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The relationship between life events and sense of coherence in adolescence. A longitudinal twin study

Eirunn Skaug [a,*], Nikolai Olavi Czajkowski [b,c], Trine Waaktaar [a], Svenn Torgersen [a]

[a] Department of Psychology, University of Oslo, Oslo, Norway
[b] PROMENTA Research Center, Department of Psychology, University of Oslo, Oslo, Norway
[c] Department of Mental Disorders, Norwegian Institute of Public Health, Oslo, Norway

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ABSTRACT

This three-wave study examined the relationship between life events and sense of coherence (SOC) throughout adolescence by using genetically informative random intercept cross-lagged panel models. We also examined the genetic and environmental contribution to variance in the measured constructs. The data come from a Norwegian population-based twin sample (N = 2,878). Life events and SOC were associated, and both showed substantial genetic variance. Negative longitudinal effects were observed from negative dependent life events to SOC, from SOC to negative dependent life events and from SOC to positive dependent life events. However, these longitudinal effects were negligible in magnitude. In summary, the associations between all three clusters of life events and SOC were almost completely accounted for by shared genetic influences.

1. Introduction

The theory of sense of coherence (SOC) was originally developed by Aaron Antonovsky, aiming to explain an individual's ability to cope with life stressors (A. Antonovsky, 1979). According to this theory, people with a strong SOC view the world as (1) *comprehensible*, (2) *manageable* and (3) *meaningful*. People with a strong SOC are therefore likely to (1) view stressors in life as clear and understandable, (2) believe that they have the necessary resources to meet the demands of the situation, and (3) find it meaningful to invest time and effort to cope with the challenges in question. People with a weak SOC, on the other hand, perceive the world as more chaotic, unmanageable and meaningless. Consequently, a strong SOC is believed to facilitate successful coping with stressful life situations (A. Antonovsky, 1993).

Conceptually, SOC may be considered as a personality characteristic characterized by a stable tendency to view the world more or less predictable, manageable and meaningful (H. Antonovsky & Sagy, 1986). Indeed, the SOC questionnaire (A. Antonovsky, 1987) includes items that are related to personality in terms of covering characteristic ways of thinking, feeling and behaving. Although SOC and the Big Five personality traits are theoretically distinct concepts, these constructs are conceptually related to each other (Feldt, Metsäpelto, Kinnunen, & Pulkkinen, 2007b). For example, individuals high on *neuroticism* are prone to feelings of hopelessness and are likely to use ineffective coping strategies, which characterizes persons with weak SOC. *Conscientious* individuals tend to plan and be organized, making it likely that they will perceive the world as structured and predictable, which are characteristic of a strong SOC. Furthermore, *extraverts* often have big social networks, and individuals who score high on *agreeableness* often get along well with other people. These characteristics may in turn increase a person's belief that he/she will receive social support when facing various stressors, a feature related to stronger SOC. In line with these considerations, empirical studies have shown that SOC is negatively related to neuroticism, and positively related to extraversion, conscientiousness, and agreeableness (Ebert, Tucker, & Roth, 2002; Feldt et al., 2007b; Hochwälder, 2012).

Antonovsky described SOC as an enduring and global way of looking at the world which develops throughout childhood and adolescence, and becomes stabilized by the end of young adulthood around the age of 30 (A. Antonovsky, 1993). Supporting this notion, a study by Feldt et al. (2007a) found that the rank-order stability of SOC was higher among persons over 30 years compared to younger adults. This is also in line with the empirical literature on the stability of personality traits, which finds that the rank-order stability increases throughout adolescence and peaks in adulthood (Costa, McCrae, & Lockenhoff, 2019). Supporting its trait-like nature, empirical studies have found moderate rank-order

* Corresponding author at: Department of Psychology, University of Oslo, Forskningsveien 3A, 0373 Oslo, Norway.
E-mail address: eirunn.skaug@psykologi.uio.no (E. Skaug).

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stability of SOC measured with a one-year interval and up to 13 years between measurements, both in adolescence and adulthood (Eriksson & Lindström, 2005; Hakanen, Feldt, & Leskinen, 2007; Honkinen et al., 2008), similar to the rank-order stability found for personality traits (Costa et al., 2019; Ferguson, 2010; Roberts & Delvecchio, 2000).

The introduction of the theory of SOC represented a paradigm shift from a pathogenic focus on risk factors and causes of disease, to factors promoting and maintaining good health (A. Antonovsky, 1993; Eriksson & Lindström, 2005). Numerous studies have investigated the relationship between SOC and health in adult populations. Two reviews of nearly 500 papers published between 1992 and 2003 showed that SOC is strongly related to perceived health and quality of life (Eriksson & Lindström, 2006, 2007). Fewer studies have examined adolescent populations, but the existing empirical evidence supports a relationship between SOC and health in adolescence as well (Buddeberg-Fischer, Klaghofer, & Schnyder, 2001; García-Moya, Rivera, & Moreno, 2013; Honkinen, Suominen, Välimaa, Helenius, & Rautava, 2005; Moksnes, Rannestad, Byrne, & Espnes, 2011; Nielsen & Hansson, 2007; Ristkari, Sourander, Rønning, Nikolakaros, & Helenius, 2008; Torsheim, Aaroe, & Wold, 2001).

With respect to the development of SOC, Antonovsky emphasized the importance of experiences in childhood, but also experiences in adolescence and young adulthood (A. Antonovsky, 1987). He pointed to putative SOC-promoting factors like consistency in life circumstances, a balance between demands in life and available resources, social support and the importance of playing an active role in life. Negative life events, on the other hand, could potentially weaken a person's SOC (A. Antonovsky, 1987). Beyond these theoretical assumptions, little is known empirically about causal factors behind the development of SOC during childhood and adolescence (Rivera, Garcia-Moya, Moreno, & Ramos, 2013).

With respect to the association between life events and SOC, most studies have examined negative life events. Hochwälder and Forsell (2011) located 10 studies that have examined the association between negative life events and SOC in adult populations. All 10 studies concluded that SOC was lowered by negative life events. However, most of the studies suffered from some methodological shortcomings that limited the possibility to determine whether negative life events actually were related to change in SOC (e.g., absence of a measure of SOC prior to the occurrence of the negative life events). Hochwälder and Forsell (2011) addressed some of these methodological issues by measuring negative life events and SOC at two time points, one and a half year apart. They found no strong evidence supporting the hypothesis that negative life events lowered SOC.

The existing literature on adolescent populations suggests that both negative and positive life events may be related to SOC. Results from a study by Ristkari et al. (2008) showed that adolescents who had experienced parental divorce, parental illness or death of a parent had lower mean levels of SOC compared to those who had not experienced such major life events. However, the differences were small. Furthermore, studies have reported weak to moderate negative associations between SOC and stressful life events such as peer pressure, pressure of schoolwork and family conflict (Marsh, Clinkinbeard, Thomas, & Evans, 2007; Moksnes et al., 2011; Natvig, Hanestad, & Samdal, 2006). Regarding life events associated with stronger SOC, studies have reported moderate associations between SOC and positive experiences such as social support (Marsh et al., 2007; Natvig et al., 2006) and positive family relationships (Olsson, Hansson, Lundblad, & Cederblad, 2006). However, all these studies on adolescent populations are cross-sectional and thus do not allow for conclusions about the direction of effect between life events and SOC.

Although some of the methodological challenges in the existing literature that have studied the association between life events and SOC could potentially be resolved by using longitudinal study designs, *confounding* still represents a serious challenge to valid inference. If a third variable (e.g., genes) affects both life events and SOC, this may create a spurious association between them. To our knowledge, only two studies have examined the heritability of SOC in particular. In these studies, the heritability of SOC was estimated to 35% (Hansson et al., 2008) and 45% (Silventoinen et al., 2014). Furthermore, measured variables that seem environmental almost by definition, such as life events, are influenced by genes (Kendler & Baker, 2007). Genetically informative studies often distinguish between 'dependent' and 'independent' life events. Dependent life events refer to life events that may be associated with an individual's own behavior (e.g., arguments with parents) whereas independent life events refer to life events that do not seem to have anything to do with an individual's own behavior (e.g., death of a family member). As expected, prior studies have shown that the heritability of dependent life events tends to be higher compared to independent life events (Bemmels, Burt, Legrand, Iacono, & McGue, 2008; Billig, Hershberger, Iacono, & McGue, 1996; Plomin, Lichtenstein, Pedersen, McClearn, & Nesselroade, 1990).

As measured life events are partly influenced by genetic factors, genetically informative studies (e.g., twin studies) are needed to examine whether the associations between life events and SOC are truly environmental in nature. Such designs make it possible to determine the relative role of genetic and environmental contributions to phenotypic correlations between variables. However, genetically informative studies on life events and SOC are lacking. The aim of the present longitudinal twin study is to contribute to a better understanding of the relationship between life events and SOC through (1) investigating the genetic and environmental contributions to phenotypic variance in life events and SOC in adolescence and (2) examining the direction and the nature of the relationship between these constructs. Specifically, this study will investigate to what extent the phenotypic associations between life events and SOC are due to real environmental influences and to what extent they are mediated by genetic influences.

2. Method

2.1. Sample and procedure

The participants were Norwegian adolescent twins taking part in the Oslo University Adolescent and Young Adult Twin Project (Torgersen & Waaktaar, 2019; Torgersen & Waaktaar, 2020). The project involved three waves of questionnaires throughout adolescence and one face-to-face interview with the twins when they were around 18 years old. All twin pairs born in Norway between 1988 and 1994 were invited to participate. The twins were identified through the Norwegian Medical Birth Registry. In the present study, we used data on SOC and life events derived from the questionnaires. A number of other variables relevant for personality in adolescence were also assessed in the project (Torgersen & Waaktaar, 2019; Torgersen & Waaktaar, 2020), such as Big Five (Kandler, Waaktaar, Mõttus, Riemann, & Torgersen, 2019), resilience (Waaktaar & Torgersen, 2011) and loneliness (Waaktaar & Torgersen, 2012). The data collection started in 2006 when the twins were 12 to 18 years old. Questionnaires were sent to the twins three times, with two years in between. Informed consent was obtained from both the twins and their parents. The mean ages at Wave−1, Wave−2 and Wave−3 were 15.2 (SD = 1.9), 16.9 (SD = 2.0) and 19.6 (SD = 1.9), respectively. In order to maximize the number of scales in the questionnaires, the complete scales were abbreviated based on results from a pilot study (Torgersen & Waaktaar, 2019). The final sample consisted of 2,878 twins (56% females) from 1,483 families, including 1,093 monozygotic (MZ) twins and 1,785 dizygotic (DZ) twins. That is, the percentage of complete pairs was close to one hundred. The study was approved by the Norwegian Data Inspectorate and the Regional Committees for Medical and Health Research Ethics. American Psychological Association ethical standards were followed in the conduct of the study.

