Journal of Affective Disorders 170 (2015) 203–212

Contents lists available at ScienceDirect

Journal of Affective Disorders

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

Research report

Temperament and depressive symptoms: What is the direction of the association?

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ARTICLE INFO

Article history:
Received 17 February 2014
Received in revised form
8 August 2014
Accepted 12 August 2014
Available online 3 September 2014

Keywords:
Temperament
EAS
Mental disorders
Cross-lagged modeling
Prospective study

ABSTRACT

Background: Temperament characteristics have been suggested to be associated with mental health outcomes, especially depression, but the direction of the association is unknown. In this study, we tested whether temperament characteristics, as defined by the Buss–Plomin adulthood emotionality–activity–sociability (EAS) temperament model, predict depressive symptoms or whether depressive symptoms predict changes in temperament characteristics.
Methods: Participants comprised a population-based sample of 719 men and 1020 women from the Young Finns study aged 20–35 years at baseline in 1997 and who responded to repeated surveys of temperament and depressive symptoms in four study phases from 1997 to 2012. The associations were tested using linear regression models, repeated cross-lagged structural equation models, parallel latent growth curve models and two-dimensional continuous-time state space model (Exact Discrete Model).
Results: Both low sociability ($\beta = -0.12$, $p < 0.001$) and high negative emotionality ($\beta = 0.34$, $p < 0.001$) predicted subsequent increased depressive symptoms, whereas earlier depressive symptoms predicted increased negative emotionality ($\beta = 0.50$, $p < 0.001$), but not low sociability.
Limitations: The depressive symptoms scale applied may not be used for measuring clinically recognized depression.
Conclusions: Our findings suggest that the direction of the association is from low sociability to depressive symptoms rather than the reverse, but the association between negative emotionality and depressive symptoms seems to be reciprocal.

1. Introduction

Depression and depressive disorders are among the leading causes of poor quality of life in Western countries (World Health Organization, 2008; Mathers and Loncar, 2006; Mathers and Loncar, 2005). Depression and depressive symptoms cause human suffering, affect families, and are associated with substantial work impairment (Brown et al., 2009; Stewart et al., 2003; Thomas and Morris, 2003). Depressive symptoms have a multifactorial etiology and temperament characteristics have been proposed to be among the risk factors (Elovainio et al., 2004; Kendler et al., 1993; Kendler, 1992; Kendler and Prescott, 2007; Kampman and Poutanen, 2011; Klein et al., 2011; Clark, 2005; Cloninger, 1986; Svrakic et al., 1992).

Temperament is suggested to reflect individual differences in the autonomic nervous system and the brain's neuroendocrinological functions (Buss, 1991; Plomin et al., 1988). The proposed biological origin and relative stability of temperament make it a suitable construct for studying the etiology of various health problems and associated processes. The existing evidence offers partial support for the association between various temperament characteristics and increased risk of depression (Rothbart and Bates, 1998; Goodyer et al., 1993; Svrakic et al., 1992).

According to Buss and Plomin (1975) and Buss (1991), the temperament dimensions may be categorized into three dimensions: emotionality, activity, and sociability (Emotionality-Activity-Sociability construct, abbreviated EAS). Emotionality refers to the tendency to experience and show frequent and intense negative emotions such as fear, distress, anger, or aggression. Activity refers

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http://dx.doi.org/10.1016/j.jad.2014.08.040
0165-0327/

to hyperactive behaviors such as speed, impatience, impulsivity, and vigorous motoric style. Sociability reflects the preference to be with people and to interact with others, as opposed to the preference of being and doing things alone.

Negative emotionality is generally defined as a temperamental sensitivity to negative stimuli (i.e. stress reactivity or proneness to general anxiety (Cloninger and Svrakic, 1993)), causing a broad range of negative moods and cognitions, such as guilt, hostility, and self-dissatisfaction (Clark et al., 1994). People with this temperament get angry, insecure, and distressed easily, especially when facing novel situations or after the occurrence of something unpleasant.

Sociability "is the tendency to prefer the presence of others" (Buss and Plomin, 1984, p. 63). Low sociability is also often defined as a dimension of behavioral inhibition (Kagan et al., 1987) that is defined as the consistent tendency to display fear and withdrawal in unfamiliar situations (Kagan et al., 1984). Children with this temperament are often reticent in social contacts. They are shy towards strangers and timid in unfamiliar situations. Sociability is not, however, the same as shyness, because shy people may desire the presence of others, but they avoid it because they tend to be stressed and anxious when surrounded by other people, especially unfamiliar people. There is substantial evidence that children with stable high levels of low sociability (McDermott et al., 2009; Williams et al., 2009) and/or high negative emotionality have a higher risk of developing mental health problems including depression (Muris and Ollendick, 2005; Vreeke and Muris, 2012). Research on the potential etiological role of temperament in health problems has concentrated mainly on children and adolescents, but negative emotionality and low sociability are likely to be high-risk temperament traits also in adulthood.

There are at least three postulated mechanisms through which temperament affects depressive symptoms. First, temperament and depression may have a shared genetic or environmental background (Kendler, 1992). Second, temperament may moderate an individual's reactions to stressful life events (Ellis et al., 2011; Belsky and Pluess, 2013; Elovainio et al., 2004) due to ineffective coping strategies (Mezulis et al., 2004). This means that if one is prone to being highly negatively reactive to new stimuli it may be difficult to maintain the positive self-evaluation needed for good mental health (Carver and Connor-Smith, 2010). Third, certain temperament characteristics may select individuals into certain environments or influence the individual's behaviors (Kendler and Prescott, 2007). In sum, substantial evidence suggests that some temperamental characteristics may predict later mental health problems, particularly depression.

However, it is reasonable to assume that depression also may alter temperamental characteristics. Long-term depression may even affect biological processes and structures that potentially impact the structural and widespread local functional abnormalities in specific brain regions (Lui et al., 2009) and reduce gray matter volume in the caudate nucleus (Kim et al., 2008) and thus may effect core, biologically rooted psychological structures such as temperament (Buss, 1991). Earlier research has also shown that individual differences in temperament are reflected in structural variances in specific brain areas. Patients with depression have also been found to have increased neuronal responses to emotional social stimuli (Cheng et al., 2010).

