AFFECTIVE SPECTRUM SYMPTOMS AND SELF-CRITICISM:

A BEHAVIORAL GENETIC APPROACH

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Highlights

• Behavioral genetic study of depressive and functional somatic symptoms (FSS).

• Greater role for genetic factors in FSS than depressive symptoms.

• Similar environmental factors were found in depressive symptoms and FSS.

• These factors were related to the environmental factor of self-criticism.

• No shared genetic factors between depressive symptoms, FSS, and self-criticism.

1. INTRODUCTION

Studies have shown that depression and so-called functional somatic symptoms (FSS; i.e., chronic pain and fatigue) are highly comorbid both at the symptom [1,2] and syndrome [[3], [4], [5], [6], [7], [8]] level. In addition, there is evidence for familial co-aggregation at both the symptom and syndrome levels [[9], [10], [11], [12]]. These findings have led researchers to suggest that functional somatic disorders and depression are part of an affective spectrum of symptoms and disorders [10,13], although it is not clear whether a common pathophysiology and similar causal pathways are implicated in disorders belonging to this spectrum [[14], [15], [16], [17], [18], [19]]. Shared genetic and environmental vulnerabilities in the development of these disorders have been suggested [[20], [21], [22], [23], [24]], but behavioral genetic studies investigating the similarity between genetic and environmental factors underlying both depressive symptoms and FSS have provided mixed results [25,26].

In this context, there is also increasing evidence that the personality trait of self-criticism, characterized by high levels of perfectionism in combination with harsh self-evaluation [27], may be an important vulnerability factor implicated in affective spectrum disorders [[28], [29], [30], [31], [32], [33], [34], [35]]. Self-criticism is increasingly conceptualized as a transdiagnostic factor that may play a key role in explaining the high comorbidity between depressive symptoms and FSS [36,37]. Specifically, self-criticism has been shown to be associated with a pattern of over-activity and persistence [[38], [39], [40], [41]], which may lead to a crash of the stress system, typical of both depression [[42], [43], [44], [45]] and functional somatic disorders [38,[46], [47], [48], [49]], because of the 'wear and tear' caused by chronic stress and over-activity. In this regard it is also important to consider that self-criticism and both depressive symptoms and FSS share important environmental factors, such as early childhood adversity [[50], [51], [52], [53]] and dysfunctional (e.g., cold and controlling) parenting [[54], [55], [56]]. However, there is also some evidence that self-criticism is partly genetically determined, although little research has directly investigated this assumption [57,58]. Hence, it remains unclear whether the relationship between self-criticism, depression, and FSS stems from shared environmental and genetic factors [9,10,59,60]. Furthermore, to date, no study

has investigated whether the genetic and environmental factors implicated in affective spectrum symptoms are also related to the genetic and environmental factors implicated in self-criticism.

The present study

Given the limitations in existing research, we conducted a behavioral genetic study using a family design with parents and their biological or internationally adopted children. More specifically, we applied analyses of variance decomposition in a Structural Equation Modelling framework. To identify the genetic component, we used the difference in heredity between families with a biologically related child (a biologically related child has 50% genetic association with each parent) and families with an adopted child (no genetic association with the parents). This type of family study may complement information obtained from more classic behavioral genetic designs such as twin studies and clinical samples [[61], [62], [63], [64]]. Indeed, twin samples have been criticized because of the possible inflated estimates of additive genetic variance, while the use of clinical samples with cut-off criteria for depression may fail to grasp the dimensional nature of this disorder [[61], [62], [63],65,66].

In line with earlier findings, we investigated the following hypotheses in this study. First, we expected that depressive symptoms, FSS, and self-criticism would each show a genetic and environmental factor (Fig. 1). Second, given the evidence of similar genetic and environmental factors in affective spectrum disorders, we expected that there would be a shared genetic and environmental factor implicated in depressive symptoms and FSS. More specifically, we investigated whether (a) the same genetic and environmental factor explained variance in both depressive symptoms and FSS (Fig. 2) or (b) whether the genetic and environmental factors in depressive symptoms and FSS would be distinct but positively correlated (Fig. 3). Third, we expected that the genetic and environmental factors implicated in affective spectrum symptoms would be related to the genetic and environmental factors involved in self-criticism (Fig. 3).

