mean ratings and confidence intervals calculated using the entire sample; therefore, our method of identifying negative events may have reduced some of the individual subjectivity and potential for “false positive” gender differences in rates of negative life events. Still, a longitudinal quantitative genetic analysis of life events as measured by a life events interview will be an important next step for future research.

Another limitation of our life events measure is its relative focus on family-related events and limited assessment of peer-related events. As discussed, the stress literature suggests that adolescents are more likely to experience and report peer-related stressors than are children (Rudolph & Hammen, 1999; Wagner & Compas, 1990), and these peer stressors are germane to stress generation and risk for psychopathology (e.g., Rudolph et al., 2000). The limited assessment of peer-related events in our life events measure may have limited our ability to capture changes in developmentally relevant stressors across time.

Although the twin design can provide valuable information regarding the timing and magnitude of genetic and environmental influences, the partitioning of phenotypic variance into A, C, and E components is necessarily simplified. For example, interactions between genes and the environment (GxE interaction) that contribute to greater similarity between MZ than DZ twins will be captured only in the genetic influences (A), inflating the magnitude of genetic contributions. Further, with data only from twins reared together, we are unable to estimate non-additive genetic influences and shared environmental influences

simultaneously. Additionally, an environmental influence that impacts both twins but leads to different outcomes will be captured in the estimate of non-shared environment (E) rather than the shared environment. However, our results were largely consistent with those in the previous literature on exposure to life events, which includes both twin studies as well as adoption studies, which are not subject to the same methodological limitations.

Conclusions

These methodological limitations notwithstanding, the current study is, to our knowledge, the first to assess the etiology of stability and change in life events from childhood to adolescence. Our results suggest that genetic factors influence dependent life events during this transition period, and that their influence increases in adolescence. In general, these genetic influences are common to all ages rather than new genes “turning on” during adolescence. In general, shared environmental influences seem to play a greater role in exposure to life events in childhood, with their influences decreasing during the transition to adolescence. Finally, we found that nonshared environmental factors influence exposure to life events consistently from childhood to adolescence and are accounted for by new influences at each age. There was some evidence for gender differences in the etiology of exposure to independent life events, which were less genetically influenced than dependent life events in boys, but not in girls. These results suggest the possibility that genetically-influenced traits, such as personality, may have a greater influence on girls’ reporting of these events during adolescence; however, replication of these results is needed before firm conclusions regarding gender differences in exposure to life events can be made.

Life events have long been considered “environmental” risk factors for psychopathology in youth. However, the results of the current study, in line with previous work in behavior genetics (e.g., Bemmels et al., 2008; Kendler et al., 1993; Plomin et al., 1990), suggest that the negative events that youths experience result, in part, from a genetic propensity to experience stressful events. The current study provides a novel longitudinal perspective on these etiological pathways. Most importantly, we find that the genetic propensity to experience negative events is to a large extent a consistent and continuing influence across development and thus, some individuals will experience multiple negative events over several years as result of their genetic propensity. Future research is needed to determine to what extent this chronic genetic predisposition might, through its consequences for successive exposure to negative life events, have a cumulative and magnified effect on the risk for psychopathology in adolescence.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Panel A.

Panel B.

Figure 1.

Genetic influences from common factor ACE model for negative dependent life events (Panel A) and Simplex ACE model for negative dependent life events (Panel B).

Note. Standardized parameter estimates are shown. Paths and parameter estimates in bold indicate statistical significance ($p < .05$). Parameters fixed to 0.0 or to 1.0 for model fit are indicated by an asterisk. LE=life events. Panel A: A=common additive genetic influence; A₉₋₁₆=age-specific additive genetic influence. Panel B: A=additive genetic influence; a=additive genetic innovation effects.

Appendix 3.

Cholesky model for genetic influences on life events from age 9 to age 16.

Note. A=additive genetic influence; LE=life events.

Table 1

Correlations for Negative Dependent Life Events (Panel A) and Negative Independent Life Events (Panel B).

