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Original Article

Long-term personality changes and predictive adaptive responses after depressive episodes

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ABSTRACT

An external or internal "predictive adaptive response" (PAR) can be defined as an adaptive change in long-term behavior or development due to an environmental exposure that triggers it. A PAR can lead to differential development among initially similar individuals, and increase evolutionary fitness. Despite many theories and empirical observations of PAR-like changes in depressive tendencies, clear empirical findings on human personality changes following depressive symptoms are lacking, possibly because these changes take a long time to develop and most follow up studies have been short. Here we show that in sufficiently long (5- and 15-year) clinical and general-population follow ups, increases can be observed in the Temperament and Character Inventory's personality trait harm avoidance as a function of temporally accumulating major depressive episodes (132 depression patients from Vantaa Depression Study) and depressive symptoms (3105 participants from Young Finns general-population sample). Personality changes did not occur in the other six personality traits of the inventory, but did in a highly similar neuroticism trait from another inventory. Even when controlling for concurrent changes in depressive symptoms from the baseline to the endpoint, depressive symptoms that occurred during the follow-up period associated with harm-avoidance changes, rendering individuals more fearful and anticipating harm. This study provides consistent, specific, and plausible dose–response and temporal gradients between accumulated depressive episodes and personality change. Effect sizes were between small to moderate, though. Altogether, the findings support the feasibility of using existing systems of personality assessment (i.e., self-report questionnaires) to study PARs, despite the multiplicity of the systems.

1. Introduction

Differential environmental conditions could lead individuals to 'choose' a unique optimal developmental trajectory, producing stable developmental differences in behavior, that is, personality differences (Allport, 1937; Wolf, van Doorn, Leimar, & Weissing, 2007; Wolf, van Doorn, & Weissing, 2008). An external or internal "predictive adaptive response" (PAR) could be such a developmental trigger, but the issue of prevalence of PARs in humans lacks theoretical clarity (Del Giudice, 2014; Nettle, Frankenhuis, & Rickard, 2013), let alone empirical verification. Roughly, in an external PAR hypothesis, ones' surroundings provide a 'weather forecast' of circumstances to come, and the development is modified accordingly, whereas in an internal PAR the surroundings inflict a lasting impact on individual's somatic state (e.g., health) and the altered state requires compensating behavioral adjustments (Nettle et al., 2013). In principle, appropriate empirical evidence could put the matter to rest, but empirical studies are complicated by the fact that many validated measures of human personality are self-assessments that cannot be easily administered in early developmental periods, and there are several systems of assessment without a clear consensus on preference ordering (Grucza & Goldberg, 2007; John, Robins, & Pervin, 2008).

A 'brute force' approach to the issue would be to detect an adulthood PAR and simply show its impact on all, or at least several, major personality systems out there. Such an approach would also require a suitably common exposure that could feasibly trigger a PAR. Depressive disorder is common, functionally disabling (Bromet et al., 2011), and strongly associated with triggering stressful life events (Kendler, Karkowski, & Prescott, 1998) and somatic changes (Mykletun et al., 2007), thereby being relevant for both internal and external PARs. So relevant, that several theoretical works have studied the possible adaptive role of depression in evolution (Hagen, 1999; Nesse, 1991, 2000, 2009; Nettle, 2004; Rosenström, 2013). Furthermore, depression researchers have long been curious

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about the possibility of personality change due to depressive episodes. A specifically depression-predisposing change in personality has been called a personality "scar" in this literature (Wichers, Geschwind, van Os, & Peeters, 2010). Personality scarring has been interesting because it might help to explain many empirical results, such as the finding that first episode of depression is more likely to be preceded by major environmental stressors than the subsequent episodes (Monroe & Harkness, 2005; Stroud, Davila, & Moyer, 2008). Empirical studies have mostly failed to find lasting personality changes that follow depressive episodes, however (Jylhä, Melartin, Rytsälä, & Isometsä, 2009; Klein, Kotov, & Bufferd, 2011; Ormel, Oldehinkel, & Vollebergh, 2004; Wichers et al., 2010). Yet, we, and others (Wichers et al., 2010), argue that it is premature to abandon this possibility to demonstrate PARs in effect.

Instead of being caused by single episodes, scars may "develop gradually along the life cycle, proportionally to the severity and duration of the depressive symptoms experienced", and therefore it is critical to re-examine the issue of personality changes in a sufficiently long follow-up study with multiple temporal sampling points (Wichers et al., 2010). Indeed, in a recent study researchers fitted several statistical models of bivariate temporal development to traits from Emotionality–Activity–Sociability system of personality and a depressive symptoms scale assessed in a 15-year follow up, showing a reciprocal temporal interaction between negative emotionality and depression (Elovainio et al., 2015). That is, also the experienced depressive symptoms appeared to causally, or at least temporally (Hill, 1965), antecedent to the personality trait emotionality, not just other way around, suggesting personality scarring due to depression in a long-term follow up. We have previously studied long-term effects of personality on depression using different systems of personality assessment (Jylhä et al., 2009, 2011; Rosenström et al., 2014), but encouraged by the aforementioned results, we present here also a brute force analysis of personality scarring, or PARs (i.e., the opposite temporality), using two different personality systems and both clinical and general-population samples. Also, we now have followed longer the patients that previously led us to conclude a lack of scar-effects, and re-examine that inference (Jylhä et al., 2009).