2.2. Zygosity determination

The zygosity of same-sex twin pairs were partially determined through a 12-item zygosity scale where questions about similarity in appearance, how often the twins have been mixed-up with each other, and whether they believe that they are monozygotic or dizygotic were asked (Torgersen, 1979). To validate the zygosity scale, cheek swabbed DNA was drawn from a subsample of twin pairs. Twin pairs with ambiguous scores on the zygosity scale were oversampled for DNA-tests. Seventeen genetic markers were tested, with an estimated probability of misclassification <0.0001. The scores on the zygosity scale were analyzed using discriminant analysis. The same-sex twins who were not gene tested were classified as MZ or DZ twins based on discriminant analysis of the zygosity scale scores.

2.3. Power analyses

Power analyses were conducted to examine how big the genetic correlations between two phenotypes in the population had to be in order to have a statistical power of 0.80 to detect it under different scenarios of heritability and shared environmental effects. The analyses were conducted in OpenMx (Neale et al., 2016), using the R functions provided by Verhulst (2017). We used the same ratio of MZ to DZ twin pairs as in the present sample (N_{MZ} = 556; N_{DZ} = 927) and assumed complete twin pairs, and continuous variables. Assuming no shared environment and a heritability of 0.30, 0.40 and 0.50, we could with a power of 0.80 detect genetic correlations of 0.57, 0.41 and 0.30, respectively. Alternatively, in the presence of a shared environmental effect of 0.10 and a heritability of 0.30, 0.40 and 0.50, we could with a power of 0.80 detect genetic correlations of 0.52, 0.36 and 0.26, respectively. With information provided by additional covariance statistics, power to detect genetic correlations will be greater in the trivariate (longitudinal) models.

2.4. Measures

2.4.1. Life events

Life events were measured by a 38-item 2-point scale in which the participants were asked about whether they had experienced any of the set of life events the past year (0 = *no*, 1 = *yes*). The scale included 29 events from the Life Event Questionnaire for Adolescents (LEQ-A; Masten, Neemann, & Andenas, 1994) translated into Norwegian. In addition, nine new life events were added (see Table A1). Life events from the LEQ-A were classified as negative dependent (e.g., "I had many arguments with my parents"), negative independent (e.g., "One of my parents died") or positive dependent (e.g., "I received a special award for something done at school") according to Masten et al. (1994). The additional life events were assigned to clusters based on evaluation of their independence (i.e., dependent or independent) and desirability (i.e., positive or negative). The sum of reported life events within each cluster was used when analyzing the data, with possible values ranging between 0 and 14 (negative dependent), 0–19 (negative independent) and 0–5 (positive dependent). The life events scale did not include positive independent life events, probably because such life events rarely appear (Kandler, Bleidorn, Riemann, Angleitner, & Spinath, 2012).

2.4.2. Sense of coherence

SOC was measured by an abbreviated 5-item version of the Sense of Coherence 13-item scale (SOC−13; A. Antonovsky, 1987) translated into Norwegian. The final scale included the following questions: "Do you have the feeling that you are being treated unfairly?", "Do you have the feeling that you are in an unfamiliar situation and don't know what to do?", "Do you have very mixed-up feeling and ideas?", "Does it happen that you have feelings inside you would rather not feel?" and "How often do you have the feeling that there's little meaning in the things you do in your daily life?". Responses were given on a 7-point Likert scale (1 = *very often*, 7 = *rarely/never*) with higher values indicating stronger SOC. For each participant an average score was calculated. A review of 127 studies using the SOC−13 showed that the Cronbach's alpha ranged from 0.70 to 0.92 (Eriksson & Lindström, 2005). In our study, the Cronbach's alpha ranged from 0.82 to 0.83 across the three study waves, supporting the reliability of the abbreviated 5-item scale.

2.5. Statistical analysis

First, phenotypic correlations were computed to examine stability across time within each of the measured constructs and to investigate the association between them. Next, cross-twin correlations were calculated to give an initial impression of the genetic and environmental contributions to variation within and covariation between the measured constructs. Twin studies make use of the knowledge that MZ twins are genetically identical while DZ twins share, on average, half of their segregating genes. These differences allow for calculations of the variance in a phenotype (and the covariance between phenotypes) caused by genetic and environmental influences. Additive genetic influences (A; i.e., genes that together operate in an additive manner, causing similarity among family members) are inferred when the MZ correlation is greater than the DZ correlation. Shared environmental influences (C; i.e., any environmental factors that contribute to similarity among family members) are inferred when the DZ correlation is more than half the magnitude of the MZ correlation. Any remaining variance in the phenotype (or covariance between the phenotypes) not accounted for by A or C is attributed non-shared environmental influences (E). The E factor thus represents any influences that contribute to phenotypic dissimilarity within both MZ and DZ twin pairs, including measurement error. Since phenotypic differences between MZ pairs can only be due to E, an initial estimate of E can be estimated through the lack of similarity between MZ pairs.

The correlation analyses were extended using biometric analyses, implemented as structural equation models. This allows us to specify and evaluate the fit of multivariate twin models, as well as calculate standard errors and confidence intervals of parameter estimates. The structural equation modeling program, OpenMx, was used for the biometric models (Neale et al., 2016). Models were fitted to raw data using full information maximum likelihood. First, we fitted Cholesky decomposition models to data from the three measurement waves for each measured construct separately to estimate the genetic and environmental contributions to variance in SOC and the three clusters of life events. The models were fitted with separate means for males and females to account for mean-level sex differences in SOC and life events. Using data from twins, a Cholesky decomposition allows us to partition the observed phenotypic variances into their latent genetic and environmental components. The Cholesky decomposition specifies as many latent factors as there are variables for each source of variance. The first latent factor loads on all of the measured variables, the second loads on all variables except the first and so on. In this way, each factor accounts for as much of the residual variance as possible, with the last factor accounting for the remaining variance in the last measured variable. For each construct, we first fitted a full ACE model, followed by reduced models (AE, CE and E). Relative model fit was determined by comparing the models' Akaike's information criterion (AIC; Akaike, 1987), with lower values indicating better model fit.

Next, genetically informative cross-lagged panel models were fitted to examine the longitudinal relationship between life events and SOC. The traditional cross-lagged panel model (CLPM) is often used to investigate causal longitudinal influences between constructs (Hamaker, Kuiper, & Grasman, 2015). In the CLPM, the autoregressive parameters reflect the rank order stability of individuals from one measurement occasion to the next. This model implicitly assumes that every person varies over time around the same mean. Thus, if there to some extent

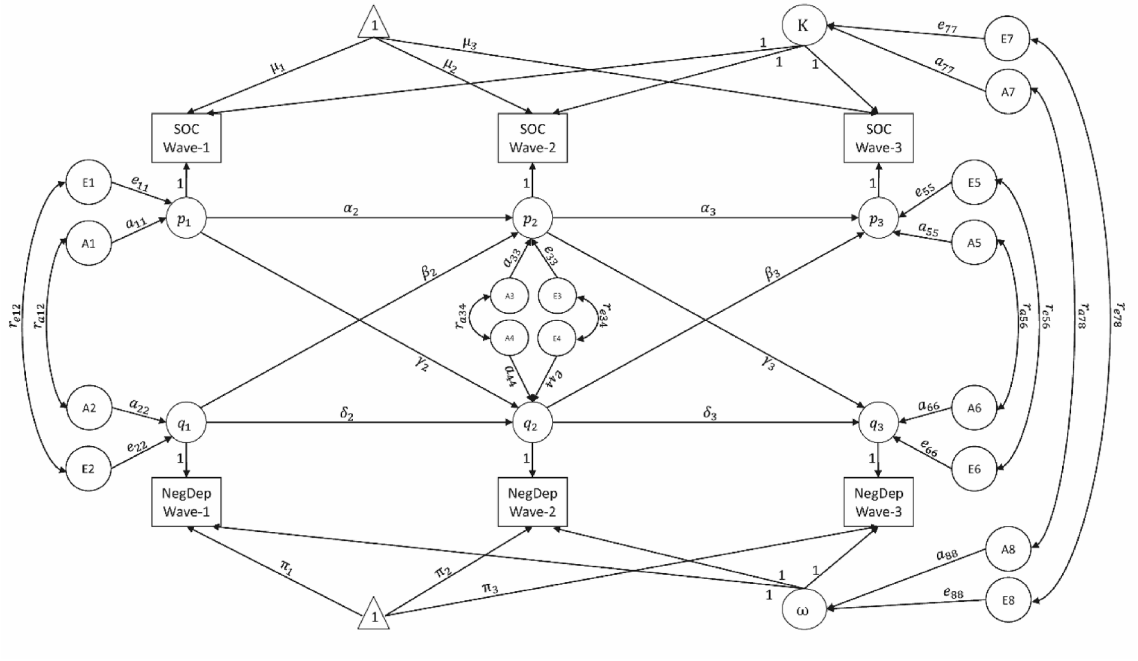

Fig. 1. Figurative Illustration of the Genetically Informative RI-CLPM for the Relationship between SOC and Negative Dependent Life Events. *Note.* For simplicity, the model is shown for one twin only and only additive genetic (A) and non-shared environmental (E) influences are shown. Triangles represent constants for the means, rectangles represent observed variables and circles represent latent variables. SOC = sense of coherence; NegDep = negative dependent life events; Wave−1, Wave−2 and Wave−3 = measurement occasions two years apart; μ_t and π_t = temporal grand means; K and ω = random intercept latent factors; p_t and q_t = within-person components; α_t and δ_t = autoregressive parameters; β_t and γ_t = cross-lagged parameters; A and a = latent additive genetic factors and paths; E and e = latent non-shared environmental factors and paths; r_a and r_e = additive genetic correlations and non-shared environmental correlations, respectively.

exist stable individual differences in mean level between persons in the phenotypes studied, the autoregressive parameters in the traditional CLPM fail to adequately account for this. This may lead to incorrect estimates of the cross-lagged parameters because the model does not separate the between-person variance from the within-person variance (Hamaker et al., 2015; Selig & Little, 2012). Hamaker et al. (2015) have proposed an alternative model, the random intercept cross-lagged panel model (RI-CLPM). This model extends the CLPM by including random intercepts that account for stable individual differences between persons (i.e., between-person variance). In this way, the cross-lagged parameters represent actual within-person processes (i.e., variance due to changes within individuals over time), which are the processes of main interest when studying reciprocal relations between variables. We first fitted three genetically informative RI-CLPMs to data, each with SOC and one cluster of life events. A graphic representation of the model with SOC and negative dependent life events is given in Fig. 1.

