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ORIGINAL PAPER

Articulation and testing of a personality-centred model of psychopathology: evidence from a longitudinal community study over 30 years

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Abstract Advances in psychopathological research advocate a personality-centred model of common mental disorders (CMD). We tested four hypotheses to test such a model. First, personality relates to critical life events; second, both personality and critical life events relate to CMD; third, interaction effects between personality and critical life events relate to CMD; fourth, neuroticism explains the majority of variance in psychopathology. We analysed data ($n = 453$) based on seven semi-structured interviews from a longitudinal epidemiologic cohort study over 30 years spanning years 1979 (age 20) to 2008 (age 50). CMD and critical life events were assessed seven times between 1979 and 2008 and personality domains of neuroticism, extraversion and aggressiveness in 1988 and 1993. Aggressiveness and neuroticism related to partnership rupture and job loss. Neuroticism related significantly to major depression, anxiety disorders, substance-use disorders (SUD) and severity of psychopathology. Both partnership rupture and job loss related to major depression and severity of psychopathology, but not to anxiety disorder or SUD. An interaction effect between neuroticism and partnership rupture pointed towards significantly increased SUD prevalence. All associations held when additionally adjusted for childhood adversity and familial socio-economic status. According to a pseudo-R^2, neuroticism explained 51% of total variance in severity of psychopathology over time, while all three personality domains along with both partnership rupture and job loss explained 59% of total variance. In conclusion, personality, especially neuroticism, relates consistently to repeated measures of psychopathology. These associations are independent of and more pervasive than the effects of partnership rupture and job loss. Partnership rupture in interaction with neuroticism may further increase the risk for SUD. We conclude that neuroticism is a fundamental aetiological factor for severe psychopathology, but further testing of this model in other longitudinal studies is required.

Keywords Personality · Internalising disorder · Psychopathology · Etiology · Epidemiology · Depression · Anxiety · Critical life events

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Introduction

Insights from personality science and evolutionary biology

Based on the evolutionary concept of harmful internal dysfunctions [1–3] and informed by research in animal ecology and evolutionary biology [4–7], we suggest that an individual's adaptiveness to the contemporary environment

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is largely influenced by inter-individual personality trait variation. Because personality trait variation is an evolved biological mechanism that substantially influences health, inter-individual functioning and life histories in humans and many other animal taxa [7–9], the question to pose is not whether, but how, personality should be incorporated in an etiological model of common mental disorders (CMD). By CMD researchers and clinicians typically refer to depression, anxiety and substance-use disorder (SUD). We will use the term CMD synonymously for the group of these disorders throughout. In particular neuroticism plays a major role in recent etiological models of CMD due to its persistent negative effects on mental health and functioning [10–13]; for reviews see [14–16]. Critical life events, i.e., environmental factors, may also independently contribute to increased prevalence of psychopathological symptoms and CMD [17–19]. However, we suggest that personality is predominant, because dysfunctional personality not only constitutes a broad vulnerability factor for severe, chronic or relapsing poly-symptomatic psychopathology [13, 20–22], but also indirectly increases the risk for mental disorders by fostering and creating critical life events [23–25]. In addition, there is compelling evidence that personality moderates the effects of critical life events [12, 17, 26] and daily stress [27, 28] on mental health, which further bolsters the primacy of personality effects. Please note that by dysfunctional personality we not only mean extreme scores (both low and high; see Ref. [9]) on basic personality traits such as neuroticism and agreeableness, but also personality disorders, which are pathological extremes along basic personality domains [29–31]. Though all these aspects have received considerable scientific attention, few attempts have been made to examine them simultaneously over extended observation periods of several years [e.g., 12, 26]. However, these valuable exceptions have generally focused on associations between neuroticism and major depression only. Therefore, we believe that this is the first original study to directly compare effects of personality, critical life events, and their interaction effects on various psychopathological outcomes including major depression, anxiety disorders, SUD, and severity of psychopathology.

Personality, environmental influences and critical life events

Findings from evolutionary biology suggest that personality trait variation enables the adaptation of a species to different ecological conditions under varying selection pressure [7, 9]. However, as traits vary substantially, not each individual is equally adapted to a given environment. Depending on specific environmental demands, animals with varying trait characteristics will, therefore, demonstrate differential fitness-related outcomes [6, 32]. In both humans [33] and non-human animals [34], the heritability of personality traits is estimated with approximately 50%. The remaining 50% are thus mainly environmental influences and gen-environment correlations. In personality science the association between personality and environment is supposed to be bidirectional, with personality having a causal effect on an individual's environment (referred to as selection processes) and the environment having a causal effect on personality trait development (referred to as socialisation processes) [24, 35, 36]. More specifically, over the last decades evidence has accumulated that personality relates to low occupational attainment and poor work satisfaction [37], partnership conflicts [25], relationship insecurity [38], and negative life events in general [23, 24]. Finally, personality traits are important predictors of socio-environmental functioning in persons with mental health problems [39–41]. Successfully coping with developmental tasks and age-specific ecological challenges such as taking responsibility for a family, forming intimate relationships or fulfilling occupational duties, is hence by large a product of personality functioning [35]. Because social functioning is crucial for the maintenance of mental health, personality stringently needs to be considered in both psychiatric practice and psychopathological research [14].

The present study

The literature briefly reviewed above suggests that it could be worthwhile to define personality as the crucial internal mechanism underlying CMD, here specifically internalising disorder (depression and anxiety) related to neuroticism [42–44]. That claim is supported by the remarkably strong association of neuroticism with the risk of comorbidity and the covariance between disorders [11, 13, 43]. As detailed above, negative life events such as job loss or divorce are also important risk factor for psychopathology [12, 18, 19] and it has been shown that the relationship between critical life events and psychopathology is moderated by personality [12, 17, 26]. It is, therefore, necessary to consider both personality traits and critical life events in the prediction of CMD. Such an environmentally contingent function of personality trait variation for individual health and functioning is also well supported by research in animal ecology and evolutionary biology [6, 7, 9]. To avoid unnecessary multiple testing and its well-known effects on the inflation of α-error probability, in this work we include only relationship rupture and job loss as critical life events, as both predictors are considered highly important for mental health and functioning [18, 19, 45].

There are four linked hypotheses to test our model:

Hypothesis 1 On the basis of the literature [25, 37, 38], personality is bi-directionally related to critical life events

through selection and socialisation processes, i.e., we expect personality and critical life events to be interrelated, though, noteworthy, such associations are typically weak [24, 36].

Hypothesis 2 Because both personality [14, 16] and critical life events [19, 46] relate to mental disorders, personality, partnership rupture and job loss relate independently to severity of psychopathology and either internalising disorder or SUD.

Hypothesis 3 Because CMD can also result from an interaction between personality and environmental factors [12, 17, 47], the interactions of personality with partnership rupture and job loss relate to either internalising disorder or SUD.

