Journal of Affective Disorders 226 (2018) 6–11

Contents lists available at ScienceDirect

Journal of Affective Disorders

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

Research paper

Can flow experiences be protective of work-related depressive symptoms and burnout? A genetically informative approach

Miriam A. Mosing[a,b,*,1], Ana Butkovic[c,1], Fredrik Ullén[a]

[a] Dept of Neuroscience, Karolinska Institutet, Stockholm, Sweden
[b] Dept of Medical Epidemiology and Biostatistics, Karolinska Institutet, Stockholm, Sweden
[c] Dept of Psychology, University of Zagreb, Zagreb, Croatia

ARTICLE INFO

Keywords:
Emotional exhaustion
Mental health
Well-being
Intervention
Twin

ABSTRACT

Background: Genetic research on depression and burnout has focused mostly on adverse factors, although various aspects in daily life related to positive coping and well-being have been shown to potentially be protective. Using a large genetically informative sample, we aim to explore the potential relationship between flow proneness and work-related depressive symptoms and burnout.

Methods: About 10,000 Swedish twins filled in the Swedish Flow Proneness Questionnaire, a subscale of the Hopkins Symptom Checklist (SCL) depression scale, and the Emotional Exhaustion subscale of the Maslach Burnout Inventory-General Survey. A higher score indicated more flow, less emotional exhaustion and less depression. The classical twin design and co-twin control analyses were applied.

Results: Phenotypic correlations were .43 between depressive symptoms and flow proneness, .34 between burnout and flow proneness, and .62 between depressive symptoms and burnout. Broad-sense heritabilities (G) ranged between 33–35% for the three variables. Associations between the variables were due to significant genetic as well as non-shared environmental influences. Co-twin control analyses showed that associations remained significant when controlling for all genetic and shared familial factors, in line with a causal relationship.

Limitations: Although the co-twin control design can test for consistency of associations with a causal relationship, it cannot unequivocally establish causality.

Conclusions: Genetic liability has a substantial influence on associations between flow proneness and emotional problems at work (depression, burnout). However, the presence of significant environmental correlations is in line with a (partly) causal relationship between flow and work related depression and burnout, which in turn may suggest that interventions which increase flow could potentially reduce emotional problems at work.

1. Introduction

According to the World Health Organisation (WHO, 2016) globally more than 350 million people of all ages suffer from depression, the leading cause of disability worldwide. Depression is a highly recurrent disorder associated with poor physical health and a negative impact on person's work and interpersonal life and is among the most prevalent of all psychiatric disorders (Gotlib and Hammen, 2014). Accordingly, there has been an exponential increase in research examining factors related to onset, course and treatment of depression.

In the last two decades another condition has been studied across the globe and characterized as a diagnosable mental health disorder by the WHO: occupational burnout. It emerged as an important concept in the 1970s and from the 1990s onwards it has been researched in North America, Western and Eastern Europe, Asia, the Middle East, Latin America, Australia, New Zealand, Africa, China, and Indian subcontinent (Schaufeli et al., 2009). While most research uses the three-dimensional Maslach Burnout Inventory (MBI; Maslach and Jackson, 1981; Maslach et al., 1996) which consists of exhaustion, cynicism, and inefficacy, others regard burnout as a one-dimensional construct with emotional exhaustion as the only hallmark.

A recent review of more than 90 studies established that occupational burnout and depression are strongly related with correlations ranging between .50 – .60 and that the emotional exhaustion component of burnout showed the strongest association with depression (Bianchi et al., 2015). Notwithstanding the multitude of studies, the association between burnout and depression and its underlying biological architecture is still not well understood.

* Correspondence to: Department of Neuroscience, Karolinska Institute, Retzius v 8, SE-171 77 Stockholm, Sweden.
E-mail address: Miriam.Mosing@ki.se (M.A. Mosing).
[1] Authors have equally contributed to this work.

http://dx.doi.org/10.1016/j.jad.2017.09.017
Received 24 August 2017; Received in revised form 12 September 2017; Accepted 13 September 2017
Available online 18 September 2017
0165-0327/

While genetic influences on depressive symptoms have thoroughly been explored and its heritability has been estimated at about 37% (for a review see Flint and Kendler, 2014), only little is known about genetic influences on burnout or emotional exhaustion. In a study on Dutch twins, their siblings and spouses, Middeldorp et al. (2005) found that family environment contributed to individual differences in burnout (measured by the emotional exhaustion subscale of the Maslach Burnout Inventory (Maslach et al., 1996)), explaining 22% of the variance, while genetic influences were not significant. However, in another study in Dutch twins and their siblings using the same measure of burnout, Middeldorp et al. (2006) reported heritabilities of 30% for males and 13% for females with an additional 15% of variance explained by family environment in females. Finally, in a sample of Swedish twins using Pines Burnout Measure, Blom et al. (2012) found a heritability of 33% in both men and women. To our knowledge, Middeldorp et al. (2006) is the only study to date that has explored the association between burnout (emotional exhaustion) and anxious depression using a genetically informative sample. The phenotypic correlation between the two variables was estimated at .40 in both sexes, with 50% and 66% of the association being due to genetic factors in males and females, respectively.