The RI-CLPMs were modelled following procedures as described by Hamaker et al. (2015). SOC_{it} and LE_{it} denote the measurements of SOC and life events at time point t for individual i. We modelled temporal grand means for SOC and life events (μ_t and π_t). Random intercepts (K_i and ω_i) were modelled to represent individuals' trait-like deviations from the temporal grand means. Factor loadings were constrained to one to reflect time-invariant effects. By including random intercepts, the RI-CLPM accounts for stable individual differences in mean levels of SOC and life events across the three measurement waves. The remaining variation in the data is attributed to within-person processes. Within-person components were modelled by specifying a latent variable for each observed variable (p_{it} and q_{it}), with all factor loadings constrained to one. These components (p_{it} and q_{it}), represent individuals' observed temporal deviations from their own expected score (i.e., $\mu_t + K_i$ and $\pi_t + \omega_i$). That is, individuals' time-specific deviations from their own stable level.

The autoregressive and cross-lagged paths were specified between the within-person components. The autoregressive parameters α_t and δ_t represent the amount of within-person carry-over effect. A positive α_t implies that individuals who experience stronger (weaker) SOC relative to their own stable level, are likely to experience stronger (weaker) SOC relative to their own stable level at the next measurement occasion as well. The same logic applies to δ_t. The cross-lagged parameters, β_t and γ_t, represent the degree by which within-person fluctuations in one construct predict fluctuations in another construct, after controlling for the carry-over stability effects. More specifically, a positive β_t implies that individuals who experience more (less) life events relative to their stable level of life events, are likely to experience stronger (weaker) SOC relative to their stable level of SOC at the next measurement occasion, after controlling for the carry-over stability effects in SOC. The same logic applies to γ_t.

Furthermore, we extended the RI-CLPM approach by partitioning the variance in the within-person components and the between-person components (i.e., random intercepts) into genetic and environmental sources of variance. All variances of the genetic and environmental latent factors were fixed to one and factor loadings were estimated. In addition, we modelled genetic and environmental correlations between within-time fluctuations in SOC and life events and between the random intercepts. At Wave−1, the genetic and environmental influences on p_1 and q_1 account for all within-person variance in SOC and life events. At Wave−2 and Wave−3, some of the within-person variance in SOC and life events are due to the individuals' previous state (i.e., influences from the previous age). In addition, the model allows for new sources of genetic and environmental influences at each follow-up assessment. The within-person variance in SOC and life events at Wave−2 and Wave−3 can be partitioned into four sources: (1) stability effects, (2) cross-lagged effects, (3) common effects and (4) residual effects. For example, within-person variance in SOC_2 can be partitioned into (1) genetic and environmental influences on within-person variance in SOC_1 contributing to within-person variance in SOC_2 (e.g., genetic influences: $\alpha_2{}^2 \times a_{11}{}^2$), (2) genetic and environmental influences unique to within-person variance in LE_1 contributing to within-person variance in SOC_2 (e.g., genetic influences: $\beta_2{}^2 \times a_{22}{}^2$), (3) genetic and environmental

Table 1
Descriptive statistics and phenotypic correlations.

Variable[a]	*n*	*M*	*SD*	1	2	3	4	5	6	7	8	9	10	11	12
1. Sex	2,878	0.56	0.50	–											
2. SOC_{W1}	2,567	5.12	1.28	−0.18***	–										
3. SOC_{W2}	1,911	4.98	1.27	−0.20***	0.46***	–									
4. SOC_{W3}	1,451	4.97	1.27	−0.15***	0.39***	0.47***	–								
5. $NegDep_{W1}$	2,567	2.32	2.06	0.19***	−0.53***	−0.32***	−0.23***	–							
6. $NegDep_{W2}$	1,915	2.49	2.12	0.21***	−0.37***	−0.48***	−0.33***	0.48***	–						
7. $NegDep_{W3}$	1,448	2.20	1.89	0.19***	−0.25***	−0.33***	−0.46***	0.33***	0.48***	–					
8. $NegInd_{W1}$	2,627	1.44	1.36	0.07***	−0.26***	−0.13***	−0.08**	0.36***	0.20***	0.12***	–				
9. $NegInd_{W2}$	1,918	1.46	1.43	0.08***	−0.19***	−0.27***	−0.19***	0.24***	0.40***	0.25***	0.22***	–			
10. $NegInd_{W3}$	1,453	1.39	1.37	0.10***	−0.14***	−0.14***	−0.25***	0.21***	0.26***	0.39***	0.18***	0.24***	–		
11. $PosDep_{W1}$	2,567	2.15	1.27	0.06**	−0.18***	−0.05*	0.00	0.31***	0.21***	0.15***	0.21***	0.07**	0.09**	–	
12. $PosDep_{W2}$	1,914	2.19	1.25	0.06*	−0.11***	−0.10***	−0.07*	0.18***	0.30***	0.19***	0.05*	0.22***	0.10***	0.27***	–
13. $PosDep_{W3}$	1,448	1.96	1.23	−0.02	0.01	−0.05	−0.07*	0.08**	0.18***	0.28***	−0.01	0.11***	0.18***	0.10***	0.29***

Note. SOC = sense of coherence; NegDep = negative dependent life events; NegInd = negative independent life events; PosDep = positive dependent life events; W1, W2, W3 = Wave−1, Wave−2 and Wave−3, respectively. [a] Sex coded 0 = male, 1 = female; SOC coded from 1 to 7; NegDep coded from 0 to 14; NegInd coded from 0 to 19; PosDep coded from 0 to 5.
* $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

Table 2
Cross-twin within-trait and cross-twin cross-trait correlations.

Variable	1	2	3	4	5	6	7	8	9	10	11	12
						MZ correlations						
1. SOC_{W1}	0.48***											
2. SOC_{W2}	0.34***	0.40***										
3. SOC_{W3}	0.29***	0.31***	0.44***									
4. $NegDep_{W1}$	−0.38***	−0.29***	−0.22***	0.57***								
5. $NegDep_{W2}$	−0.29***	−0.33***	−0.23***	0.43***	0.55***							
6. $NegDep_{W3}$	−0.12*	−0.24***	−0.26***	0.31***	0.32***	0.39***						
7. $NegInd_{W1}$	−0.21***	−0.09*	−0.05	0.28***	0.12**	0.06	0.63***					
8. $NegInd_{W2}$	−0.16***	−0.17***	−0.11*	0.22***	0.28***	0.10*	0.16***	0.54***				
9. $NegInd_{W3}$	−0.03	−0.01	−0.16**	0.04	0.15**	0.22***	0.05	0.10*	0.48***			
10. $PosDep_{W1}$	−0.17***	−0.12**	−0.05	0.29***	0.22***	0.13**	0.21***	0.11**	0.06	0.53***		
11. $PosDep_{W2}$	−0.09*	−0.13***	−0.06	0.19***	0.24***	0.16**	0.04	0.16***	0.04	0.26***	0.51***	
12. $PosDep_{W3}$	0.12*	−0.02	−0.05	0.00	0.05	0.11*	−0.08	0.03	0.02	0.10*	0.25***	0.42***
						DZ correlations						
1. SOC_{W1}	0.22***											
2. SOC_{W2}	0.10**	0.07*										
3. SOC_{W3}	0.15***	0.15***	0.19***									
4. $NegDep_{W1}$	−0.18***	−0.10**	−0.14***	0.27***								
5. $NegDep_{W2}$	−0.15***	−0.08*	−0.15***	0.20***	0.21***							
6. $NegDep_{W3}$	−0.09*	−0.14***	−0.18***	0.16***	0.24***	0.34***						
7. $NegInd_{W1}$	−0.14***	−0.05	−0.07*	0.19***	0.13***	0.08*	0.49***					
8. $NegInd_{W2}$	−0.13***	−0.10**	−0.11**	0.16***	0.17***	0.19***	0.14***	0.41***				
9. $NegInd_{W3}$	−0.06	−08*	−0.16***	0.04	0.11**	0.22***	0.11**	0.17***	0.43***			
10. $PosDep_{W1}$	−0.08**	0.01	−0.02	0.11***	0.10**	0.06	0.12***	0.03	0.06	0.28***		
11. $PosDep_{W2}$	−0.05	−0.02	−0.07	0.07*	0.09**	0.11**	0.00	0.09**	0.09*	0.10**	0.27***	
12. $PosDep_{W3}$	0.00	0.03	−0.03	0.00	0.06	0.11**	−0.04	0.01	0.11**	0.01	0.08*	0.21***

Note. SOC = Sense of coherence; NegDep = negative dependent life events; NegInd = negative independent life events; PosDep = positive dependent life events; W1, W2, W3 = Wave−1, Wave−2 and Wave−3, respectively.
* $p < 0.05$. ** $p < 0.01$. *** $p < 0.001$.

influences common to within-person variance in both SOC_1 and LE_1 (e.g., genetic influences: $2 \times [\alpha_2 \times a_{11} \times r_{a12} \times a_{22} \times \beta_2]$) and (4) genetic and environmental influences unique to within-person variance in SOC_2 (e.g., genetic influences: $a_{33}{}^2$).