Hypothesis 4 As neuroticism is the predominant factor underlying general psychopathological vulnerability [14, 16, 42], neuroticism will account for most of the variance explained in the severity of psychopathology.

Methods

Participants and sampling procedure

The Zurich Cohort Study originally comprised a cohort of 4547 subjects ($m = 2201$; $f = 2346$) representative of the canton of Zurich in Switzerland, who were screened in 1978 with the Symptom Checklist 90-R (SCL-90-R) [48] when the men were 19 and the women 20-years-old. In Switzerland, every male citizen must undertake a military screening test at the age of 19. Therefore, conscripts within a defined catchment area comprise its respective, complete male age group. With the consent of military authorities, but independent of their screening procedure, we randomly screened 50% of all male conscripts of the canton of Zurich of this age group. The refusal rate was 0.3%. Since, with the exception of severely disabled persons, all Swiss men had to undergo military conscription at that time, drawing a random sample from conscripts allowed for the most representative male sample possible. As women were not obliged to serve in the army, female participants were identified from the complete electoral register of the canton of Zurich. Again, 50% of them were randomly selected and received questionnaires by mail, of which 75% responded. To increase the probability of the development of psychiatric syndromes, a stratified subsample of 591 persons ($m = 292$; $w = 299$) was selected for comprehensive interview, with two-thirds consisting of high-scorers [defined by the 85th percentile or more of the Global Severity Index (GSI) of the SCL-90-R] and one-third being a random sample of subjects with scores below that 85th percentile. This study was approved by the ethics committee of the canton of Zurich and all participants gave their fully informed written consent. A detailed description of the sampling method has been provided elsewhere [49].

Altogether seven interview waves have been conducted, specifically, in 1979 ($n = 591$), 1981 ($n = 456$), 1986 ($n = 457$), 1988 ($n = 424$), 1993 ($n = 407$), 1999 ($n = 367$) and 2008 ($n = 335$). The corresponding attrition rates were 0, 22.8, 22.7, 28.3, 31.1, 37.9, and 43.3%. That is, even after 30 years of study duration, more than half of all participants continued to participate. The causes of attrition involved dropouts due to decease, lost contact, or refusal to participate. Unfortunately, it was not possible to allocate a specific reason to each dropout, because in many cases contacting was simply not answered. The initial allocation to the two groups, that is, high-scorers on GSI versus all others, remained stable throughout the study. The dropouts were more frequent among the extremely high and extremely low GSI scorers [50]. We repeated the attrition analyses after the most recent interview, but we found no significant difference between subjects who had left the study and those who remained with regard to socio-economic status and education as measured at the study outset, nor in their initial psychopathological impairment according to the nine SCL-90-R subscales. However, there was a moderate gender bias, with more dropouts among men (OR 1.82; 95% CI 1.31–2.53; $p < 0.001$).

Instruments and measures

Interviews were conducted using the "Structured Psychopathological Interview and Rating of the Social Consequences of Psychological Disturbances for Epidemiology" (SPIKE) [49]. This semi-structured interview collects data on socio-demography, somatic syndromes, psychopathology, substance-use, medication, health services, impairment and social activity. Its good reliability and validity have been reported previously [51]. Twelve-month prevalence rates of CMD were diagnosed according to DSM-III, DSM-III-R and subsequently DSM-IV criteria, except for obsessive–compulsive disorder, which was diagnosed according to DSM-III-R criteria throughout [52]. Dysthymia and minor depression were not included in the present study. For the present study we condensed major depression, GAD, agoraphobia, social phobia, specific phobia, panic disorder and obsessive–compulsive disorder into an umbrella diagnosis of internalising disorder. GAD, agoraphobia, social phobia, specific phobia, panic disorder and

obsessive–compulsive disorder were further subsumed as broad anxiety disorder. Alcohol as well as drug abuse and dependence were subsumed under the broad umbrella diagnosis of SUD. A third outcome, termed severity of psychopathology, comprised the sum of all concurrent diagnoses as delineated above, computed separately for each assessment wave.

Partnership rupture was defined as separation or divorce from an intimate partner within the past 12 months, and job loss as unintended dismissal from work, as evaluated at each assessment wave with the SPIKE. As further covariates we included variables that ascertained early environmental adversity, including severe family problems during childhood and adolescence such as broken home, conflicts between parents, or psychological problems in parents (for more information, see [53]), low parental income and a low participant's education level. Partnership rupture and job loss were chosen a priori because they are considered highly important for mental health and functioning [18, 19, 45, 54] and because they were sufficiently frequent in our data to provide reliable estimates (as opposed to, e.g., severe forms of sexual abuse).

In 1988, when participants were 29/30 years old, we examined the participants' personality traits using the Freiburg Personality Inventory (FPI) [55]. In a majority of participants (82%) personality was re-assessed in 1993, when participants were 34/35 years old. When possible the mean score across measurements was computed to reduce the influence of state-effects on personality characteristics. Participants who dropped-out after the first assessment of personality did not differ in their trait-scores from participants who remained in the study. When personality was first assessed in 1988, the FPI was a widely used German personality inventory depicting personality traits on the three higher-order domains of masculinity, extraversion and neuroticism, and on nine distinct primary scales. These traits are (1) nervousness, (2) irritability, (3) depressiveness, (4) impulsivity, (5) sociability, (6) resilience, (7) aggressive dominance, (8) inhibition, and (9) frankness. However, these primary traits are rather outdated nowadays. For the present study we thus applied the empirically derived domain of aggressiveness (comprising facets of proneness to violence, callousness and lack of self-control), extraversion (comprising facets of being outgoing, cheerful and self-confident) and neuroticism (comprising facets of emotional lability, somatisation and worry). These three superordinate domains were extracted through extensive factor-analytic examination on item-level in a very large sample of $n > 5000$ and replication in six random subsamples thereof [56]. They were then further validated in a more recent paper [10]. FPI neuroticism and extraversion items were constructed according to Eysenck's biological model of personality [57] and thus correlate strongly with the Eysenck Personality Inventory (EPI) scales of extraversion and neuroticism [55]. The broad FPI domains also differentiate reliably between distinct mental disorder diagnoses and sub-threshold conditions [56, 58, 59]. Because neuroticism and extraversion items were adapted from Eysenck's original definition, they tap into the presumed biological core of these broad trait domains. With respect to neuroticism, Eysenck's theory links neuroticism to lower neurobiological activation thresholds in the sympathetic and limbic system, which cause excessive physiological responsiveness [55]. Neuroticism items, therefore, mostly capture psychosomatic aspects of hyperarousability such as having frequent headaches, getting easily dizzy, having frequently nausea or vomiting, or getting frequently short of breath. Other items involve feeling miserable for no reasons, feeling low without really knowing why, or being frequently lost in thought. For more information, see also [10, 56].