The aim of this study was to assess if temporal accumulation of depressive or dysphoric episodes lead to changes, or scars, in personality traits using longitudinal data. In order to cover a sufficient time span for detecting changes in self-representation, we studied 5-year clinical follow-up data from the Vantaa Depression Study (VDS) (Holma, Holma, Melartin, Rytsälä, & Isometsä, 2008; Melartin et al., 2002) and a 15-year follow-up of general-population sample from Young Finns Study (YFS) (Raitakari et al., 2008). The aim was also to study consistency of findings across measures and clinical versus non-clinical populations. Whereas we previously found support for scarring using Buss's and Plomin's Emotionality–Activity–Sociability system of personality assessment (Buss, 1991; Elovainio et al., 2015), we now study also Cloninger's Psychobiological system of personality (Cloninger, Svrakic, & Przybeck, 1993; Cloninger, Przybeck, Svrakic, & Wetzel, 1994) and Eysenck's personality system (Eysenck & Eysenck, 1964), a predecessor of the well-known big five system of personality assessment (John, Naumann, & Soto, 2008). If all the personality systems and all the studied populations show consistent evidence for personality changes due to depressive episodes, that would constitute a strong argument for adulthood PARs. In contrast, if only sporadic and temporally ambiguous associations between baseline depression and personality change exist, that would suggest the lack of PAR-like causation between depressive episodes and commonly used measures of human personality (Hill, 1965).

2. Methods

2.1. Participants

2.1.1. Patients from Vantaa Depression Study (VDS)

Participants of the VDS were screened from all psychiatric patients aged 20–59 years who were inhabitants of the City of Vantaa in Finland (population 169 000 in 1997), and seeking treatment, receiving it, or referred to treatment in the Department of Psychiatry of the Helsinki University Central Hospital (former Peijas Medical Care District). Study inclusion criterion was Diagnostic and Statistical Manual of Mental Disorders IV (DSM-IV) defined major depressive disorder (MDD) with a new depressive episode (American Psychiatric Association, 1994). Patients provided information at the baseline in 1997, after 6 months, after 18 months, and after a five years' time from the baseline using a life-chart methodology (see Measures). Patients with bipolar spectrum, schizophrenia, and/or substance-induced mood disorders were excluded, as well as the patients who switched to other than MDD diagnosis during the five-year follow-up.

Originally, altogether 806 psychiatric patients were screened for depressive symptoms during an eighteen-month period. Of those 703 who had symptoms, 542 (77%) agreed to participate. They gave their written informed consents, and the study was approved by the ethics committee of the Healthcare District of Helsinki and Uusimaa. Then, a researcher using World Health Organization Schedules for Clinical Assessment in Neuropsychiatry diagnosed 269 patients out of the 542 as having DSM-IV defined MDD (judging from the 20 videotaped interviews, κ-coefficient for MDD was 0.86 and the observers agreed at 95% rate).

At the final follow up, a life chart of the entire five-year follow-up period was constructed individually for 142 patients by two interviewers, using all available medical and psychiatric records to complement the information. Finally, 132 patients provided the TCI at the final follow up. Further details of the sample can be found from previous publications (Holma et al., 2008; Melartin et al., 2002) and online supplement (available on the journal's Website at www.ehbonline.org). Examples of reasons for dropping out of the study are withdrawal of consent, or unreachable, dead, or living too far away subjects (Holma et al., 2008).

2.1.2. General-population sample from the Young Finns Study (YFS)

The YFS is an ongoing prospective study that began in 1980 (Raitakari et al., 2008). The original sample consists of 3596 healthy Finnish children and adolescents (1832 women, 1764 men) sampled from six birth cohorts with approximately equal frequency. In order to select a broadly sociodemographically representative sample, Finland was divided into five areas according to locations of university cities with a medical school. In each area, urban and rural boys and girls were randomly selected on the basis of their unique personal social security number. All participants gave written informed consent, and the study was approved by the local ethics committees. The sample has been followed subsequently in 8 data collection waves, but mainly data from the four latter waves (years 1997, 2001, 2008, and 2012) were used herein, as they contained relevant measures. Participants' ages were 20–35 at baseline, with the prospective follow-up period spanning 15 years.

Altogether 868 participants (301 men and 567 women) had provided complete data relevant for the analyses in the YFS data, but multiple-imputation methods made it possible to use information from 3105 participants (1432 men, 1673 women). Supplementary on-line material (available on the journal's Website at www.ehbonline.org) presents details of the imputation analysis, and also studies the sensitivity of the findings to the imputations.

2.2. Measures

2.2.1. Measures of depression

Depression can be conceptualized as a sum of severity-levels of depressive symptoms, or as a state of exceeding certain total severity or symptom count. The former way seems to be closer to natural reality (Haslam, Holland, & Kuppens, 2012), but the latter is more common in clinical practice and has the benefit of allowing one to compute the time an individual has spent in a disordered or dysphoric state. This study does not aim to solve what definition is best, but instead uses typical measures for both types of definitions and aims for conclusions that

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generalize over different definitions. While existing definitions of depression may ultimately change (Fried & Nesse, 2015; Keller, Neale, & Kendler, 2007; Nesse, 2009), it is also likely that researchers continue to draw from the vast pool of accumulated knowledge on the existing definitions, whenever they permit generalizable results; especially so, because clear directions for changing the definitions are lacking (Fried & Nesse, 2015; van Loo, de Jonge, Romeijn, Kessler, & Schoevers, 2012; but see Wakefield & Schmitz, 2013; Zimmerman, Ellison, Young, Chelminski, & Dalrymple, 2014).

