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

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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


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. Especially during adolescence, characterized by a range of normative biological and social changes, self-control is key to a successful transition into adulthood. 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. Because low self-control is a powerful predictor of health, wealth, and public safety across the lifespan, it is important to identify factors shaping its development, including contextual factors such as family conflict.

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, others find that adolescents’ low self-control predicts family conflict, and again others suggest a reciprocal relationship.

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. Findings from longitudinal studies demonstrate that children exposed to chronic, hostile, or poorly resolved family conflicts exhibit lower self-control. In addition, family conflict may have an indirect effect on children’s self-control, mediated through other family processes such as poor parenting practices, insecure parent—child relationships, and chaotic household conditions.
Alternatively, evidence suggests that low self-control predicts conflict. Individuals with low self-control are more likely to behave more aggressively towards strangers and their romantic partner. Individuals with low self-control trigger distrust within relationships and are less successful in de-escalating conflict. Also, low self-control is a modest yet consistent predictor of victimization, suggesting that low self-control may evoke aggression in others.

Longitudinal studies investigating a reciprocal association between family conflict and self-control in adolescence are scarce and yield inconsistent results. One study tracked the development of family conflict and low self-control across early adolescence (from age 12 to age 13 years, N = 120). 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 = 2,450). 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. 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. 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. 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). 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. 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. 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.

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. 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. 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). The scale consists of items assessing the direction of causality (DoC). In a series of saturated models, we tested equality of means and variances across zyosity 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. 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. 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. 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 Rg), rather than a nonshared correlation (denoted as Re) 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 \( \chi^2 \) tests. The \( \chi^2 \) statistic was computed by subtracting \(-2LL\) (log-likelihood) for the full model from that for a reduced model (\( \chi^2 = -2LL(\text{full}) - (-2LL(\text{reduced})) \)). If a \( p \) value higher than 0.01 was obtained from the \( \chi^2 \) 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 \( \Delta \chi^2(\text{df} = 1) = 109.94, p < .001 \), 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.*
self-control, the higher heritability of self-control (50%—60%) 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 \( \Delta \chi^2 (df = 1, N = 5,176 \text{ pairs}) = 2.50, p = .11 \). 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 \( \Delta \chi^2 (df = 1, N = 5,176 \text{ pairs}) = 33.23, p < .001 \). 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 \( \Delta \chi^2 (df = 1, N = 5,176 \text{ pairs}) = 2.63, p = .10 \). 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
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). 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 ($0.46$, $95\% \text{ CI} = 0.34–0.57$), including common genetic influences ($R_g = 0.56$, $95\% \text{ CI} = 0.12–0.99$), 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). Supporting theoretical models, we found that family conflict predicts low self-control in adolescence. More specifically, in line with the self-control strength model of family violence, 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.
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

<table>
<thead>
<tr>
<th>Model</th>
<th>Estimated Parameters</th>
<th>AIC</th>
<th>AIC weights</th>
</tr>
</thead>
<tbody>
<tr>
<td>Correlational</td>
<td>73</td>
<td>158,094.98</td>
<td>0.00</td>
</tr>
<tr>
<td>Bidirectional</td>
<td>73</td>
<td>158,067.31</td>
<td>0.26</td>
</tr>
<tr>
<td>Low self-control → family conflict</td>
<td>72</td>
<td>158,131.77</td>
<td>0.00</td>
</tr>
<tr>
<td>Family conflict → low self-control</td>
<td>72</td>
<td>158,070.57</td>
<td>0.05</td>
</tr>
<tr>
<td>Family conflict → low self-control (with Rg)</td>
<td>73</td>
<td>158,065.41</td>
<td>0.68</td>
</tr>
</tbody>
</table>

Note: Rg = genetic correlation.
strategies could be promising.\(^4^7\) 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.\(^4^8\)

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).\(^4^7\) 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.\(^3^9,^5^0\)

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.

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