

VU Research Portal

Out of Control

Willems, Yayouk E.; de Zeeuw, Eveline L.; van Beijsterveldt, Catharina E.M.; Boomsma, Dorret I.; Bartels, Meike; Finkenaue, Catrin

published in

Journal of the American Academy of Child and Adolescent Psychiatry
2020

DOI (link to publisher)

[10.1016/j.jaac.2019.02.017](https://doi.org/10.1016/j.jaac.2019.02.017)

document version

Publisher's PDF, also known as Version of record

document license

Article 25fa Dutch Copyright Act

[Link to publication in VU Research Portal](#)

citation for published version (APA)

Willems, Y. E., de Zeeuw, E. L., van Beijsterveldt, C. E. M., Boomsma, D. I., Bartels, M., & Finkenaue, C. (2020). Out of Control: Examining the Association Between Family Conflict and Self-Control in Adolescence in a Genetically Sensitive Design. *Journal of the American Academy of Child and Adolescent Psychiatry*, 59(2), 254-262. <https://doi.org/10.1016/j.jaac.2019.02.017>

General rights

Copyright and moral rights for the publications made accessible in the public portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognise and abide by the legal requirements associated with these rights.

- Users may download and print one copy of any publication from the public portal for the purpose of private study or research.
- You may not further distribute the material or use it for any profit-making activity or commercial gain
- You may freely distribute the URL identifying the publication in the public portal ?

Take down policy

If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

E-mail address:

vuresearchportal.ub@vu.nl

Out of Control: Examining the Association Between Family Conflict and Self-Control in Adolescence in a Genetically Sensitive Design

Yayouk E. Willems, MSc, Eveline L. de Zeeuw, PhD, Catharina E.M. van Beijsterveldt, PhD, Dorret I. Boomsma, PhD, Meike Bartels, PhD, Catrin Finkenauer, PhD

Objective: Family conflict is associated with low self-control in adolescence. Thus far research about the direction of this association is inconclusive. In this study, we sort out whether this association reflects a causal effect or whether it is explained by a common underlying cause, including genetic factors.

Method: In twin data, we fitted a series of causal models, and compared models for the association of family conflict and self-control including reciprocal causation, unidirectional causation from family conflict to low self-control, unidirectional causation from low self-control to family conflict, and common genetic susceptibility. We included data from a large sample of twins aged 14 years ($N = 9,173$), all enrolled in the Netherlands Twin Register.

Results: The results suggested a unidirectional pathway model in which family conflict leads to low self-control in adolescence, with genetic factors also playing a role in explaining the association.

Conclusion: Adolescents experiencing family conflict are at risk for showing hampered self-control capacities, with family conflict being a robust predictor of low self-control through common genetic factors but also through direct causal influences.

Key words: family conflict, self-control, twins, environment, genetics

J Am Acad Child Adolesc Psychiatry 2020;59(2):254–262.  

Adolescence is marked by a range of self-control challenges. Adolescents have to finish their homework while being tempted to check social media feeds, conforming to parental rules while striving for independence, and regulating insecurities when exposed to picture-perfect Instagram posts. Not being able to exert self-control—the inability to alter unwanted impulses and behavior, to bring them into agreement with goal-driven responses—places adolescents at risk for myriad negative outcomes.^{1,2} Especially during adolescence, characterized by a range of normative biological and social changes, self-control is key to a successful transition into adulthood.³ For example, youth who exhibit low self-control are more likely to fail in school, to drink alcohol, to be arrested for crimes, and to develop psychiatric disorders.^{4–6} Because low self-control is a powerful predictor of health, wealth, and public safety across the lifespan,^{7,8} it is important to identify factors shaping its development, including contextual factors such as family conflict.^{9,10}

Growing evidence indicates that family conflict—relational escalations in which one or more family members

engage in physical and/or verbal aggression—is associated with self-control problems. However, adolescents are not passive recipients to their environment, and the ways in which family conflict and low self-control are associated are complex. Although some association studies find that family conflict predicts low self-control,¹¹ others find that adolescents' low self-control predicts family conflict,¹² and again others suggest a reciprocal relationship.^{13,14}

How Are Family Conflict and Low Self-Control Associated?

Relational escalations and the coinciding unsafe and unpredictable family environment can undermine children's ability to regulate and to alter undesirable impulses, behaviors, and emotions.^{1,9,15} Findings from longitudinal studies demonstrate that children exposed to chronic, hostile, or poorly resolved family conflicts exhibit lower self-control.^{16,17} In addition, family conflict may have an indirect effect on children's self-control, mediated through other family processes such as poor parenting practices,¹⁸ insecure parent–child relationships,¹⁵ and chaotic household conditions.¹⁹

Alternatively, evidence suggests that low self-control predicts conflict. Individuals with low self-control are more likely to behave more aggressively towards strangers²⁰ and their romantic partner.²¹ Individuals with low self-control trigger distrust within relationships and are less successful in de-escalating conflict.²² Also, low self-control is a modest yet consistent predictor of victimization, suggesting that low self-control may evoke aggression in others.²³

Longitudinal studies investigating a reciprocal association between family conflict and self-control in adolescence are scarce and yield inconsistent results.¹¹ One study tracked the development of family conflict and low self-control across early adolescence (from age 12 to age 13 years, $N = 120$).¹² The investigators found that low self-control was linked to conflict 1 year later, but conflict was not related to lower self-control 1 year later. Another study assessed family conflict and low self-control repeatedly over 5 years from middle childhood into adolescence ($N = 2450$).¹³ They found reciprocal effects, with earlier poor self-control predicting later conflict and earlier conflict predicting later poor self-control. In contrast, another study following adolescents from age 11 to age 16 years ($N = 473$), illustrated no significant bidirectional effects between family conflict (eg, verbal hostility and punitive communication) and low self-control.²⁴ This indicates that uncertainty still exists about the direction of causation between family conflict and low self-control.