The significance of the autoregressive and cross-lagged paths was tested by fixing all members of the parameter sets α, δ, γ and β to zero, one set at a time (i.e., four reduced models). The reduced models were compared to the full RI-CLPM by likelihood ratio χ^2 (chi square) tests. The difference in −2 log likelihood (−2ll) between two nested models is asymptotically distributed as a χ^2 with degrees of freedom equal to the difference between the number of estimated parameters in the full and in the restricted model. A non-significant χ^2 difference indicates that the restricted model should be accepted over the full model (i.e., the restricted model does not lead to a substantial loss of fit).

Finally, we fitted three genetically informative CLPMs to data, each with SOC and one cluster of life events. The CLPM can be obtained by removing the random intercepts from the RI-CLPM. Comparison of model fit between the models was examined using AIC (Akaike, 1987) and BIC (Raftery, 1995), with lower values indicating better model fit. Absolute model fit was evaluated by inspecting the comparative fit index (CFI), the Tucker-Lewis index (TLI) and the root mean square error of approximation (RMSEA). CFI and TLI values greater than 0.95 and RMSEA values < 0.06 were considered as indicating good model fit (Hu & Bentler, 1999).

3. Results

3.1. Descriptive statistics and correlations

Descriptive statistics and Pearson correlations between variables are presented in Table 1. Means, standard deviations and number of participants for the study variables by zygosity are provided in Table A2.

Table 3
Fit statistics of the Cholesky decomposition models.

Model	Model fit (AIC)[a]			
	ACE	AE	CE	E
Sense of coherence	6727.0	**6715.0**	6760.8	6927.4
Negative dependent life events	12089.5	**12082.6**	12139.3	12464.3
Negative independent life events	**7938.1**	7993.4	7947.9	8728.2
Positive dependent life events	7050.5	**7040.8**	7088.2	7427.0

Note. A = additive genes; C = shared environment; E = non-shared environment.
[a] Akaike's information criterion for the univariate Cholesky ACE, AE, CE and E models, with the best fitting model indicated in bold.

Table 4
Parameter estimates (95% CI) from the best fitting Cholesky models.

Measure	Additive genetic effects (A)	Shared environmental effects (C)	Non-shared environmental effects (E)
Sense of coherence$_{W1}$	0.47 (0.41, 0.53)	–	0.53 (0.47, 0.59)
Sense of coherence$_{W2}$	0.31 (0.23, 0.39)	–	0.69 (0.61, 0.77)
Sense of coherence$_{W3}$	0.40 (0.30, 0.48)	–	0.60 (0.52, 0.70)
Negative dependent life events$_{W1}$	0.55 (0.50, 0.60)	–	0.45 (0.40, 0.50)
Negative dependent life events$_{W2}$	0.50 (0.43, 0.56)	–	0.50 (0.44, 0.57)
Negative dependent life events$_{W3}$	0.47 (0.38, 0.54)	–	0.53 (0.46, 0.62)
Negative independent life events$_{W1}$	0.22 (0.09, 0.36)	0.40 (0.28, 0.50)	0.38 (0.34, 0.44)
Negative independent life events$_{W2}$	0.25 (0.06, 0.43)	0.29 (0.14, 0.43)	0.46 (0.40, 0.53)
Negative independent life events$_{W3}$	0.12 (0.00, 0.35)	0.39 (0.20, 0.52)	0.49 (0.41, 0.58)
Positive dependent life events$_{W1}$	0.52 (0.46, 0.57)	–	0.48 (0.43, 0.54)
Positive dependent life events$_{W2}$	0.50 (0.43, 0.57)	–	0.50 (0.43, 0.57)
Positive dependent life events$_{W3}$	0.43 (0.34, 0.51)	–	0.57 (0.49, 0.66)

Note. W1, W2, W3 = Wave−1, Wave−2 and Wave−3, respectively.

SOC and negative dependent life events were moderately stable over time, whereas negative independent life events and positive dependent life events showed less stability. As to the associations between life events and SOC, there were moderate negative within-time correlations between negative dependent life events and SOC, whereas the cross-time correlations were lower. Weak negative correlations were observed between negative independent life events and SOC, both cross-sectionally and over time. The correlations between positive dependent life events and SOC were even weaker, and surprisingly, these correlations were also negative.

When excluding the events not included in the LEQ-A (i.e., only analyzing the events from the LEQ-A), the correlations between the three clusters of life events and SOC were similar but slightly lower compared to those provided in Table 1 (see Table A3). We also calculated a sum score of all negative life events, both dependent and independent. The correlations between this total score of negative life events and SOC are provided in Table A4. Overall, these correlations were slightly lower than the correlations between negative dependent life events and SOC provided in Table 1.

Cross-twin correlations are presented in Table 2. The correlations of main interest for the genetic analyses include the cross-twin within-trait correlations and the cross-twin cross-trait correlations between SOC and the three clusters of life events. Overall, the considerably stronger resemblance within MZ pairs than DZ pairs, with the DZ correlations about half the size of the MZ correlations, suggest additive genetic influences with negligible influence of shared environmental factors. An exception is negative independent life events, which seem to have a substantial influence of shared environmental factors.

3.2. Cholesky decomposition models

Cholesky decomposition models were fitted to data to estimate the genetic and environmental contributions to variance in the measured constructs. Table 3 presents the results of fitting these models. Consistent with the pattern of cross-twin correlations, the AE model could be accepted over the full ACE model (i.e., indicated by the lowest AIC value) for SOC, negative dependent life events and positive dependent life events. For negative independent live events, an ACE model provided the best fit of data.

Standardized parameter estimates from the best fitting models are presented in Table 4. SOC and dependent life events were moderately heritable. Negative independent life events also seem to be somewhat heritable, but a substantial proportion of individual differences in negative independent life events was due to shared environmental influences. Of note, measurement error is also included in the estimates of the non-shared environmental influences, which may lead to an underestimation of the heritability estimates (and possibly the estimates of the shared environment).

Table 5
Fit Statistics of the Genetically Informative Cross-Lagged Panel Models.

Model	*df*	*ep*	AIC	BIC	RMSEA (95% CI)	CFI	TLI
SOC and NegDep							
RI-CLPM	11,821	46	17698.0	−45405.3	0.015 (0.007, 0.021)	0.986	0.987
CLPM	11,827	38	17825.9	−45309.4	0.028 (0.023, 0.033)	0.949	0.955
SOC and NegInd							
RI-CLPM	11,885	50	14453.2	−48991.8	0.007 (0.000, 0.016)	0.995	0.995
CLPM	11,892	41	14542.5	−48939.9	0.022 (0.016, 0.027)	0.954	0.958
SOC and PosDep							
RI-CLPM	11,820	46	13776.5	−49321.4	0.008 (0.000, 0.016)	0.992	0.992
CLPM	11,826	38	13863.9	−49266.1	0.022 (0.016, 0.027)	0.938	0.945

Note. SOC = sense of coherence; NegDep = negative dependent life events; NegInd = negative independent life events; PosDep = positive dependent life events; RI-CLPM = random intercept cross-lagged panel model; CLPM = cross-lagged panel model; *df* = degrees of freedom associated with the model; *ep* = number of parameters estimated; AIC = Akaike's information criterion; BIC = Bayesian information criterion; RMSEA = Root Mean Square Error of Approximation; CFI = Comparative Fit Index; TLI = Tucker-Lewis Index.

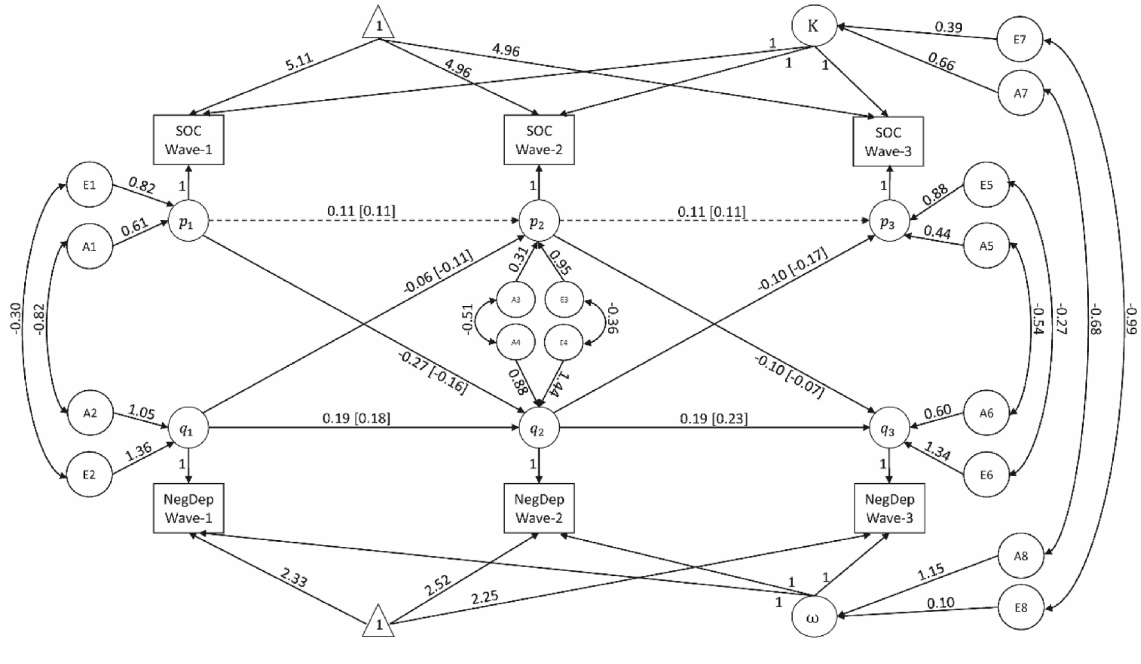

Fig. 2. Genetically Informative RI-CLPM with Unstandardized Coefficients for the Longitudinal Relationship between SOC and Negative Dependent Life Events. *Note.* Standardized coefficients are given in square brackets. Dashed lines indicate non-significant paths. SOC = sense of coherence; NegDep = negative dependent life events. See Fig. 1 for a more detailed description of model parameters.