Panel A.												
	y9LE	y10LE	y11LE	y12LE	y13LE	y14LE	y15LE	y16LE				
y9LE	1.0 .498 .351											
y10LE	.427 .270 .288	1.0 .472 .424										
y11LE	.365 .340 .221	.567 .441 .369	1.0 .621 .599									
y12LE	.351 .325 .196	.437 .385 .314	.536 .452 .281	1.0 .566 .279								
y13LE	.370 .306 .221	.383 .312 .285	.491 .417 .310	.601 .530 .339	1.0 .635 .380							
y14LE	.380 .371 .327	.392 .245 .325	.399 .321 .321	.461 .419 .217	.555 .372 .245	1.0 .596 .324						
y15LE	.337 .382 .245	.333 .297 .208	.370 .339 .225	.517 .478 .096	.565 .429 .196	.632 .455 .183	1.0 .678 .195					
y16LE	.335 .285 .232	.279 .269 .217	.366 .331 .223	.433 .418 .272	.509 .447 .292	.543 .462 .206	.589 .446 .149	1.0 .586 .288				

Panel B.												
	y9LE		y10LE		y11LE		y12LE		y13LE		y14LE	
	M	F	M	F	M	F	M	F	M	F	M	F
y9LE	1.0 .214 .026	1.0 .637 .447										
y10LE	.243 .288 .217	.419 .303 .171	1.0 .362 .503	1.0 .405 .322								
y11LE	.239 -.02 .000	.239 .244 .178	.319 .255 .246	.249 .178 .124	1.0 .422 .312	1.0 .313 .358						

Panel B.																
	y9LE		y10LE		y11LE		y12LE		y13LE		y14LE		y15LE		y16LE	
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
y12LE	.161	.213	.304	.099	.376	.320	1.0	1.0								
	.244	.285	.206	.110	.231	.185	.423	.378								
	.059	-.03	.242	-.06	.280	.153	.590	.082								
y13LE	.105	.252	.213	.169	.243	.185	.399	.378	1.0	1.0						
	-.02	.347	.063	.244	.106	.111	.135	.320	.452	.492						
	.190	.297	.226	.144	-.03	.351	.270	.216	.289	.466						
y14LE	.136	.184	.092	.132	.294	.023	.321	.186	.420	.470	1.0	1.0				
	.143	.286	.180	.079	.140	.001	.354	.241	.407	.398	.465	.623				
	-.01	.132	.033	.149	.066	.148	.136	.107	.337	.202	.238	.368				
y15LE	.123	.424	.023	.225	.175	.158	.254	.249	.406	.300	.270	.419	1.0	1.0		
	-.06	.316	-.08	.183	.118	.043	.145	.330	.163	.484	.187	.517	.302	.562		
	.017	.123	.166	.239	-.07	.031	.109	-.08	.104	.095	.285	.309	.247	.053		
y16LE	.046	.140	.023	.068	.141	.283	.219	.252	.136	.235	.271	.252	.184	.288	1.0	1.0
	.003	.149	.014	.120	.165	.229	.075	.216	.074	.232	.139	.181	.259	.277	.409	.594
	.018	.150	-.08	-.04	.082	.190	-.09	.163	.059	.143	.139	.069	.008	-.04	-.01	.408

Note. On-diagonal cells: (1) Phenotypic, (2) MZ within-trait cross-twin, (3) DZ within-trait cross-twin. Off-diagonals cells: (1) Phenotypic, (2) MZ cross-trait cross-twin, (3) DZ cross-trait cross-twin. M=male, F=female, LE=life events.

Table 2

Model Fit Statistics for Dependent and Independent Life Events

Model	χ^2	df	p	CFI	TLI	RMSEA
Dependent Events						
1. Cholesky	584.18	568	.31	.996	.996	.016
2. Common Factor A	599.49	585	.33	.996	.997	.015
3. Common Factor C	599.97	588	.36	.997	.997	.013
4. Common Factor E	615.34	597	.29	.995	.996	.016
5. Common Factor ACE	649.45	626	.25	.993	.995	.019
6. Simplex A	603	591	.36	.997	.997	.013
7. Simplex C	603.1	591	.36	.997	.997	.013
8. Simplex E	607.46	593	.33	.996	.997	.015
9. Simplex ACE	649.33	633	.32	.996	.997	.015
Independent Events						
1. Cholesky	533.16	503	.17	.966	.968	.023
11. Common Factor A	547.35	525	.24	.975	.977	.019
12. Common Factor C	523	512	.35	.987	.988	.014
13. Common Factor E	558.66	536	.24	.975	.977	.019
14. Common Factor ACE	523.48	523	.48	.999	1	.003
15. Simplex A	451.27	455	.54	1	1	0
16. Simplex C	55.26	529	.25	.977	.979	.018
17. Simplex E	558.66	536	.24	.975	.978	.019
18. Simplex ACE	52.38	525	.55	1	1	0