2.   METHOD

Participants and Procedures

Data from the Gene-environment Interactions in Families with Adolescents study was used. This longitudinal study focuses on the role of gene–environment interactions in predicting adolescent development. Two groups were included in this study: a group of parents with their biologically related adolescents (recruited in 2014) and a group of parents with their adopted adolescents (recruited in 2014 and 2015). Inclusion criteria were Belgian Dutch-speaking families with a biologically related or internationally adopted adolescent between the age of 12 and 18 years. Only one adolescent from each family was allowed to participate. Exclusion criteria for both groups were families with members with a serious medical illness (e.g., cancer, recent physical injury, physical disability). Further, children that were adopted after their first birthday were excluded from the study to reduce the risk of strong differences in environmental quality between the biological and adoption groups during the first year of child development [64,67]. This study was approved by the ethical committee of the KU Leuven and Ghent University.

The final sample comprised 266 biological families and 73 adoptive families (see online supplement A1). The two samples were very similar in terms of parental education (the majority had a higher education level), adolescent education (the majority followed a broad general education which prepares the student for higher education), adolescent age (M = 15.05 years, SD = 7.97), and adolescent gender distribution (53% female). The only difference was in the age of the parents, with the adoptive parents being significantly older than the biological parents (Table 1, online supplement A1). This finding was not unexpected, given the typically long duration of the adoption process [68,69].

Measures

Depressive symptoms. Depressive symptoms were measured in adolescents using the Children's Depression Inventory (CDI) [70] and in adults using the Beck Depression Inventory-II (BDI-II) [71] (Table 1). Both the CDI and the BDI-II showed good reliability, with Cronbach's alpha of 0.89 for BDI-II and 0.83 for CDI. Due to the confounding presence of items measuring somatic complaints on

depression scales [72,73], a depressive symptoms score was created by excluding such items from the total depression symptoms score to prevent overlap with FSS, as we have done in previous studies (see online supplement A2). For the BDI-II, item 11 and items 15 to 21 (i.e., Agitation, Loss of energy, Changes in sleeping pattern, Irritability, Changes in appetite, Tiredness or fatigue, and Loss of interest in sex) were excluded; the score with these items excluded is here termed the BDI-IIc. For the CDI, items 16 to 19 (i.e., Sleep disturbance, Fatigue, Loss of appetite, and Negative somatic preoccupation) were excluded, and the score with these items excluded is here termed the CDIc. Both the CDIc and BDI-IIc showed good reliability, with Cronbach's alpha of 0.85 for the BDI-IIc and 0.81 for the CDIc.

FSS. Among both adolescents and parents, FSS were measured using the 33-item Somatic Symptoms Questionnaire (SSQ) [74], assessing five types of frequent FSS: fatigue-related complaints, pain symptoms, respiratory complaints, gastrointestinal problems, and tension-related problems (Table 1, see online supplement A3). The SSQ showed good reliability, with Cronbach's alpha's of 0.88 for parents and 0.88 for adolescents.

Self-criticism. Self-criticism was measured using the Depressive Experiences Questionnaire (DEQ) for adults [75] and an age-appropriate version of the DEQ for adolescents [76] (Table 1). The DEQ has shown good internal consistency (identical solutions of confirmatory factor analyses), test–retest reliability, and validity with other scales [76].

Data analyses

Preliminary analyses were performed investigating the influence of the demographic variables (i.e., gender, adoption versus biological group status, educational level, age) on the study variables using a series of analyses of variance (ANOVAs) and repeated measures ANOVAs (see online supplement A4 for preliminary analyses with demographic variables). For all measures, there were no mean-level differences between parents and adolescents of the biological and adoption groups. Moreover, mean levels of the measures within a family (i.e., across mother, father, and adolescent) also did not differ between the biological and adoption groups. For parents, gender and the interaction between gender and age were related to BDI-IIc and DEQ scores. For both parents and adolescents, age was positively

related to FSS (r = −0.12, p = .002 and r = .20, p < .001, respectively). In subsequent analyses we thus controlled for gender and age (online supplement A4). All scores were transformed to Z-scores using means and standard deviations calculated separately for the adoption and biological groups (see online supplement A5 for correlation tables).