Genetic Contributions to Family Conflict and Self-Control

Importantly, caution is warranted when interpreting these effects because the findings are likely to be confounded by genetic factors that influence both exposure and outcome.²⁵ Adolescent twin studies show that individual differences in family conflict and self-control, respectively, are partly influenced by genetic factors. Heritability estimates range between 18% and 31% for family conflict, based on adolescent self-report, and between 44% and 64% for self-control, based on adolescent self-report.²⁶⁻²⁸ Given the known genetic contributions to both, it may thus well be that observed associations are explained by common genetic factors that simultaneously influence both family conflict and low self-control rather than by a direct relation.

Current Study

A design taking into account genetic and environmental sources of variance while simultaneously modelling the direction of effect is the “direction of causality” model (DoC model).^{29,30} The DoC model predicts different cross-sectional, cross-twin, cross-trait correlations (ie, the

correlation between family conflict in twins with self-control in his/her co-twin) depending on differences in heritability between two traits, allowing one to make a prediction considering the direction of the effect. This model has been applied successfully to address directionality in earlier studies.^{31,32} Thereby, this method allows us to statistically test whether the cross-twin cross-trait correlations reflect (1) a unidirectional effect whereby family conflict predicts low self-control, (2) a unidirectional effect whereby low self-control predicts family conflict, (3) a reciprocal effect, whereby family conflict and low self-control influence each other bidirectionally, or (4) a common genetic factor driving the association between family conflict and low self-control. In the present study, we apply the direction of causality model to elucidate the relationship between family conflict and self-control in a large sample of adolescent twins.

METHOD

Sample and Procedure

The Netherlands Twin Register (NTR) is a population-based study initiated in 1987 in the Netherlands, following twins and their families from birth until adulthood with age-specific assessments. In the current study, we include data of 14-year-old twins who, upon parental consent, received questionnaires with questions on physical and psychological well-being and family functioning.³³ Data collection was approved by the Medical Ethical Committee at the Vrije Universiteit medical center (2003/182).

The sample consisted of 9,173 twins aged 14 years (mean age = 14.66, $SD = 0.64$; 57.6% female participants; complete twin pairs: 85%; monozygotic twin pairs (MZ): $n = 1,861$; dizygotic twin pairs (DZ): $n = 3,315$). Participants came from all regions of the Netherlands, both rural and urban areas, and were primarily of white ethnicity. For 28.1% of the same-sex twin pairs, zygosity was determined based on DNA typing or blood group. For the remaining same-sex pairs, zygosity was determined based on items concerning physical similarities rated by their parents. Earlier research showed these items allowed for accurate determination of zygosity in 93% of the cases.³⁴

Measures

Family Conflict. This study used the Dutch translation of the Conflict subscale from the Family Environment Scale (FES) to assess adolescents' perception of family conflict.³⁵ This subscale consists of 11 items, measuring the amount of conflict, aggression, and openly expressed anger within the family, yielding a Cronbach α coefficient of 0.72. For example, the scale included statements such as “we argue a lot at home” and “sometimes family members get so angry,

they throw things.” Participants were asked to indicate if these statements were applicable to their family (1 = No, 2 = Yes), with higher scores indicating more conflict.

Self-Control. We used the eight-item ASEBA Self-Control Scale (ASCS) to assess self-control of adolescents.²⁸ The ASCS is a psychometrically sound construct, with solid construct validity (one-factor structure), acceptable internal consistency (Cronbach’s alpha coefficient of 0.73), and good test–retest reliability (test–retest correlation of 0.55).²⁸ The scale consists of items of the aggression problem scale, attention problem scale, and rule breaking scale of the ASEBA such as “I fail to finish things that I start” and “I am inattentive or easily distracted.”³⁶ The response format of the items is a 3-point scale, with response options “not true” (coded 0), “somewhat or sometimes true” (coded 1), and “very true or often true” (coded 2). An overall score for aggression, attention, and rule-breaking problems, respectively, was created. These scores tap into self-control problems, with higher scores reflecting lower overall levels of self-control. From earlier research, we know that the combination of genetic effects and unique environmental effects on the ASCS is similar to the estimates of other aspects of self-regulation such as effortful control, impulsivity, and attentional control.^{28,37}

Statistical Analyses

The association between family conflict and low self-control was tested in three consecutive steps, with each model that was tested forming the basis for the next step. All analyses were performed in *Mplus*.³⁸ We applied robust maximum likelihood as an estimator, providing less-biased standard errors.³⁹ The model’s *Mplus* syntax is provided in Supplement 1, available online.

Measurement Model. Although the measurement model of the ASCS has been studied previously,²⁸ the factor structure of the family conflict scale has not yet been tested in our sample. Earlier studies reported a unidimensional structure of family conflict.³⁵ Accordingly, we conducted a confirmatory factor analysis in our data confirming the unidimensional structure. Next, we applied a correlational model, including measurement models for both traits, to assess the phenotypic correlation between family conflict and low self-control. To correct for the dependency of the observations due to clustering in families, we applied a sandwich estimator.⁴⁰

Direction of Causality. The model including both measurement models formed the basis for the subsequent analyses: assessing the direction of causality (DoC).^{29,30} In a series of saturated models, we tested equality of means and

variances across zygosity and gender. The DoC design is built on the classical twin method with the premise that differences in the resemblance between monozygotic twins (sharing approximately 100% of their segregating genes) and dizygotic twins (sharing 50% of their segregating genes on average) can be used to parse phenotypic trait variance into genetic and environmental variance. If monozygotic twins are more alike than dizygotic twins, genetic influences are indicated. Often, the total variance of a trait and the covariance between traits is decomposed into additive genetic (A, additive effects of alleles at multiple loci), dominance genetic (D), or common environment (C, the part of the variance that is shared by members of family), and nonshared environment (E, the part of the total variance that is unique to a certain individual) variances. Residual (co)variances of the items were also decomposed into genetic and environmental effects.