Most behaviour genetics research on depression and burnout has focused on adverse factors, such as negative life events (Mather et al., 2014; Wichers et al., 2012), rumination (Johnson et al., 2014) and anxiety sensitivity (Waszczuk et al., 2015). However, various factors related to positive coping and well-being in daily life have been shown to be negatively associated with depression and burnout. If this relationship is not entirely due to shared underlying genetic aetiology, possibly lifestyle factors promoting enjoyment and intrinsic motivation may be protective for development of depressive symptoms and burnout and as such may serve as a good starting point for interventions. Only two studies to date have explored this using a genetically informative design. Waszczuk et al. (2015) reported a significant negative association between mindfulness, commonly defined as the state of being attentive to and aware of what is taking place in the present (Brown and Ryan, 2003), and depression ($r = -.34$). Most of this association (60%) could be explained by overlapping genetic factors. Whisman et al. (2014) looked at another possible protective factor for depression: experience of pleasant events. Phenotypic correlations between the different measures of experience of pleasant events (frequency, enjoyment and obtained pleasure) with depressive symptoms were all negative and significant (ranging between $-.31$ to $-.44$) and were largely due to shared genetic factors (65%).

Here, we explore individual differences in the proneness to have psychologícal flow experiences (flow proneness) as a possible protective factor for depression and emotional exhaustion. Flow is defined as a subjective state that has the following characteristics: intense and focused concentration on what one is doing in the present moment, merging of action and awareness, loss of reflective self-consciousness, a sense that one can control one's actions, distortion of temporal experience, and experience of the activity as intrinsically rewarding (Nakamura and Csikszentmihalyi, 2002). A series of studies on different Japanese samples have shown that flow experiences are associated with better health in elderly people (Hirao et al., 2012), that students who experienced flow more often in their daily lives are more likely to show higher self-esteem and lower anxiety, use active coping strategies more often and passive coping strategies less often, and they also report more Jujitsu-kan, a Japanese sense of fulfilment, and greater satisfaction with their lives (Asakawa, 2010). Further, unemployed adults who experienced more flow in daily life had significantly higher health-related quality of life (Hirao and Kobayashi, 2013).

In the present study, using a large, genetically informative sample of Swedish twins, we aim to: (1) explore the potentially protective influence of flow proneness on emotional exhaustion and depressive symptoms; (2) estimate genetic and environmental influences on individual differences in emotional exhaustion; and (3) understand the genetic and environmental aetiology underlying the relationship between the three variables.

2. Methods

2.1. Participants

Participants in the study were twins from the Swedish Twin Registry (STR), one of the largest registries of its kind (Lichtenstein et al., 2002, 2006), born between 1959 and 1985. The participants were part of the STAGE cohort (Lichtenstein et al., 2006), which has been approached several times, and the data for the present study were collected between 2012 and 2013 as part of a web survey (Mosing et al., 2014). The full sample included 10,120 twins with a score for at least one of the studied variables, with 2337 full twin pairs (1114 monozygotic (MZ) and 1223 dizygotic (DZ) pairs) and 7783 single twins without the co-twin participating. Their age was between 27 and 54 years ($M = 40.7$, $SD = 7.75$). Single twin individuals contribute to the estimation of means, variances, and covariate effects and were therefore included in the analyses. Zygosity was determined based on questions about intra-pair similarities and has subsequently been confirmed in 27% of the twins in the STR using genotyping confirming that the questionnaire based zygosity determination was correct for more than 98% of twin pairs. For further details on the STAGE cohort and zygosity determination in the STR see (Lichtenstein et al., 2002, 2006). All participants gave informed consent to participate and the study was approved by the Regional Ethics Review Board in Stockholm (Dnr 2011/570-31/5, 2011/1425-31, and 2012/1107/32).