Next, behavioral genetic models were fitted in a Structural Equation Modelling framework using Mplus software [77], based on previous research using parent–offspring designs [[78], [79], [80]]. In the first set of models, a model with one genetic and environmental factor was fitted for all measures separately using the model specifications detailed in Fig. 1. Second, we combined the model of depressive symptoms and the model of FSS from step 1 in one model to evaluate whether depressive symptoms and FSS could be best explained by one underlying genetic and environmental factor (Fig. 2) or whether each phenotype could be better explained by a separate genetic and environmental factor (Fig. 3). Finally, based on the previous models, all three phenotypes (depressive symptoms, FSS, and self-criticism) and their respective genetic and environmental factors were fitted in one model.

The presented results include the standardized regression estimate β with 95% confidence interval (95%CI) and p-value. The explained variance of the environmental and genetic factor (which is the square of the standardized beta), together with the 95%CI, is also presented. Further, model fit was evaluated using the Chi-square (χ^2) statistic (which should be non-significant), the Root Mean Square Error of Approximation (RMSEA; acceptable values <0.06), the Standardized Root Mean Square Residual (SRMR; acceptable values <0.09), and the Tucker-Lewis Index (TLI; acceptable values ≥0.95). Models were compared for parsimony using the Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC; lower values of AIC and BIC indicate better fit) [[81], [82], [83], [84]]. A χ^2-difference test was used to compare nested models; in other cases AIC and BIC were used.

A post-hoc power analysis for multiple regression suggested that statistical power was well above conventional criteria (≥0.8) for all of the observed R2 in a model with 3 predictors (three observed

variables from mother, father, and adolescent data), probability level of $p \leq .05$, and a sample size of 1017 participants (339 families).

3. RESULTS

Genetic base models

For depressive symptoms, the variance due to genetic effects was 7% (95%CI = [0%; 32%]), which was non-significant, while the environmental factor was significant and accounted for 92% (95%CI = [77%; 100%]) of the variance (Table 2, Fig. 1).1 For FSS, both the genetic and environmental factors were significant, accounting for 31% (95%CI = [16%; 50%]) and 68% (95%CI = [53%; 88%]) of the variance, respectively. For self-criticism, there was also a significant genetic and a significant environmental factor, which accounted for 13% (95%CI = [2%; 34%]) and 86% (95%CI = [70%; 100%]) of the variance, respectively.

Functional somatic and depressive symptoms

The model with one underlying genetic and environmental factor for depressive symptoms and FSS (Fig. 2) did not provide a good fit ($\chi2$ (50) = 97.43, p < .001; RMSEA = 0.08; SRMR = 0.13; TLI = 0.87; AIC = 5542.45; BIC = 5557.77). In contrast, the model where each phenotype was explained by a separately modeled genetic and environmental factor (Fig. 3) had a good fit ($\chi2$ (48) = 40.11, p = .784; RMSEA = 0.00; SRMR = 0.08; TLI = 1.00) and was more parsimonious (AIC = 5489.13; BIC = 5512.11). In this model, the covariance between the genetic factors of depressive symptoms and FSS was non-significant (β_{AA} = 0.51, 95%CI = [−0.17; 1.18], p = .142). However, the covariance between the environmental factors was significant (β_{EE} = 0.42, 95%CI = [0.28; 0.57], p < .001). Deleting the covariance between the genetic factors did not result in a significantly worse fit ($\Delta\chi2$diff (1) = 1.42, p = .233; RMSEA = 0.00; SRMR = 0.08; TLI = 1.00; AIC = 5488.55; BIC = 5507.70). The resulting model showed a non-significant variance due to the genetic factor in depressive symptoms (β = 0.16, 95%CI = [−0.28; 0.59], p = .478, 2% of variance, 95%CI = [0%; 34%]), while the genetic factor in FSS accounted for a significant 26% (95%CI = [12%;

43%]) of variance ($\beta = 0.51$, 95%CI = [0.36; 0.66], p < .001). In addition, the variance due to environmental effects was significant in both depressive symptoms and FSS ($\beta = 0.99$, 95%CI = [0.92; 1.06], p < .001, 98% of variance, 95%CI = [84%; 100%] and $\beta = 0.86$, 95%CI = [0.77; 0.95], p < .001, 73% of variance, 95%CI = [59%; 90%], respectively), and there was significant covariance between the environmental factors ($\beta EE = 0.49$, 95%CI = [0.41; 0.57], p < .001).