Based on the literature, we know that differences in family conflict are due mainly to differences in environment.²⁷ This is reflected in the twin correlations of family conflict, with the DZ correlation being close to the MZ correlation. On the contrary, self-control is more heritable than family conflict, with the MZ correlation approximately twice as high as the DZ correlation.²⁸ This difference in the pattern of the MZ and DZ correlation is used by the DoC model to make a prediction regarding the direction of the effect. Instead of focusing on the univariate twin correlations, the DoC model examines the cross-twin cross-trait correlations (ie, the correlation between family conflict in twins with self-control in his/her co-twin), and tests specific predictions regarding the pattern of the MZ and DZ cross-twin cross-trait correlations.

If family conflict (low heritability) unidirectionally predicts self-control (high heritability), the cross-twin cross-trait correlations should reflect a DZ correlation that is close to the MZ correlation, mirroring the genetic architecture of family conflict. If self-control unidirectionally predicts family conflict, the cross-twin cross-trait correlations should reflect MZ correlations that are approximately twice as high as the DZ correlations, mirroring the genetic architecture of self-control. If the association is bidirectional, the cross-twin cross-trait correlations reflect a combination of the MZ and DZ pattern of family conflict and self-control.³⁰ Structural equation modelling allows us to assess which of these directional models fits the cross-trait cross-twin correlations best.

Bivariate Genetic Correlational Model. Considering the heritability of family conflict and low self-control, their association might be explained by a common underlying genetic factor instead of a causal effect. Therefore, we also

applied a bivariate genetic correlational model to investigate the relative contributions of genetic and environmental factors to the variance in family conflict and self-control and their covariance. We opted for adding a genetic correlation (denoted as R_g), rather than a nonshared correlation (denoted as R_e) because of the major contribution of genetics on individual differences in self-control.²⁸

Assessing Model Fit. Several indices were applied to assess which of the aforementioned models was most likely to be reflected by the data. Goodness of fit was evaluated using the root mean square error of approximation (RMSEA) and the comparative fit index (CFI), adopting the cut-off scores defined elsewhere.⁴¹ Nested submodels (eg, bidirectional versus unidirectional models) were compared by hierarchic χ^2 tests. The χ^2 statistic was computed by subtracting $-2LL$ (log-likelihood) for the full model from that for a reduced model ($v2 = -2LL1 - (-2LL0)$). If a p value higher than 0.01 was obtained from the χ^2 test, the fit of the constrained model was not significantly worse than the fit of the more complex model. In this case, the constrained model was kept as the most parsimonious and best-fitting model. To compare nonnested models (eg, direction of causation models versus bivariate genetic correlational model), we applied Akaike's Information Criterion (AIC). The AIC addresses the trade-off between the descriptive accuracy and parsimony of the model, with lower AIC indicating a better fit of the model to the observed data. To better understand AICs of competing models, we computed AIC weights (in R, with the Multi-Model Inference "MuMIn" Package).⁴² AIC weights are ratios that reflect differences in AIC with respect to the AIC of the best candidate model, thereby obtaining estimates of the relative likelihood of the model.⁴³ The convenience of AIC weights is that they are distributed according to relative probability, translated to percentages, so they have interpretable meaning, ranging between 0% = very unlikely to 100% = very likely that the model represents the true model. This allowed us to quantify the amount of statistical confidence for each of the models, providing insights into the relative advantage of competing models.⁴³

RESULTS

Descriptive Statistics

There were no significant mean or variance differences in family conflict and self-control between monozygotic and dizygotic twins, or between boys and girls. On average, adolescents scored a mean of 14.34 (minimum = 11, maximum = 22, SD = 2.45) on family conflict, and a mean of 4.23 (minimum = 0, maximum = 16, SD = 2.76) on self-control. All MZ correlations were higher than DZ

correlations, suggesting a role of genetic effects for both family conflict and self-control (Table 1). For family conflict, DZ correlations were close to MZ correlations, implying a role of the shared environment. For self-control, MZ correlations were twice as high as DZ correlations, implying a role of dominant genetic influences.

Measurement Model

Applying a one-factor model for the family conflict scale suggested room for improvement (RMSEA = 0.038; CFI = 0.930; estimated parameters [EP] = 33). Upon inspection of the modification indices, we allowed the residuals of items "hitting" and "swearing" to correlate. The correlation between these items suggests that both tap into more expressive forms of family conflict. This revised model fit significantly better than the initial model $\Delta\chi^2(df = 1) = 109.94$, $p < .001$, and showed good overall model fit for the one-factor model (RMSEA = 0.031; CFI = 0.95; EP = 34; see Figure S1, available online). The one-factor structure of the self-control scale has been tested elaborately in our data published in earlier work,²⁸ based on sum scores of "attention problems," "aggression problems," and "rule breaking" items, showing good fit (RMSEA = 0.00; CFI = 1.00; EP = 9; see Figure S2, available online). For the phenotypic association between family conflict and low self-control, we extended the measurement models by correlating family conflict and low self-control (Figure 1), showing good overall model fit and a moderate to strong correlation (RMSEA = 0.028; CFI = 0.96; EP = 44; $r = 0.61$; 95% CI = 0.58–0.64).