2.2. Measures

2.2.1. Flow

Flow proneness (FP) was measured with the Swedish Flow Proneness Questionnaire (SFPQ; Ullén et al., 2012) which measures FP in three domains: work, leisure and maintenance. In addition, a subscale measuring flow proneness in the musical domain, i.e. how frequently the participant experienced flow during musical activities, was included. Note that for this reason, the FP questions in leisure activities explicitly excluded musical activities such as playing an instrument or singing. All four sub-scales consisted of seven items each rated on a five-point Likert scale ('never' to 'every or almost every day'). Global FP was calculated as the mean score of flow proneness in work, leisure and maintenance or as the mean score of any other two areas if the participant were missing a score in one of the three areas (see Mosing et al., 2012b). Cronbach alpha reliabilities in this study were .76, .86, .79 and .86 for FP in work, leisure, maintenance, and music, respectively.

2.2.2. Depressive symptoms

Depressive symptoms were measured with a six item subscale of the Hopkins Symptom Checklist (SCL) depression scale, which has previously been used in Swedish and Danish population studies Magnusson Hanson et al., 2014). The items are graded from 0 to 4 giving a range of full scores from 0 to 24. Note that the scale is reversed scored, so that a higher score indicates less depressive symptoms. The Cronbach alpha reliability in present study was .89.

2.2.3. Bournout

Emotional exhaustion was measured with the Emotional exhaustion subscale of the Maslach Burnout Inventory-General Survey (MBI-GS; Schaufeli et al., 1996) which was developed to measure burnout in occupations without direct personal contact with service recipients or with only casual contact with people. The scale has five items (e.g. “I feel tired when I get up in the morning and have to face another day on the job”). In this study items were scored on a 6-point frequency rating scale as in some previous studies in Sweden (e.g. Magnusson Hanson et al., 2008), ranging from 'every day ' (1) to 'a few times per year or

less/never ' (6), with higher scores indicating less emotional exhaustion. The Cronbach alpha reliability was .88 in the present study.

2.3. Statistical analyses and genetic modelling

All variables were converted to z-scores and Winsorised (Dixon, 1960) at three standard deviations from the mean to reduce the effect of possibly spurious outliers. Phenotypic analyses included all single twins and one twin of each pair to account for relatedness of the twins and were conducted in SPSS (IBM Corp, 2013). Pearson correlations were calculated between all variables.

The classical twin design makes use of the differences in genetic sharing between MZ and DZ twins, with the former sharing 100% of their segregating genes and the latter only sharing 50% on average, to partition variance in a trait and covariance between traits into that due to genes (*additive (A)* and *non-additive (D))* and environment (*common (C)* – all influences shared between the twins and making the pair more alike to each other, *and unique (E)* – all influences not shared between the twins and making them more different including measurement error). With the use of structural equation modelling the combination of ACE/ADE influences that best explains the population variance in a trait or the covariance between traits can be estimated. In classical twin studies only three of those influences (ACE or ADE) can be fitted at a time, with non-shared (E) environmental influences always included as it contains the error variance. Whether to fit an ACE or ADE model depends on the pattern of MZ-DZ correlations. MZ correlations less than twice the size of DZ correlations strongly suggest the importance of shared environment, and DZ correlations less than half the MZ correlations indicate the importance of non-additive genetic influences. Using maximum likelihood (ML) modelling procedures, parameter estimates for the saturated model can be derived and subsequently specific hypotheses regarding the significance of particular parameters can be tested statistically. This is done by comparing the goodness-of-fit to the observed data (distributed as $\chi 2$) of various models using the minus two times log-likelihood ($-2LL$) statistic. If the change in $\chi 2$ ($\Delta\chi 2$) is not significant, the more parsimonious model is the one of choice.

Genetic analyses were done in the statistical program Mx (Neale et al., 2006; Neale and Maes, 2004). After assumption testing, univariate sex-limitation models, allowing for the estimates to differ between males and females, were fitted to the data in order to estimate A, C/D and E influences for each variable, i.e. an ADE model was fitted for global flow and depressive symptoms and ACE model for emotional exhaustion. Based on the results of the univariate analyses, a trivariate GE Cholesky decomposition was fitted to estimate the contribution of genetic and environmental influences to the covariation between the three variables. Nested reduced univariate and multivariate models were compared to the full models in order to test which parameters were significant.