Statistical analysis

The longitudinal associations between personality and repeated measures of partnership rupture, job loss and CMD were estimated using generalized estimating equations (GEE). These statistical models were introduced to fit regression analyses that account for within-subject correlation, which is an inherent part of longitudinal studies that rely on repeated outcome measures [60]. The GEE approach uses weighted combinations between a predictor variable and repeated outcomes that account for varying observations, e.g., a disorder being present versus absent, within a person across time. GEE use all available data and estimate missing values under the assumption of Missing Completely at Random (MCAR) [61]. Prerequisite to the application of GEE was, therefore, a thorough missing value analysis, which revealed that all outcomes of interest met the criteria of MCAR according to Little's MCAR test. Personality measured as the average score of the assessments at age 29/30 and 34/35 and the critical life events, repeatedly assessed between ages 20 and 50, were entered as the predictor variables. The broad diagnoses of internalising disorder, major depression, anxiety disorder and SUD repeatedly measured between ages 20 and 50 were successively entered as the dependent variable. Owing to the dichotomous distribution of the DSM-based diagnoses we computed these models with a binomial distribution and logit link-function. Severity of psychopathology conformed to an over-dispersed Poisson distribution, which is why this outcome was modelled with a negative binomial distribution and log link-function. The within-subject covariance was specified with the "unstructured" correlation type to avoid having any constraints on the covariance structure. A robust sandwich estimator was used to reduce the effects

of outliers and influential observations. The proportion of total variance explained in severity of psychopathology was computed according to a pseudo-R^2 formula proposed by Twisk [61], according to which $R^2 = 1 - \left(S^2_{\text{model}}/S^2_Y\right)$, where S^2_{model} is the variance of the model (also known as the scale parameter) and S^2_Y is the variance of the outcome variable Y, calculated over all available data. Results were reported with odds ratios (OR), which referred to a 1 standard deviation increase with respect to the standardized continuous personality measures. The exact model pertaining to each hypothesis is specified in the results section. All analyses were performed with SPSS 23 for Windows.

Table 1 Association between personality assessed at age 29/30 and 34/35 and repeated measures of critical life events between age 20 and 50 (participants: $n = 453$; data points: $n = 2759$)

Outcome	Predictors	OR (95% CI)
Partnership rupture	Aggressiveness	1.16 (1.04–1.29)**
	Extraversion	1.07 (0.95–1.20)
	Neuroticism	1.10 (0.99–1.22)
Job loss	Aggressiveness	1.42 (1.11–1.81)**
	Extraversion	1.01 (0.79–1.30)
	Neuroticism	1.36 (1.08–1.70)**

** $p < 0.01$

Results

Descriptive statistics

The prevalence rates of CMD have been shown in detail in previous publications [e.g., 51, 52]. The cumulative 12-month incidence rates from age 20 to 50 were as follows: 32.5% for major depression, 6.8% for agoraphobia, 12.6% for social phobia, 26.9% for specific phobia, 6.1% for panic disorder, 20.8% for GAD, 9.2% for OCD, 29.3% for alcohol use disorder and 16.8% for drug use disorder [52]. The proportion of participants who experienced a job loss or a partnership rupture is indicated in the supplementary Table 1. Further included in that supplementary Table are the frequency and the distribution of the covariates included in the fully adjusted models, i.e., severe family problems during childhood/adolescence, parental income, and education level. The correlations between personality traits were as follows: aggressiveness with extraversion $r = -.094$, $p = .047$; aggressiveness with neuroticism $r = .497$, $p < .001$; and extraversion with neuroticism $r = -.381$, $p < .001$. Inter-correlations within covariates as well as between personality domains and these covariates were small (all $r < .3$), suggesting that multicollinearity is not an issue and that all predictors can be entered simultaneously in a multivariable model.

Hypothesis 1: personality relates to both job loss and partnership rupture

To test our first hypothesis we conducted a series of simple bivariate models where partnership rupture and job loss were separately included as the dependent variable and personality domains successively as the predictor variables. Personality traits were examined separately in this model because we anticipated weak associations that would not hold in a multivariable model where all personality traits are adjusted for each other. As indicated in Table 1, partnership rupture was weakly albeit significantly related to aggressiveness, while job loss related weakly to both aggressiveness and neuroticism. Hence, hypothesis 1 was confirmed.

Hypothesis 2: Personality, partnership rupture and job loss all relate independently to psychopathology

To test our second hypothesis we conducted a separate model for each dependent variable, that is, internalising disorders, major depression, anxiety disorder, SUD and severity of psychopathology. Personality traits, partnership rupture and job loss, were entered simultaneously as the predictor variables. The results are indicated in Table 2. Internalising disorder was significantly predicted by extraversion (negative association), neuroticism, partnership rupture and job loss. Major depression was associated with neuroticism, partnership rupture and job loss, whereas anxiety disorder related to extraversion (negatively) and neuroticism only. SUD, too, related significantly to extraversion and neuroticism only. Finally, severity of psychopathology was significantly predicted by neuroticism, partnership rupture and job loss. All those associations held when additionally adjusted for sex, severe family problems during childhood/adolescence, low parental income and low education degree at age 20/21. Note that the odds ratio (OR) for personality and critical life events do not directly compare, as in the former the OR refers to a one standard deviation increase while in the latter the OR refer to the absence of the respective life event (i.e., present vs. absent). Hypothesis 2 was fully confirmed as concerns personality, but only partially with respect to critical life events, because both partnership rupture and job loss did not relate to anxiety disorders and SUD.

Hypothesis 3: Interactions of personality with partnership rupture and job loss relate to psychopathology

To test our third hypothesis we added an interaction term between significant personality traits and both partnership

Table 2 Effect of personality assessed at age 29/30 and 34/35 and critical life events on repeated occurrence of mental disorders between age 20 and 50 (participants: $n = 453$ and $n = 367$; data points: $n = 2759$ and $n = 2312$, respectively, for crude and adjusted models)