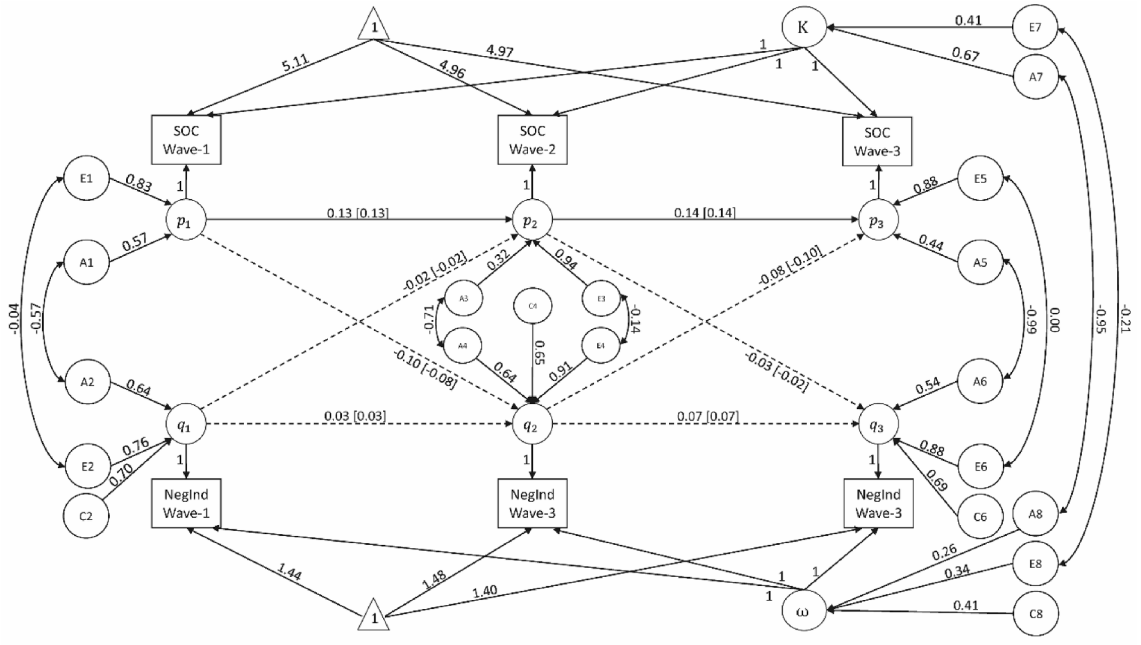

Fig. 3. Genetically Informative RI-CLPM with Unstandardized Coefficients for the Longitudinal Relationship between SOC and Negative Independent Life Events. *Note.* Standardized coefficients are given in square brackets. Dashed lines indicate non-significant paths. SOC = sense of coherence; NegInd = negative independent life events. See Fig. 1 for a more detailed description of model parameters.

3.3. Genetically informative cross-lagged panel models

Based on the best fitting Cholesky models, we included only A and E influences in the variance decomposition of SOC, negative dependent life events and positive dependent life events. In the variance decomposition of negative independent life events, we estimated all three sources of variance (i.e., A, C and E). Table 5 presents fit statistics from the genetically informative cross-lagged panel models. The RI-CLPMs provided a better fit to data compared to the traditional CLPMs (i.e., based on the lowest AIC and BIC values, the lowest RMSEA values and the highest CFI and TLI values). This indicates that there are stable individual differences between persons in SOC and/or life events, implying that it is important to account for stable between-person differences in the measured constructs before examining the reciprocal relations between them. Thus, only the results of the RI-CLPMs will be presented and discussed.

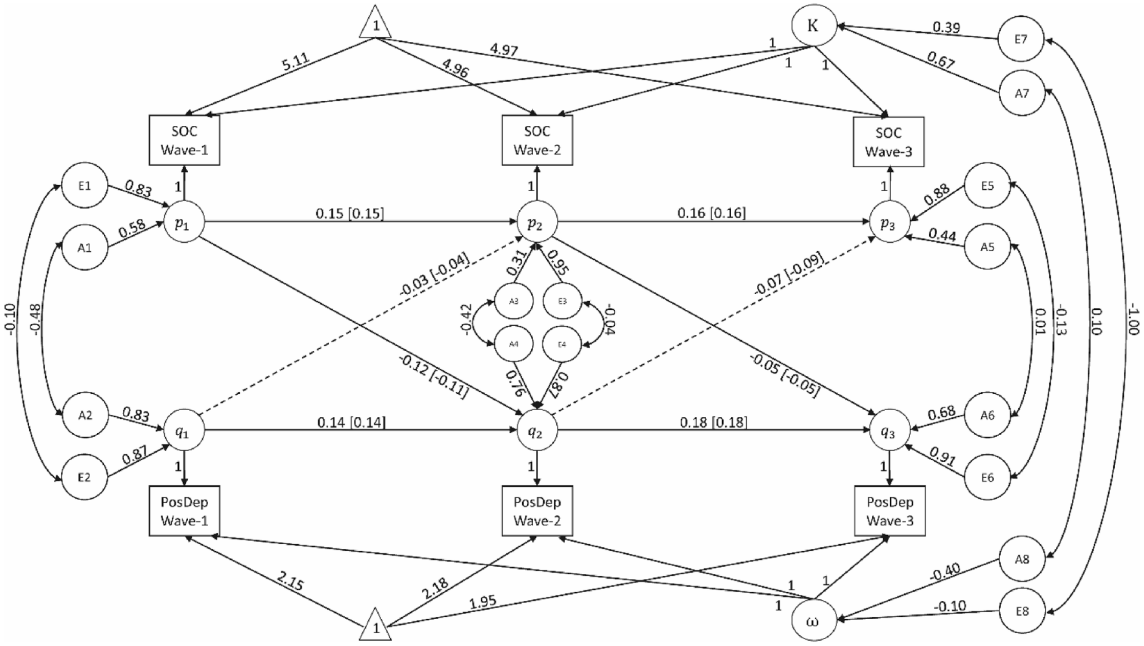

Fig. 4. Genetically Informative RI-CLPM with Unstandardized Coefficients for the Longitudinal Relationship between SOC and Positive Dependent Life Events. *Note.* Standardized coefficients are given in square brackets. Dashed lines indicate non-significant paths. SOC = sense of coherence; PosDep = positive dependent life events. See Fig. 1 for a more detailed description of model parameters.

Table 6
Proportion of variance in the measured constructs explained by the stable trait factors.

Measure	Wave−1	Wave−2	Wave−3
Sense of coherence	36.9 %[a]	37.1 %[a]	37.4 %[a]
Negative dependent life events	31.1%	29.9%	36.8%
Negative independent life events	19.2%	17.4%	18.5%
Positive dependent life events	10.5%	10.8%	11.3%

Note. [a] Mean based on the three RI-CLPMs.

Table 7
Proportion of variance in the stable trait factors due to additive genetic, shared environmental and non-shared environmental influences.

Measure	Additive genetic influences (A)	Shared environmental influences (C)	Non-shared environmental influences (E)
Sense of coherence	73.9%[a]	–	26.1%[a]
Negative dependent life events	99.2%	–	0.8%
Negative independent life events	19.2%	47.9%	32.9%
Positive dependent life events	94.1%	–	5.9%

Note. [a] Mean based on the three RI-CLPMs.

Unstandardized estimates derived from the genetically informative RI-CLPMs for the relationship between SOC and the three clusters of life events are displayed in Fig. 2, Fig. 3 and Fig. 4 (unstandardized estimated with standard errors are provided in Table A5). Standardized estimates of the autoregressive and cross-lagged paths are displayed in square brackets to enable comparison.

3.3.1. Stability in SOC and life events

Table 6 displays the proportion of variance in the measured constructs at each measurement wave explained by the stable trait factors (i.e., random intercepts). To calculate this, we divided the squared variance in the random intercept by the squared total variance in the measured construct at the given measurement wave. At Wave−1, the variance in p_1 and q_1 and the variance in the random intercepts (K and ω) contribute to the total variance (e.g., proportion of variance in SOC at Wave−1 accounted for by the random intercept = $a_{77}^2 + e_{77}^2 / a_{11}^2 + e_{11}^2 + a_{77}^2 + e_{77}^2$, see Fig. 1). At Wave−2 and Wave−3, stability effects, cross-lagged effects and common effects also contribute to the total variance (for further explanation, see Section 2.5. Statistical Analysis). As expected from the cross-time within-trait correlations, both SOC and negative dependent life events were moderately stable over time, whereas negative independent life events and positive dependent life events showed less stability.

Table 7 displays the proportion of variance in the stable trait factors attributable to additive genetic, shared environmental and non-shared environmental influences. To calculate this, we divided the squared genetic (and environmental) variance in the random intercept by the squared total variance in the random intercept (e.g., proportion of variance in the stable trait factor of SOC due to additive genetic influences = $a_{77}^2 / a_{77}^2 + e_{77}^2$, see Fig. 1). As expected from the cross-twin correlations, most of the stability in SOC and dependent life events were attributable to additive genetic influences whereas the stability in negative independent life events was mainly due to shared environmental influences.

3.3.2. The relationship between SOC and life events

With respect to the relationship between SOC and negative dependent life events, there was a strong negative genetic correlation between stable traits of SOC and negative dependent life events (−0.68), indicating shared genetic influences on the stability of these constructs. The non-shared environmental correlation between the two stable traits was estimated to −0.99. However, this correlation has no practical meaning because the non-shared environmental influences on the stable trait factor of negative dependent life events were negligible (0.8%, see Table 7). Furthermore, there were statistically significant negative cross-lagged effects from negative dependent life events to SOC and vice versa (i.e., significant changes in χ^2 when dropping these parameters from the

model). This indicates that individuals who experienced more (less) negative dependent life events than they typically do (i.e., quantitative deviations from the persons 'stable' amount of negative dependent life events) were likely to score lower (higher) on SOC than they typically do at the next assessment, and vice versa. Although the cross-lagged coefficients were significant, the effects were weak in magnitude. By squaring the standardized cross-lagged regression coefficients, fluctuations in negative dependent life events explained 1.2% (Wave−1 to Wave−2) and 2.9% (Wave−2 to Wave−3) of the fluctuations in SOC at the next measurement occasion whereas fluctuations in SOC explained 2.6% (Wave−1 to Wave−2) and 0.5% (Wave−2 to Wave−3) of the fluctuations in negative dependent life events at the next assessment.