In addition to potential physiological mechanisms linking depression to temperament, cognitive and behavioral mediators are also possible. Individuals suffering from depression process information about the world, the future, and especially themselves in a maladaptive fashion compared with healthy individuals (APA, 2000). According to several theoretical models of depression, such as Beck's cognitive model (Beck et al., 1979; Haaga and Beck, 1995; Disner et al., 2011), Seligman's learned helplessness model (Seligman, 1972), and more recent cognitive neuropsychological models (Clark et al., 2009; Roiser et al., 2012), maladaptive cognitive biases are central to the development and maintenance of depression. Thus, depressed people tend to view the world and themselves in an excessively pessimistic or hopeless light (Stratta et al., 2014). Depressed individuals may judge themselves and their temperament characteristics harshly and underestimate their positive attributes, possessing a self-disparaging and critical view. Such preoccupations may even result in individuals withdrawing from social situations, further affecting their self-image. In the long run, all of these may affect an individual's self-perceptions of temperament characteristics, including sociability, activity, and negative emotionality.

Based on the current evidence, we hypothesized that (A) temperament characteristics, especially low sociability and negative emotionality, are risk factors for depressive symptoms, (B) depressive symptoms may impact temperament, and (C) depressive symptoms and temperament characteristics have a reciprocal association, reinforcing each other. We therefore explicitly tested both directions of the causality hypothesis between temperament and depressive symptoms using linear regression analyses.

2. Methods

2.1. Participants

The Cardiovascular Risk in Young Finns Study is an ongoing population-based follow-up study of coronary heart disease risk factors in Finnish children, adolescents, and young adults (Raitakari et al., 2008). The first cross-sectional study was conducted in 1980 when age cohorts of 3-, 6-, 9-, 12-, 15-, and 18-year-olds were randomly sampled on the basis of social security numbers, resulting in a total of 3596 participants. The measurement of EAS temperament and depressive symptoms was performed in 1997 (the baseline of this study) and repeated in subsequent follow-ups in 2001, 2007, and 2012. In the final study phase, participants had reached an age of 38–41 years. The data used in this study consist of 1739 participants (718 men, 1020 women) with complete information about depressive symptoms from the follow-up assessment in 2012. The participants in this sample were more often women ($p<0.001$), were slightly older ($p<0.001$), had a lower body mass index (BMI) ($p=0.003$), had a higher education ($p<0.001$), smoked less ($p<0.001$), consumed less alcohol ($p=0.005$), and used more medication ($p<0.001$) than those who dropped out. The characteristics of the study sample are presented in Table 1.

2.2. Procedure

The study was approved by the local ethics committee. Moreover, the study protocol of each study phase corresponded to the proposal by the World Health Organization and conformed to the Helsinki declaration. All participants gave written informed consent, and their treatment complied with APA ethical standards.

2.3. Temperament dimensions

Temperament was self-reported in all study phases by the participants using the Emotionality-Activity-Sociability Temperament Survey presented by Buss (1991). The questionnaire consisted of 27 items on a five-point scale of (1) totally disagree to (5) totally agree. According to the temperament theory by Buss and Plomin (1984) and Buss (1991), emotionality consists of two components: anger and fear. Anger was assessed using seven items (e.g., "I often feel like a powder keg ready to explode";

Table 1
Characteristics of the study sample ($n=1739$)[a]; the Young Finns Study.

Characteristics	Percentage or mean (standard deviation)			
	All ($n=1739$)	Men ($n=719$)	Women ($n=1020$)	p-Value
Childhood characteristics (1980)				
Age (years)	10.8 (5.0)	11.1 (5.1)	10.7(5.0)	0.10
Parental occupational status				
Manual	39	42	37	
Lower non-manual	43	38	46	
Higher non-manual	18	20	17	0.03
Adulthood risks in 2001 or 2007[a]				
Age (years)	27.8 (5.1)	28.1 (5.1)	27.7 (5.0)	0.10
Body Mass Index (kg/m^2)[a]	25.8 (4.6)	26.6 (4.0)	25.2 (4.9)	<0.001
Physical Activity Index	9.6 (2.3)	9.7 (2.5)	9.5 (2.1)	0.16
Smoking (current)	21	26	17	<0.001
Alcohol consumption (high consumption)	11	20	5	<0.001
Own occupational status				
Manual	31	42	23	
Lower non-manual	44	25	57	
Higher non-manual	25	33	20	<0.001
Medication use				
Yes	29	14	40	
No	71	86	60	<0.001
Social support (measured in 1997)	4.2(0.7)	3.9 (0.8)	4.4 (0.7)	<0.001

[a] Sample with data on depressive symptoms at follow-up.

"Sometimes people bother me just by being around"), and fear was assessed using five items (e.g., "I am easily frightened"; "I tend to be nervous in new situations"). Activity was divided into the components of vigor and tempo, both of which were assessed by five items (examples of vigor: "My gestures tend to be emphatic"; "When I knock on a door, I usually knock hard", examples of tempo: "I usually seem to be in a hurry"; "My life is fast-paced"). Sociability was assessed using five items (e.g., "I like to be with people"; "I prefer working with others rather than alone"). The Cronbach alphas of the scales ranged from 0.81 to 0.78 for emotionality, from 0.70 to 0.66 for activity, and from 0.78 to 0.72 for sociability. Previous confirmatory factor analyses have shown that this temperament construct has good fit with the Cardiovascular Risk in Young Finns data (Katainen et al., 1999). The EAS Temperament Survey has shown an acceptable psychometric properties also in other settings among adult populations (Braithwaite et al., 1984; Naerde, Roysamb, and Tambs, 2004).