Note. Models allowing for gender differences are italicized. df=degrees of freedom; p=significance value of chi square test; CFI=Comparative Fit Index; TLI=Tucker-Lewis Index; RMSEA=Root Mean Square Error of Approximation; A=additive genetic influence; C=shared environmental influence; E=nonshared environmental influence.

Table 3

Proportions of Variance for Negative Dependent Life Events (NDLEs)

Panel A.						
Year	ComA	ComC	ComE	SpecA	SpecC	SpecE
9	.14	.12	.04	.16	.08	.47
10	.05	.43	.08	.00	.00	.44
11	.14	.37	.04	.00	.14	.32
12	.35	.16	.02	.03	.00	.44
13	.41	.10	.05	.10	.02	.31
14	.35	.05	.21	.20	.01	.20
15	.48	.01	.17	.15	.00	.18
16	.41	.02	.07	.15	.00	.35
Panel B.						
	Genetic		Shared Environment		Nonshared Environment	
9	.295 (.0+.295)		.203 (.0+.203)		.503 (.0+.503)	
10	.172 (.137+.035)		.309 (.088+.221)		.518 (.017+.501)	
11	.204 (.204+0)		.425 (.266+.159)		.371 (.018+.353)	
12	.393 (.393+0)		.121 (.081+.040)		.486 (.016+.470)	
13	.429 (.429+0)		.130 (.103+.027)		.442 (.014+.428)	
14	.342 (.342+0)		.217 (.070+.147)		.441 (.019+.422)	
15	.626 (.626+0)		.020 (.005+.015)		.354 (.098+.256)	
16	.437 (.413+.024)		.135 (.135+0)		.427 (.052+.375)	

Note. Panel A presents the common factor model, Panel B presents the simplex model. In Panel B, the first value in a cell represents the variance in life events explained by the factor (A, C, or E). The first and second values in parentheses represent variance explained by continuation and innovation effects, respectively. ComA=common additive genetic influence; ComC=common shared environmental influence; ComE=common nonshared environmental influence; SpecA=age-specific additive genetic influence; SpecC=age-specific shared environmental influence; SpecE=age-specific nonshared environmental influence.

Table 4

Proportions of Variance for Negative Independent Life Events (NILEs)

Panel A.						
BOYS						
Year	ComA	ComC	ComE	SpecA	SpecC	SpecE
9	.00	.09	.05	.07	.00	.79
10	.03	.42	.04	.00	.00	.51
11	.00	.13	.21	.22	.07	.37
12	.05	.19	.18	.00	.29	.29
13	.21	.10	.10	.11	.03	.45
14	.36	.10	.03	.00	.00	.51
15	.17	.01	.11	.07	.05	.59
16	.08	.00	.05	.27	.00	.59
GIRLS						
9	.17	.21	.01	.22	.04	.36
10	.04	.30	.02	.00	.05	.60
11	.08	.03	.44	.00	.24	.21
12	.26	.00	.09	.00	.05	.60
13	.40	.02	.00	.00	.14	.44
14	.40	.00	.08	.11	.11	.30
15	.37	.04	.00	.10	.00	.49
16	.17	.00	.04	.20	.22	.37
Panel B.						
BOYS						
	Genetic	Shared Environment	Nonshared Environment			
9	.07 (.00 + .07)	.09 (.00 + .09)	.84 (.00 + .84)			
10	.03 (.03 + .00)	.42 (.42 + .00)	.55 (.00 + .55)			
11	.33 (.10 + .23)	.08 (.07 + .02)	.58 (.01 + .57)			
12	.11 (.04 + .07)	.43 (.20 + .23)	.46 (.03 + .43)			
13	.10 (.10 + .00)	.32 (.07 + .25)	.57 (.04 + .53)			
14	.31 (.31 + .00)	.16 (.16 + .00)	.54 (.00 + .54)			
15	.19 (.12 + .07)	.10 (.08 + .02)	.71 (.00 + .71)			

