

Meeting risk with resilience: high daily life reward experience preserves mental health

Citation for published version (APA):

Geschwind, N., Peeters, F., Jacobs, N., Delespaul, P., Derom, C., Thiery, E., van Os, J., & Wichers, M. (2010). Meeting risk with resilience: high daily life reward experience preserves mental health. *Acta Psychiatrica Scandinavica*, 122(2), 129-138. <https://doi.org/10.1111/j.1600-0447.2009.01525.x>

Document status and date:

Published: 01/08/2010

DOI:

[10.1111/j.1600-0447.2009.01525.x](https://doi.org/10.1111/j.1600-0447.2009.01525.x)

Document Version:

Publisher's PDF, also known as Version of record

Document license:

Taverne

Please check the document version of this publication:

- A submitted manuscript is the version of the article upon submission and before peer-review. There can be important differences between the submitted version and the official published version of record. People interested in the research are advised to contact the author for the final version of the publication, or visit the DOI to the publisher's website.
- The final author version and the galley proof are versions of the publication after peer review.
- The final published version features the final layout of the paper including the volume, issue and page numbers.

[Link to publication](#)

General rights

Copyright and moral rights for the publications made accessible in the public portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognise and abide by the legal requirements associated with these rights.

- Users may download and print one copy of any publication from the public portal for the purpose of private study or research.
- You may not further distribute the material or use it for any profit-making activity or commercial gain
- You may freely distribute the URL identifying the publication in the public portal.

If the publication is distributed under the terms of Article 25fa of the Dutch Copyright Act, indicated by the "Taverne" license above, please follow below link for the End User Agreement:

www.umlib.nl/taverne-license

Take down policy

If you believe that this document breaches copyright please contact us at:

repository@maastrichtuniversity.nl

providing details and we will investigate your claim.

Meeting risk with resilience: high daily life reward experience preserves mental health

Geschwind N, Peeters F, Jacobs N, Delespaul P, Derom C, Thiery E, van Os J, Wichers M. Meeting risk with resilience: high daily life reward experience preserves mental health.

Objective: To examine prospectively whether high reward experience (the ability to generate positive affect boosts from pleasurable daily events) protects against affective symptoms and whether environmental or genetic risk factors moderate protective effects.

Method: At baseline, 498 female twins participated in an experience sampling study measuring reward experience in daily life. They also completed questionnaires on childhood adversity and recent stressful life events (SLE). Affective symptoms were measured at baseline and at four follow-ups using SCL-90 anxiety and depression subscales. Co-twin affective symptoms were used as indicators of genetic risk.

Results: Baseline reward experience did not predict follow-up affective symptoms, regardless of level of genetic risk. However, high reward experience was associated with reduced future affective symptoms after previous exposure to childhood adversity or recent SLE.

Conclusion: High daily life reward experience increases resilience after environmental adversity; modification of reward experience may constitute a novel area of therapeutic intervention.

**N. Geschwind¹, F. Peeters¹,
N. Jacobs^{1,2}, P. Delespaul¹,
C. Derom³, E. Thiery⁴, J. van Os^{1,5},
M. Wichers¹**

¹Department of Neuropsychiatry and Psychology, South Limburg Mental Health Research and Teaching Network, EURON, Maastricht University, Maastricht, the Netherlands, ²Department of Clinical Psychology, Open University of the Netherlands, Heerlen, the Netherlands, ³Department of Human Genetics, University Hospital Gasthuisberg, Catholic University of Leuven, Leuven, Belgium, ⁴Association for Scientific Research in Multiple Births, Ghent, Belgium and ⁵Division of Psychological Medicine, Institute of Psychiatry, London, UK

Key words: affective symptoms; resilience; psychological; risk factors; longitudinal studies; twin study

Nicole Geschwind, Department of Neuropsychiatry and Psychology, Maastricht University, PO Box 616 (VIJV-SN2), 6200 MD Maastricht, the Netherlands.
E-mail: n.geschwind@sp.unimaas.nl

Accepted for publication December 2, 2009

Significant outcomes

- The ability to generate positive affect boosts (reward experience) from pleasant daily life events preserves mental health, but only in case of high childhood adversity or recent stressful life events.
- High daily life reward experience may thus represent a mechanism of resilience in people at risk for depression and related disorders.
- Novel treatments for the prevention of affective disorders should focus on enhancing daily life reward experience in people who experienced early or late environmental adversity. Discovering how to enhance reward experience remains a challenge for future research.

Limitations

- The observations of the experience sampling data rely on participants' compliance.
- Childhood trauma and recent stressful life events were measured retrospectively.
- Because participants were female only, the results may not be generalizable to men.