Outcome	Predictors	Crude OR (95% CI)	Adjusted[a] OR (95% CI)
Internalising disorder	Aggressiveness	0.97 (0.85–1.10)	0.96 (0.84–1.09)
	Extraversion	0.87 (0.76–0.98)*	0.87 (0.76–0.99)*
	Neuroticism	1.61 (1.41–1.84)**	1.55 (1.35–1.79)**
	Partnership rupture	1.56 (1.20–2.03)**	1.82 (1.37–2.43)**
	Job loss	1.90 (1.14–3.18)*	2.06 (1.24–3.41)**
Major depression	Aggressiveness	0.86 (0.72–1.02)	0.86 (0.73–1.03)
	Extraversion	1.06 (0.91–1.23)	1.08 (0.92–1.26)
	Neuroticism	1.65 (1.38–1.98)**	1.57 (1.30–1.89)**
	Partnership rupture	2.66 (1.96–3.63)**	3.09 (2.19–4.37)**
	Job loss	2.87 (1.71–4.84)**	2.51 (1.45–4.34)**
Anxiety disorder	Aggressiveness	1.04 (0.91–1.20)	1.04 (0.91–1.20)
	Extraversion	0.81 (0.71–0.93)**	0.82 (0.71–0.95)**
	Neuroticism	1.59 (1.39–1.82)**	1.57 (1.36–1.83)**
	Partnership rupture	1.02 (0.75–1.37)	1.07 (0.78–.47)
	Job loss	1.40 (0.81–2.43)	1.64 (0.98–2.74)
SUD	Aggressiveness	1.09 (0.89–1.34)	1.07 (0.85–1.35)
	Extraversion	1.30 (1.07–1.57)**	1.33 (1.08–1.64)**
	Neuroticism	1.41 (1.14–1.75)**	1.45 (1.12–1.88)**
	Partnership rupture	1.14 (0.80–1.63)	1.20 (0.82–1.75)
	Job loss	1.46 (0.86–2.48)	1.33 (0.72–2.47)
Severity of psychopathology	Aggressiveness	1.01 (0.91–1.11)	1.01 (0.91–1.12)
	Extraversion	1.00 (0.91–1.09)	1.00 (0.90–1.10)
	Neuroticism	1.53 (1.38–1.69)**	1.49 (1.33–1.67)**
	Partnership rupture	1.37 (1.13–1.65)**	1.48 (1.21–1.79)**
	Job loss	1.49 (1.05–2.12)*	1.51 (1.13–2.01)**

SUD substance-use disorder

* $p < 0.05$; ** $p < 0.01$

[a] Adjusted for severe family problems during childhood/adolescence, sex, low parental income and low education degree at age 20/21

rupture and job loss to their main effects as detailed above. A significant interaction term between personality and critical life events emerged in relation to major depression and SUD (see Table 3). With respect to the former, we found a negative interaction effect between neuroticism and job loss on the occurrence of major depression, which replicates findings from Vinkers and colleagues [26]. Further inspection of the data based on median split revealed that in persons scoring high on neuroticism, job loss did increase the unconditionally high depression risk by 8% points (from 14 to 26%), whereas in persons scoring low on neuroticism the risk increase attributable to job loss was 23% points (from 8 to 31%). However, due to power failure this negative interaction effect did not reach statistical significance in the adjusted model and is, therefore, presented with reservation. As for SUD, in the crude model the main effect of neuroticism and the interaction term between neuroticism and partnership rupture related significantly to SUD. Persons scoring low on neuroticism with and without partnership rupture revealed the same risk for SUD (both 11%), while in persons scoring high on neuroticism the risk increase attributable to partnership rupture was 7% points (from 15 to 22%). This interaction effect also held in the fully adjusted model. Hypothesis 3 was, therefore, confirmed.

Hypothesis 4: Neuroticism accounts for the majority of variance explained in the severity of psychopathology

Related to our fourth hypothesis and according to our pseudo-R^2 estimate, neuroticism explained 51% of total variance in the repeatedly assessed severity of psychopathology over time. Adding the other two personality domains did not improve the proportion of total variance explained, while adding partnership rupture and job loss to all three personality domains accounted for 59% of total variance explained, that is, an absolute increment of 8% points. Hypothesis 4 was, therefore, fully confirmed. To further demonstrate the strong association between personality and psychopathology we estimated the projected

Table 3 Interaction effect between personality assessed at age 29/30 and 34/35 and critical life events on repeated occurrence of major depression and SUD between age 20 and 50 (participants: $n = 453$ and $n = 367$; data points: $n = 2759$ and $n = 2312$, respectively, for crude and adjusted models)

Outcome	Predictors	Crude OR (95% CI)	Adjusted[a] OR (95% CI)
Major depression	Neuroticism	1.57 (1.35–1.82)**	1.47 (1.24–1.74)**
	Partnership rupture	2.65 (1.91–3.69)**	3.07 (2.12–4.45)**
	Job loss	3.42 (2.10–5.57)**	2.94 (1.73–4.99)**
	Neuroticism by partnership rupture	0.97 (0.74–1.27)–	0.98 (0.73–1.34)
	Neuroticism by job loss	0.60 (0.37–0.96)*	0.64 (0.37–1.09)
SUD	Extraversion	1.21 (0.99–1.47)	1.28 (1.04–1.58)*
	Neuroticism	1.34 (1.11–1.62)**	1.34 (1.07–1.67)*
	Partnership rupture	0.91 (0.61–1.37)	0.97 (0.62–1.50)
	Job loss	1.31 (0.68–2.49)	1.21 (0.54–2.72)
	Extraversion by partnership rupture	1.42 (0.96–2.10)	1.29 (0.87–1.90)
	Neuroticism by partnership rupture	1.60 (1.09–2.36)*	1.69 (1.14–2.52)**
	Extraversion by job loss	1.10 (0.56–2.19)	0.92 (0.49–1.74)
	Neuroticism by job loss	1.14 (0.66–1.97)	1.19 (0.69–2.04)

SUD, substance-use disorder

* $p < 0.05$; ** $p < 0.01$

[a] Adjusted for severe family problems during childhood/adolescence, sex, low parental income and low education degree at age 20/21

Table 4 Cumulative prevalence of common mental disorders from age 20 to 50 in relation to personality profiles assessed at age 29/30 and 34/35 (participants: $n = 453$; data points: $n = 2759$)

Personality profile	Trait level[a]	Internalising disorder %	SUD %
Dysfunctional	Aggressiveness: +2 Extraversion: −2 Neuroticism: +2	49.8	15.3
Neutral	Aggressiveness: 0 Extraversion: 0 Neuroticism: 0	23.1	11.3
Functional	Aggressiveness: −2 Extraversion: +2 Neuroticism: −2	8.4	8.2

[a] All traits have a mean = 0.0 and a standard deviation = 1.0

30-year cumulative prevalence of internalising disorder and SUD based on specific personality profiles (see Table 4). In particular with respect to internalising disorder, the cumulative 30-year risk differed substantially between personality profiles. Persons scoring high on aggressiveness and neuroticism and low on extraversion had an approximately 6 times increased risk for internalising disorder compared to persons scoring low on aggressiveness and neuroticism and high on extraversion.

Discussion

In this longitudinal community cohort study we articulated and tested a personality-centred model of CMD. To the best of our knowledge, this is the first original work to focus simultaneously on the relative effects of critical life events and personality traits and their interactions on various psychopathological outcomes using long-term longitudinal data. The results revealed that personality was associated weakly with critical life events and substantially with CMD, directly and in interaction with critical life events. Dysfunctional personality profiles, comprising extremely high neuroticism and aggressiveness in combination with extremely low extraversion, related to a manifold increased risk of internalising disorders and in particular neuroticism explained a very large amount of variance in the severity of psychopathology. As there is a dearth of longitudinal studies examining interaction effects between personality and environmental conditions, this work makes an important contribution to the literature. Moreover, we further believe that this is the first longitudinal long-term study that aimed at quantifying the independent contribution of personality traits on long-term psychopathology by providing a pseudo-R^2 for the global severity of psychopathology from age 20 to 50 and by estimating the 30-year cumulative risk of internalising disorder and SUD related to different personality profiles.