Relations to self-criticism

The model combining the genetic base models of all three phenotypes showed a good fit ($\chi 2$ (96) = 85.03, p = .781; RMSEA = 0.00; SRMR = 0.07; TLI = 1.00; AIC = 8022.27; BIC = 8068.22). However, there were non-significant covariances between the genetic factors of depressive symptoms and FSS ($\beta AA = 0.52$, 95%CI = [−0.16; 1.20], p = .136) and FSS and self-criticism ($\beta AA = 0.20$, 95%CI = [−0.36; 0.75], p = .487). When removing these non-significant covariances, the covariance between depressive symptoms and self-criticism also became non-significant ($\beta AA = 1.18$, 95%CI = [−1.47; 3.82], p = .382).[2] Removing all non-significant covariances did not influence the model fit ($\Delta\chi 2$diff (3) = 2.85, p = .415; RMSEA = 0.00; SRMR = 0.08; TLI = 1.00) and this model was also more parsimonious (AIC = 8019.13; BIC = 8053.59). The final model thus included only significant covariances between the environmental factors (depressive symptoms and FSS: $\beta EE = 0.49$, 95%CI = [0.41; 0.56], p < .001; depressive symptoms and self-criticism: $\beta EE = 0.53$, 95%CI = [0.47; 0.59], p < .001; FSS and self-criticism: $\beta EE = 0.33$, 95%CI = [0.25; 0.41], p < .001; Table 3).

Results from this final model were similar to the results of the genetic base models from step 1 and to separate results of (a) depressive symptoms and self-criticism and (b) FSS and self-criticism (see online supplement A5). For depressive symptoms, the variance due to genetic effects was non-significant, and the environmental factor accounted for all of the variance. For both FSS and self-criticism, the genetic factor accounted for a significant 26% (95%CI = [13%; 43%]) and 6% (95%CI = [0%; 23%]) of variance, respectively, and the environmental factor accounted for a significant 73% (95%CI = [59%; 88%]) and 94% (95%CI = [82%; 100%]) of variance, respectively.

4. DISCUSSION

The current study used a behavioral genetic design comparing parents and their biologically related adolescents with parents and their internationally adopted adolescents to investigate whether affective spectrum symptoms (i.e., depressive symptoms and FSS) share genetic and environmental factors [22,36,85,86]. We further investigated whether the environmental and genetic factors of depressive symptoms and FSS are also related to the genetic and environmental factors of self-criticism, which has an important role in transdiagnostic vulnerability for affective spectrum disorders [29,32,35].

First, we found that the variance in depressive symptoms was mostly due to environmental effects (92%, 95%CI = [77%; 100%]), with a small and non-significant variance due to genetic effects (7%, 95%CI = [0%; 32%], p = .070). The relatively modest estimate of genetic loading for depressive symptoms may be somewhat surprising, considering that previous behavioral genetic studies have generally yielded heritability estimates ranging between 30 and 40% [62,87]. However, these higher estimates were primarily reported in studies using twins and clinical samples. The current behavioral genetic study used a family-based design with biological and adopted children, and investigated symptoms of depression (rather than major depressive disorder). Considering that depression is a familial disorder and that this disorder is also dimensionally distributed [65,66,88,89], the current study offers important and complementary information about the heritability of depression compared with more classical studies [[61], [62], [63], [64]]. Indeed, the lower genetic estimate found in our study is in line with research suggesting that the genetic liability depends in part on the severity of the depressive condition that is assessed (with major depression having a higher heritability compared to minor depression) [[89], [90], [91]]. A study with a very similar design to the present study, investigating the influence of genetic factors on depressive symptoms in families, reported a similar non-significant heritability index of 9% (95%CI = [0%; 57%] of variance) [61]. Interestingly, a study with a sample of Swedish twins (mean age = 81 years), and using the Center for Epidemiological Studies Depression scale [92], also found a comparable and non-significant 0 to 7% of variance due to