In the VDS data, all collected information was integrated into a graphic life chart together with the patient. In addition to symptom ratings, change points in psychopathological states were inquired using probes related to important life-events in order to improve the accuracy of the assessment. Proportions of time in the five-year follow-up during which the participants fulfilled DSM-IV criteria for MDD (5 or more of the 9 symptoms) were computed from the life charts. See previous report for full details about the life-chart methodology (Holma et al., 2008). Accuracy of life charts must considerably exceed simple interpolation from face-to-face follow-up assessments, but cannot be quantified further, as the patients were not under continuous surveillance. In addition to the life chart, Beck's Depression Inventory (BDI) was completed by the participants in all follow-ups (Beck & Steer, 1993; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961).

A modified version of the Beck's Depression Inventory (mBDI) was completed by the YFS participants in all the four follow-up assessments used herein. Beck's Depression Inventory is a sum score of severity levels for 21 depressive-symptom descriptions. The modified version has been comprehensively described in the online supplement (available on the journal's Website at www.ehbonline.org) and previous publications (Rosenström et al., 2014).

2.2.2. Personality measures

A personality trait is a continuous measure for individual differences occurring along a certain dimension of behavior and thought. These dimensions are typically (and herein) defined by summing several questionnaire items into a single score that reflects a given general tendency to behave and think. Our aim was not a detailed study of single system of personality assessment, but rather integration of generalizable evidence across personality systems. Therefore, only the main personality traits that showed significant effects are shortly described in Results and Discussion sections, when needed. All the studied traits are further described in the online supplement (available on the journal's Website at www.ehbonline.org) and preceding publications (Cloninger et al., 1993, 1994; Enns & Cox, 1997; Eysenck & Eysenck, 1964).

Personality as defined by the psychobiological model of temperament and character (Cloninger, 1987; Cloninger et al., 1993) was assessed in the final follow-up of the VDS and both baseline and endpoint of the YFS. The VDS used revised version (an official 5-point formulation of original yes/no items) of Temperament and Character Inventory (TCI; Cloninger et al., 1994), and the YFS used an unofficial, but much studied, 5-point formulation of the original items, also referred to as TCI (e.g., Josefsson et al., 2013; Rosenström et al., 2014). Both, highly similar, inventories can be used to assess the same seven major traits in the psychobiological model (listed in Tables 1 and 2). In addition, Eysenck's Neuroticism and Extraversion Personality traits were assessed at the baseline and endpoint of the VDS (Enns & Cox, 1997; Eysenck & Eysenck, 1964).

2.3. Statistics

Statistical methods were chosen to succinctly describe the effect of temporally accumulating depression on the personality at the endpoint of the follow up, or to personality change from baseline to endpoint, and the analytic choices reflect both this aim and the restrictions of the data. Long research projects are subject to naturally changing conditions, peoples, and scientific ideas. Thus, they often suffer from missing values or variables and changing methods of measurement.

Fig. 1 illustrates the temporal structure of the datasets and the variables we had at our disposal, and in below, we will describe the analytic strategy (next paragraphs and on-line supplement, available on the journal's Website at www.ehbonline.org, provides technical details). First, we assessed whether clinical patients' time spent in MDEs associated with TCI personality traits at the end of the follow up. Although this does not allow direct inferences about personality changes as a function of time spent in major depressive episodes (MDE), as we lacked the baseline TCI measures for the patients, the associations logically tend towards the changes. These associations were compared to general-population results, wherein the baseline assessments were available, and to results on clinical patients' neuroticism and extraversion that were also measured both at baseline and at the endpoint. Second, we regressed the population-dwellers' TCI values at the endpoint (year 2012) onto standardized sum of depression scores from the years 2001 and 2008, while controlling for preceding TCI levels, observed in year 1997. Any significant associations between endpoint TCI and preceding depression scores that are independent of the even more preceding TCI values are likely to be due to depression-related changes in personality. Third, when possible, we assessed partial correlations among the observed changes in relevant personality traits and changes

Table 1
Regression coefficients (95% CI) from beta inflated regression of TCI-personality traits at 5-year follow up onto preceding time spent in major depressive episode by the depression patients.