2.4. Depressive symptoms

Depressive symptoms were assessed in 1997, 2001, 2007, and 2012 using a modified version of Beck's Depression Inventory (Katainen et al., 1999; Beck and Steer, 1984; Beck et al., 1988). Subjects were asked to rate 21 items (e.g., "I often feel sad") on a five-point scale ranging from (1) totally disagree to (5) totally agree. The Cronbach alphas of the scale ranged from 0.89 to 0.92. In the original version of the BDI, subjects were asked to choose between four alternative descriptions of 21 items, with the descriptions of each item ranging from minimal to severe symptoms of depression. In the present study, the participants were asked to rate the second mildest descriptions of the original 21 items (e.g., "I often feel sad") on a five-point scale ranging from (1) totally disagree to (5) totally agree. For instance, an original BDI item might have the following four response options: (0) I do not feel sad, (1) I feel sad, (2) I am sad all the time and I cannot snap out of it, and (3) I am so sad or unhappy that I cannot stand it. In our modified version, we would select response option (1) and ask the participants to rate their agreement with it on a five-point Likert scale. Originally, these second mildest items were selected because they were expected to most accurately measure depressive symptoms among the normal population (Rosenström et al., 2014).

2.4.1. Covariates

The parental level of education was reported in 1980. Parental occupational status was divided into three categories: manual, lower non-manual, and higher non-manual (Elovainio et al., 2012; Pulkki-Råback et al., 2011). Social support was assessed at study phase 1 (in 1997) with the Perceived Social Support Scale-Revised (PSSS-R) consisting of 12 items ($\alpha=0.94$) measuring social support received from family and friends. Responses were given on a five-point scale ranging from (1) strongly disagree to (5) strongly agree. The participants' own occupational status was measured at phase 2 (in 2001), and it was divided into the same categories as the occupational status of their parents (manual, lower non-manual, and higher non-manual). Current smoking (yes/no) was reported at phase 2 (in 2001). Information on alcohol consumption was assessed at phase 2 (in 2001) by frequency of intoxication (drinking six or more units of alcohol) on a seven-point scale ranging from daily to never. The physical activity index ($\alpha=0.76$) was collected at phase 2 (in 2001) from five items enquiring about the frequency, intensity, average duration, hours of physical activity, and participation in a guided sport (range 5–16) (Telama et al., 1997). High scores on physical activity index (PAI) indicate high physical activity. Body mass index (BMI) was calculated by dividing participants' weight in kilograms by their squared height in meters. BMI was measured at phase 3 (in 2007). Other measures included age, sex, and self-reported use of antidepressant medication.

All of these potential confounders have been associated with both temperament or personality characteristics and depressive symptoms (Blanch and Aluja, 2013; Bergmeier et al., 2013; Espana-Romero et al., 2013; Wiltink et al., 2013).

2.5. Data analyses

To explore the direction of the relationship between temperament and depressive symptoms, we adopted a four-tier data analytic approach to complement the typical problems of each individual method and to get a complete picture of the

relationship between temperament characteristics and depression. We thus did not repeat multiple similar models to test multiple associations, but tested basically the same associations with multiple modeling approaches.

First, two linear models were fitted where the effects of baseline temperament on depressive symptoms at the final follow-up (in 2012) were modeled, adjusting for all potential confounders, to see whether the association would be accounted for by some third factor. After this, the linear regression model was adjusted additionally for depressive symptoms at baseline (1997) to detect the association between temperament and depressive symptoms change. Similar two-step modeling was performed to test the effects of baseline depressive symptoms on temperament at the final follow-up, adjusting for temperament at baseline. Because no gender interactions were found in the association between temperament characteristics and depressive symptoms, the associations between temperament and depressive symptoms were tested with men and women combined. All associations between depressive symptoms and each temperament characteristic were tested separately.

We ran the linear regression models using the imputed data to retain all participants with data on depressive symptoms in 2012. Thus, multiple imputed values were generated for the missing data from the variables used in the analysis, by means of STATA MI IMPUTE procedure. Twenty datasets were randomly selected, and these datasets were analyzed using STATA MI ESTIMATE option. This procedure takes into account the uncertainty in the imputation as well as uncertainty due to random variation (as in all multivariable analyses) (Royston, 2004).

The linear regression modeling only tests one direction of the association and basically uses data from only two data collection points. Thus, the ability of standard regression analyses to give a full picture of potential reciprocity between two factors is limited. To overcome these problems and to estimate the two-way antecedent–consequence relationships simultaneously, we fitted repeated cross-lagged structural equation models (SEM) separately for (negative) emotionality and (low) sociability (depicted in Fig. 1) using all four measurement points. Second, we examined the associations between depressive symptoms and temperament using parallel latent growth curve models (depicted in Fig. 2). The advantage of parallel growth models is that they allow the examination of individual trajectories of temperament and depression over time in a parallel fashion, divide the variation over time in the level (intercept) and the development (slope), and enable testing of the associations between both levels and slopes. The advantage of growth curve models is that they allow examination of the shape of development over time.

In all SEM models, the contribution of the potential confounding factors to the relationships between temperament and depressive symptoms was taken into account by using adjusted values of temperament characteristics and depressive symptoms predicted by the linear regression models. The growth curves (and the cross-lagged analyses) were applied to all available data and not just for individuals who responded at all five phases. The maximum likelihood (ML) algorithm is conditioned on complete data for the independent variables and adjusts estimates for non-response in the dependent variables if data are missing at random. The analyses were performed using STATA/SE v.12.0® (StataCorp 2005).

Goodness-of-fit of the SEM models was evaluated using the following fit indices: Chi-square, the root mean square error of approximation (RMSEA), Bayesian Information Criterion (BIC), comparative fit index (CFI), and Tucker–Lewis Index (TLI). A non-significant Chi-square value indicates that the model fits the data. However, Chi-square is highly sensitive to sample size. RMSEA values of less than 0.05 and 0.10 represent a good and acceptable fit, respectively, whereas CFI values above 0.90 and 0.95 indicate an acceptable and good fit, respectively (Byrne, 1998). In comparing alternative models, a statistically significant improvement in the Chi-square value indicated a better fit of the model.

Fourth, because different time intervals between follow-ups may preclude accurate estimation of discrete-time autoregressive models, we also estimated a two-dimensional continuous-time state space model (called Exact Discrete Model, EDM) that described the concurrent time-evolution of depressive symptoms score and temperament traits (Oud and Jansen, 2000; Delsing and Oud, 2008).Estimation of the EDM was done using OpenMx software (Boker et al., 2011).