As to the relationship between SOC and negative independent life events, there was a strong genetic correlation between stable traits of SOC and negative independent life events (−0.95). This may indicate shared genetic influence on the stability of these constructs, but the practical importance of this correlation is modest because most of the stability in negative independent life events was attributable to shared environmental influences (see Table 7). None of the cross-lagged coefficients was statistically significant, indicating that fluctuations in SOC were not predicted by fluctuations in negative independent life events two years earlier, or vice versa.

In addition to analyze negative dependent and negative independent life events separately, we also fitted a model to SOC and all negative life events considered together (see Fig. A1). Of course, the results from this analysis provided a less nuanced picture of the stability of negative life events and the relative proportion of genetic and environmental factors influencing this stability of reoccurrence of life events. However, results from this model showed similar results regarding the relationship between SOC and negative life events as when analyzing dependent and independent life events separately. That is, the cross lagged effects were weak in magnitude, only explaining a negligible proportion of the within-person variance in the measured constructs at each measurement occasion.

The genetic correlation between stable traits of SOC and positive dependent life events was almost zero (0.10), indicating that different genes are operating creating stability in these constructs. The non-shared environmental correlation between the two stable traits was estimated to −1.00. However, the practical importance of this correlation is negligible as the non-shared environmental influences on the stable trait factor of positive dependent life events were very weak in magnitude, explaining only 6% of the variance (see Table 7). The cross-lagged effects from SOC to positive dependent life events were statistically significant, whereas the cross-lagged effects from positive dependent life events to SOC were statistically non-significant. Although this might indicate a unidirectional effect from SOC to positive dependent life events, the effects were weak in magnitude. Fluctuations in SOC only explained 1.2% (Wave−1 to Wave−2) and 0.3% (Wave−2 to Wave−3) of the fluctuations in positive dependent life events two years later.

Finally, in all three models the concurrent genetic correlations between SOC and life events within each measurement occasion were greater in magnitude compared to the non-shared environmental correlations, indicating that the within-time correlations between life events and SOC were mainly due to shared genetic influences.

4. Discussion

The main purpose of the present study was to examine the longitudinal relationship between life events and SOC. Previous studies have shown that measured environments like life events are partly influenced by genetic factors (Kendler & Baker, 2007). Similarly, we found substantial genetic variance in measured life events, with heritability estimates across the three study waves ranging from 47% to 55% for negative dependent life events, from 43% to 52% for positive dependent life events and from 12% to 25% for negative independent life events. These results corroborate prior work, finding higher heritability of dependent life events compared to independent life events (Bemmels et al., 2008; Billig et al., 1996; Plomin et al., 1990). The heritability of SOC ranged from 31% to 47% across the three study waves, which are similar to the heritability estimates of SOC found in prior studies (Hansson et al., 2008; Silventoinen et al., 2014) and to heritability estimates reported for human traits in general (Polderman et al., 2015).

The rationale behind a classification of life events into 'dependent' and 'independent' events is that the association between dependent life events and some outcome variable is assumed to be confounded by a person's behavior, which may be genetically influenced. Therefore, although dependent life events may be causally related to a certain outcome, the association between dependent life events and the outcome may be confounded by genetic influences affecting them both. Independent life events, on the other hand, are considered outside a person's control and are therefore more likely to have direct/causal effects on the outcome in question (Kendler, Karkowski, & Prescott, 1999).

Genetic influences on environmental exposures such as life events give rise to gene-environment correlations. That is, a person's genetically influenced behaviors may play a role in the person's choice of environments and exposure to life events (i.e., active gene-environment correlation) or elicit certain reactions from the environment (i.e., evocative gene-environment correlation). For example, people with a genetic predisposition to more difficult temperament may select into risky environments where negative life events are more likely to occur or elicit negative reactions from parents resulting in many conflicts. Finding genetic influence on life events classified as independent may reflect the difficulty of finding clear criteria for categorization. It may also suggest that most life events are not exclusively independent. In addition, the same event may for some be dependent (e.g., arguments with a sibling due to the interviewee's behavior) and for others independent (e.g., arguments with a sibling due to the sibling's oppositional behavior). However, analyzing dependent and independent life events separately is still important, both conceptually for the reasons described in the paragraph above, and because dependent life events, potentially only genetically related to an outcome of interest, may obscure potential environmental effects of independent events on the outcome.

Results from the genetically informative RI-CLPM analyses suggest that SOC is relatively stable in adolescence. More specifically, nearly 40% of the total variance in SOC at each measurement occasion was explained by the stable trait factor. The amount of negative life events the participants experienced also seem to be somewhat stable, with approximately 30% (negative dependent) and 20% (negative independent) of the total variance at each measurement occasion being explained by the stable trait factor. Positive dependent life events showed less stability, with only about 10% of the total variance at each measurement occasion being explained by the stable trait factor. Most of the stability in SOC and dependent life events was attributable to additive genetic influences, i.e., genetic influences are the main reason why SOC is stable and why dependent life events reoccur. In contrast, shared environmental influences explained most of the stability of negative independent life events. The proportion of stable variance in life events found in this study and the finding that the recurrence of life events is mainly due to genetic influences corroborate findings from a German twin study (Kandler et al., 2012).

If we look at the within-person fluctuations in SOC, the results indicate that both genetic and non-shared environmental influences contribute to time-specific changes. The findings that most of the stability in SOC was due to genetic influences and that both genetic and environmental factors contribute to change in SOC are in line with studies that have examined stability and change in personality in adolescence (Blonigen, Carlson, Hicks, Krueger, & Iacono, 2008; Bratko & Butkovic, 2007; Kawamoto & Endo, 2015, 2019).

As to the relationship between SOC and life events, our results indicate that life events do not seem to predict change in SOC to a substantial degree, or vice versa. Although the analyses revealed statistically significant negative reciprocal longitudinal effects between negative dependent life events and SOC and statistically significant

negative unidirectional longitudinal effects from SOC to positive dependent life events, the effects were weak in magnitude. More specifically, fluctuations in negative dependent life events explained at most 2.9% of fluctuations in SOC two years later, and fluctuations in SOC explained at most 2.6% (negative dependent) and 1.2% (positive dependent) of fluctuations in life events two years later. The negative association between SOC and positive dependent life events may seem strange at first sight. However, consistent with previous studies, the correlation analysis showed that all three clusters of life events were positively correlated with each other, indicating that individuals who reported more life events of one kind, also tended to report more life events of another kind (Kandler et al., 2012; Magnus, Diener, Fujita, & Pavot, 1993; Plomin et al., 1990). Thus, experience of more positive life events also means experience of more negative life events. For example, a person who is active, outgoing and open to new experiences may experience more life events, both positive and negative (Magnus et al., 1993). Finding genetic influences on measured life events may explain a part of this covariance between life events clusters, in which genetic factors influencing a person's level of activity and openness to new experiences increase the frequency of exposure to both positive and negative life events (Kandler et al., 2012).

Overall, the nature of the phenotypic associations between life events and SOC seems mainly to be accounted for by shared genetic influences. More specifically, the phenotypic associations between negative dependent life events and SOC seem almost exclusively to be explained by the fact that they share common genes that influence the stability of both constructs. In addition, within-time fluctuations in negative dependent life events and SOC were correlated with each other, and these concurrent associations were to a greater extent explained by shared genetic influences compared to shared non-shared environmental influences. That is, people who experienced more (less) negative dependent life events than they typically do at a specific time point, also tended to experience weaker (stronger) SOC relative to their stable level at the same time point, and these concurrent associations were largely attributable to shared genetic influences. Such within-time associations were also observed between SOC and both negative independent and positive dependent life events. Similarly, these concurrent associations were mainly due to shared genetic influences. To sum up, SOC and life events share common genetic influences. For example, some people may be more likely to experience and/or report negative life events due to their genetic predisposition to perceive the world as chaotic and unmanageable which characterizes a weak SOC.

To our knowledge, this is the first genetically informative study of the relationship between life events and SOC. Hence, it is important to validate the current findings by replicating the results in studies with data from other populations (e.g., other countries and age groups). Future studies may also expand on these results by measuring other life events to see whether the results generalize to different life events. However, genetically informative studies on the relationship between life events and related constructs like personality have reported similar results as the results in the present study. Several studies have suggested that genetically influenced personality traits may be potential candidates creating genetic variance in life events (Billig et al., 1996; Kandler et al., 2012; Saudino, Pedersen, Lichtenstein, McClearn, & Plomin, 1997). These studies and the results from the present study suggest that we need to think differently about the causal structure of life events. Measures of the environment, like life events, are influenced by genes and we need to start looking for the genetics behind the observed environment.

4.1. Limitations and strengths

The results of this study should be considered in light of some possible limitations. First, related to the discussion regarding the categorization of life events above, some of the life events may not be clearly classified as independent or dependent. For example, "moving schools" may for some individuals be classified as an independent life event whereas for others moving schools may be a dependent life event. However, the life events were classified according to classifications used in previous studies (Masten et al., 1994) and to the best of our knowledge. Second, the finding that life events do not explain much variance in SOC (i.e., the cross-lagged effects were small in magnitude) does not mean that people experiencing extreme life events do not change their SOC. For example, an extreme but infrequent life event may have a huge effect on SOC, but only a small proportion of the variance in SOC is explained by this infrequent but important life event.

