Journal of Psychosomatic Research xxx (xxxx) xxx–xxx

Contents lists available at ScienceDirect

Journal of Psychosomatic Research

journal homepage: www.elsevier.com/locate/jpsychores

Affective spectrum symptoms and self-criticism: A behavioral genetic approach

Dries Bleys[a,*], Patrick Luyten[a,b], Stephan Claes[c], Bart Soenens[d]

[a] Faculty of Psychology and Educational Sciences, KU Leuven, Tiensestraat 102, 3000 Leuven, Belgium
[b] Faculty of Brain Sciences, University College London, 1-19 Torrington Place, London WC1E 7HB, United Kingdom
[c] Psychiatry Research Group, KU Leuven, Herestraat 49, 3000 Leuven, Belgium
[d] Department of Developmental, Personality and Social Psychology, H. Dunantlaan 2, Ghent University, 9000 Ghent, Belgium

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 [3–8] level. In addition, there is evidence for familial co-aggregation at both the symptom and syndrome levels [9–12]. 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 [14–19]. Shared genetic and environmental vulnerabilities in the development of these disorders have been suggested [20–24], 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 [28–35]. 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 [38–41], which may lead to a crash of the stress system, typical of both depression [42–45] and functional somatic disorders [38,46–49], 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 [50–53] and dysfunctional (e.g., cold and controlling) parenting [54–56]. 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.

1.1. 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 [61–64]. 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 [61–63,65,66].

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).

* Corresponding author at: KU Leuven, Department of Clinical Psychology, Tiensestraat 102, Box 3720, 3000 Leuven, Belgium.
E-mail address: dries.bleys@kuleuven.be (D. Bleys).

https://doi.org/10.1016/j.jpsychores.2018.03.172
Received 11 January 2018; Received in revised form 30 March 2018; Accepted 30 March 2018

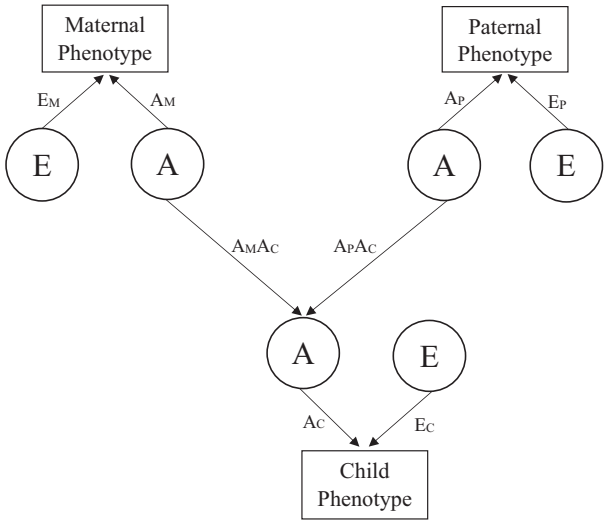

Fig. 1. Figure detailing relations between measures using Structural Equation Modelling framework in step 1. A denotes the genetic effect, E denotes the environmental effect, AA the regression between child and parent genetic effect, and subscripts after these letters denotes the family member (i.e., 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., $A_MA_C = A_PA_C = 0$) and biological group (i.e., $A_MA_C = A_PA_C = 0.5$). The starting value for A_M, A_P, A_A, E_M, E_P, and E_A was 0.5.

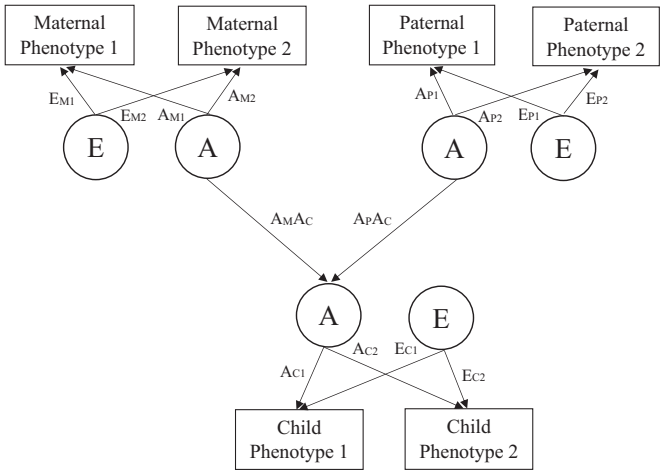

Fig. 2. Figure detailing relations between measures using Structural Equation Modelling framework in step 2 and 3. A denotes the genetic effect, E denotes the environmental effect, AA the regression between child and parent genetic effect, and subscripts after these letters denotes the family member 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., $A_MA_C = A_PA_C = 0$) and biological group (i.e., $A_MA_C = A_PA_C = 0.5$). The starting values for each parameter were based on the model results of step 1.

2. Method

2.1. 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].

2.2. Measures

2.2.1. 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.

2.2.2. 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.

2.2.3. 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].

2.3. Data analyses

Preliminary analyses were performed investigating the influence of