We compared and ranked the fit of SEM models (including EDM) using Akaike's Information Criterion (AIC) and Bayesian Information Criterion (BIC) that favor models that fit well to observed data but also punish from complexity and thus aim to favor the most "parsimonious" model. Both AIC and BIC aim for this same goal, but AIC tends to be a better approximation for optimal performance when true data-generating model is not included among the studied models and BIC when it is (Kass and Raftery, 1995; Vrieze, 2012). AIC and BIC use the same measure for model fit, but BIC penalizes much more heavily from model complexity (proportionally to $k \ln(n)$, where model has k parameters and n observations are available) than AIC (proportionally to $2k$). The model with lowest AIC (resp. BIC) is favored by the criterion.

3. Results

Characteristics of the study sample are presented in Table 1. The mean age at baseline for men was 11.1 years and for women 10.7 years. In adulthood, men had higher BMI, smoked more, and used more alcohol than women. Women more often reported using medication and experienced more social support than men. Women

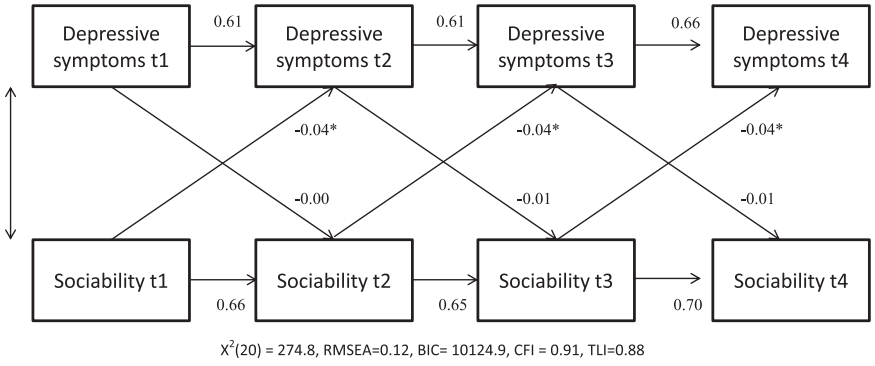

Fig. 1. The repeated cross-lagged model of the temporal associations between depressive symptoms and temperament characteristics sociability in four study phases (t1–t4).

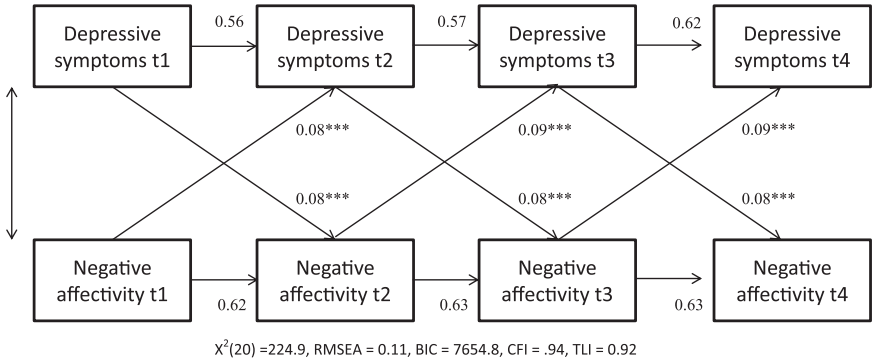

Fig. 2. The repeated cross-lagged model of the temporal associations between depressive symptoms and temperament characteristics negative affectivity in four study phases (t1–t4).

Table 2
Linear regression[a] (based on the 20 imputed data sets) of the longitudinal relationship of the Emotionality-Activity-Sociability temperament characteristics at baseline (in 1997) with depressive symptoms at follow-up (in 2012).

Depressive symptoms at follow-up in 2012								
Temperament characteristics at baseline in 1997	**Model 1**[a]				**Model 2**[b]			
	n	**B (95% CI)**	***t*-value**	***p*-value**	***n***	**B (95% CI)**	***t*-value**	***p*-value**
Sociability	1739	−0.11 (−0.16 to −0.06)	−4.73	<0.001	1739	−0.04 (−0.09 to −0.02)	−2.05	0.041
Negative affectivity	1739	0.34 (0.29 to 0.40)	12.71	<0.001	1739	0.04 (−0.03 to 0.10)	1.15	0.25
Activity	1739	0.01 (−0.06 to 0.06)	0.04	0.97	1739	0.01 (−0.05 to 0.06)	0.25	0.81

[a] Adjusted for age, sex, parental occupational status, own occupational status, health behavior, medication, and social support.
[b] Additionally adjusted for depressive symptoms at baseline.

scored their sociability (mean 3.6−3.3) and negative affectivity (mean 2.8−2.5) higher (all p-values < 0.001) in every data collection phase than men (mean 3.3−3.1/2.5−2.4), and their activity slightly higher in 1997 and in 2001 (the two first data collection phases). Women also reported more depressive symptoms (mean 2.2/2.1) than men (mean 2.0/2.0) in all data collection phases, except the last one (in 2012).

3.1. One-way effects between temperament and depressive symptoms

The results of the longitudinal linear regression analyses of the association between temperament characteristics at baseline and depressive symptoms at follow-up are shown in Table 2. In the first step, without taking into account the effect of baseline depressive symptoms, both sociability ($B=-0.11$, $p<0.001$) and negative affectivity ($B=0.34$, $p<0.001$) were associated with depressive symptoms at follow-up. The association attenuated and the association between negative affectivity and depression was no longer statistically significant when the models were adjusted for baseline depressive symptoms. The longitudinal regression analyses of the association between depressive symptoms at baseline and temperament characteristics at follow-up (Table 3) revealed that baseline depressive symptoms were a predictor of low sociability ($B=-0.13$, $p<0.001$) and negative affectivity ($B=0.41$, $p<0.001$). Only the association between earlier depressive symptoms and negative affectivity was robust to adjustment for baseline temperament characteristics at baseline. No associations were found between depressive symptoms and activity.