Variable	Model I	Model II	Model III
Intercept	–	–	−1.27 (−1.97, −0.57)***
Age	–	–	0.00 (−0.02, 0.01)
Sex (male)	–	–	0.35 (−0.05, 0.75)
BDI bl	0.25 (0.08, 0.42)**	–	0.14 (−0.03, 0.31)
BDI 5y	0.68 (0.51, 0.85)***	–	0.55 (0.33, 0.77)***
Novelty seeking 5y	−0.06 (−0.26, 0.14)	−0.01 (−0.18, 0.16)	0.14 (−0.08, 0.36)
Harm avoidance 5y	0.50 (0.30, 0.70)***	0.24 (0.04, 0.44)*	0.35 (0.08, 0.62)**
Reward dependence 5y	−0.15 (−0.35, 0.05)	0.00 (−0.17, 0.17)	0.02 (−0.22, 0.26)
Persistence 5y	−0.20 (−0.40, 0.00)*	−0.12 (−0.29, 0.05)	−0.04 (−0.24, 0.16)
Self-directedness 5y	−0.30 (−0.50, −0.20)	−0.01 (−0.21, 0.19)	0.18 (−0.09, 0.45)
Cooperativeness 5y	−0.16 (−0.33, 0.01)	−0.04 (−0.21, 0.13)	−0.06 (−0.31, 0.19)
Self-transcendence 5y	0.17 (−0.03, 0.37)	0.02 (−0.15, 0.19)	0.09 (−0.11, 0.29)

Note: "BDI" = Beck's Depression Inventory; "bl" = variable in the baseline; "5y" = variable in the end of the 5-year follow up; "Model I" = regression models with only indicated personality trait and age and sex covariates; "Model II" = multivariate regression models with age, sex, indicated personality trait and baseline and endpoint BDI; "Model III" = multivariate regression model with age, sex, all personality traits, baseline BDI, and BDI at the 5-year endpoint. Wald-type 95% confidence interval is provided in parentheses after the regression coefficients.

* p-value less than 0.05.
** p-value less than 0.01.
*** p-value less than 0.001.

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Table 2
Coefficients (95% CI) from linear regression of TCI-personality trait endpoints on depressive symptoms in middle of the 15-year YFS follow-up, adjusting for baseline personality and baseline and endpoint depressive symptoms.

Personality trait	Model A	Model B
Novelty seeking	−0.04 (−0.11, 0.03)	−0.01 (−0.07, 0.05)
Harm avoidance	0.10 (0.05, 0.15)***	0.07 (0.03, 0.11)***
Reward dependence	0.02 (−0.04, 0.07)	−0.00 (−0.05, 0.05)
Persistence	−0.00 (−0.08, 0.07)	0.02 (−0.05, 0.10)
Self-directedness	−0.11 (−0.16, −0.05)***	−0.07 (−0.11, −0.02)***
Cooperativeness	−0.02 (−0.09, 0.05)	0.03 (−0.02, 0.09)
Self-transcendence	0.03 (−0.05, 0.11)	0.02 (−0.06, 0.10)

Note: Regression coefficients are for a standardized sum of 2001 and 2008 depression scores, with a 2012 personality trait as an outcome. Other "adjusting" covariates were the same personality trait in 1997, age cohort, sex, and depression score in 1997, and depression score in 2012. Model(s) A include only single trait as an outcome, whereas the model(s) B include the indicated trait as an outcome but add also all other personality traits at the 2012 endpoint to the adjusting covariates. Wald-type 95% confidence interval is given in parentheses for each regression coefficient.
* p-value less than 0.05.
** p-value less than 0.01.
*** p-value less than 0.001.

in depression scores from 1997 to 2012, and the intervening years' depression (2001 and 2008). Whereas adjusting by multiple linear regression (semi-partial correlation) assesses the specific effects of each independent variable on the dependent variable, partial correlation is designed to eliminate the effect of other variables on two variables of interest (i.e., it is an estimate of conditional correlation; Abdi, 2007). Also possible measurement errors behave differently in the second and third case, and hence the analyses provide complementary information. Finally, we studied dose–response between number of intervening dysphoric episodes (none, one either in 2001 or 2008, or two) and personality chance from 1997 to 2012.

Together, these models allow a comprehensive picture of depression-associated personality change, provided that consistent changes exist. In regression models, we present both univariate effects on personality traits (models I, II, and A) and multivariate effects of the traits when controlled for the other traits (models III and B). This allows the reader to see both how individual traits predict the outcome and what they add to predictions by the other traits. Variance inflation factors were below 3.3 in all basic regression models, typically much less, thus excluding the possibility of serious statistical bias due to multicollinearity (Mitra, 2007).

Regarding the VDS, a proportion of time in MDEs was modeled by inflated beta regression (Stasinopoulos & Rigby, 2007), as previously done (Rosenström et al., 2014). It is a basic regression model for proportion variables that can have zero- and unit-proportions. When assessing effect sizes on changes in Eysenck's Personality Inventory, we instead Logit-transformed the MDE proportion, dropping the few zero- and unit-proportions (Rosenström et al., 2014), and provide details and a sensitivity analysis in the online supplement (available on the journal's Website at www.ehbonline.org). Ordinary linear regression with multiple imputation by chained equations (Li, Raghunathan, & Rubin, 1991; White, Royston, & Wood, 2011) was used for regression analyses in the YFS data. A correlation matrix was computed using the expectation maximization algorithm for missing-data modeling ("norm" R package), and normal-distribution theory was used to compute partial correlations from the inverse correlation matrix (see, e.g., Theorem 2.3 in Rue & Held, 2005). These alternative methods for missing-data modeling were used for convergent evidence. All estimates of uncertainty represent 95% confidence intervals (CI). Bias-corrected and accelerated bootstrap CIs were derived for correlations and partial correlations from 10 000 bootstrap resamples (Efron & Tibshirani, 1993). For regression coefficients, the usual Wald-type CI is given. All results derived by missing-data methods are studied in sensitivity analyses without missing-data modeling in the online supplement (available on the journal's Website at www.ehbonline.org), and the supplement also