Introduction

'The foolish man seeks happiness in the distance, the wise grows it under his feet' (James Oppenheim). This quote refers to the ability to enjoy the moment instead of living in the future. Some people are able to generate an abundance of positive emotions from

pleasant everyday events like going to a birthday party, seeing an inspiring movie or eating tasty food. In other words, they have the ability to use pleasant events to boost their mood, at least temporarily, be it deliberate or non-deliberate. Does this ability make them more resilient in the long run? Here we examine prospectively whether the ability to gener-

ate boosts in positive affect (PA) in response to pleasurable daily life events is associated with more favourable mental health outcomes.

In general, PA is a strong candidate for resilience against stress-related disorders, because it reduces stress-induced psychiatric and physiological symptoms (1–5). In daily life, experiencing PA during moments of minor daily life stress is associated with lowered sensitivity to stress. In other words, the usual increase in negative affect (NA) (in reaction to stressful events) is less pronounced when people experience PA (6). Furthermore, Fredrickson and colleagues recently found that experiencing more PA predicted increased personal resources (like social support and purpose in life), which in turn predicted decreased affective symptoms (4). Similarly, a retrospective study showed that maintaining near-normal levels of positive emotions during the aftermath of the September 11 terrorist attacks was associated with less affective symptoms a couple of weeks later (7). These studies show that PA buffers the effects of stressful events.

Apart from that, PA also reduces the expression of genetic vulnerability for affective disorders. Individuals with genetic vulnerability for depression are more stress sensitive than individuals without genetic vulnerability (8). However, in situations of high PA, the difference in stress sensitivity between genetically vulnerable and non-vulnerable individuals is reduced (6). A study using direct genetic information found similar results: experiencing positive emotions neutralizes the difference in stress sensitivity caused by the BDNF val66met genotype (9).

Thus, the existing literature demonstrates that PA preserves, and possibly improves, mental health. Preserving mental health becomes particularly important in the presence of environmental or genetic risk. Childhood adversity (10, 11) as well as recent stressful life events (SLE) (12–14) are important environmental risk factors, because they increase daily life stress sensitivity as well as the risk for major depression and related disorders (15, 16).

Given the many benefits of PA, we expect that the ability to generate boosts in PA from pleasant daily life situations (hereafter: *high reward experience*) will contribute to the preservation of mental health. We predict that high reward experience, measured at baseline, will predict low affective symptoms at follow-up, especially when someone is at risk for low mood (due to either environmental adversity like childhood adversity or recent SLE or genetic risk), see Fig. 1. We tested these hypotheses in a prospective twin study, using the experience sampling method (ESM). ESM is optimally equipped to test these hypotheses, because it measures people's

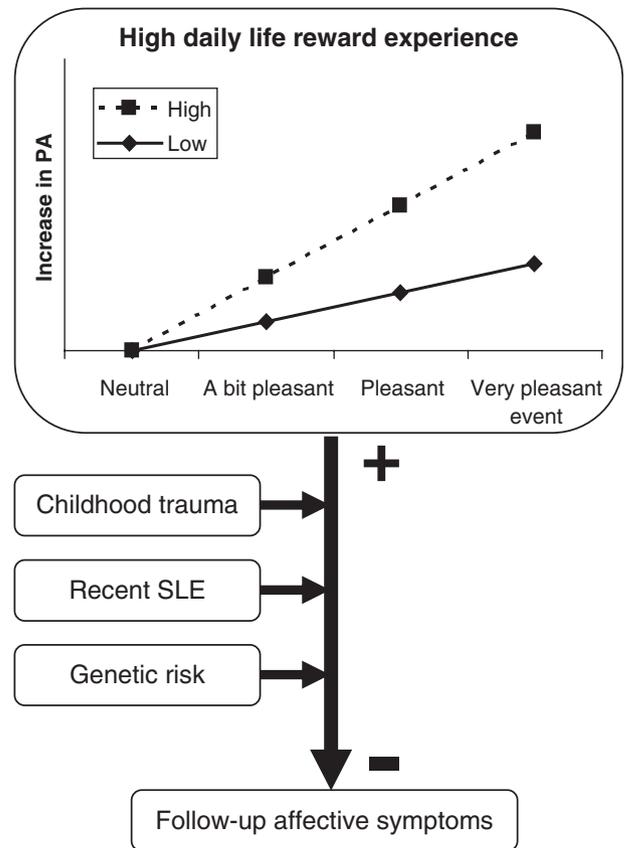


Fig. 1. Daily life reward experience and expected relations with follow-up affective symptoms. People with high reward experience are able to generate stronger positive affect (PA) boosts in response to pleasant events than people with low reward experience. Higher daily life reward experience is hypothesized to be associated with less follow-up affective symptoms, especially in people at risk for affective symptoms, i.e. those with higher levels of childhood trauma, recent stressful life events (SLE) and genetic risk.

experiences in their own daily life context, not only prospectively but also repetitively, catching variability in the flow of daily life. This type of measurement is necessary to reliably capture such a dynamic concept as daily life reward experience.