Proximate aetiological explanations

Overall, our findings stress the fundamental role of personality, mainly neuroticism, for the occurrence, persistence and severity of psychopathology [10, 11, 13, 43]. Our data further highlight that the relationship between critical life events and psychopathology is at least in part moderated by personality [12, 17, 26]. Nevertheless, we would like

to stress that the studies referenced above, including our own, do not provide a stringent test of causation, which is typically very difficult to deduce in observational studies. We further recognise that the proximate aetiopathological mechanisms underlying a personality-psychopathology association are mostly unknown and that different aetiological models may account for varying degrees of both phenotypic and genetic covariance [62, 63]. For instance, one may suggest that neuroticism scores in middle adulthood might be increased due to preceding mental disorders occurring during adolescence (scarring effect). However, meta-analytic research consistently revealed that personality traits are highly stable during adolescence and early adulthood in both community and clinical samples [64], which argues against the differential impact of adolescent psychopathology on personality trait-scores. Moreover, scarring effects appear to be transient [65] or, when detected as enduring, they are shown to be very weak (adjusted $r \leq .16$) [66] and hence practically almost irrelevant. Also consider that anti-depressive treatments significantly reduce neuroticism scores [67, 68], which further counters the logic of scarring effects.

Anusic and Schimmack [69] recently estimated that 83% of the reliable variance underlying personality was due to stable influences. Thus, even if the remaining 17% were attributable to psychopathology, which is very unlikely due to the very low stability of the change component [69], it would account for only a small amount of variance. We, therefore, contend that neuroticism high-scores at age 30 are not a consequence of preceding mental disorders and suggest that problematic personality predates the onset of severe and recurrent psychopathology [70]. Such a notion corresponds to a growing body of evidence demonstrating that persons who develop psychopathological symptoms during adolescence and adulthood have premorbid childhood personality characteristics such as high stress reactivity and impulsivity that markedly deviate from same-age controls with no subsequent adolescent/adult psychopathology [22, 71, 72]. Finally, common genetic factors also account for a substantial amount of shared variance between neuroticism and internalising psychopathology [15, 16], but that genetic correlation is perhaps best explained by an ultimate evolutionary model (see below).

An evolutionary perspective

Recently, Durbin and Hicks [62] stated that research on personality-psychopathology associations is stagnant and in need of development. They identified the established scarring, continuum and predisposition models as the major source of stagnancy and advocated for a developmental perspective. Here we extend their notion and propose an evolutionary developmental theory informed by animal ecology and evolutionary biology. A possible ultimate explanation that may account for personality-psychopathology associations is life history theory, a subdivision of evolutionary biology aimed at explaining variation in fitness components such as growth rate, timing of reproduction, number of offspring, and lifespan [73, 74]. Life history theory postulates that organisms, to increase inclusive fitness, must allocate finite resources to competing demands. These specifically comprise somatic efforts, i.e., investments in psychobiological growth and health maintenance, versus reproductive efforts, i.e., investments in mating and parenting. Because resources such as time and energy are inherently limited, trade-offs emerge so that investment towards one life history trait diminishes resources available for the others [73, 74]. As a result, fast growth rate and high early fecundity traded-off against a long lifespan and increased late-life fecundity produce a fast life history strategy, whereas the opposite pattern defines a slow life history strategy [73, 75]. In line with theoretical and empirical research in animal evolutionary biology [4, 75, 76], human personality trait variation is closely connected to distinct life history strategies [77, 78]. Specifically, humans scoring high on personality domains of negative affectivity, impulsivity, antagonism and aggressiveness pursue a fast life history strategy [79–81], which is naturally selected for by harsh environments indicative of increased extrinsic morbidity-mortality [73, 82, 83]. This means that stably high levels of impulsive risk-taking and aggression are evolutionary adaptive in hostile environments. Being altruistic and cooperative does not pay off when life expectancy is low, because the individual would not live long enough to benefit from future fitness returns [76, 84, 85]. This largely genetically determined, but environmentally plastic fast life history strategy further involves early reproduction at the cost of psychosomatic growth, mating efforts at the cost of parenting efforts, and rule-breaking, opportunism and risk-taking at the cost of cooperation, interpersonal stability and health promotion [77, 86]. Findings from ecology and evolutionary biology have additionally revealed that personality is closely linked to an individual's environmental responsiveness [87, 88]. In general, bold-aggressive animals appear to be less environmentally responsive than docile-sociable animals, i.e., they adapt poorly to fluctuating environments and show high routine formation [8, 87, 89]. These findings might help to explain why humans scoring high on traits of negative affectivity and aggressiveness experience more negative life events [23, 24] and why they are more affected by these experiences [17, 28]. As a result, and in correspondence with all these findings, psychopathology has been shown to relate predominantly to a fast life history strategy [86, 90, 91]. A fast life history strategy relates to important proximate mechanisms underlying psychopathology, including insecure attachments [92] and high stress reactivity [93],

which are clinical characteristics often seen in patients with severe mental disorders. The very high proportion of stable (genetic) factors underlying inter-individual variation in human personality traits [69], therefore, bolsters the notion that life history traits may constitute a core component of personality. These findings stress the importance of evolutionary biology for biomedicine and the health sciences [94, 95], including psychiatry and psychopathology [3, 96]. Further research along these lines would certainly help to improve our understanding of developmental psychopathology and vulnerability to CMD.

Limitations

We acknowledge the following limitations: firstly, due to parsimony we included only partnership rupture and job loss as critical life events, though these certainly do not cover the whole range of stressful life experiences. Secondly, all information used in this study relied on self-reports, therefore, we may not exclude a certain bias due to selective disclosure and social desirability. Thirdly, personality was first assessed in 1988, thus associations between personality and psychopathology are merely retrospective from 1979 to 1986. Nevertheless, we contend that these data are informative because the differential stability of personality is very high across adulthood, with corrected test–retest correlations around r = 0.8 in both clinical and community samples [64]. We are thus confident that persons who scored high on given trait in 1988 also scored high on the same trait in 1979. In the present sample the attenuated 5-year differential stability of personality traits (i.e., uncorrected test–retest correlation for the assessments of 1988 and 1993) was as follows: r = .65 for aggressiveness, $r = .78$ for extraversion, and $r = .70$ for neuroticism (disattenuated coefficients are all $>r = .8$). These estimates further bolster the high differential stability of personality traits over time. Nevertheless, repeated personality assessments during adolescence and early adulthood would be necessary to rule out the notion of reversed causation, i.e., the assumption that psychopathology during adolescence causes high neuroticism in adulthood. More longitudinal research on the personality-psychopathology association in adolescents is thus worthwhile. Fourthly, our pseudo-R^2 provides only a vague estimation of the total variance explained [61]. Further exploration using for instance latent state-trait analysis (e.g. [97]) is, therefore, required. Unfortunately, such modelling was not feasible with these data due to the low frequency of particular disorders at specific assessment waves. However, please note that even if our pseudo-R^2 would overestimate the proportion of total variance explained by absolutely 20% points, the resulting 31% of variance explained would still be impressive and in accord with our hypotheses. Fifthly, we are not aware of any original work that linked the FPI to personality disorders and pathological personality traits. We therefore do not exactly know whether our three domains of aggressiveness, extraversion and neuroticism converge with personality pathology at their extreme poles. Moreover, our three personality domains do not provide a complete coverage of personality. Replication based on the five-factor model of personality is thus worthwhile.