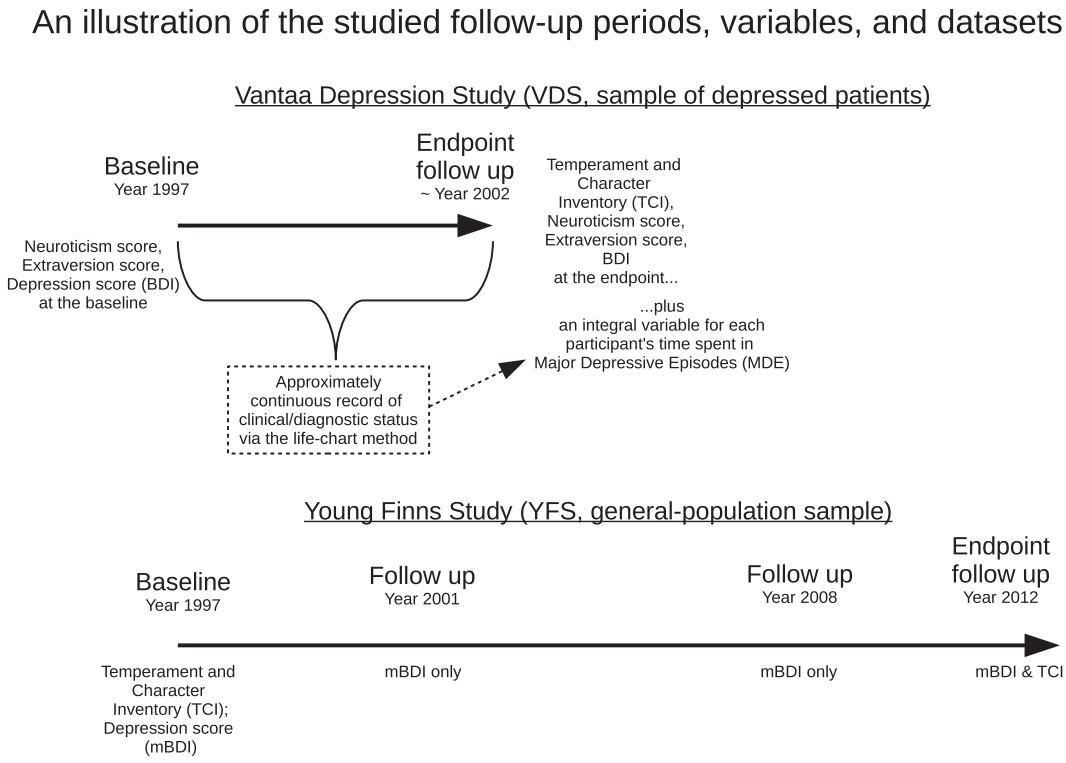

Fig. 1. Timeline-based illustration of the available data in the present study.

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provides further information on the statistical methodology. All computations were performed in 64-bit R software (Linux) version 2.15.3.

3. Results

3.1. Depression patients

Extensive 5-year life charts were constructed from medical records and four interviews (baseline, 6-month, 18-month, and 5-year follow ups) for the 132 treatment-seeking or -referred VDS depression patients (ages 20–59 years) who had completed the TCI at the end of the follow-up (Cloninger et al., 1994). The proportion of follow-up time under DSM-IV-defined MDE was regressed onto the individual TCI personality traits measured at the endpoint and age and sex covariates (Table 1, model I). Mainly the traits harm avoidance and self-directedness were clearly associated with the preceding MDE burden. They reflect tendencies to respond intensely to signals of aversive stimuli (harm avoidance) and to self-determine life goals and to attribute causes of external events to oneself rather than to others (self-directedness). Only harm avoidance's effect remained statistically significant after adjusting for baseline and endpoint BDI scores (Table 1, model II), and it also withstood further adjusting for all the other TCI traits (Table 1, model III). Therefore, our clinical data suggested the trait harm avoidance, encoding inhibition of behavior in response to aversive stimuli and frustrative non-reward, as the strongest candidate (personality dimension) for PARs or scar formation. Although the clinical follow up lacked baseline measurement of TCI, both baseline and endpoint data existed for two other traits, extraversion and neuroticism, from the Eysenck Personality Inventory (Enns & Cox, 1997; Eysenck & Eysenck, 1964).

We then regressed the 5-year change in extraversion and neuroticism onto (Logit of) the proportion of time in MDE, adjusting for the 5-year change in BDI score and for age and sex. Changes in extraversion were unrelated to accumulated MDE time after taking BDI changes into account ($p = 0.385$), but changes in neuroticism were larger for those who had greater temporal MDE burden ($p = 0.005$, $\Delta R^2 = 5.2\%$ corresponding to a conventional small- to medium-sized effect; $R^2 = 12.7$ without any covariate adjusting). Neuroticism is a very similar construct to harm avoidance, implying greater likelihood for interpreting ordinary situations as threatening. Together these results established that accumulated MDEs did associate with changes in personality traits of the clinical patients. A more detailed analysis of neuroticism is available in the online supplementary material (available on the journal's Website at www.ehbonline.org), and the findings for the general population are discussed next.