3.2. Cross-lagged effects between temperament and depression

The repeated cross-lagged structural equation modeling (Figs. 1 and 2) was conducted only for sociability and negative affectivity. The model suggesting that earlier sociability would predict later depressive symptoms showed an acceptable fit ($\chi^2(21)=274.9$, RMSEA=0.12, BIC=10118.2, CFI=0.91, TLI=0.88). The association between sociability and depression was weak (standardized coefficient −0.04, $z=-2.27$, $p=0.024$). The model testing the association only in the direction from earlier depressive symptoms to later sociability had a slightly worse fit ($\chi^2(21)=279.9$, RMSEA=0.12, BIC=10123.3, CFI=0.91, TLI=0.88), and the association between depressive symptoms and later sociability was not statistically significant (standardized coefficient −0.01, $z=-0.35$, $p=0.73$). The model testing both directions simultaneously (from earlier sociability to later depressive symptoms and from earlier depressive symptoms to later sociability) was no better than the first model ($\chi^2(20)=274.8$, RMSEA=0.12, BIC=10124.9, CFI=0.91, TLI=0.88) (Fig. 1).

Negative affectivity predicted later depressive symptoms (standardized coefficient −0.12, $z=5.84$, $p<0.001$), and the model had an acceptable fit ($\chi^2(21)=245.9$, RMSEA=0.11, BIC=7668.9, CFI=0.94, TLI=0.92). The model testing only the associations from depressive symptoms to later negative affectivity was, however, slightly better ($\chi^2(21)=240.5$, RMSEA=0.11, BIC=7555.1, CFI=0.94, TLI=0.92), and the coefficient from depressive symptoms was significant (standardized coefficient=0.11, $z=6.29$, $p<0.001$). The best-fitting model was the one testing both directions of the associations (from negative affectivity to depressive symptoms and from depressive symptoms to negative affectivity) ($\chi^2(20)=224.9$, RMSEA=0.11, BIC=7654.8, CFI=0.94, TLI=0.92). The coefficient from earlier negative affectivity to later depressive symptoms was 0.09 (z-value 3.95, $p=0.01$), and the coefficient from earlier depressive symptoms to later negative affectivity was 0.08 (z-value 4.57, $p<0.01$) (Fig. 2).

3.3. Associations between intercepts and slopes of temperament and depressive symptoms

The results of the parallel latent growth curve (Figs. 3 and 4) analyses showed that the level of depressive symptoms (intercept mean 2.12, intercept variance 0.29; slope mean −0.03, slope

Table 3
Linear regression* (based on the 20 imputed data sets) of the longitudinal relationship of depressive symptoms at baseline (in 1997) with the Emotionality-Activity-Sociability (EAS) temperament characteristics at follow-up (in 2012).

	EAS temperament characteristics at follow-up in 2012			
	n	**B (95% CI)**	***t*-value**	***p*-value**
	Sociability			
Depressive symptoms at baseline in 1997				
Model 1	1739	−0.13 (−0.19 to −0.07)	−4.42	<0.001
Model 2	1739	−0.03 (−0.08 to 0.02)	−1.18	0.28
	Negative affectivity			
Depressive symptoms at baseline in 1997				
Model 1	1739	0.41 (0.36 to 0.45)	17.88	<0.001
Model 2	1739	0.11 (0.05 to 0.17)	3.89	<0.001
	Activity			
Depressive symptoms at baseline in 1997				
Model 1	1739	−0.05 (−0.10 to 0.01)	−1.86	0.06
Model 2	1739	−0.04 (−0.08 to 0.01)	−1.94	0.06

Model 1: Adjusted for age, sex, parental occupational status, own occupational status, health behavior, medication, and social support.
Model 2: Additionally adjusted for the corresponding temperament characteristics at baseline.

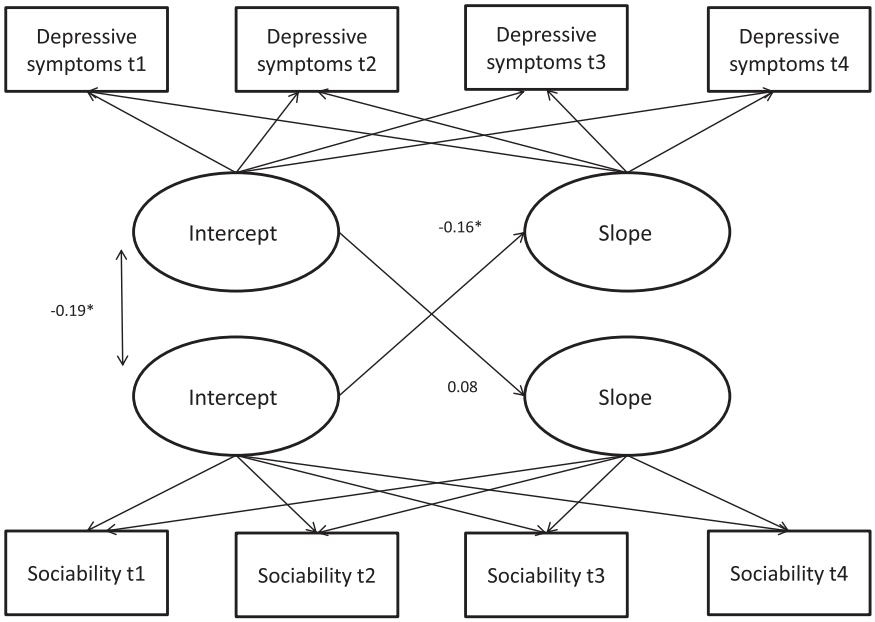

Fig. 3. The parallel latent growth curve model of the association between depressive symptoms and temperament characteristics sociability in four study phases (t1–t4). Intercepts weights are fixed at 1. Slope weights are fixed at 1,2,3,4. ($\chi^2(21)=160.17$, $p=0.003$, RMSEA=0.09, BIC=9161.08, CFI=0.95, TLI=0.93).

variance 0.02) was not associated with the development (slope mean −0.07, slope variance 0.02) of sociability (standardized coefficient 0.08, $z=1.05$, $p=0.29$). However, the level of sociability (intercept mean 3.46, intercept variance 0.40) was associated with the development of depressive symptoms (standardized coefficient −0.16, $z=-2.22$, $p=0.026$). The model incorporating both paths (from depressive symptoms to sociability and from sociability to depressive symptoms) offered a reasonably good fit to the data ($\chi^2(21)=160.17$, $p=0.003$, RMSEA=0.09, BIC=9161.08, CFI=0.95, TLI=0.93) (Fig. 3).