Furthermore, there are several assumptions related to the classical twin design, potentially threatening the validity of twin studies. First, the equal environment assumption (EEA) assumes that MZ and DZ twins are exposed to shared environmental factors to the same degree. If MZ twins are being treated more similarly or spend more time together than DZ twins, the EEA may be violated. If the EEA has not been met, the higher correlations in MZ twins compared to DZ twins may be due to environmental influences rather than genetic influences, thus overestimating the effect of the genetic influences. However, existing empirical studies have shown that the EEA generally holds (Derks, Dolan, & Boomsma, 2006; Kendler, 1993; Kendler, Neale, Kessler, Heath, & Eaves, 1993; Plomin, DeFries, Knopik, & Neiderhiser, 2013; Tambs, Harris, & Magnus, 1995). A second assumption in twin modelling is that DZ twins share half of their segregating genes. This assumption is based on random mating (i.e., parents do not share genes beyond what is expected by random chance). However, people tend to fall in love and have children with people that resemble themselves on domains like education, religion, attitudes and socioeconomic status (Neale & Maes, 2004). If this assortative mating leads to genetic similarity in parents, DZ twins would share more than 50 % of their segregating genes, thus increasing the genetic similarity between MZ and DZ twins. Consequently, assortative mating tends to overestimate shared environmental effects and underestimate genetic effects. A third assumption is that there is no gene-environment interaction. Gene-environment interaction occurs when effects of the environment depend on an individual's genotype. Depending on the nature of the gene-environment interaction, its presence can lead to biased estimates of both additive genetic, shared environmental and non-shared environmental factors (Posthuma et al., 2003). Gene by non-shared environment interactions will overestimate the effects of the non-shared environment, whereas gene by shared environment interactions will overestimate the effects of both additive genetic and shared environmental factors.

This study also has several strengths, including the use of a longitudinal design with a genetically informative sample. Twin studies represent a powerful design to partition the phenotypic variance into genetic and environmental influences, and thereby examination of the nature (i.e., genetic and/or environmental) of the association between phenotypes of interest. The large sample size in this study provided statistical power to run separate analyses of clusters of life events. From previous studies, we know that various life events may have different effects on an outcome variable, depending on event independence. Thus, grouping of life events with regards to event independence may be crucial when studying the effect of life events on an outcome variable. Furthermore, the data come from a population-based sample. This strengthens the possibility of generalization of the results. The validity of conclusions drawn from twin studies rely on the assumption that twins are representative of the general population. Several studies have confirmed this assumption. Twins have been found not to differ from singletons with regards to personality, cognitive abilities, lifestyle characteristics and both mental and somatic health (Johnson, Krueger, Bouchard, & McGue, 2012; Kendler, Martin, Heath, & Eaves, 1995; Nilsen, Bergsjø, & Nome, 1984; Posthuma, Geus, Bleichrodt, & Boomsma, 2000). Participation bias also represents a threat to generalization of findings. For instance, persons with poorer health are shown to be less likely to participate in population-based health studies (Knudsen, Hotopf, Skogen, Øverland, & Mykletun, 2010). However,

participation bias is probably more problematic for the validity of studies of prevalence compared to studies like this which focus on associations between variables (Knudsen et al., 2010). Moreover, the analysis of recruitment and dropout of the twin material used in this study showed that attrition did not influence the heritability estimates (Torgersen & Waaktaar, 2019).

Authors' Contributions.

Eirunn Skaug conducted the data analyses and wrote the original draft. Nikolai O. Czajkowski contributed to the data analyses, interpretation of data and provided critical feedback on drafts. Trine Waaktaar collected the data and provided critical feedback on drafts. Svenn Torgersen collected the data, contributed to the interpretation of data and provided critical feedback on drafts. All authors approved the final version of the manuscript for submission.

Author note

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Authors' Contributions

Eirunn Skaug conducted the data analyses and wrote the original draft. Nikolai O. Czajkowski contributed to the data analyses, interpretation of data and provided critical feedback on drafts. Trine Waaktaar collected the data and provided critical feedback on drafts. Svenn Torgersen collected the data, contributed to the interpretation of data and provided critical feedback on drafts. All authors approved the final version of the manuscript for submission.

Data Availability

Data collection was preapproved in 2005 by the Norwegian Data Protection Authority (DPA) under a clause of 20 years individual data protection and subsequent data deletion or anonymization. Thus, anonymized data may be requested after 2025.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgements

None.

Appendix

Table A1
The life events scale.

Negative dependent life events
I had an important change in physical appearance, which upset me (acne, glasses, physical development, etc.)[a]
I was a victim of violence (mugging, sexual abuse, robbery)[a]
I was disappointed by a friend
I was disappointed by someone in the family
I did not get into a group or activity that I wanted to get into (music group, sports team, theater, etc.)[a]
I had major problems with a teacher
I did much worse than I expected in an important exam or course[a]
I had less contact with one of my parents[a]
I had many arguments with my siblings[a]
I had many arguments with my parents[a]
I was bullied by other pupils/adolescents
I broke up with a girlfriend/boyfriend[a]
I had an abortion (girls) / my girlfriend had an abortion (boys)
I lost a close friend[a]
Negative independent life events
I lost a pet
I changed schools[a]
I became seriously ill or was injured[a]
At least one parent or another family member became seriously ill or was injured[a]
One of my parents died[a]
A brother or sister died[a]
Another family member died[a]
One of my close friends died[a]
Mom or Dad's friend moved in with us[a]
A member of my family ran away from home[a]
My parents divorced, moved apart[a]
One of my parents had problems at work[a]
One parent lost his or her job[a]
My mother began to work[a]
There has been a change in a parent's job so that my parent is away from home more often[a]
The family financial situation was difficult[a]
There was some damage or loss of family property (such as apartment, house, car or bike)[a]
There were many arguments between the adults[a]
Someone in the family had problems with the police[a]
Positive dependent life events
I received a special award (trophy, diploma etc.) for something done at school[a]
I became more popular with my friends
I joined a fun group of friends
I got a boyfriend/girlfriend[a]
I got a new friend

Note. [a] Question from the Life Event Questionnaire for Adolescents (LEQ-A; Masten et al., 1994). The wording in some of the questions were slightly changed from the LEQ-A.

Table A2
Means and number of participants for the study variables, by sex and zygosity.

Measure	MZM	DZM	MZF	DZF	DZOS
SOC_{W1}	5.45(1.10)	5.43(1.18)	5.03(1.33)	4.78(1.32)	5.08(1.27)
	$n = 391$	$n = 370$	$n = 587$	$n = 481$	$n = 738$
SOC_{W2}	5.32(1.14)	5.30(1.19)	4.75(1.34)	4.73(1.31)	5.00(1.21)
	$n = 266$	$n = 263$	$n = 452$	$n = 359$	$n = 571$
SOC_{W3}	5.16(1.22)	5.46(1.06)	4.87(1.30)	4.81(1.31)	4.88(1.26)
	$n = 192$	$n = 185$	$n = 366$	$n = 302$	$n = 406$
$NegDep_{W1}$	1.82(1.92)	1.84(1.77)	2.59(2.09)	2.73(2.07)	2.35(2.14)
	$n = 389$	$n = 370$	$n = 587$	$n = 480$	$n = 741$
$NegDep_{W2}$	1.90(1.92)	1.79(1.77)	2.92(2.25)	2.89(2.22)	2.49(2.04)
	$n = 267$	$n = 263$	$n = 453$	$n = 359$	$n = 573$
$NegDep_{W3}$	1.83(1.73)	1.55(1.54)	2.29(1.89)	2.64(2.12)	2.25(1.82)
	$n = 190$	$n = 185$	$n = 365$	$n = 302$	$n = 406$
$NegInd_{W1}$	1.31(1.34)	1.33(1.26)	1.53(1.42)	1.56(1.38)	1.40(1.34)
	$n = 403$	$n = 385$	$n = 593$	$n = 486$	$n = 760$
$NegInd_{W2}$	1.37(1.47)	1.31(1.40)	1.53(1.40)	1.73(1.52)	1.36(1.36)
	$n = 267$	$n = 262$	$n = 453$	$n = 360$	$n = 576$
$NegInd_{W3}$	1.27(1.34)	1.12(1.14)	1.39(1.40)	1.61(1.41)	1.40(1.40)
	$n = 191$	$n = 187$	$n = 365$	$n = 303$	$n = 407$
$PosDep_{W1}$	1.97(1.28)	2.03(1.31)	2.13(1.30)	2.26(1.16)	2.23(1.27)
	$n = 389$	$n = 370$	$n = 587$	$n = 480$	$n = 741$
$PosDep_{W2}$	2.12(1.26)	1.92(1.30)	2.18(1.28)	2.33(1.20)	2.26(1.23)
	$n = 267$	$n = 262$	$n = 453$	$n = 359$	$n = 573$
$PosDep_{W3}$	1.95(1.17)	1.87(1.22)	1.83(1.22)	1.96(1.27)	2.11(1.23)
	$n = 190$	$n = 185$	$n = 365$	$n = 302$	$n = 406$

Note. Standard deviations in parentheses; n = number of participants; MZM = monozygotic male; DZM = dizygotic male; MZF = monozygotic female; DZM = dizygotic female; DZOS = dizygotic opposite sex; SOC = Sense of coherence; NegDep = negative dependent life events; NegInd = negative independent life events; PosDep = positive dependent life events; W1, W2, W3 = Wave−1, Wave−2 and Wave−3, respectively.

Table A3
Correlations between SOC and life events, including only the life events from the LEQ-A.

Variable	SOC_{W1}	SOC_{W2}	SOC_{W3}
$NegDep_{W1}$	-0.48^{***}	-0.27^{***}	-0.21^{***}
$NegDep_{W2}$	-0.32^{***}	-0.41^{***}	-0.28^{***}
$NegDep_{W3}$	-0.23^{***}	-0.28^{***}	-0.37^{***}
$NegInd_{W1}$	-0.26^{***}	-0.13^{***}	-0.09^{**}
$NegInd_{W2}$	-0.19^{***}	-0.27^{***}	-0.19^{***}
$NegInd_{W3}$	-0.14^{***}	-0.13^{**}	-0.25^{***}
$PosDep_{W1}$	-0.11^{***}	0.01	0.03
$PosDep_{W2}$	-0.07^{**}	−0.04	0.00
$PosDep_{W3}$	−0.04	−0.04	−0.01

Note. LEQ-A = Life Event Questionnaire for Adolescents (Masten et al., 1994); SOC = Sense of coherence; NegDep = negative dependent life events; NegInd = negative independent life events; PosDep = positive dependent life events; W1, W2, W3 = Wave−1, Wave−2 and Wave−3, respectively.
$^{**}p < 0.01$. $^{***}p < 0.001$.