Conclusions

The present study revealed that personality, in particular neuroticism, is a fundamental behavioural phenotype underlying the occurrence and severity of CMD. We also provide a rationale, specifically life history theory [73], that may explain the persistent association between personality and mental health. This interpretation of our data is in accordance with an increased recognition of evolutionary biology in psychiatry [96] and general medicine [95], which, as we believe, can substantially advance our understanding of psychopathology and inform aetiological models of CMD. Integration of an evolutionary account may further stipulate research on life history strategies and psychopathology. More work is required though, especially with respect to the proximate neurobiological and endocrinological pathomechanisms underlying this ultimate explanation. Future research should also try to expand the scheme of environmental factors and include further internal mechanisms such as for instance cognitive ability [98] and immune function [99], which both are closely tied to human evolution and psychopathology. A strong association between personality and psychopathology may imply causation, but this is not necessarily true. Genetically and environmentally informed long-term prospective studies beginning in childhood are, therefore, required to better understand the causal role of personality traits in the occurrence and course of severe psychopathology.

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Compliance with ethical standards

Conflict of interest None

References

1. Wakefield JC (1992) The concept of mental disorder. On the boundary between biological facts and social values. Am Psychol 47(3):373–388

2. Wakefield JC (2007) The concept of mental disorder: diagnostic implications of the harmful dysfunction analysis. World Psychiatry 6(3):149–156
3. Nesse RM, Stein DJ (2012) Towards a genuinely medical model for psychiatric nosology. BMC Med 10:5
4. Biro PA, Stamps JA (2008) Are animal personality traits linked to life-history productivity? Trends Ecol Evol 23(7):361–368
5. Reale D, Reader SM, Sol D, McDougall PT, Dingemanse NJ (2007) Integrating animal temperament within ecology and evolution. Biol Rev 82(2):291–318
6. Sih A, Cote J, Evans M, Fogarty S, Pruitt J (2012) Ecological implications of behavioural syndromes. Ecol Lett 15(3):278–289
7. Wolf M, Weissing FJ (2012) Animal personalities: consequences for ecology and evolution. Trends Ecol Evol 27(8):452–461
8. Carere C, Caramaschi D, Fawcett TW (2010) Covariation between personalities and individual differences in coping with stress: converging evidence and hypotheses. Curr Zool 56(6):728–740
9. Nettle D (2006) The evolution of personality variation in humans and other animals. Am Psychol 61(6):622–631
10. Hengartner MP, Ajdacic-Gross V, Wyss C, Angst J, Rössler W (2016) Relationship between personality and psychopathology in a longitudinal community study: a test of the predisposition model. Psychol Med 46(8):1693–1705
11. Hengartner MP, Kawohl W, Haker H, Rossler W, Ajdacic-Gross V (2016) Big Five personality traits may inform public health policy and preventive medicine: evidence from a cross-sectional and a prospective longitudinal epidemiologic study in a Swiss community. J Psychosom Res 84:44–51
12. Kendler KS, Kuhn J, Prescott CA (2004) The interrelationship of neuroticism, sex, and stressful life events in the prediction of episodes of major depression. Am J Psychiatry 161(4):631–636
13. Khan AA, Jacobson KC, Gardner CO, Prescott CA, Kendler KS (2005) Personality and comorbidity of common psychiatric disorders. Br J Psychiatry 186:190–196
14. Hengartner MP (2015) The detrimental impact of maladaptive personality on public mental health: a challenge for psychiatric practice. Front Psychiatry 6:87
15. Lahey BB (2009) Public health significance of neuroticism. Am Psychol 64(4):241–256
16. Ormel J, Jeronimus BF, Kotov R, Riese H, Bos EH, Hankin B et al (2013) Neuroticism and common mental disorders: meaning and utility of a complex relationship. Clin Psychol Rev 33(5):686–697
17. Hengartner MP, van der Linden D, Bohleber L, von Wyl A (2017) Big five personality traits and the general factor of personality as moderators of stress and coping reactions following an emergency alarm on a Swiss University Campus. Stress Health 33:35–44
18. Breslau J, Miller E, Jin R, Sampson NA, Alonso J, Andrade LH et al (2011) A multinational study of mental disorders, marriage, and divorce. Acta Psychiatr Scand 124(6):474–486
19. Pevalin DJ, Goldberg DP (2003) Social precursors to onset and recovery from episodes of common mental illness. Psychol Med 33(2):299–306
20. Hengartner MP, De Fruyt F, Rodgers S, Müller M, Rössler W, Ajdacic-Gross V (2014) An integrative examination of general personality dysfunction in a large community sample. Pers Ment Health 8(4):276–289
21. Tyrer P, Seivewright H, Johnson T (2004) The Nottingham study of neurotic disorder: predictors of 12-year outcome of dysthymic, panic and generalized anxiety disorder. Psychol Med 34(8):1385–1394
22. Wilson S, Vaidyanathan U, Miller MB, McGue M, Iacono WG (2014) Premorbid risk factors for major depressive disorder: are they associated with early onset and recurrent course? Dev Psychopathol 26(4 Pt 2):1477–1493
23. Jeronimus BF, Riese H, Sanderman R, Ormel J (2014) Mutual reinforcement between neuroticism and life experiences: a five-wave, 16-year study to test reciprocal causation. J Pers Soc Psychol 107(4):751–764
24. Ludtke O, Roberts BW, Trautwein U, Nagy G (2011) A random walk down university avenue: life paths, life events, and personality trait change at the transition to university life. J Pers Soc Psychol 101(3):620–637
25. Robins RW, Caspi A, Moffitt TE (2002) It's not just who you're with, it's who you are: personality and relationship experiences across multiple relationships. J Pers 70(6):925–964
26. Vinkers CH, Joels M, Milaneschi Y, Kahn RS, Penninx BWJH, Boks MPM (2014) Stress exposure across the life span cumulatively increases depression risk and is moderated by neuroticism. Depress Anxiety 31(9):737–745
27. Bolger N, Schilling EA (1991) Personality and the problems of everyday life—the role of neuroticism in exposure and reactivity to daily stressors. J Pers 59(3):355–386
28. Gunthert KC, Cohen LH, Armeli S (1999) The role of neuroticism in daily stress and coping. J Pers Soc Psychol 77(5):1087–1100
29. Hengartner MP, Ajdacic-Gross V, Rodgers S, Muller M, Rössler W (2014) The joint structure of normal and pathological personality: further evidence for a dimensional model. Compr Psychiatry 55(3):667–674
30. Tyrer P, Reed GM, Crawford MJ (2015) Classification, assessment, prevalence, and effect of personality disorder. Lancet 385(9969):717–726
31. Widiger TA, Livesley WJ, Clark LA (2009) An integrative dimensional classification of personality disorder. Psychol Assess 21(3):243–255
32. Dingemanse NJ, Reale D (2005) Natural selection and animal personality. Behaviour 142:1159–1184
33. Bouchard TJ Jr, Loehlin JC (2001) Genes, evolution, and personality. Behav Genet 31(3):243–273
34. Dochtermann NA, Schwab T, Sih A (2015) The contribution of additive genetic variation to personality variation: heritability of personality. P R Soc B Biol Sci 282:1798
35. Caspi A, Roberts BW, Shiner RL (2005) Personality development: stability and change. Annu Rev Psychol 56:453–484
36. Specht J, Egloff B, Schmukle SC (2011) Stability and change of personality across the life course: the impact of age and major life events on mean-level and rank-order stability of the Big Five. J Pers Soc Psychol 101(4):862–882
37. Roberts BW, Caspi A, Moffitt TE (2003) Work experiences and personality development in young adulthood. J Pers Soc Psychol 84(3):582–593
38. Neyer FJ, Asendorpf JB (2001) Personality-relationship transaction in young adulthood. J Pers Soc Psychol 81(6):1190–1204
39. Goodwin RD, Hoven CW, Lyons JS, Stein MB (2002) Mental health service utilization in the United States. The role of personality factors. Soc Psychiatry Psychiatr Epidemiol 37(12):561–566
40. Michon HW, ten Have M, Kroon H, van Weeghel J, de Graaf R, Schene AH (2008) Mental disorders and personality traits as determinants of impaired work functioning. Psychol Med 38(11):1627–1637
41. ten Have M, Oldehinkel A, Vollebergh W, Ormel J (2005) Does neuroticism explain variations in care service use for mental health problems in the general population? Results from the Netherlands Mental Health Survey and Incidence Study (NEMESIS). Soc Psychiatry Psychiatr Epidemiol 40(6):425–431