3.2. General population

The 3105 YFS participants represent the general Finnish adult population, with baseline personality and depressive symptoms having been assessed in the year 1997, endpoints in 2012, and with two temporally intervening depression assessments in the years 2001 and 2008. Because we were particularly interested in whether depression accumulation during follow-up period causes personality changes independently of the baseline and endpoint situation, we regressed endpoint personality onto the standardized sum of the year 2001 and year 2008 mBDI depression scores, adjusting for age, sex, baseline personality, and baseline and endpoint depression scores (Table 2, model A). An independent contribution of intervening depression accumulation on the traits harm avoidance and self-directedness was found again. These effects remained after further adjusting for the other six personality traits (Table 2, model B).

To further isolate how long-term changes in personality and depression were affected by the accumulated experiences, we present correlations and partial correlations between the changes from year 1997 to year 2012 and the sum of the symptoms observed in the intervening years 2001 and 2008 (Table 3). Clearly, the changes in personality correlated strongly with changes in depressive symptoms, but in addition, the intervening symptoms made a contribution to the changes in harm avoidance trait that was independent from the total changes in depressive symptoms across the same period. Furthermore, the sign of the partial correlation was the same as for the correlation (which did not hold for self-directedness). This finding represents a personality change in harm avoidance, caused by the depressive symptoms or their co-occurring life events or physiological changes during the intervening years.

Finally, in order to demonstrate a dose–response relationship, Fig. 2 displays the change in the traits harm avoidance and self-directedness as a function of the number of observed intervening dysphoric episodes [defined as depression score exceeding a pre-specified cut off (Rosenström et al., 2014)]. A dose–response relationship between personality change and the number of dysphoric episodes can be seen for harm avoidance, whereas self-directedness changes show less clear relationship to the episodes. Results based on multiple imputation were shown here, but substantial differences compared to listwise deletion of missing data were not found in the sensitivity analyses (see online supplement, available on the journal's Website at www.ehbonline.org).

4. Discussion

This study observed personality changes in response to accumulated depressive episodes in 5- and 15-year long clinical-patient and general-population follow ups, respectively. The changes showed in the harm avoidance trait of the Cloninger's Temperament and Character Inventory (patients and population dwellers) and neuroticism trait of the Eysenck's Personality Inventory (patients, data not available for the general population). As we have previously reported increases in Buss's and Plomin's negative emotionality trait following depressive symptoms (Elovainio et al., 2015), and both harm avoidance and neuroticism are highly correlated with and conceptually overlapping with negative emotionality (De Fruyt, van De Wiele, & van Heeringen, 2000; John, Naumann, et al., 2008; e.g., respective correlations ~0.54 and ~0.62 with negative emotionality in our data; details not shown), the picture that emerges is the one where adverse experiences and depression lead individuals to adjust their behavior towards caution, or to become sensitized to such experiences, in the long run. This aligns with theories of PARs (Del Giudice, 2014; Nettle et al., 2013) and environmental or somatic sensitization (Monroe & Harkness, 2005; Stroud et al., 2008).

The observed personality changes are to a direction that predisposes for future depression (Elovainio et al., 2015; Rosenström et al., 2014), and thus fulfill the definition of "personality scar" (Wichers et al., 2010). The continuously-varying trait harm avoidance represents a bias to respond intensely to aversive stimuli and to inhibit behaviors. A "harm avoidant" person would be "fearful and anticipating harm even in reassuring and supportive circumstances", pessimistic, inhibited, and fatigable (Cloninger, 1987; Cloninger et al., 1993). A person high in harm avoidance typically is "neurotic", "introverted", and not very "open to new experiences" (De Fruyt et al., 2000). Here, as in a previous study (Kendler, Neale, Kessler, Heath, & Eaves, 1993), scarring was implicated for neuroticism, but not for extra-/introversion. Altogether, findings about personality scarring turn out surprisingly consistent provided that long-term temporal accumulation rather than single episodes was studied. Personality is frequently assessed via probes of typical (i.e., long-term) behavior, and may not change much as a function of relatively common and passing single states, but rather as a function of their temporal integrals (i.e., accumulation). Indeed, our findings seem to confirm the suspicion that past studies have expected too fast and dramatic changes, thus not detecting the scarring (Wichers et al., 2010).

Provided that past experiences carry information about current and future environmental statistics, an optimally behaving organism should make use of that information (Del Giudice, 2014; Fawcett et al., 2014;

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Table 3
General-population correlations and partial correlations of 15-year changes in TCI-assessed personality and depression and sum of depressive symptoms in the intervening years.

Cor\pCor	ΔHarm avoidance	ΔSelf-directedness	ΔmBDI	$\Sigma_{intermediate}$ mBDI
ΔHarm avoidance	–	−0.32 (−0.37, −0.26)***	0.32 (0.26, 0.38)***	0.09 (0.02, 0.16)*
ΔSelf-directedness	−0.53 (−0.57, −0.48)***	–	−0.43 (−0.48, −0.37)***	0.07 (0.00, 0.14)
ΔmBDI	0.54 (0.49, 0.58)***	−0.59 (−0.63, −0.54)***	–	0.07 (−0.01, 0.13)
$\Sigma_{intermediate}$ mBDI	0.11 (0.05, 0.18)**	−0.02 (−0.08, 0.04)	0.09 (0.03, 0.15)*	–

Note: Correlations (below diagonal) and partial correlations (above diagonal) for the changes in harm avoidance, self-directedness, and depressive symptoms (mBDI) from 1997 baseline to 2012 final follow up, and for the sum of the two mBDI scores assessed in the intervening years 2001 and 2008. Partial correlations estimate conditional correlation given the other variables. In other words, partial correlation (linearly) approximates what pairwise correlation would be in samples that have been stratified according to the levels of other variables. Bias-corrected and accelerated 95% bootstrap confidence intervals are shown in parentheses.
* p-value less than 0.05.
** p-value less than 0.01.
*** p-value less than 0.001.