2.3.1. Within-pair analyses in identical twins

To test whether the association of flow proneness with burnout and depressive symptoms, respectively, could entirely be explained by familial (genetic and environmental) factors shared within the twin pairs, within-pair (co-twin control) analyses in the identical pairs were conducted. In genetically identical twin pairs differing in their flow proneness, the twin of a pair with more flow experiences would be expected to be less likely to suffer from depressive symptoms and burnout than the co-twin with less flow experiences. As such, we would expect that in MZ pairs the within-twin pair difference in flow proneness (twin 1 minus twin 2) would be significantly associated with the within-pair difference in the mental health measure (i.e. burnout or depressive symptoms), unless the association observed in the full sample (between twin pairs) is largely due to shared underlying aetiology. This is as identical twins share all their segregating genes as well as their family environment. In other words, if flow experience is protective of depressive symptoms and burnout as we observed in the between-twin analyses above, we would expect that a twin with higher flow proneness would have lower risk for adverse mental health (as measured here) than his or her co-twin with less flow proneness. For within-pair analyses we calculated the within-pair difference score for flow proneness and calculated Pearson correlations with the within-pair difference score of depressive symptoms and burnout, respectively, in the full MZ-sample (Npairs = 1129).

Table 1
Descriptive statistics and phenotypic correlations between studied variables.

Variable	N	M	SD	(1)	(2)	(3)	(4)	(5)	(6)
(1) Flow work	7195	26.78	3.89	1					
(2) Flow maintenance	7641	25.27	4.06	.40*	1				
(3) Flow leisure	7597	25.87	4.02	.48*	.42*	1			
(4) Flow global	7640	25.91	3.21	.77*	.80*	.81*	1		
(5) Flow music	5454	22.41	5.48	.17*	.17*	.21*	.23*	1	
(6) Depressive symptoms	6326	17.79	5.10	.45*	.28*	.31*	.43*	.07*	1
(7) Emotional exhaustion	6339	22.37	6.08	.36*	.24*	.23*	.34*	.03	.62*

Note. Depressive symptoms and emotional exhaustion scales are reversed scored, so that a higher score indicates less depressive symptoms and less emotional exhaustion.
* $p < .001$.

3. Results

Descriptive statistics for all variables and their inter-correlations are presented in Table 1. FP work, maintenance, leisure, as well as global FP correlated significantly with depressive symptoms and emotional exhaustion with correlations ranging between .23 and .45 (with the strongest associations with FP work and global FP). In contrast, correlations with FP music were close to zero. Finally, as expected the correlation between depressive symptoms and emotional exhaustion was high ($r = .62$, $p < .001$).

Twin correlations are presented in Table 2. Estimates from the univariate sex-limitation models are presented in Table 3. Parameters could be equated between the sexes for global flow and emotional exhaustion, but not for depressive symptoms. Broad-sense heritability (G), which includes both additive and non-additive genetic influences, was 33% for global flow for both males and females. Heritability of emotional exhaustion was also 33% when equated between sexes. Although parameters could not be equated between sexes for depressive symptoms, due to sex differences in A and D estimates, broad-sense heritability was very similar for males (33%) and females (35%).

Since ADE models fitted best for global flow and depressive symptoms, but ACE showed the best fit for emotional exhaustion, a trivariate GE Cholesky decomposition was fitted, estimating broad-sense heritability. As univariate models showed little sex differences between genetic (A + D) and environmental (E or C) influences for all three variables, the trivariate GE Cholesky decomposition was fitted with the sexes equated (Fig. 1). Modelling results (Fig. 1) show that the phenotypic association between depressive symptoms and emotional exhaustion was due to both genetic (see the significant path from the G1 factor to emotional exhaustion) and environmental (see the significant path from the E1 factor to emotional exhaustion) overlap between those two constructs. The genetic correlation between depressive symptoms and emotional exhaustion was $r_g = .83$, while environmental correlation was $r_e = .51$. This indicates that of the phenotypic association between depressive symptoms and emotional exhaustion, 46% is explained by genetic factors and 54% by non-shared environmental factors.

Of the genetic influences on global FP, 59% were unique (as indicated by the path from G3 to global flow) with the remaining 41% being shared with genetic influences on depressive symptoms and emotional exhaustion. E influences on global FP were mainly unique

Table 2
Twin correlations with 95% confidence intervals for global flow, depressive symptoms and emotional exhaustion.