The baseline (intercept) of depressive symptoms was associated with the slope (mean −0.09, variance 0.02) of negative affectivity (standardized coefficient −0.29, $z=-4.37$, $p<0.001$), and the intercept of negative affectivity (mean 2.69, variance 0.27) was also associated with the development of depressive symptoms (standardized coefficient −0.25, $z=-4.44$, $p<0.001$). The fit of the model was close to acceptable ($\chi^2(21)=678.9$, RMSEA=0.29, BIC=7398.75, CFI=0.83, TLI=0.80) (Fig. 4).

3.4. Continuous concurrent time-evolution of temperament and depressive symptoms

The results of EDM basically supported the results of the cross-lagged SEM. Table 4 summarizes model fit for the continuous-time models. For sociability and negative affectivity, the EDM seemed to provide good descriptions of the observed data. Cross-lagged effects from sociability to depressive symptoms were of very similar magnitude as those from depressive symptoms to sociability, and both variables reduced each other. Cross-lagged effects from negative affectivity to depressive symptoms were slightly larger than those from depressive symptoms to negative affectivity (~23 percentage units after few years lag), but both variables induced each other according to the model estimate. The cross-lagged effects exhibit a peak at a certain lag (~1.5 years for sociability and ~1.8 years for negative affectivity). Autoregressive effects were quite similar among the personality traits and depressive symptoms (Fig. 5).

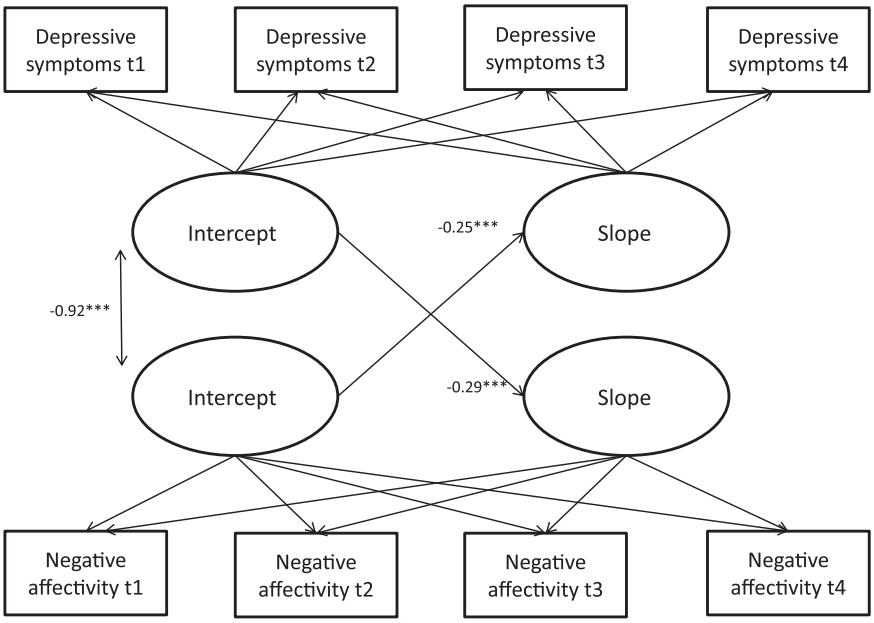

Fig. 4. The parallel latent growth curve model of the association between depressive symptoms and temperament characteristics sociability in four study phases (t1–t4). Intercepts weights are fixed at 1. Slope weights are fixed at 1,2,3,4. ($\chi^2(21)=678.9$, RMSEA=0.29, BIC=7398.75, CFI=0.83, TLI=0.80).

Table 4
Comparison of co-development (Exact Discrete Model, EDM) and Saturated ('Null'/non-linear co-development) models.

Covariate	Model	AIC	BIC	χ^2	d.f.	*p*-Value
Negative affectivity	EDM	−5471.35	−40425.85	60.13	27	$2.5 \cdot 10^{-4}$
	Saturated	−5477.48	−40284.51	–	–	–
Sociability	EDM	−3043.21	−38003.17	36.36	27	0.108
	Saturated	−3025.57	−37838.06	–	–	–

p-value indicates whether the EDM model differs significantly from the Saturated model.
AIC=Akaike's Information Criterion
BIC=Bayesian Information Criterion

4. Discussion

Our study examined the direction of the association between depressive symptoms and the three temperament components described by the temperament model of Buss and Plomin (Buss, 1991; Plomin et al., 1988), negative emotionality, activity, and sociability. Over a period of 15 years among a large population-based sample of Finnish men and women, we found that higher levels of both low sociability and high negative emotionality predicted subsequent more depressive symptoms, whereas earlier high depressive symptoms predicted only higher negative emotionality. Therefore, our findings suggest that the direction of the long-term association is from low sociability to depressive symptoms rather than the reverse. However, the association between negative emotionality and depressive symptoms seem to be reciprocal. Thus, it may be that negative emotionality and depressive symptoms affect each other, forming a kind of vicious circle of increasing risk of depressive symptoms and psychological vulnerability to such symptoms.

There are several potential mechanisms explaining the association between temperament and depressive symptoms. The relationship between temperament and depression has been suggested to be explained by shared genetic factors that predispose an individual to both experiencing depressive symptoms and being high in a certain temperament dimension. A Swedish study including 20 692 twins showed that an association between neuroticism and depression resulted largely from shared genetic background, which influenced both neuroticism and depression (Kendler, 1992). The genetic liability that is shared between personality and depression is suggested to be about 50% (Kendler et al., 1993). Thus, higher risk for depressive symptoms among those low in sociability and high in negative affectivity may be dependent on the same genetic structures that determine both individual differences in temperament characteristics and risk of depressive symptoms. Consequently, one possible interpretation of our findings is that low sociability or high negative affectivity do not cause depressive symptoms, but are markers of genetic or some other biological or structural vulnerability to future depressive symptoms.