42. Barlow DH, Sauer-Zavala S, Carl JR, Bullis JR, Ellard KK (2014) The nature, diagnosis, and treatment of neuroticism: back to the future. Clin Psychol Sci 2(3):344–365
43. Griffith JW, Zinbarg RE, Craske MG, Mineka S, Rose RD, Waters AM et al (2010) Neuroticism as a common dimension in the internalizing disorders. Psychol Med 40(7):1125–1136
44. Tyrer P, Tyrer H, Guo B (2016) The general neurotic syndrome: a re-evaluation. Psychother Psychosom 85(4):193–197
45. Paul KI, Moser K (2009) Unemployment impairs mental health: meta-analyses. J Vocat Behav 74(3):264–282
46. Kessler RC (1997) The effects of stressful life events on depression. Annu Rev Psychol 48:191–214
47. Brugha TS, Weich S, Singleton N, Lewis G, Bebbington PE, Jenkins R et al (2005) Primary group size, social support, gender and future mental health status in a prospective study of people living in private households throughout Great Britain. Psychol Med 35(5):705–714
48. Derogatis LR (1977) Symptom Checklist 90, R-Version manual I: scoring, administration, and procedures for the SCL-90. Johns Hopkins Press, Baltimore
49. Angst J, Dobler-Mikola A, Binder J (1984) The Zurich study—a prospective epidemiological study of depressive, neurotic and psychosomatic syndromes. I. Problem, methodology. Eur Arch Psychiatry Neurol Sci 234(1):13–20
50. Eich D, Ajdacic-Gross V, Condrau M, Huber H, Gamma A, Angst J et al (2003) The Zurich Study: participation patterns and Symptom checklist 90-R scores in six interviews, 1979–99. Acta Psychiatr Scand Suppl 108(s418):11–14
51. Angst J, Gamma A, Neuenschwander M, Ajdacic-Gross V, Eich D, Rössler W et al (2005) Prevalence of mental disorders in the Zurich Cohort Study: a twenty year prospective study. Epidemiol Psichiatr Soc 14(2):68–76
52. Angst J, Paksarian D, Cui L, Merikangas KR, Hengartner MP et al (2016) The epidemiology of common mental disorders from age 20 to 50: results from the prospective Zurich Cohort Study. Epidemiol Psychiatr Sci 25(1):24–32
53. Angst J, Gamma A, Rossler W, Ajdacic V, Klein DN (2011) Childhood adversity and chronicity of mood disorders. Eur Arch Psychiatry Clin Neurosci 261(1):21–27
54. Hengartner MP, Müller M, Rodgers S, Rössler W, Ajdacic-Gross V (2014) Occupational functioning and work impairment in association with personality disorder trait-scores. Soc Psychiatry Psychiatr Epidemiol 49(2):327–335
55. Fahrenberg J, Hampel R, Selg H (1984) Das Freiburger Persönlichkeitsinventar FPI. Revidierte Fassung FPI-R und teilweise geänderte Fassung FPI-A1. 4., revidierte Auflage. Hogrefe, Göttingen
56. Angst J, Clayton P (1986) Premorbid personality of depressive, bipolar, and schizophrenic patients with special reference to suicidal issues. Compr Psychiatry 27(6):511–532
57. Eysenck HJ (1963) Biological basis of personality. Nature 199:1031–1034
58. Angst J, Gamma A, Benazzi F, Ajdacic V, Rössler W (2007) Melancholia and atypical depression in the Zurich study: epidemiology, clinical characteristics, course, comorbidity and personality. Acta Psychiatr Scand Suppl 433:72–84
59. Merikangas KR, Avenevoli S, Acharyya S, Zhang H, Angst J (2002) The spectrum of social phobia in the Zurich cohort study of young adults. Biol Psychiatry 51(1):81–91
60. Zeger SL, Liang KY, Albert PS (1988) Models for longitudinal data: a generalized estimating equation approach. Biometrics 44(4):1049–1060
61. Twisk JWR (2003) Applied longitudinal data analysis for epidemiology: a practical guide. Cambridge University Press, Cambridge
62. Durbin CE, Hicks BM (2014) Personality and Psychopathology: a stagnant field in need of development. Eur J Pers 28(4):362–386
63. Widiger TA (2011) Personality and psychopathology. World Psychiatry 10(2):103–106
64. Ferguson CJ (2010) A meta-analysis of normal and disordered personality across the life span. J Pers Soc Psychol 98(4):659–667
65. Ormel J, Oldehinkel AJ, Vollebergh W (2004) Vulnerability before, during, and after a major depressive episode: a 3-wave population-based study. Arch Gen Psychiatry 61(10):990–996
66. De Bolle M, Beyers W, De Clercq B, De Fruyt F (2012) General personality and psychopathology in referred and nonreferred children and adolescents: an investigation of continuity, pathoplasty, and complication models. J Abnorm Psychol 121(4):958–970
67. De Fruyt F, Van Leeuwen K, Bagby RM, Rolland JP, Rouillon F (2006) Assessing and interpreting personality change and continuity in patients treated for major depression. Psychol Assess 18(1):71–80
68. Tang TZ, DeRubeis RJ, Hollon SD, Amsterdam J, Shelton R et al (2009) Personality change during depression treatment: a placebo-controlled trial. Arch Gen Psychiatry 66(12):1322–1330
69. Anusic I, Schimmack U (2016) Stability and change of personality traits, self-esteem, and well-being: introducing the meta-analytic stability and change model of retest correlations. J Pers Soc Psychol 110(5):766–781
70. Tyrer P (2015) Personality dysfunction is the cause of recurrent non-cognitive mental disorder: a testable hypothesis. Pers Ment Health 9(1):1–7
71. Hicks BM, Iacono WG, McGue M (2014) Identifying childhood characteristics that underlie premorbid risk for substance use disorders: socialization and boldness. Dev Psychopathol 26(1):141–157
72. Moffitt TE, Arseneault L, Belsky D, Dickson N, Hancox RJ et al (2011) A gradient of childhood self-control predicts health, wealth, and public safety. Proc Natl Acad Sci U S A 108(7):2693–2698
73. Ellis BJ, Figueredo AJ, Brumbach BH, Schlomer GL (2009) Fundamental dimensions of environmental risk. The impact of harsh versus unpredictable environments on the evolution and development of life history strategies. Hum Nat 20(2):204–268
74. Roff DA (2002) Life history evolution. Sinauer, Sunderland
75. Reale D, Garant D, Humphries MM, Bergeron P, Careau V et al (2010) Personality and the emergence of the pace-of-life syndrome concept at the population level. Philos T R Soc B 365(1560):4051–4063
76. Wolf M, van Doorn GS, Leimar O, Weissing FJ (2007) Life-history trade-offs favour the evolution of animal personalities. Nature 447(7144):581–584
77. Figueredo AJ, Vasquez G, Brumbach BH, Schneider SMR, Sefcek JA et al (2006) Consilience and life history theory: from genes to brain to reproductive strategy. Dev Rev 26(2):243–275
78. Van Linden D, Dunkel CS, Petrides KV (2016) The general factor of personality (GFP) as social effectiveness: review of the literature. Pers Individ Differ 101:98–105
79. Dunkel CS, Kim JK, Papini DR (2012) The general factor of psychosocial development and its relation to the general factor of personality and life history strategy. Pers Individ Differ 52(2):202–206
80. Figueredo AJ, Vasquez G, Brumbach BH, Sefcek JA, Kirsner BR et al (2005) The K-factor: individual differences in life history strategy. Pers Individ Differ 39(8):1349–1360
81. Gladden PR, Figueredo AJ, Jacobs WJ (2009) Life history strategy, psychopathic attitudes, personality, and general intelligence. Pers Individ Differ 46(3):270–275