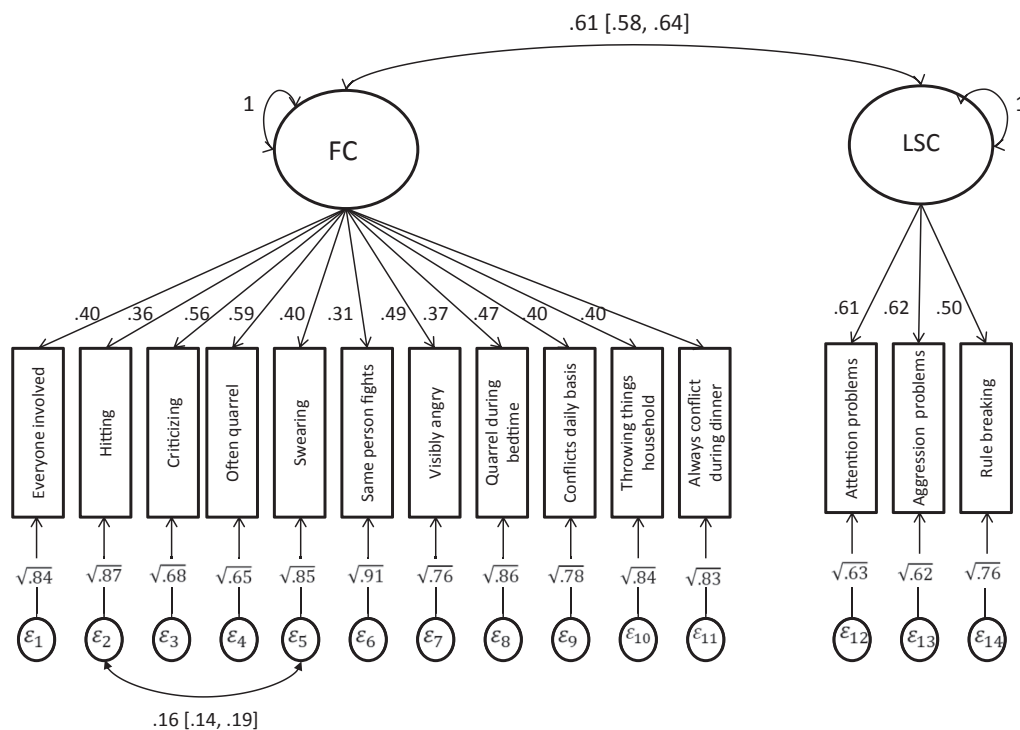
Direction of Causation Model

The direction of causality model (DoC) works well when the phenotypic correlation between traits is robust, the traits differ in their heritability, and measurement error is accounted for with a measurement model.^{29,30} Considering the large phenotypic correlation between family conflict and

TABLE 1 Twin Correlations and Cross-Twin Cross-Trait Correlations

	MZ	DZ
Twin Correlations		
Family Conflict	0.73 [0.69, 0.77]	0.62 [0.58, 0.66]
Low Self-control	0.60 [0.58, 0.63]	0.32 [0.25, 0.38]
Cross-Twin Cross-Trait Correlations		
Family Conflict – Low Self-control	0.46 [0.41, 0.50]	0.33 [0.29, 0.37]

Note: DZ = dizygotic twin pairs; MZ = monozygotic twin pairs.

FIGURE 1 Measurement Model

Note: FC = family conflict; LSC = low self-control.

self-control, the higher heritability of self-control (50%–60%)²⁸ as compared to family conflict (30%–40%),²⁷ the application of measurement models, and the large sample size (>9,000 twins), we were confident for the model to work well.

We decomposed the phenotypic twin correlations into the A, C (for family conflict), or D (for self-control), and E variance components. It is not possible to estimate both C and D in the same model. Based on the previous literature and on the twin correlations, we therefore estimated an ACE model for family conflict and an ADE model for self-control. Considering that previous work on the same data found no sex differences in heritability for family conflict nor self-control, we did not consider the genetic and environmental components to differ between boys and girls.^{27,44} In line with earlier studies applying direction of causality models,^{31,32} residual variances and correlating residuals (for the family conflict scale) of the measurement model were also decomposed into genetic and environmental variance (see Table S1, available online, for the estimates). The contribution of D to low self-control was not significant. Therefore, we omitted this path, resulting in a constrained, and parsimonious model that was not significantly worse than the fit of the more complex model $\Delta\chi^2$ ($df = 1$, $N = 5,176$ pairs) = 2.50, $p = .11$. This bidirectional direction of

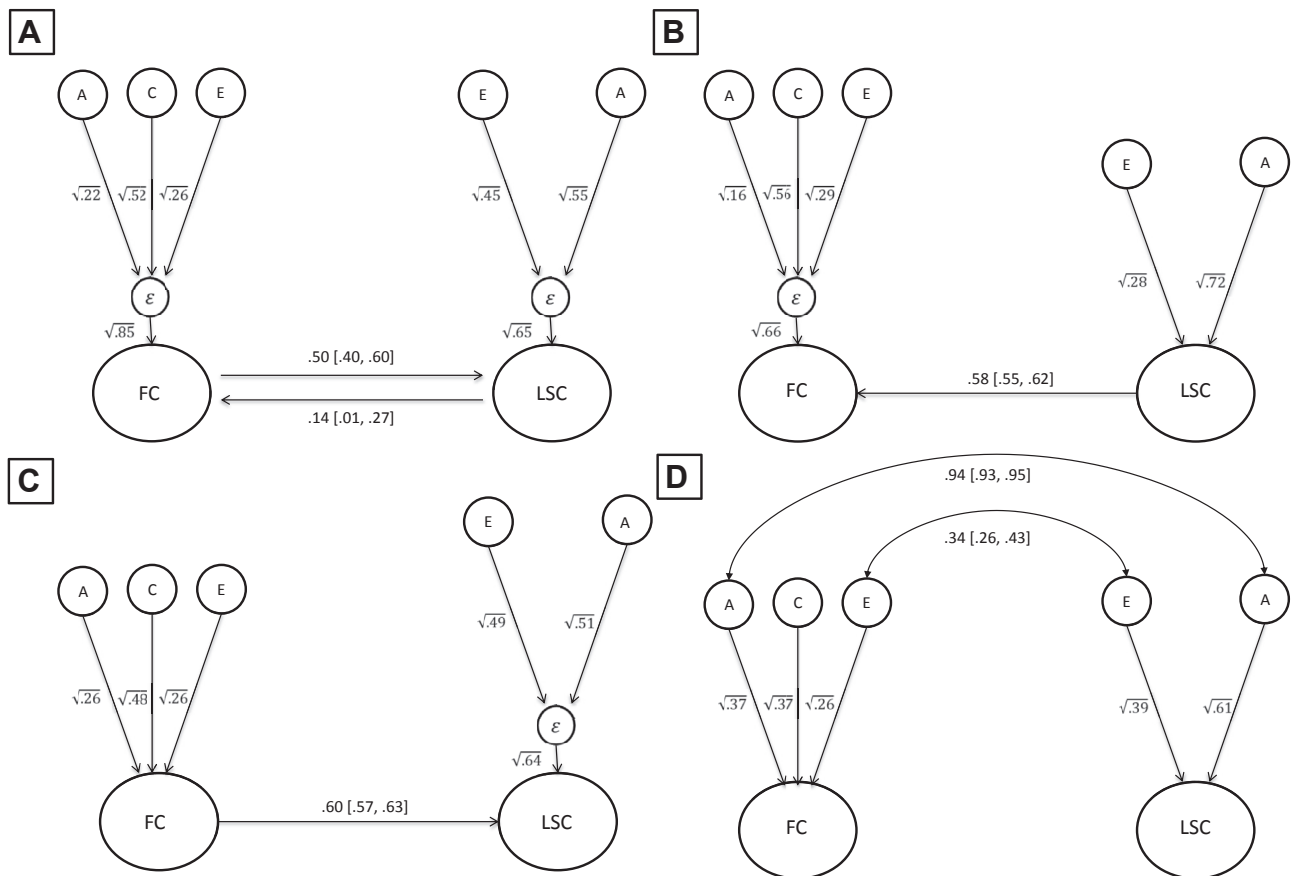
causation model showed good model fit (RMSEA = 0.02; CFI = 0.95) (Figure 2A).