Another potential explanation is derived from a "differential susceptibility" hypothesis that suggests that an individual's characteristics, such as temperament, moderate the individual's reactions to stressful life events (Ellis et al., 2011; Belsky and Pluess, 2013; Elovainio et al., 2004). This hypothesis proposes that individuals possessing certain temperament characteristics (such as high negative emotionality) are emotionally more reactive and tend to cope with negative experiences in unproductive ways (Carver and Connor-Smith, 2010). This in turn may make it difficult to maintain the positive self-evaluation needed for optimal mental health. Children who are temperamentally fearful have been shown to be likely to develop negative thinking styles when faced with external stresses (Mezulis et al., 2004). Our findings thus suggest that individuals with low sociability and high negative emotionality are at higher risk of depressive symptoms partly because they are more vulnerable to adverse experiences.

A third explanation suggests that inherited temperament selects individuals into certain environments and temperament

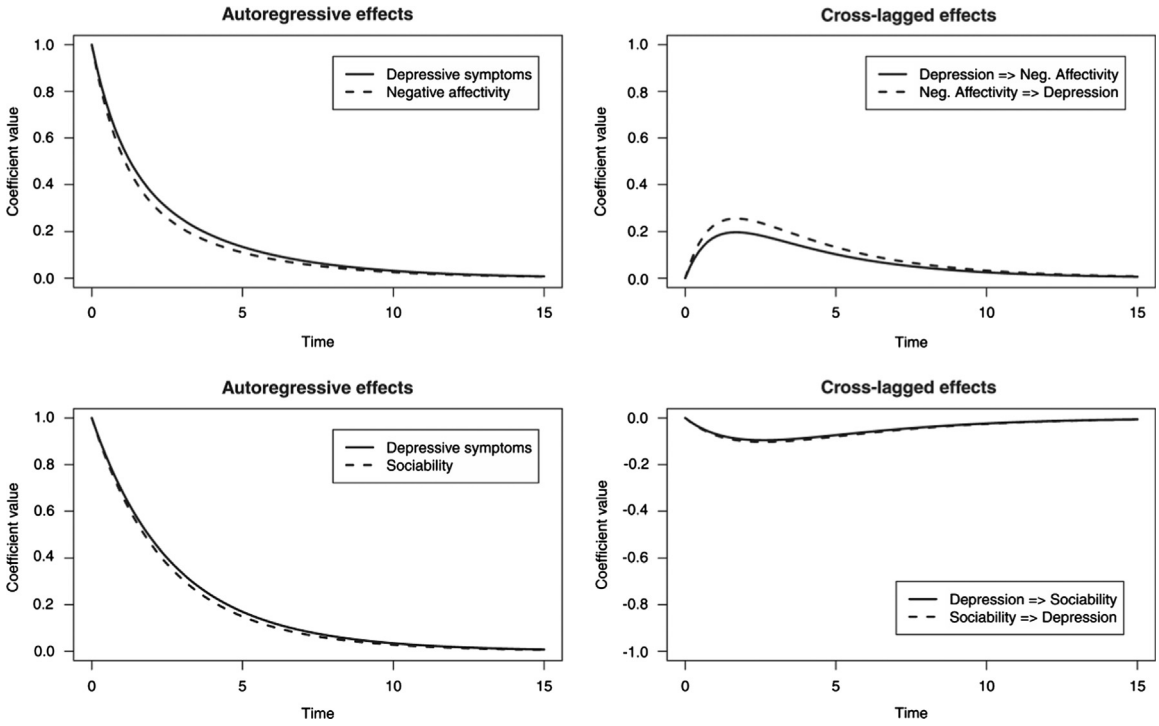

Fig. 5. Exact Discrete (depression co-evolution) Model fits for the Negative-Affectivity and Sociability traits. Both autoregressive (1st column of panels) and cross-lagged (2nd column) effects are shown for the co-evolution of Negative Affectivity and depressive symptoms (1st row) and for the co-evolution of Sociability and depressive symptoms (2nd row). The graphs show the discrete-time effect as a function of measurement-time distance, as predicted from the estimated continuous-time model.

modifies these environments. According to this view, environment is influenced by the individual's own behaviors (Kendler and Prescott, 2007). Individuals with low levels of sociability are likely to withdraw from social interactions, and thereby, they may face a less supportive environment, which in turn may lead to higher risk of depressive symptoms. They may also have less social skills, therefore being less able to form supportive relationships with others. Although sociability indicates an individual's need for social contacts, it has been argued that the core of the sociability dimension is nevertheless affective and that persons high in sociability frequently experience positive feelings (Clark et al., 1994).

It is also reasonable to assume that depressive symptoms may predict changes in temperamental characteristics. Functional neuroimaging studies have repeatedly shown that especially major depression manifests as widespread activity abnormalities in certain brain regions such as the prefrontal cortex, limbic areas, and subcortical structures (Mah et al., 2007; Scott et al., 2012). In individuals with an inhibited temperament relative to those with an uninhibited temperament, differences have been demonstrated in amygdala and hippocampus function using functional magnetic resonance imaging (fMRI). For example, individuals with an inhibited temperament show multiple differences in amygdala function relative to those with an uninhibited temperament, including faster amygdala responses to novel faces, sustained amygdala responses to recently familiarized faces, and greater amygdala responses to novel faces (Schwartz et al. 2003). Recent findings in non-human primates demonstrate that increased hippocampus activation is a key neural signature of anxious temperament (Oler et al. 2010), a construct similar to inhibited temperament in humans. Our results are only partly in agreement with the idea that depression may alter temperament through potential changes in brain structures and connectivity (Buss, 1991; Cloninger and Svrakic, 1993). Earlier depressive symptoms predicted only changes in later negative emotionality, not sociability.