82. Belsky J, Steinberg L, Houts RM, Halpern-Felsher BL (2010) The development of reproductive strategy in Females: early maternal harshness → earlier menarche → increased sexual risk taking. Dev Psychol 46(1):120–128
83. Wilson M, Daly M (1997) Life expectancy, economic inequality, homicide, and reproductive timing in Chicago neighbourhoods. BMJ 314(7089):1271–1274
84. Dunkel CS, Mathes E, Beaver KM (2013) Life history theory and the general theory of crime: life expectancy effects on low self-control and criminal intent. J Soc Evol Cult Psychol 7(1):12–23
85. McNamara JM, Barta Z, Fromhage L, Houston AI (2008) The coevolution of choosiness and cooperation. Nature 451(7175):189–192
86. Ellis BJ, Del Giudice M, Dishion TJ, Figueredo AJ, Gray P et al (2012) The evolutionary basis of risky adolescent behavior: implications for science, policy, and practice. Dev Psychol 48(3):598–623
87. Koolhaas JM, de Boer SF, Coppens CM, Buwalda B (2010) Neuroendocrinology of coping styles: towards understanding the biology of individual variation. Front Neuroendocrinol 31(3):307–321
88. Wolf M, van Doorn GS, Weissing FJ (2008) Evolutionary emergence of responsive and unresponsive personalities. Proc Natl Acad Sci U S A 105(41):15825–15830
89. David JT, Cervantes MC, Trosky KA, Salinas JA, Delville Y (2004) A neural network underlying individual differences in emotion and aggression in male golden hamsters. Neuroscience 126(3):567–578
90. Figueredo AJ, Vasquez G, Brumbach BH, Schneider SM (2007) The K-factor, covitality, and personality: a psychometric test of life history theory. Hum Nat 18(1):47–73
91. Hurst JE, Kavanagh PS (2017) Life history strategies and psychopathology: the faster the life strategies, the more symptoms of psychopathology. Evol Hum Behav 38(1):1–8
92. Del Giudice M (2009) Sex, attachment, and the development of reproductive strategies. Behav Brain Sci 32(1):1–21 **(discussion 21-67)**
93. Del Giudice M, Ellis BJ, Shirtcliff EA (2011) The adaptive calibration model of stress responsivity. Neurosci Biobehav Rev 35(7):1562–1592
94. Gluckman PD, Low FM, Buklijas T, Hanson MA, Beedle AS (2011) How evolutionary principles improve the understanding of human health and disease. Evol Appl 4(2):249–263
95. Stearns SC, Nesse RM, Govindaraju DR, Ellison PT (2010) Evolutionary perspectives on health and medicine. Proc Natl Acad Sci U S A 107(Suppl 1):1691–1695
96. Brune M, Belsky J, Fabrega H, Feierman HR, Gilbert P et al (2012) The crisis of psychiatry—insights and prospects from evolutionary theory. World Psychiatry 11(1):55–57
97. Rössler W, Hengartner MP, Ajdacic-Gross V, Haker H, Angst J (2013) Deconstructing sub-clinical psychosis into latent-state and trait variables over a 30-year time span. Schizophr Res 150(1):197–204
98. Barnett JH, Salmond CH, Jones PB, Sahakian BJ (2006) Cognitive reserve in neuropsychiatry. Psychol Med 36(8):1053–1064
99. Berk M, Williams LJ, Jacka FN, O'Neil A, Pasco JA et al (2013) So depression is an inflammatory disease, but where does the inflammation come from? BMC Med 11:200