Next, we compared the unidirectional model low self-control *to* family conflict (Figure 2B) to the bidirectional model (Figure 2A), resulting in a large deterioration in fit $\Delta\chi^2$ ($df = 1$, $N = 5,176$ pairs) = 33.23 $p < .001$. This indicates that the unidirectional model from low self-control to family conflict shows a worse fit to the data than the bidirectional model. Alternatively, we compared the unidirectional model family conflict *to* low self-control (Figure 2C) to the bidirectional model (Figure 2A). Results showed that the unidirectional model was not significantly worse than the bidirectional model $\Delta\chi^2$ ($df = 1$, $N = 5,176$ pairs) = 2.63, $p = .10$. This indicates that a unidirectional model from family conflict to low self-control shows a better fit to the data than the bidirectional model.

Bivariate Genetic Correlational Model

Fitting the bivariate genetic correlational model (Figure 2D) resulted in an AIC increase of 24.42 compared to the unidirectional model from family conflict to low self-control (Figure 2C), indicating that the unidirectional causal model fits the data better than the genetic correlational model.

Considering the high genetic correlation between the two traits (see Figure 2D), we subsequently tested the best

FIGURE 2 Direction of Causality

Note: (A) bidirectional model, (B) unidirectional model low self-control to family conflict, (C) unidirectional model family conflict to low self-control, and (D) bivariate genetic correlational model. In the interest of space, only the top parts of the models are shown. FC = family conflict; LSC = low self-control.

fitting model of the direction of causation tests (unidirectional model from family conflict to self-control), and added a common genetic correlation (Figure 3). The AIC of the model family conflict to low self-control including the genetic correlation (AIC = 158065.41) was lower than the model family conflict to low self-control excluding the genetic correlation (AIC = 158070.565), indicating that adding a genetic correlation fits the data well.

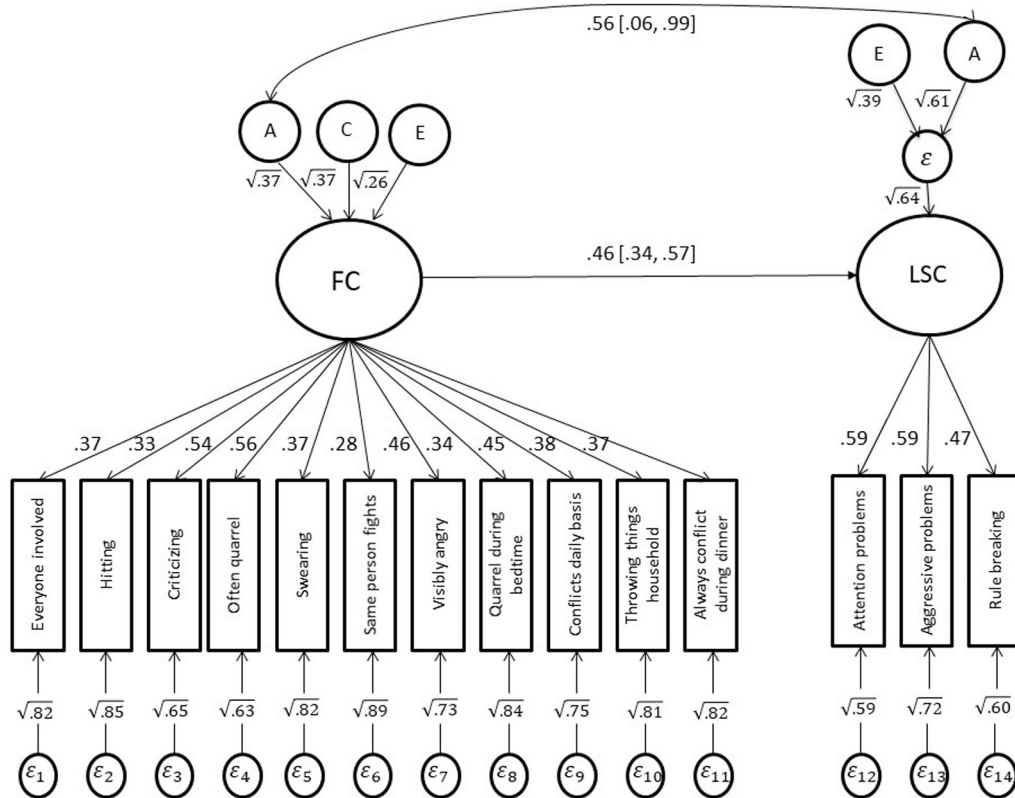
Although it is current practice to accept a single model based on the lowest AIC value, differences in models based on AIC values are difficult to unambiguously interpret. Calculating AIC weights allow for a more straightforward interpretation (Table 2).⁴³ The AIC weights showed that indeed the statistical confidence for the last model was stronger (Figure 3, probability of 68%) than the statistical confidence for the bidirectional model (Figure 2A, probability of 26%). Thus, the unidirectional model from family conflict to self-control (0.46, 95% CI = 0.34–0.57), including common genetic influences ($R_g = 0.56$, 95%

CI = 0.12–0.99), is most likely to be supported by the data.