genetic effects for depressive symptoms (excluding somatic items), and a significant 19% of variance due to genetic effects for FSS [93]. This latter finding is consistent with the current study, which showed a significant genetic factor in FSS, accounting for 31% of variance (95%CI = [16%; 50%]). Existing studies generally suggest comparable genetic estimates for FSS (i.e., between 19 and 54%) [[94], [95], [96], [97]]. Taken together, these findings suggest that FSS may be more heritable than cognitive-depressive symptoms [98].

Second, our findings suggest that the co-occurrence of depressive symptoms and FSS is more likely due to a shared environment than a shared genetic liability. Indeed, the strong covariation between the environmental factors ($\beta EE = 0.49$) suggests that there is considerable overlap between both conditions in terms of environmental causes. Yet, this also means that there remains considerable room for non-shared environmental factors, as there was no evidence for a model with one underlying environmental and genetic factor explaining variance in both depressive symptoms and FSS. Further, our findings are in line with previous behavioral genetic studies that found little genetic association between depressive symptoms and FSS [26,63,99]. However, other studies did find a genetic association between affective spectrum disorders or reported one genetic factor underlying affective spectrum disorders [25,100]. It is clear that more research in this area is needed.

Results of this study suggest that self-criticism has a small but significant genetic factor that accounts for 13% of the variance (95%CI = [2%; 34%]), and a larger environmental factor that accounts for 86% of the variance (95%CI = [70%; 100%]). Only a few studies have investigated the genetic basis of self-criticism or related traits, but the genetic estimates in these studies (with twin samples) generally are slightly higher, explaining between 23% and 42% of variance [57,58,[101], [102], [103]]. Furthermore, we found that the model with separate genetic and environmental factors best explained the relationship among depressive symptoms, FSS, and self-criticism. In this model, there were significant covariations between the environmental factors of depressive symptoms and FSS ($\beta EE = 0.49$), depressive symptoms and self-criticism ($\beta EE = 0.53$), and FSS and self-criticism ($\beta EE = 0.33$), yet no significant covariations between their respective genetic factors. There was one exception, namely for self-criticism and depressive symptoms (see online supplement A5), but there

was no significant effect of the genetic factor in depressive symptoms. These findings suggest that self-criticism and affective spectrum symptoms are associated through partly shared environmental factors, but not through a shared genetic factor. As noted, studies have shown identical environmental factors, such as early adversity [53,104] and parenting [[54], [55], [56]], to be implicated in self-criticism and affective spectrum disorders. Interestingly, studies have shown that self-criticism mediates the relationship between stressful environmental circumstances and affective spectrum symptoms [36,46,105].

Although we found little evidence for genetic covariation between depressive symptoms, FSS, and self-criticism, it is important to note that the current study investigated whether variance in adolescents' affective spectrum symptoms and self-criticism can be explained by an intergenerationally transmitted heritable genetic vulnerability. In this regard, the field of molecular studies has shown that gene–environment interactions (i.e., genetic effects that are moderated by environmental circumstances) might be especially important in stress-related psychopathology [88,106]. However, such a mechanism will not be apparent in the heritability estimate of the current study; rather, it will be part of the variance explained by the environmental component. Such gene–environment interactions have already been implicated in depression [107,108] and perhaps also in FSS [[109], [110], [111]]..