Zygosity	*N*	Global flow	*N*	Depressive symptoms	*N*	Emotional exhaustion
MZ	3810	.33 (.28–.38)	3077	.35 (.31–.41)	3084	.35 (.29–.40)
DZ	5993	.13 (.07–.18)	4735	.10 (.04–.17)	4747	.14 (.07–.20)
MZF	2299	.36 (.29–.42)	1876	.37 (.30–.44)	1881	.35 (.28–.42)
MZM	1511	.29 (.20–.37)	1201	.31 (.19–.41)	1203	.35 (.25–.44)
DZF	1665	.08 (−.02 to .17)	1339	.16 (.04–.27)	1342	.23 (.11–.33)
DZM	1283	.22 (.10–.33)	1014	.02 (−.13 to .17)	1018	.21 (.04–.36)
DZOS	3045	.12 (.04–.20)	2382	.10 (.00–.19)	2387	.05 (−.04 to .15)

Note. DZ = dizygotic twins, F = female, M = male, MZ = monozygotic twins, OS = opposite sex.

Table 3
Univariate estimates with 95% confidence intervals from sex-limitation models for females (f) and males (m).

	Global flow (ADE)	Depressive symptoms (ADE)*	Emotional exhaustion (ACE)
Af	.11 (.00–.37)	.25 (.00–.41)	.24 (.00–.39)
Df/Cf	.24 (.00–.39)	.10 (.00–.41)	.08 (.00–.30)
Ef	.65 (.59–.71)	.65 (.58–.71)	.68 (.61–.74)
Am	.31 (.02–.39)	.03 (.00–.36)	.31 (.00–.47)
Dm/Cm	.01 (.00–.31)	.30 (.00–.43)	.09 (.00–.41)
Em	.68 (.61–.77)	.67 (.57–.79)	.60 (.51–.71)

* Female and male parameters could not be equated without significant deterioration of model fit.

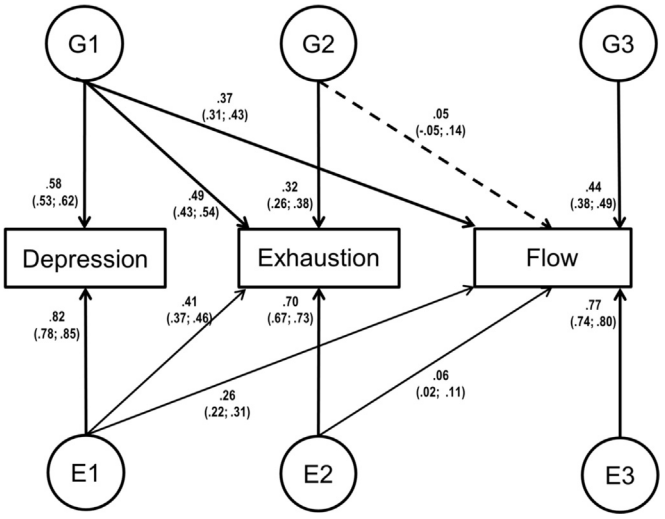

Fig. 1. Standardized estimates with confidence intervals based on the trivariate GE Cholesky decomposition with non-significant pathways indicated with a dashed line.

(88% as indicated by the path from E3 to global FP) with only 12% overlapping with E influences on depressive symptoms and emotional exhaustion. The genetic correlation between FP and depressive symptoms was r_g = .64, and the environmental correlation r_e = .32, while the genetic correlation between flow and emotional exhaustion was r_g = .58, and the environmental correlation r_e = .23. This means that 50% of phenotypic association between flow and depressive symptoms is due to overlapping genetic influences, with the remainder being due to overlapping environmental influences, while 57% of phenotypic association between flow and emotional exhaustion is due to overlapping genetic influences, and 43% to overlapping environmental influences.

Results of the co-twin control analyses in identical twins showed that flow proneness was still significantly associated with less symptoms of depression (r = .33; p < .001) and emotional exhaustion (r = .23; p < .001) even when controlling for all shared genetic and familial factors. In other words, the twin with higher flow proneness was significantly less likely to experience symptoms of emotional exhaustion or depression, than his or her co-twin who experienced less flow.

4. Discussion

The present study explored the association between flow proneness, depression and emotional exhaustion and their underlying genetic aetiology. Both depression and emotional exhaustion (burnout) are increasing health problems around the world meriting research on related factors and their operating mechanisms.

4.1. Phenotypic associations between flow, emotional exhaustion and depressive symptoms

First, we explored the phenotypic associations between flow proneness, depressive symptoms and emotional exhaustion. As expected, higher global FP was associated with less emotional exhaustion (.34) and less depressive symptoms (.43), suggesting that individuals experiencing flow more often (regardless of domain), also experience less depressive symptoms and less emotional exhaustion. This finding suggests that, unless the association was entirely explained by shared genetic influences, enhancing an individual's flow proneness could potentially reduce the risk for health problems, such as depression and emotional exhaustion (see further discussion below). The global flow measure used here was not domain specific and included flow experiences in different areas such as work, leisure and maintenance, suggesting that overall FP regardless of domain seems to be protective. This is in line with the finding that FP music (restricted to only one specific domain) showed little to no association with our mental health measures.