DISCUSSION

Studies consistently find that poor decisions made during adolescence due to insufficient self-control ensnare adolescents in lifestyles that have effects lasting into adulthood (eg, poor physical health, overweight, financial issues, and substance use).^{7,8} Supporting theoretical models,^{9,10} we found that family conflict predicts low self-control in adolescence. More specifically, in line with the self-control strength model of family violence,^{9,10} we see impairments of self-control in the wake of family conflict. To our knowledge, this is the first study to investigate this association in a large, genetically sensitive design (>9,000 twins) allowing us to identify the direction of effect while controlling for genetic confounds. Our results suggest a unidirectional path whereby family conflict predicts lower self-control in adolescence, with genetic factors also playing a role in explaining the association.

FIGURE 3 Unidirectional Model With Genetic Correlation



Note: In the interest of space, decomposition of residual errors, and correlation between residuals, into genetic and environmental variance is not illustrated in this figure (see Table S1, available online, for the estimated parameters). Akaike's Information Criterion weights show that this model supports the data best. FC = family conflict; LSC = low self-control.

This finding indicates that families are at risk because they share the same genes, with the same genes influencing the presence of family conflict and the risk for having low self-control. Crucially, the findings suggest a directional effect of family conflict on self-control. The exertion of self-controlled behavior requires energy and resources.²¹ It is possible that

family conflict diminishes an individual's limited resources, because it requires attention and vigilance, for example, which cannot be invested in engaging in self-control and resisting temptations.²¹ Alternatively, the presence of conflict and subsequent emotional activation may impair prefrontal cortex functioning, decreasing the ability to engage self-regulatory processes.⁴⁵ It is also possible that conflict gets in the way of social support and guidance necessary to develop and strengthen self-control.¹⁸ There may also be other reasons, such as that family conflict possibly creates unpredictability that makes the exertion of self-control and delay of gratification disadvantageous or risky for the individual.⁴⁶

One possible implication of our results is that practitioners and professionals should be aware that low self-control may result from the experience of conflict in the home environment rather than treating them as separate problems. In addition, both of the underlying pathways explaining the association between family conflict and self-control—the contextual risk of family conflict and the genetic similarities within the family—manifest at the family level. This suggests that family-based approaches for intervention or prevention

TABLE 2 Akaike's Information Criterion (AIC) Weights of the Competing Models

Model	Estimated Parameters	AIC	AIC weights
Correlational	73	158094.98	0.00
Bidirectional	73	158067.31	0.26
Low self-control → family conflict	72	158131.77	0.00
Family conflict → low self-control	72	158070.57	0.05
Family conflict → low self-control (with Rg)	73	158065.41	0.68

Note: Rg = genetic correlation.

strategies could be promising.⁴⁷ Such approaches, targeting family conflict, might break the potentially vicious cycle of maladaptive self-control development. Empirical research with controlled trials would be needed to confirm this suggestion. Importantly, we need to acknowledge the complexity of family conflict and the environmental factors associated with it, and solely targeting family conflict is unlikely to be the one and only way to help those families at risk.

There are some limitations in this study. Establishing causality is a complex endeavor. Although our results suggest causality, we cannot infer it with full certainty. Despite its powerful design, our study is based on cross-sectional data and does not explicitly model person–environment transactions from earlier time points. Future research is needed to apply complementary research designs with longitudinal data (to see how childhood experiences shape adolescence) or observational designs (to further examine underlying mechanisms). In addition, both family conflict and low self-control measures relied on self-reports. An important extension of our study would therefore be to include a multiple-rater approach. However, one advantage of using adolescents' self-reports is that they reflect their subjective experience, and the way their psychological reality influences their behavior may be at least as important as parental perceptions of family functioning.⁴⁸

Moreover, our results show a common genetic pathway between family conflict and self-control, potentially indicating the presence of gene–environment correlation (rGE, when there is a correlation between the genotype the adolescent inherits and the environment in which the adolescent is raised). Unfortunately, our model does not allow us to distill whether the genetic pathway reflects genetic pleiotropy or, if present, which specific gene–environment correlation (eg, passive, evocative, or active gene–environment correlation).⁴⁷ Future research applying adoption data or children of twin data is strongly recommended, as this would allow us to further unravel the dynamic processes underlying the family conflict–self-control link.^{49,50}

To conclude, most adolescents develop well and find their way into society without many problems, but not all adolescents do. Poor decisions and reckless behaviors due to

low self-control in adolescence are concurrently and longitudinally costly,^{7,8} and revealing possible factors contributing to individual differences in self-control is necessary. Applying a genetically sensitive design, this study points to the existence of a directional effect, in the presence of a genetic correlation, of family conflict on low self-control in adolescence. Future intervention and prevention practices should take this mechanism into account, when aiming to target adolescents at risk for developing low self-control.

Accepted March 5, 2019.

Drs. de Zeeuw, van Beijsterveldt, Boomsma, Bartels, Finkenauer, and Ms. Willems are with Vrije Universiteit Amsterdam, The Netherlands. Drs. Drs. de Zeeuw, van Beijsterveldt, Boomsma, Bartels, and Ms. Willems are also with the Amsterdam Public Health Research Institute, Vrije Universiteit Amsterdam, The Netherlands. Drs. Boomsma and Bartels are also with the Neuroscience Campus Amsterdam, The Netherlands. Dr. Finkenauer and Ms. Willems are also with Universiteit Utrecht, The Netherlands.