Panel A.						
BOYS						
Year	ComA	ComC	ComE	SpecA	SpecC	SpecE
16	.36 (.36 + .00)		.01 (.01 + .00)		.63 (.00 + .63)	
GIRLS						
9	.54 (.00 + .54)		.13 (.00 + .13)		.34 (.00 + .34)	
10	.19 (.19 + .00)		.22 (.00 + .22)		.59 (.01 + .58)	
11	.10 (.10 + .00)		.25 (.00 + .25)		.65 (.02 + .63)	
12	.19 (.08 + .11)		.08 (.07 + .01)		.73 (.01 + .72)	
13	.46 (.46 + .00)		.03 (.03 + .00)		.51 (.00 + .51)	
14	.54 (.34 + .20)		.02 (.02 + .00)		.43 (.02 + .41)	
15	.59 (.59 + .00)		.01 (.01 + .00)		.40 (.06 + .34)	
16	.35 (.14 + .21)		.24 (.24 + .00)		.41 (.00 + .41)	

Note. Panel A presents the common factor model. In Panel B, the first value in a cell represents the variance in life events explained by the factor (A, C, or E). The first and second values in the parentheses represent variance explained by continuation and innovation effects, respectively. ComA=common additive genetic influence; ComC=common shared environmental influence; ComE=common nonshared environmental influence; SpecA=age-specific additive genetic influence; SpecC=age-specific shared environmental influence; SpecE=age-specific nonshared environmental influence.

Appendix 1

LESA items identified as negative and dependent.

- 1 broke up with a boyfriend/girlfriend
- 2 falling out/broke up with a close friend
- 3 falling out/broke up with my best friend
- 4 physical fight with a friend
- 5 locked out of home/apartment
- 6 getting along worse than before with mother
- 7 getting along worse than before with father
- 8 getting along worse than before with brothers/sisters
- 9 had quarrels or disagreements with your parents about friends
- 10 had quarrels or disagreements with your parents about dress
- 11 had quarrels or disagreements with your parents about going places by yourself (like concerts, parties, the mall, etc.)
- 12 had quarrels or disagreements with your parents about staying out
- 13 had quarrels or disagreements with your parents about things you do at home (music you listen to, TV, telephone)
- 14 had quarrels or disagreements with your parents about cleaning up
- 15 had quarrels or disagreements with your parents about the way you talk (swearing, slang, talking back)
- 16 had a big disappointment at school
- 17 got in trouble with the teacher
- 18 got in trouble with the principal
- 19 parents were called about school problem

LESA Items identified as negative and independent

- 1 got picked on by any of your friends
 - 2 your group of friends got picked on by other groups
 - 3 home was broken into
 - 4 had something stolen at home
 - 5 had something stolen at school
 - 6 had something stolen on the way home
 - 7 was sick for over a week
 - 8 spent at least one night in the hospital
 - 9 was hurt or very ill
 - 10 pet died
-

Appendix 2

Binned ordinal variable for negative dependent and independent life events, percent of sample in each bin, and sample size at each time point.

	Bin	Year 9	Year 10	Year 11	Year 12	Year 13	Year 14	Year 15	Year 16
Negative Dependent									
0 events	0	10.8%	13.7%	13.2%	10.4%	14.0%	12.5%	17.3%	9.3%
1-2 events	1	27.9%	34.4%	35.9%	29.3%	34.3%	31.0%	27.7%	29.4%
3-5 events	2	33.2%	32.3%	30.9%	36.5%	34.3%	36.8%	35.3%	36.4%
6 events	3	28.1%	19.5%	20.1%	23.7%	17.3%	19.8%	19.8%	24.8%
Negative Independent									
0 events	0	15.8%	21.6%	25.6%	29.0%	38.6%	43.1%	52.9%	40.2%
1 event	1	24.7%	30.1%	30.7%	30.8%	28.9%	31.5%	29.1%	32.7%
2 events	2	59.5%	48.3%	43.6%	40.2%	32.4%	25.4%	18.0%	27.1%
Total N		798	764	722	758	722	642	556	813