Table A4
Correlations between SOC and negative life events, without considering event-dependence.

Variable	SOC_{W1}	SOC_{W2}	SOC_{W3}
Negative life events$^{a}_{W1}$	-0.51^{***}	-0.30^{***}	-0.21^{***}
Negative life events$^{a}_{W2}$	-0.35^{***}	-0.47^{***}	-0.33^{***}
Negative life events$^{a}_{W3}$	-0.24^{***}	-0.30^{***}	-0.44^{***}

Note. SOC = Sense of coherence. [a] The negative life events score included all negative life events, both those considered dependent and independent.

Table A5
Unstandardized parameter estimates from the RI-CLPMs.

Parameters	Model		
	SOC and NegDep	SOC and NegInd	SOC and PosDep
Autoregressive parameters			
$SOC_{W1} \rightarrow SOC_{W2}$ (α_2)	0.11 (0.05)	0.13 (0.05)	0.15 (0.05)
$SOC_{W2} \rightarrow SOC_{W3}$ (α_3)	0.11 (0.05)	0.14 (0.05)	0.16 (0.05)
$LE_{W1} \rightarrow LE_{W2}$ (δ_2)	0.19 (0.04)	0.03 (0.04)	0.14 (0.04)
$LE_{W2} \rightarrow LE_{W3}$ (δ_3)	0.19 (0.04)	0.07 (0.04)	0.18 (0.04)
Cross-lagged parameters			
$SOC_{W1} \rightarrow LE_{W2}$ (γ_2)	−0.27 (0.07)	−0.10 (0.05)	−0.12 (0.05)
$SOC_{W2} \rightarrow LE_{W3}$ (γ_3)	−0.10 (0.07)	−0.03 (0.05)	−0.05 (0.04)
$LE_{W1} \rightarrow SOC_{W2}$ (β_2)	−0.06 (0.03)	−0.02 (0.03)	−0.03 (0.03)
$LE_{W2} \rightarrow SOC_{W3}$ (β_3)	−0.10 (0.03)	−0.08 (0.03)	−0.07 (0.04)
Random intercept variance			
A SOC (a_{77})	0.66 (0.04)	0.67 (0.04)	0.67 (0.04)
A LE (a_{88})	1.15 (0.06)	0.26 (0.17)	−0.40 (0.06)
C LE (c_{88})	–	0.41 (0.09)	–
E SOC (e_{77})	0.39 (0.06)	0.41 (0.06)	0.39 (0.06)
E LE (e_{88})	0.10 (0.10)	0.34 (0.06)	−0.10 (0.07)
Random intercept correlations			
A SOC ↔ LE (r_{a78})	−0.68 (0.05)	−0.95 (0.61)	0.10 (0.13)
E SOC ↔ LE (r_{e78})	−0.99 (1.10)	−0.21 (0.20)	−1.00 (1.06)
Within-person variance			
A SOC_{W1} (a_{11})	0.61 (0.05)	0.57 (0.05)	0.58 (0.05)
E SOC_{W1} (e_{11})	0.82 (0.04)	0.83 (0.04)	0.83 (0.04)
A LE_{W1} (a_{22})	1.05 (0.08)	0.64 (0.11)	0.83 (0.04)
C LE_{W1} (c_{22})	–	0.70 (0.08)	–
E LE_{W1} (e_{22})	1.36 (0.04)	0.76 (0.03)	0.87 (0.03)
A SOC_{W2} (a_{33})	0.31 (0.10)	0.32 (0.09)	0.31 (0.10)
E SOC_{W2} (e_{33})	0.95 (0.04)	0.94 (0.04)	0.95 (0.04)
A LE_{W2} (a_{44})	0.88 (0.09)	0.64 (0.15)	0.76 (0.05)
C LE_{W2} (c_{44})	–	0.65 (0.12)	–
E LE_{W2} (e_{44})	1.44 (0.05)	0.91 (0.04)	0.87 (0.03)
A SOC_{W3} (a_{55})	0.44 (0.08)	0.44 (0.07)	0.44 (0.08)
E SOC_{W3} (e_{55})	0.88 (0.04)	0.88 (0.04)	0.88 (0.04)
A LE_{W3} (a_{66})	0.60 (0.12)	0.54 (0.19)	0.68 (0.06)
C LE_{W3} (c_{66})	–	0.69 (0.12)	–
E LE_{W3} (e_{66})	1.34 (0.05)	0.88 (0.04)	0.91 (0.04)
Within-person correlations			
A $SOC_{W1} \leftrightarrow LE_{W1}$ (r_{a12})	−0.82 (0.07)	−0.57 (0.16)	−0.48 (0.09)
E $SOC_{W1} \leftrightarrow LE_{W1}$ (r_{e12})	−0.30 (0.05)	−0.04 (0.06)	−0.10 (0.06)
A $SOC_{W2} \leftrightarrow LE_{W2}$ (r_{a34})	−0.51 (0.21)	−0.71 (0.31)	−0.42 (0.22)
E $SOC_{W2} \leftrightarrow LE_{W2}$ (r_{e34})	−0.36 (0.05)	−0.14 (0.05)	−0.04 (0.05)
A $SOC_{W3} \leftrightarrow LE_{W3}$ (r_{a56})	−0.54 (0.22)	−0.99 (0.43)	0.01 (0.17)
E $SOC_{W3} \leftrightarrow LE_{W3}$ (r_{e56})	−0.27 (0.06)	0.00 (0.06)	−0.13 (0.06)
Means			
SOC_{W1} (μ_1)	5.11 (0.03)	5.11 (0.03)	5.11 (0.03)
SOC_{W2} (μ_2)	4.96 (0.03)	4.96 (0.03)	4.96 (0.03)
SOC_{W3} (μ_3)	4.96 (0.03)	4.97 (0.03)	4.97 (0.03)
LE_{W1} (π_1)	2.33 (0.05)	1.44 (0.03)	2.15 (0.03)
LE_{W2} (π_2)	2.52 (0.05)	1.48 (0.04)	2.18 (0.03)
LE_{W3} (π_3)	2.25 (0.05)	1.40 (0.04)	1.95 (0.04)

Note. Standard errors in parentheses. LE refers to the respective life event cluster. SOC = sense of coherence; NegDep = negative dependent life events; NegInd = negative independent life events; PosDep = positive dependent life events; W1, W2, W3 = Wave−1, Wave−2 and Wave−3, respectively; A and a = additive genetic influences; C and c = shared environmental influences; E and e = non-shared environmental influences.

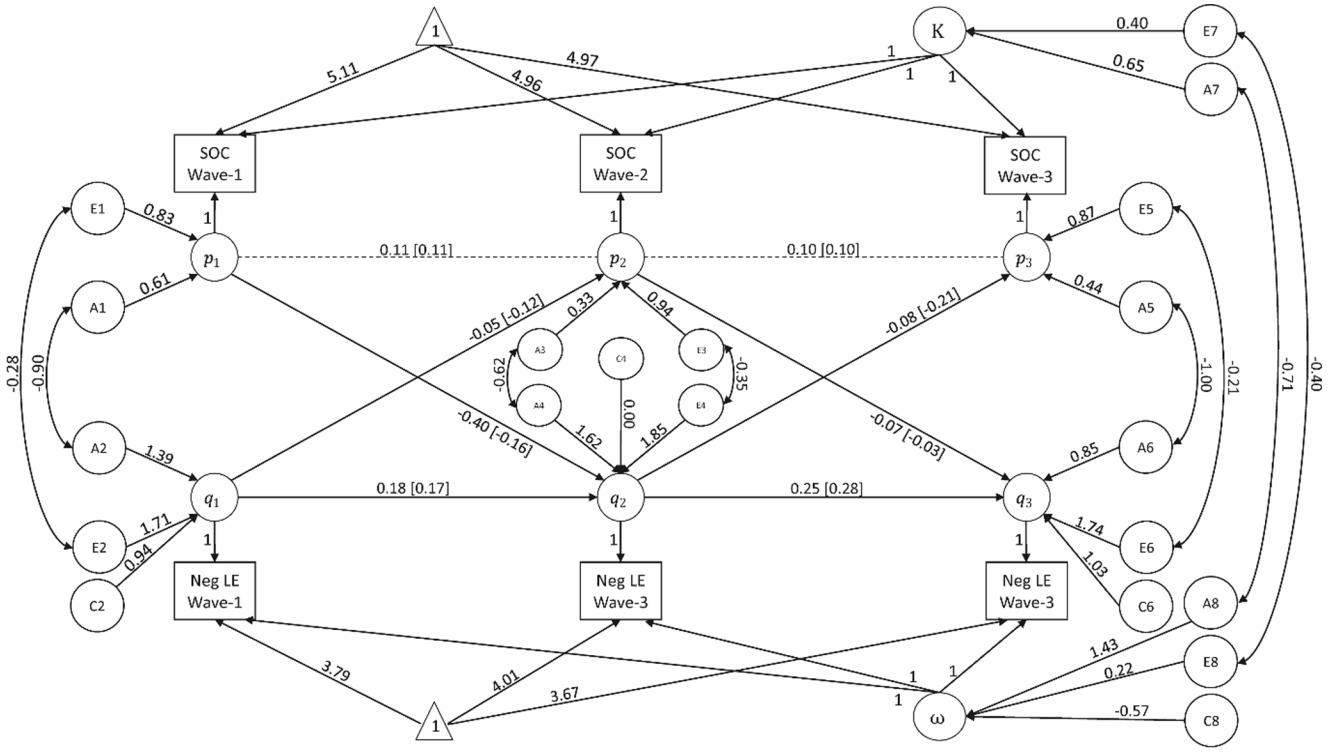

Fig. A1. Genetically Informative RI-CLPM with Unstandardized Coefficients for the Longitudinal Relationship between SOC and Negative Life Events. *Note.* Standardized coefficients are given in square brackets. Dashed lines indicate non-significant paths. SOC = sense of coherence; Neg LE = negative life events. See Fig. 1 for a more detailed description of model parameters.

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