This work was supported by the following grants: Database Twin Register [Netherlands Organisation for Scientific Research (NWO) 575-25-006]; Spinozapremie (NWO/SPI 56-464-14192); a Twin-Family Database for Behavior Genetics and Genomics Studies (NWO 480-04-004); BBMRI –NL: Biobanking and Biomolecular Resources Research Infrastructure (NWO 184.021.007); Genetics of Mental Illness: European Research Council (ERC-230374); Genetic and Family Influences on Adolescent Psychopathology and Wellness (NWO 463-06-001); a Twin-Sib Study of Adolescent Wellness (NWO-VENI 451-04-034); Genetic Influences on Stability and Change in Psychopathology From Childhood to Young Adulthood (NWO/ZonMW 91210020); Amsterdam Public Health Research Institute (former EMGO+); Neuroscience Amsterdam Research Institute (former NCA); Developmental Study of Attention Problems in Young Twins (NIMH RO1 MH58799-03); Determinants of Adolescent Exercise Behavior (NIH 1R01DK092127-01); Netherlands Twin Registry Repository: Researching the Interplay Between Genome and Environment (NWO-large investment 480-15-001/674); Consortium on Individual Development (CID), which is funded through the gravitation program of the Dutch Ministry of Education, Culture and Science and the Netherlands Organization for Scientific Research (NWO 0240-001-003); ACTION funded by the European Union Seventh Framework Program (FP7/2007-2013, grant no. 602768); and the Research Talent Fund (NWO 406-15-132).

Drs. de Zeeuw and Bartels and Ms. Willems served as the statistical experts for this research.

The authors warmly thank all NTR participants who participated in this study. They would also like to thank Elsje van Bergen, PhD, for sharing her elaborate analytic scripts and Michel Nivard, PhD, both of Vrije Universiteit Amsterdam, for his invaluable statistical insights.

Disclosure: Drs. de Zeeuw, van Beijsterveldt, Boomsma, Bartels, and Finkenauer, and Ms. Willems have reported no biomedical financial interests or potential conflicts of interest.

Correspondence to author Yayouk E. Willems, MSc, van der Boechorststraat 1, 1081 BT, Amsterdam, The Netherlands; e-mail: y.e.willems@vu.nl

0890-8567/\$36.00/©2019 American Academy of Child and Adolescent Psychiatry

<https://doi.org/10.1016/j.jaac.2019.02.017>

REFERENCES

1. Bridgett DJ, Burt NM, Edwards ES, Deater-Deckard K. Intergenerational transmission of self-regulation: a multidisciplinary review and integrative conceptual framework. *Psychol Bull.* 2015;141:602-654.
2. Nigg JT. Annual research review: on the relations among self-regulation, self-control, executive functioning, effortful control, cognitive control, impulsivity, risk-taking, and inhibition for developmental psychopathology. *J Child Psychol Psychiatry.* 2017;58:361-383.
3. Crone EA, Dahl RE. Understanding adolescence as a period of social-affective engagement and goal flexibility. *Nat Rev Neurosci.* 2012;13:636-650.
4. Duckworth AL, Seligman MEP. Self-discipline outdoes IQ in predicting academic performance of adolescents. *Psychol Sci.* 2012;16:939-944.
5. Finkenauer C, Engels R, Baumeister R. Parenting behaviour and adolescent behavioural and emotional problems: the role of self-control. *Int J Behav Dev.* 2005;29:58-69.
6. Vazsonyi AT, Mikuska J, Kelley EL. It's time: a meta-analysis on the self-control-deviance link. *J Crim Justice.* 2017;48:48-63.
7. Moffitt TE, Arseneault L, Belsky D, et al. A gradient of childhood self-control predicts health, wealth, and public safety. *Proc Natl Acad Sci.* 2011;108:2693-2698.