Houston & McNamara, 1999; Nowak & Sigmund, 2004). For example, generally passive behavior could be adaptive after repeated failures, when no information regarding reasons of the failures is available. Optimality criteria are powerful theoretical tools where applicable (Fawcett et al., 2014; Houston & McNamara, 1999; Nowak & Sigmund, 2004), several applications to depressive disorders and human behavioral traits already exist (Hagen, 1999, 2003, 2011; Nesse, 1991, 2000, 2009; Nettle, 2004, 2006; Rosenström, 2013), and they have been promoted as an opportunity to install models of normal functioning to the foundations of psychiatry (Nesse & Stein, 2012). Hence, albeit not large in statistical effect size, we consider our findings important in establishing the empirical feasibility of the many suggested ideas of environmental and evolutionary plasticity in human personality.

This one and other similar studies (e.g., Elovainio et al., 2015; Jeronimus, Ormel, Aleman, Penninx, & Riese, 2013) examine aggregate-level changes in broad personality traits and depressive-symptom presence. Yet, depression patients differ in their symptom presentation (Fried & Nesse, 2015; Zimmerman et al., 2014), and different life events may elicit different depressive symptoms (Keller et al., 2007). The PAR hypothesis can connect these levels of explanation, because successive life events are temporally (i.e., auto-) correlated: many life events, such as losing a job, can lead to an increased incidence of subsequent stressful life events, such as divorce and financial problems. Furthermore, changes in global environment, such as wars, may cause temporal clustering of stressful life events. In the PAR framework, events can then serve as a 'weather forecast' for conditions to come, and initiate anticipatory long-lasting behavioral strategies, or 'social roles', adaptive for events that are likely to follow. Indeed it has been observed that negative life events are followed by (small) long-lasting neuroticism elevations mediated by depressive symptoms, whereas positive life events are followed by (small) long-lasting neuroticism decreases that are independent of depressive symptoms (Jeronimus et al., 2013).

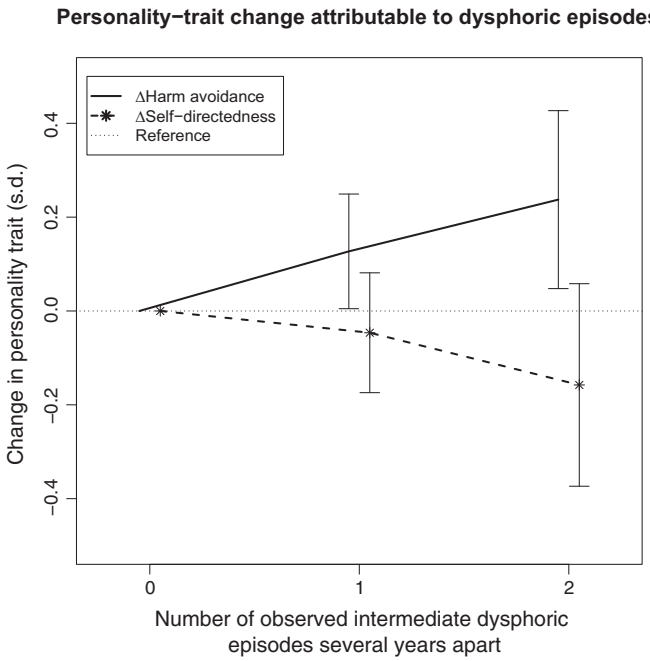

Fig. 2. Age cohort- and sex-adjusted change in personality traits harm avoidance and self-directedness from the 1997 baseline to the final follow up in 2012 as a function of observed number of intervening dysphoric or mildly depressive episodes (in the years 2001 and/or 2008). Whiskers represent 95% Wald-type confidence intervals, computed from pooled standard errors across multiple imputations.

There are two ways how the PAR framework could explain our results; the first one assumes that depressive symptoms themselves can provide a situation-dependent fitness increase, whereas the second one does not need such assumption. On one hand, if depressive episodes are reactions that have provided a selective advantage, then a PAR to an environmental change towards the reactions' environments of evolutionary adaptedness would be to tune precisely those personality traits that are most related to the original adaptive reactions (the episodes) rather than the other traits. We did observe changes in personality traits conceptually most close to depression, following depressive episodes, and did not observe changes in the other traits; a logical result in light of external PAR framework. On the other hand, if the depressive episodes associate with a process that weakens the individual physiologically and/or psychologically, then it is only logical to increase cautiousness and harm avoidance to match the decreased coping capabilities; an internal PAR consistent with the present results would ensue.