Aims of the study

The aims of our study are to examine prospectively whether high reward experience (the ability to generate positive affect boosts from pleasant events) contributes to resilience against affective disorders, and how this protective effect is moderated by environmental and genetic risk factors.

Material and methods

Sample

The study sample consisted of 621 female participants (age: 18–61) from Flanders (Belgium). The

participants were general population twin pairs and 46 of their non-twin sisters. Two-hundred and eighteen of these twin pairs were recruited from the East-Flanders Prospective Twin Survey (EFPTS), which has prospectively recorded all multiple births in the province of East-Flanders since 1964 (17, 18). Twin zygosity was determined at birth for EFPTS participants. For the other participants, zygosity was determined through a sequential analysis based on sex, foetal membranes, blood groups, DNA fingerprints, and interviews (19). The study was approved by the Ethics Committee of Maastricht University, and all participants provided written informed consent.

Experience sampling method

Experience sampling method is a structured diary technique to assess participants in their daily living environment. ESM has been validated for the use of studying the immediate effects of daily life situations on mood (20–23). Participants received a digital wristwatch and a set of ESM self-assessment forms collated in a booklet for each day. The wristwatch was programmed to emit a signal ('beep') at an unpredictable moment in each of ten 90-min time blocks between 7:30 and 22:30, on five consecutive days, resulting in a maximum of 50 beeps per person. After each beep, participants were asked to stop their activity and fill out the ESM self-assessment forms, with regard to thoughts, mood, current context (activity, persons present and location) and appraisals of current situation and, finally, the most important event since the last beep. All self-assessments were rated on 7-point Likert scales. Trained research assistants with ample experience in momentary assessment techniques explained the ESM procedure to the participants during an initial briefing session, and participants completed an ESM practice form to confirm that they understood the 7-point Likert scale. Participants could call a telephone number in case they had questions or problems during the ESM sampling period. Participants were told to complete their reports immediately after the beep, thus minimizing memory distortion, and to record the time at which they completed the form. In order to know whether the participants had completed the form within 15 min of the beep, the time at which participants indicated they completed the report was compared to the actual time of the beep. All reports not filled in within 15 min after the beep were excluded from the analysis, since previous work (22) has shown that reports completed after this interval are less reliable and consequently less valid. In addition,

previous work has shown that participants who have valid reports for at least one-third of all measurements can be included since their missing data does not distort the results, whereas measures of individuals with fewer than 30% of completed reports are less reliable (22). Therefore, participants with fewer than 17 valid reports (out of 50) were excluded from the analysis.

Design

Participants were assessed at baseline (ESM, childhood adversity, recent SLE and affective symptoms) and at four follow-up time points (affective symptoms). The average number of days between baseline and first follow-up was 132, between first and second follow-up 91, between second and third follow-up 116 and between third and fourth follow-up 91.

Measurements

Affective symptoms. Participants completed the Symptom Checklist (SCL-90R) (24, 25) at baseline and at four follow-up assessments. The SCL-90R is a comprehensive self-report symptom inventory, multidimensional in nature and oriented to screen for a broad range of psychological problems and psychopathology in community respondents. It contains 90 items, scored on a 5-point severity scale, measuring nine primary symptom dimensions named 'Somatization', 'Obsessive-Compulsive', 'Interpersonal Sensitivity', 'Depression', 'Anxiety', 'Hostility', 'Phobic Anxiety', 'Paranoid Ideation' and 'Psychoticism'.

Per participant, SCL-90R depression and anxiety scales were averaged for each measurement occasion, resulting in a combined score reflecting affective symptoms.

Childhood adversity. Childhood adversity was measured using the shortened version (26) of the 70-item Childhood Trauma Questionnaire [Jeugd Trauma Vragenlijst (Dutch version), A. Arntz and I. Wessel, unpublished data; 27]. At the request of the Twin Registry, the most explicit items concerning sexual and physical abuse were omitted; less explicit items were retained. The questionnaire thus consisted of 21 items with statements concerning early life experiences, such as 'I was abused', 'There was not enough food' and 'I was neglected'. Items were scored on a scale of 1 (never true) to 5 (very often true). Cronbach's alpha for this 21-item questionnaire was 0.93. The sum score of all items was used as a continuous measure of childhood adversity.