8. Caspi A, Houts RM, Belsky DW, *et al.* Childhood forecasting of a small segment of the population with large economic burden. *Nat Hum Behav.* 2016;1:pii:0005.
9. Finkenauer C, Buyukcan-Tetik A, Baumeister RF, Schoemaker K, Bartels M, Vohs KD. Out of control: identifying the role of self-control strength in family violence. *Curr Dir Psychol Sci.* 2015;24:261-266.
10. Finkenauer C, Buyukcan-Tetik A, Schoemaker K, Willems YE, Bartels M, Baumeister R. Examining the role of self-regulatory strength in family violence. In: de Ridder DTD, Adriaanse KF, eds. *The Routledge International Handbook of Self-Control in Health and Well-Being.* London, UK: Routledge; 2017.
11. Willems YE, Li J-B, Hendriks A, Bartels M, Finkenauer C. The relationship between family violence and self-control in adolescence: a multi-level meta-analysis. *Int J Env Res Pub Health.* 2018;15:2468.
12. Brody GH, Ge X. Linking parenting processes and self-regulation to psychological functioning and alcohol use during early adolescence. *J Fam Psychol.* 2001;15:82-94.
13. Hallquist MN, Hipwell AE, Stepp SD. Poor self-control and harsh punishment in childhood prospectively predict borderline personality symptoms in adolescent girls. *J Abnorm Psychol.* 2015;124:549-564.
14. Tiberio SS, Capaldi DM, Kerr DCR, Bertrand M, Pears KC, Owen L. Parenting and the development of effortful control from early childhood to early adolescence: a transactional developmental model. *Dev Psychopathol.* 2016;28:837-853.
15. Davies PT, Cummings EM. Marital conflict and child adjustment: an emotional security hypothesis. *Psychol Bull.* 1994;116:387-411.
16. Davies PT, Cicchetti D, Martin MJ. Toward greater specificity in identifying associations among interparental aggression, child emotional reactivity to conflict, and child problems. *Child Dev.* 2012;83:1789-1804.
17. Sturge-Apple ML, Davies PT, Cicchetti D, Hentges RF, Coe JL. Family instability and children's effortful control in the context of poverty: sometimes a bird in the hand is worth two in the bush. *Dev Psychopathol.* 2016;29:685-696.
18. Krishnakumar A, Buehler C. Interparental conflict and parenting behaviors: a meta-analytic review. *Fam Relat.* 2000;49:25-44.
19. Whitesell CJ, Teti DM, Crosby B, Kim B-R. Household chaos, sociodemographic risk, coparenting, and parent-infant relations during infants' first year. *J Fam Psychol.* 2015;29:211-220.
20. DeWall CN, Baumeister RF, Stillman TF, Gailliot MT. Violence restrained: effects of self-regulation and its depletion on aggression. *J Exp Soc Psychol.* 2007;43:62-76.
21. Finkel EJ, DeWall CN, Slotter EB, Oaten M, Foshee VA. Self-regulatory failure and intimate partner violence perpetration. *J Pers Soc Psychol.* 2009;97:483-499.
22. Righetti F, Finkenauer C. If you are able to control yourself, I will trust you: the role of perceived self-control in interpersonal trust. *J Pers Soc Psychol.* 2011;100:874-886.
23. Pratt TC, Turanovic JJ, Fox KA, Wright KA. Self-control and victimization: a meta-analysis. *Criminology.* 2014;52:87-116.
24. Moilanen KL, Rasmussen KE, Padilla-Walker KL. Bidirectional associations between self-regulation and parenting styles in early adolescence. *J Res Adolesc.* 2015;25:246-262.
25. D'Onofrio BM, Lahey BB, Turkheimer E, Lichtenstein P. Critical need for family-based, quasi-experimental designs in integrating genetic and social science research. *Am J Public Health.* 2013;103:S46-S55.
26. Beaver KM, Ratchford M, Ferguson CJ. Evidence of genetic and environmental effects on the development of low self-control. *Crimin Justice Behav.* 2009;36:1158-1172.
27. van der Aa N, Boomsma DI, Rebollo-Mesa I, Hudziak JJ, Bartels M. Moderation of genetic factors by parental divorce in adolescents' evaluations of family functioning and subjective wellbeing. *Twin Res Hum Genet.* 2010;13:143-162.
28. Willems YE, Dolan CV, van Beijsterveldt CEM, *et al.* Genetic and environmental influences on self-control: assessing self-control with the ASEBA self-control scale. *Behav Genet.* 2018;48:135-146.
29. Duffy DL, Martin NG. Inferring the direction of causation in cross-sectional twin data: theoretical and empirical considerations. *Genet Epidemiol.* 1994;11:483-502.
30. Heath AC, Kessler RC, Neale MC, Hewitt JK, Eaves LJ, Kendler KS. Testing hypotheses about direction of causation using cross-sectional family data. *Behav Genet.* 1993;23:29-50.
31. Gillespie NA, Zhu G, Neale MC, Heath AC, Martin NG. Direction of causation modeling between cross-sectional measures of parenting and psychological distress in female twins. *Behav Genet.* 2003;33:383-396.
32. van Bergen E, Snowling MJ, de Zeeuw EL, van Beijsterveldt CEM, Dolan CV, Boomsma DI. Why do children read more? The influence of reading ability on voluntary reading practices. *J Child Psychol Psychiatry.* 2018;59:1205-1214.
33. van Beijsterveldt CEM, Groen-Blokhuis M, Hottenga JJ, *et al.* The Young Netherlands Twin Register (YNTR): longitudinal twin and family studies in over 70,000 children. *Twin Res Hum Genet.* 2013;16:252-267.
34. Rietveld MJ, van der Valk JC, Bongers IL, Stroet TM, Slagboom PE, Boomsma DI. Zygosity diagnosis in young twins by parental report. *Twin Res.* 2000;3:134-141.
35. De Coole RL, Jansma JBM. G.K.S. Gezinsklimaatschaal. Lisse: Swets and Zeitlinger; 1983.
36. Achenbach T, Rescorla L. *Manual for the ASEBA school-age forms and profiles: an integrated system of multi-informant assessment.* Burlington, VT: University of Vermont Research Center for Children, Youth, and Families; 2001.
37. Fagnani C, Medda E, Alessandri G, *et al.* The genetic architecture of effortful control and its interplay with psychological adjustment in adolescence. *J Res Pers.* 2017;68:5-14.
38. Muthén LK, Muthén BO. *Mplus User's Guide.* Los Angeles, CA: Muthén & Muthén; 2012.
39. Maydeu-Olivares A. Maximum likelihood estimation of structural equation models for continuous data: standard errors and goodness of fit. *Struct Equat Model A Multidiscip J.* 2017;24:383-394.
40. Rebollo I, de Moor MHM, Dolan CV, Boomsma DI. Phenotypic factor analysis of family data: correction of the bias due to dependency. *Twin Res Hum Genet.* 2006;9:367-376.
41. Hu L, Bender PM. Cutoff criteria for fit indexes in covariance structure analysis: conventional criteria versus new alternatives. *Struct Equat Model A Multidiscip J.* 1999;6:1-55.
42. Barton K. *MuMIn: Multi-Model Inference.* 2018. Available at: <https://cran.r-project.org/web/packages/MuMIn/MuMIn.pdf>. Accessed June 6, 2019.
43. Wagenmakers E-J, Farrell S. AIC model selection using Akaike weights. *Psychol Bull Rev.* 2004;11:192-196.
44. Boisvert D, Wright JP, Knopik V, Vaske J. A twin study of sex differences in self-control. *Justice Q.* 2013;30:529-559.
45. Maier SU, Makwana AB, Hare TA. Acute stress impairs self-control in goal-directed choice by altering multiple functional connections within the brain's decision circuits. *Neuron.* 2015;87:621-631.
46. Ellis BJ, Bianchi J, Griskevicius V, Frankenhuis WE. Beyond risk and protective factors: an adaptation-based approach to resilience. *Perspect Psychol Sci.* 2017;12:561-587.
47. Harold GT, Leve LD, Sellers R. How can genetically informed research help inform the next generation of interparental and parenting interventions? *Child Dev.* 2017;88:446-458.
48. Hannigan LJ, McAdams TA, Plomin R, Eley TC. Etiological influences on perceptions of parenting: a longitudinal, multi-informant twin study. *J Youth Adolesc.* 2016;45:2387-2405.
49. Leve LD, Neiderhiser JM, Shaw DS, Ganiban J, Natsuaki MN, Reiss D. The early growth and development study: a prospective adoption study from birth through middle childhood. *Twin Res Hum Genet.* 2013;16:1832-4274.
50. McAdams TA, Hannigan LJ, Eilertsen EM, Gjerde LC, Ystrom E, Rijdsdijk FV. Revisiting the children-of-twins design: improving existing models for the exploration of intergenerational associations. *Behav Genet.* 2018;48:397-412.