The phenotypic association between depressive symptoms and emotional exhaustion was moderate to high (r = .62) which is in line with Bianchi and colleague's review (2015) on burnout-depression overlap, reporting correlations around .60.

4.2. Heritability of emotional exhaustion

Since past studies report mixed findings in terms of genetic and environmental influences on burnout, our second aim was to establish the heritability of emotional exhaustion, a commonly used indicator of burnout, using a large Swedish twin cohort. The heritability of emotional exhaustion was around 30% for males and females which is in line with the findings reported by Middeldorp et al. (2006) and Blom et al. (2012), with the remaining variance in emotional exhaustion being due to unique environmental influences. The heritability of flow proneness in this sample and the STR has been reported and discussed elsewhere (Butkovic et al., 2015; Mosing et al., 2012a, b) and the broad-sense heritability of depressive symptoms was around 35%, which is in line with results of previous studies (e.g. Kendler et al., 1994).

4.3. Genetic aetiology of the associations

Our third aim was to explore the genetic and environmental

aetiology of the relationship between emotional exhaustion, depressive symptoms and flow proneness. In the present sample, about half of the phenotypic association between flow and depressive symptoms, as well as between flow and emotional exhaustion was explained by overlapping genetic factors with the remainder being due to environmental factors. This was further confirmed by the co-twin control results in identical twins, suggesting that the protective effect of flow proneness was still present when controlling for all shared genetic and familial confounding. These findings are in line with a causal relationship between flow proneness and the mental health measures, suggesting that possibly environmental interventions increasing flow experiences could also have a positive effect on an individual's risk for burnout (McGue et al., 2010).

Further, also close to half (46%) of the phenotypic association between depressive symptoms and emotional exhaustion was explained by shared genetic factors, with the remainder being due to unique environmental factors. Although, unlike Middeldorp et al. (2006) we did not find sex-differences in the genetic aetiology between these two variables, our findings are in line with their estimates, in that 50% of the association was explained by shared genetic influences in men and 66% in women. The sex differences reported by Middeldorp et al. (2006) could be due to the fact that they, unlike in the present sample, found sex differences in the heritability of emotional exhaustion as well as depressive symptoms.

4.4. Implications and limitations of the study

This is the first study exploring the potential protective effect of flow experiences on burnout and depression using a genetically informative sample. Our findings indicate that indeed, flow proneness has a moderate protective influence on burnout (emotional exhaustion) and depression. Further, we could show that about half of these associations were due to genes while the other half was explained by environmental influences. This finding is interesting as it may suggest that interventions aiming to increase flow experience, may also be beneficial to reduce the risk of burnout or depression. For example, it seems straightforward for employers to encourage their employees to each identify and engage in the activities most intrinsically motivating and conducive to flow (Csikszentmihalyi, 1990, 1998). Further, over recent years many employers have adopted policies which allow staff to engage in physical activities during working hours, e.g. one hour a week, as this has been shown to be associated with health benefits (World Health Organisation, 2017). Similarly, instead of just allowing for physical exercise, employers could expand this to any activity which induces flow in their individuals (if applicable). This could potentially help prevent stress related health problems. Further, medical practitioners presented with individuals already experiencing work related stress and health problems could explicitly help patients to identify flow inducing activities, encourage them to engage in those more actively and keep a skill-challenge balance, i.e. taking on new challenges when skills rise, avoiding under-stimulation/boredom and over-challenge/stress (Csikszentmihalyi, 1990, 1998). If also supported by the employer, this could serve as a good starting point for an intervention. However, to date little is known about flow-enhancing prevention and intervention strategies and whether such would have long-lasting effect. Further research is needed to investigate the potential of such interventions.

Although our findings are in line with a causal model, i.e. FP causally influencing emotional exhaustion and depression, we cannot rule out more complex mechanisms underlying these associations. The MZ co-twin control results are also consistent with reverse and reciprocal causality (i.e. emotional exhaustion/depression influencing flow proneness), as well as with simultaneous non-shared environmental effects on both variables. Furthermore, it is possible that the observed effects are influenced by gene environment correlation and interactions, which were not analysed here. Nevertheless, the finding that a large part of the association is due to environmental influences gives hope that the right intervention could be beneficial. Further, since we only had twin data, we could not disentangle C and D influences, but instead applied a multivariate GE model. However, given that this is the first study to examine the aetiology of the relationship between flow, depressive symptoms and emotional exhaustion, we believe it adds valuable knowledge to the field. Finally, as usual, our findings cannot be generalized beyond the measures employed and the sample used, namely middle-aged Swedes.