Limitations

While the current study used a family design as an alternative to other behavioral genetic samples, it is important to consider that this strategy also has its limitations [64]. First, it might be expected that when comparing a biological sample to an adopted sample, the role of the environment might be inflated, considering the selective placement of adopted children into a positive environment. Second, when using adolescents to identify the genetic component, it is important to keep in mind that the genetic effect might be different in adults and adolescents, with genetic estimates in younger samples generally being lower [98]. Third, in a behavioral genetic study, partner resemblance (i.e., so-called assortative mating) can inflate behavioral genetic estimates [112]. In the current sample, depressive symptoms ($r = 0.16$, $p = .003$) and functional somatic symptoms ($r = 0.18$, $p = .001$) in particular

showed significant, although modest, correlations between partners. Finally, it is important to note that in the current study, the size of the adoption sample was relatively small, which perhaps could explain the large confidence intervals of the genetic factor of depressive symptoms. However, confidence intervals of FSS and self-criticism were smaller. Furthermore, the only estimate that differed between the biological and adoption groups was the regression path between the parental genetic factor and the adolescent genetic factor (all other estimates were fixed to be identical across groups, with a combined sample size of 339 families).

Conclusion

In the current sample of parents and their adoptive or biologically related adolescents, we found only environmental effects in depressive symptoms, and both genetic and environmental effects in FSS and self-criticism. Our evidence suggests that shared environmental circumstances in particular predict the co-aggregation of depressive symptoms and FSS, and, moreover, that similar environmental circumstances can be found in the etiology of self-criticism, an important vulnerability factor for affective spectrum symptoms. The current study did not find evidence of one underlying genetic or environmental factor predicting affective spectrum symptoms and self-criticism. These findings provide important avenues for further research aimed at disentangling similarities and differences between depressive symptoms and FSS.

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Table 1. Descriptive features of participants in the biological and adoption sample.

	Description	Biological sample	Adoption sample
Parents			
Age	M (SD) in years	46.76 (4.59)	49.75 (4.85)
Education	Secondary degree (versus higher)	34%	22%
BDI-II [a]	M (SD)	6.00 (4.61)	6.39 (4.74)
	Mild to severe depression	10%	11%
SSQ [b]	M (SD) frequency of symptoms	8.23 (5.75)	8.85 (5.42)
	Reporting at least one daily symptom	29%	36%
DEQ	M (SD)	−1.10 (0.90)	−1.17 (0.73)
Adolescents			
Age	M (SD) in years	15.13 (1.97)	14.78 (1.96)
Education	Preparing for higher education	61%	60%
CDI [c]	M (SD)	9.05 (5.59)	8.96 (5.85)
	At risk for depression	12%	14%
SSQ [b]	M (SD) frequency of symptoms	13.25 (5.97)	12.12 (5.75)
	Reporting at least one daily symptom	45%	52%
DEQ	M (SD)	−0.24 (0.82)	−0.26 (0.75)

Note. BDI-II = Beck Depression Inventory-II, SSQ = Somatic Symptoms Questionnaire, DEQ = Depressive Experiences Questionnaire, CDI = Children's Depression Inventory. Additional sample information is given in online supplement A1.

[a] Theoretical range 0–63 and cut off criteria: <14 versus ≥14. Items measuring somatic complaints were excluded from BDI-II scores for the behavioral genetic analyses.

[b] Theoretical range 0–33.

[c] Theoretical range 0–54 and cut-off criteria: <16 versus ≥16. Items measuring somatic complaints were excluded from CDI scores for the behavioral genetic analyses.

Table 2. Results of the AE-genetic base models.

Measure	Model fit					Model parsimony		Loadings (β)			
	X^2	p-value	RMSEA	SRMR	TLI	AIC	BIC	A 95%CI[;]	p-value	E 95%CI[;]	p-value
Depressive symptoms	11.68	0.766	0.00	0.06	1.00	2843.51	2851.17	0.27 [−0.02; 0.57]	0.070	0.96 [0.88; 1.05]	<0.001
FSS	19.42	0.247	0.04	0.08	0.95	2831.03	2838.68	0.56 [0.40; 0.71]	<0.001	0.83 [0.73; 0.94]	<0.001
Self-criticism	8.06	0.947	0.00	0.05	1.00	2835.65	2843.30	0.37 [0.16; 0.59]	0.001	0.93 [0.84; 1.02]	<0.001

Note. Degrees of freedom for all models = 16. Loadings A and E are standardized regression estimates for the parent variables of the genetic component and environmental component, respectively. Additionally, the 95% Confidence Interval is shown (95%CI[;]). FSS = Functional Somatic Symptoms.