Although we demonstrated the empirical feasibility of PARs regarding temporal development of personality, demonstrating the selective advantage of depressive episodes per se was left for other studies (e.g., see Hagen, 1999, 2003, 2011; Nesse, 1991, 2000, 2009; Nettle, 2004; Rosenström, 2013). In principle, the present findings could represent purely dysfunctional scarring or stress-sensitization as well (Monroe & Harkness, 2005; Stroud et al., 2008; Wichers et al., 2010), although other findings, such as situation-dependency and social contagiousness of the symptoms (Keller et al., 2007; Keller & Nesse, 2005; Rosenquist, Fowler, & Christakis, 2010), are difficult to explain from that vantage point. Both internal and external PAR effects could coexist, however.

One might question the suggested importance of our results based on "construct overlap" that is common in psychological research (Ryff, 2008). In essence, problem of construct overlap refers to the possibility of constructing differently titled complex measurement instruments that nonetheless assess pretty much the same thing, only mislead by differing naming conventions. Although construct overlap seems a salient possibility regarding measures of depression and negative-emotionality traits, we do not think it renders the present findings trivial for the following reasons. First, we did adjustments for concurrent depression changes, and they did not attenuate the personality changes due to intermediate depression accumulation. Second, personality traits must reflect slower temporal variations than depression scores: baseline depression predicts better a depression assessment at single future time point than baseline personality, but baseline personality predicts

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better the future accumulation of depressive episodes than baseline depression (Rosenström et al., 2014). In general, state effects cannot fully account for the association between personality and depression, although depression and negative emotionality share etiological factors (Klein et al., 2011). What comes to different methods of personality assessment, a great degree of construct overlap is likely (Grucza & Goldberg, 2007). We believe our findings across personality systems, study populations, and definitions for depression tap to same underlying sets of biological and social mechanisms, in part at least.

4.1. Strengths and limitations

Clear strengths of this study are the extensive longitudinal follow ups of large samples of both clinical patients and population-dwelling individuals, and the use of several personality measures. The reported findings satisfy several of the Bradford Hill's classic criteria of causal relevance (Hill, 1965). The association between accumulated depressive episodes and personality change was consistent across samples and methods of assessment. It was specific to traits related to negative emotionality, being absent in other traits despite their correlations with negative emotionality. We were able to establish temporality, that is, demonstrated that the suggested effect occurred after the cause. Temporality was explicitly studied for neuroticism and extraversion of the clinical patients and TCI traits in general population, because these data had corresponding baseline measurements available, and results without the baseline assessment suggested that analogous phenomena occurred for TCI in clinical data. We found a biological gradient, with more episodes being associated with greater change in harm avoidance (Fig. 2), and the findings were to a highly plausible trait. A major criterion not satisfied was *strength of association*, as the effect sizes ranged from small to moderate. In this case, the criterion of strength should not be over-emphasized, because there are no consensus on best measures for the exposure (Fried & Nesse, 2015; Wakefield & Schmitz, 2013) nor for the outcome (Grucza & Goldberg, 2007; John, Robins, et al., 2008), and measurement errors are known to attenuate observed effect sizes.

Regarding limitations, it was not possible to infer whether the observed personality changes were due to the experienced depressive symptoms, due to the causes of the symptoms, or both. Furthermore, only self-reports of personality were assessed, leaving out implicit aspects of personality (Back, Schmukle, & Egloff, 2009). Self-reports of personality do predict actual behavior independently of emotional valence, however (Back et al., 2009). Furthermore, we were not so much interested in differentiating between internal versus external PARs (Del Giudice, 2014; Nettle et al., 2013), as in demonstrating the general empirical feasibility of adaptive theories of personality change in response to adversity and depression. This has been far from self-evident so far (Wichers et al., 2010).

Another limitation of the present study is shared by most lengthy follow-up studies. Namely, study attrition cannot be avoided, and the projects are not able to initially include every measure they later wish for. The VDS originally included only extraversion and neuroticism from the Eysenck's Personality Inventory, as these had been the most studied traits in the context of depression research (Enns & Cox, 1997). It lacked baseline assessment for the TCI, but had assessed it in the final 5-year follow-up. The convergent evidence with the YFS data nonetheless strongly supported the interpretation that similar harm-avoidance changes apply to both data sets. Furthermore, by reference to our previous study (Elovainio et al., 2015), we can provide strikingly consistent total picture across three different systems of personality assessment: accumulated depression has a consistent effect on changes in negative emotionality and, at most, inconsistent effects to other personality traits. The limitation of study attrition was overcome to the maximum extent by the use of statistical imputation methods, and sensitivity analyses did not indicate significant failures in the methodology (see online supplement, available on the journal's Website at www.ehbonline.org). The same main qualitative findings were obtained with or without the imputation methods.

5. Conclusion

Pertaining widely applied self-reported measures of personality, this study found that personality changes do form in response to accumulated depressive episodes both in clinical and general-population samples, and in all tested personality-trait systems, although the changes are difficult to detect in short follow-up studies. The total picture was that accumulated depression has a consistent effect on changes in any traits assessing negative emotionality and, at most, inconsistent effects to other personality traits. The effect sizes were small to moderate, but several classic indications for causality were present. Although much remains to be understood, the study provides much needed empirical support for the many adaptive theories of personality and behavior in response to adversity and depression.

Supplementary materials

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.evolhumbehav.2015.01.005.

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