In summary, depression and emotional exhaustion (burnout) are huge burdens in our society, meriting research to establish possible protective factors. Results of the present study indicate that flow experiences are associated with depression and burnout and that these associations are partly due to environmental influences. These findings are in line with a causal model suggesting a potentially protective effect of flow, giving hope that flow enhancing interventions could potentially also reduce the risk for depression and burnout. However, more studies are needed to establish whether an individual's flow proneness can indeed be enhanced resulting in the desired protective effect.

Financial support

This work was supported by the Bank of Sweden Tercentenary Foundation (M11-0451:1), the Swedish Scientific Council (521–2010-3195), and the Sven and Dagmar Salén Foundation.

Ethical standards

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

Acknowledgments

The authors would like to thank the twins for their participation.

References

Asakawa, K., 2010. Flow experience, culture, and well-being: how do autotelic Japanese college students feel, behave, and think in their daily lives? J. Happiness Stud. 11, 205–223.
Bianchi, R., Schonfeld, I.S., Laurent, E., 2015. Burnout-depression overlap: a review. Clin. Psychol. Rev. 36, 28–41.
Blom, V., Bergstrom, G., Hallsten, L., Bodin, L., Svedberg, P., 2012. Genetic susceptibility to burnout in a Swedish twin cohort. Eur. J. Epidemiol. 27, 225–231.
Brown, K.W., Ryan, R.M., 2003. The benefits of being present: mindfulness and its role in psychological well-being. J. Personal. Social. Psychol. 84, 822–848.
Butkovic, A., Ullén, F., Mosing, M.A., 2015. Personality related traits as predictors of music practice: underlying environmental and genetic influences. Personal. Individ. Differ. 74, 133–138.
Csikszentmihalyi, M., 1990. Flow: the Psychology of Optimal Experience. HarperCollins Publishers, New York.
Csikszentmihalyi, M., 1998. Finding Flow: the Psychology of Engagement with Everyday Life. Basic Books.
Dixon, W.J., 1960. Simplified estimation from censored normal samples. Ann. Math. Stat. 31, 385–391.
Flint, J., Kendler, K.S., 2014. The genetics of major depression. Neuron 81, 484–503.
Gotlib, I.H., Hammen, C.L., 2014. Introduction. In: Gotlib, I.H., Hammen, C.L. (Eds.), Handbook of Depression, 3 ed. Guilford Press, New York, pp. 1–4.
Hirao, K., Kobayashi, R., 2013. Health-related quality of life and sense of coherence among the unemployed with autotelic, average, and non-autotelic personalities: a cross-sectional survey in Hiroshima, Japan. PLoS One 8, e73915.
Hirao, K., Kobayashi, R., Okishima, K., Tomokuni, Y., 2012. Flow experience and health-related quality of life in community dwelling elderly Japanese. Nurs. Health Sci. 14, 52–57.
IBM Corp, 2013. IBM SPScows, 22.0 ed. IBM Corp, Armonk, NY.
Johnson, D.P., Whisman, M.A., Corley, R.P., Hewitt, J.K., Friedman, N.P., 2014. Genetic and environmental influences on rumination and its covariation with depression. Cogn. Emot. 28, 1270–1286.
Kendler, K.S., Walters, E.E., Truett, K.R., Heath, A.C., Neale, M.C., Martin, N.G., Eaves, L.J., 1994. Sources of individual differences in depressive symptoms: analysis of two samples of twins and their families. Am. J. Psychiatry 151, 1605–1614.
Lichtenstein, P., De Faire, U., Floderus, B., Svartengren, M., Svedberg, P., Pedersen, N.L., 2002. The Swedish Twin Registry: a unique resource for clinical, epidemiological and