Table 3. Structural loadings of the final AE-genetic model including self-criticism, somatic symptoms, and depressive symptoms.

Measure	Loadings (β)			
	A95%CI[;]	p-value	E95%CI[;]	p-value
Depressive symptoms	0.00 [−0.51; 0.51]	0.999	1.00 [1.00; 1.00]	<0.001
FSS	0.51 [0.37; 0.66]	<0.001	0.86 [0.77; 0.94]	<0.001
Self-criticism	0.25 [0.02; 0.48]	0.034	0.97 [0.91; 1.03]	<0.001

Note. Loadings A and E are standardized regression estimates for the parent variables of the genetic component and environmental component, respectively. Additionally, the 95% Confidence Interval is shown (95%CI[;]). FSS = Functional Somatic Symptoms.

Fig. 1. Figure detailing relations between measures using Structural Equation Modelling framework in step 1. A denotes the genetic effect, E denotes the environmental effect, AA the regression between child and parent genetic effect, and subscripts after these letters denotes the family member (i.e., M = maternal, P = paternal, C = child). In general, all parameters were set equal across family members and between the adoption and the biological group (i.e., $E_M = E_P = E_A$; $A_M = A_P = A_C$). However, the genetic component was made identifiable by fixing the difference in heredity of genetic factors between the adoption (i.e., $A_M A_C = A_P A_C = 0$) and biological group (i.e., $A_M A_C = A_P A_C = 0.5$). The starting value for A_M, A_P, A_A, E_M, E_P, and E_A was 0.5.

Fig. 2. Figure detailing relations between measures using Structural Equation Modelling framework in step 2 and 3. A denotes the genetic effect, E denotes the environmental effect, AA the regression between child and parent genetic effect, and subscripts after these letters denotes the family member and the phenotypic measure (i.e., M = maternal, P = paternal, C = child, 1 = phenotypic measure 1, 2 = phenotypic measure 2). Again, all parameters within one phenotypic measure were set equal across family members and between the adoption and the biological group (i.e., $E_{M1} = E_{P1} = E_{C1}$; $A_{M1} = A_{P1} = A_{C1}$; $E_{M2} = E_{P2} = E_{C2}$; $A_{M2} = A_{P2} = A_{C2}$), and there was a fixed difference in heredity of genetic factors between the adoption (i.e., $A_M A_C = A_P A_C = 0$) and biological group (i.e., $A_M A_C = A_P A_C = 0.5$). The starting values for each parameter were based on the model results of step 1.

Fig. 3. Figure detailing relations between measures using Structural Equation Modelling framework in step 2 and 3. A denotes the genetic effect, E denotes the environmental effect, AA the regression between child and parent genetic effect, rEE the covariance between the environmental effects, rAA the covariance between the genetic effects, and subscripts after these letters denotes the family member and the phenotypic measure (i.e., M = maternal, P = paternal, C = child, 1 = phenotypic measure 1, 2 = phenotypic measure 2). All parameters within one phenotypic measure were set equal across family members and between the adoption and the biological group (i.e., $E_{M1} = E_{P1} = E_{C1}$; $A_{M1} = A_{P1} = A_{C1}$; $E_{M2} = E_{P2} = E_{C2}$; $A_{M2} = A_{P2} = A_{C2}$; $rE_{M1}E_{M2} = rE_{P1}E_{P2} = rE_{C1}E_{C2}$; $rA_{M1}A_{M2} = rA_{P1}A_{P2} = rA_{C1}A_{C2}$), and there was a fixed difference in heredity of genetic factors between the adoption (i.e., $A_{M1}A_{C1} = A_{M2}A_{C2} = A_{P1}A_{C1} = A_{P2}A_{C2} = 0$) and biological group (i.e., $A_{M1}A_{C1} = A_{M2}A_{C2} = A_{P1}A_{C1} = A_{P2}A_{C2} = 0.5$). The starting values for each parameter were based on the model results of step 1.