Negative affectivity is a temperament trait that is, by definition, closely related to emotional reactions, such as fear, distress, and anger, which are often experienced in depressive episodes. Thus, it is reasonable to assume that there may be common biological or environmental factors explaining both disposition and emotional problems, and a bidirectional association may exist between the two. Sociability defined as a preference to be with people and to interact with others may be more indirectly related to depressive symptoms and thus to emotional reactions. Depressive symptoms may make people less social, and in the long run may affect one's sense of self as a social person.

In this study, we tested the direction of the association between temperament characteristics and depressive symptoms using four statistical modeling procedures that were aimed at complementing the potential shortcomings of each individual procedure. We used longitudinal linear regression analyses as a standard tool and repeated the analyses using cross-lagged structural equation modeling, parallel latent growth curve modeling, and exact discrete modeling. Applying repeated cross-lagged modeling, we were able to simultaneously test both directions of the associations using an authentic prospective design with several repeated measurements over a 15-year interval. Using latent growth curve analyses, we were able to divide both temperament and depressive symptoms into baseline (intercept) and change (slope) and model the reciprocal associations between the two factors. Using exact discrete modeling, we were able to take into account the various follow-up times between repeated longitudinal measures and the potentially attenuating effects over the 15 years that may produce differences in various discrete follow-up times. With repeated follow-up, autoregressive effects can only decline and cross-lagged effects exhibit a peak at a certain time-window, and thus, using different time-frames in discrete modeling may show arbitrarily different associations.

Although the standard linear regression procedure produced slightly different results than the other procedures, the results

over various statistical modeling were consistent. Availability of long-term repeated data from more than two occasions and at shorter intervals helped to reduce some of the potential limitations and to further illuminate the lag time necessary for depressive symptoms to appear and be detected. Such data also help to eliminate the possibility that differences in temporal stability between temperament and depressive symptoms would have biased the observed direction of the associations between the two (if temperament were more stable than depressive symptoms over time).

When interpreting the results, some limitations must be noted. First, the final analyses were conducted using participants with complete data on depressive symptoms at the final follow-up. This meant that more than half of the original population was excluded. Some differences existed in baseline characteristics between included and excluded subjects, and thus, selective sample attrition may have affected our results. However, the main findings of this study were reproducible in the entire cohort with missing values imputed using multivariate multiple imputation methods. Second, the estimates of the associations between depressive symptoms and temperament characteristics were weak, and thus, the explained variance was relatively modest. A large sample, such as the one used here, usually means smaller (but more accurate) effect sizes and almost inevitably worse fit indices of the statistical models. Thus, the smaller differences between the tested theoretical models and the data become statistically significant. Depressive symptoms are multifactorial and we consider our results suggesting that temperament and depressive symptoms are related to each other partly in a reciprocal way and that the association throughout the life course is moderate are reasonable. One limitation of the study is that the baseline of the sample covers an age where many individuals have already manifested depressive symptoms or even episodes of major depression. Thus, the results are less related to the first onset of depression and more to the course of depressive symptoms during adulthood.

The depressive symptoms scale used was not a measure of clinically recognized psychiatric disorder and did not indicate the severity or the chronicity of depression (Josefsson et al., 2011). Whether our measure of depressive symptoms can be considered an indicator of early stages of clinically diagnosed depression remains to be ascertained. The possibility that subthreshold symptoms may be important indicators of future risk was suggested in a recent study where subthreshold depressive symptoms predicted a 6-fold risk of developing a depressive disorder within 2 years in a general population (Karsten et al., 2010). If this proves to be the case in our sample, our results would indicate that low sociability and high negative affectivity may play roles as risk factors of depression through their contribution to progression of depressive symptoms.

The EAS model is, of course, only one of the many temperament models measuring adult temperament. One may criticize the EAS model for not covering all of the characteristics of adult temperament, e.g. persistence/adaptability, or for including too many characteristics in large dimensions . However, none of the temperament models has achieved general consensus among the scientific community. The EAS model includes the central dimensions of risk factors for depressive symptoms. It is reasonable to assume that low sociability and low activity and negative emotionality as defined by the EAS temperament model may all be related to depressive symptoms, including negative moods, lack of energy, and withdrawing from social contacts and activities (DSM-V).

Strengths of the present investigation include a large study sample that provides sufficient statistical power to detect associations. We used repeated perceptions of temperament and depressive symptoms from four repeated surveys with sufficient self-assessment. We were also able to adjust for many of the potential confounders, such as sex, socioeconomic position, and various physical health risks, potentially affecting relationships between temperament and psychosocial environmental factors and mental health.

In summary, findings over a 15-year period from a large-scale prospective Finnish cohort support a predictive association from temperament (as assessed with both low sociability and high negative affectivity) to depressive symptoms. This relationship did not appear to be attributable to potential confounders measured in this study. Furthermore, the association between negative affectivity and depressive symptoms seemed to be, to some extent, bidirectional, representing a vicious circle of negative emotionality and depressive symptoms that may potentiate each other over time.

There are two clinical implications of our findings. It is important to detect individuals at excess risk of developing later depressive symptoms, especially those with high negative affectivity and low sociability. The reciprocal association between depressive symptoms and negative affectivity suggests that treatment of depressive symptoms and not just clinical depression is important not only because depressive symptoms often predict more severe depressive episodes, but also because depressive symptoms may alter in a negative manner a relatively stable way of reacting and behaving. This negative reactivity may, in turn, provoke further depressive symptoms, forming a vicious cycle leading to more severe symptomology.

Role of funding source

None of the funders played any role in data collection, designing the study, analyzing or interpreting the results, or reporting the findings.

Conflict of interest

All authors declare no conflict of interest.

Acknowledgments

The research is funded by Academy of Finland (projects 258711, 265 977, 265869, 128002, 132944), Signe and Ane Gyllenberg Foundation the Finnish Work Environment Fund, the EU New OSH ERA research programme, The Juho Vainio Foundation (L.P.R) and the Finnish Cultural Foundation (C.H.)

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.jad.2014.08.040.

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