genetic studies. J. Intern. Med. 252, 184–205.
Lichtenstein, P., Sullivan, P.F., Cnattingius, S., Gatz, M., Johansson, S., Carlström, E., Björk, C., Svartengren, M., Wolk, A., Klareskog, L., de Faire, U., Schalling, M., Palmgren, J., Pedersen, N.L., 2006. The Swedish Twin Registry in the third millennium: an update. Twin Res. Human. Genet. 9, 875–882.
Magnusson Hanson, L.L., Theorell, T., Oxenstierna, G., Hyde, M., Westerlund, H., 2008. Demand, control and social climate as predictors of emotional exhaustion symptoms in working Swedish men and women. Scand. J. Public Health 36, 737–743.
Magnusson Hanson, L.L., Westerlund, H., Leineweber, C., Rugulies, R., Osika, W., Theorell, T., Bech, P., 2014. The Symptom Checklist-core depression (SCL-CD6) scale: psychometric properties of a brief six item scale for the assessment of depression. Scand. J. Public Health 42, 82–88.
Maslach, C., Jackson, S.E., 1981. The measurement of experienced burnout. J. Organ. Behav. 2, 99–113.
Maslach, C., Jackson, S.E., Leiter, M.P., 1996. Maslach Burnout Inventory. Consulting Psychologists Press, Palo Alto, CA.
Mather, L., Blom, V., Svedberg, P., 2014. Stressful and traumatic life events are associated with burnout-a cross-sectional twin study. Int. J. Behav. Med. 21, 899–907.
McGue, M., Osler, M., Christensen, K., 2010. Causal inference and observational research: the utility of twins. Perspect. Psychol. Sci. 5, 546–556.
Middeldorp, C.M., Cath, D.C., Boomsma, D.I., 2006. A twin-family study of the association between employment, burnout and anxious depression. J. Affect. Disord. 90, 163–169.
Middeldorp, C.M., Stubbe, J.H., Cath, D.C., Boomsma, D.I., 2005. Familial clustering in burnout: a twin-family study. Psychol. Med. 35, 113–120.
Mosing, M.A., Madison, G., Pedersen, N.L., Kuja-Halkola, R., Ullén, F., 2014. Practice does not make perfect: No causal effect of musical practice on musical ability. Psychol. Sci. 25, 1795–1803.
Mosing, M.A., Magnusson, P.K.E., Pedersen, N.L., Nakamura, J., Madison, G., Ullén, F., 2012a. Heritability of proneness for psychological flow experiences. Personal. Individ. Differ. 53, 699–704.
Mosing, M.A., Pedersen, N.L., Cesarini, D., Johannesson, M., Magnusson, P.K., Nakamura, J., Madison, G., Ullen, F., 2012b. Genetic and environmental influences on the relationship between flow proneness, locus of control and behavioral inhibition. PLoS One 7, 2.
Nakamura, J., Csikszentmihalyi, M., 2002. The concept of flow. In: Snyder, C.R., Lopez, S.J. (Eds.), Handbook of Positive Psychology. Oxford Univ Press, Oxford, pp. 89–105.
Neale, M.C., Boker, S.M., Xie, G., Maes, H.H., 2006. Mx: Statistical Modeling, 7th ed. Department of Psychiatry, Virginia Commonwealth University, Richmond, VA.
Neale, M.C., Maes, H.H.M., 2004. Methodology for Genetic Studies of Twins and Families. Kluwer Academic Publishers, Dordrecht, The Netherlands.
Schaufeli, W.B., Leiter, M.P., Maslach, C., 2009. Burnout: 35 years of research and practice. Career Dev. Int. 14, 204–220.
Schaufeli, W.B., Leiter, M.P., Maslach, C., Jackson, S.E., 1996. The Maslach Burnout inventory-general survey. In: Maslach, C., Jackson, S.E., Leiter, M.P. (Eds.), Maslach Burnout Inventory. Consulting Psychologists Press, Palo Alto, CA.
Ullén, F., de Manzano, Ö., Almeida, R., Magnusson, P.K.E., Pedersen, N.L., Nakmura, J., Csíkszentmihályi, M., Madison, G., 2012. Proneness for psychological flow in everyday life: associations with personality and intelligence. Personal. Individ. Differ. 52, 167–172.
Waszczuk, M.A., Zavos, H.M.S., Antonova, E., Haworth, C.M., Plomin, R., Eley, T.C., 2015. A multivariate twin study of trait mindfuless, depressive symptoms, and anxiety sensitivity. Depress. Anxiety 32, 254–261.
Whisman, M.A., Johnson, D.P., Rhee, S.H., 2014. A behavior genetic analysis of pleasant events, depressive symptoms, and their covariation. Clin. Psychol. Sci.: a J. Assoc. Psychol. Sci. 2, 535–544.
Wichers, M., Maes, H.H., Jacobs, N., Derom, C., Thiery, E., Kendler, K.S., 2012. Disentangling the causal inter-relationship between negative life events and depressive symptoms in women: a longitudinal twin study. Psychol. Med. 42, 1801–1814.
World Health Organisation, 2016. Depression.
World Health Organisation, 2017. Physical Activity.