Recent stressful life events. Our inventory of recent SLE was based on the event list of the interview for recent life events (28). Participants reported whether any of 61 events happened in the past 6 months and indicated how unpleasant the experience was to them (from 1 = very pleasant to 5 = very unpleasant). SLE were divided into 10 categories: work, education, finance, health, bereavement, migration, courtship and cohabitation, legal, family and social relationships, and marital relationships, all representing datable occurrences involving changes in the external social environment. Only SLE rated as unpleasant (i.e. with a score of 4, 'unpleasant,' or a score of 5, 'very unpleasant') were included in the analysis. The number of such unpleasant events in the past 6 months was used as a continuous measure of recent SLE (19).

Genetic risk for affective symptoms. Genetic risk for affective symptoms was assessed indirectly using information on the affective health of the co-twin. The continuous variable 'co-twin affective symptoms' was constructed, representing baseline affective symptoms of the participant's co-twin. To obtain an indicator of genetic risk for depression, this variable was paired with information about twin zygosity (mono- or dizygotic).

Daily life events, affect and reward experience. Measures of daily life events and affect were extracted from the ESM framework, participants rated their current mood on 7-point Likert scales ranging from 0 (not at all) to 7 (very much). The mean of the PA items (I feel 'cheerful', 'content', 'energetic' and 'enthusiastic') formed the PA scale (Cronbach's alpha = 0.86 over the participant mean). Negative affect was assessed with six mood adjectives (I feel 'insecure', 'lonely', 'anxious', 'low', 'guilty' and 'suspicious') and had a Cronbach's alpha = 0.76 over the participant mean. After rating their affect, participants reported the most important event that happened between the current and the previous beep and rated that event on a 7-point bipolar scale (-3 = very unpleasant, 0 = neutral, 3 = very pleasant). Reward experience was conceptualized as the effect of pleasant daily life events (i.e. only those events rated as 0 (neutral) to 3 (very pleasant)) on PA (29). For each participant reporting at least four pleasant events, a reward experience variable was constructed by regressing beep-moment PA on the pleasantness score of the most recently reported daily life event (29). This variable thus reflects the extent to which the pleasantness of daily life events translates into increases in PA, see Fig. 1. Some

people, for example, experience just a small increase in PA after an event they rated as very pleasant, say a birthday party. Others experience a much larger increase in PA, suggesting that they can use the birthday party more effectively to generate positive emotions. Stress sensitivity was conceptualized likewise as NA reactivity to unpleasant daily life events, see (8).

Analysis

This study consisted of five measurement occasions. Most of the participants were twins. This means that the data were clustered: the five measurement occasions were clustered within participants, who were clustered in twin pairs. Clustering induces correlation in the data: measurements within participants are more alike than measurements between participants, and measurements within twin pairs are more alike than measurements between twin pairs. Appropriate analysis techniques are necessary to handle the correlations induced by the clustered data (30). We used the STATA XT MIXED multilevel linear regression command.

All analyses were corrected for baseline SCL-90R affective symptoms to ensure that associations between follow-up affective symptoms and baseline variables reflected effects on *change* in affective symptoms. All variables included in the analyses were standardized, yielding standardized effect sizes.

To examine our hypotheses, we ran the following regression analyses: the main effect of reward experience on follow-up affective symptoms was examined by regressing affective symptoms on reward experience. Regarding the hypothesis that risk factors moderate the effect of reward experience on affective symptoms, follow-up affective symptoms were regressed on the interaction between the risk factor and reward experience. This was done separately for each of the three risk factors (childhood adversity, recent SLE and genetic risk). For childhood adversity and recent SLE, this resulted in regressions with a two-way interaction (childhood adversity \times reward experience/recent SLE \times reward experience). Significant interactions were followed up by dose-response analyses. Dose-response relationships were examined by dividing the distribution of values for childhood adversity and recent SLEs by their tertiles using the STATA XTILE command. The STATA LINCOM command was used subsequently to calculate effect sizes of the differences between the low risk group and the medium or high risk group respectively. For genetic risk,

the regression contained a three-way interaction (co-twin affective symptoms \times zygosity \times reward experience). The STATA LINCOM command was used to calculate stratified effect sizes for mono- and dizygotic twins.

When risk factors were significantly associated with each other, the independence of their effects was checked by simultaneously entering their interactions (with reward experience) into the regression model.

Finally, the analyses were repeated to correct for a number of potential confounders. First, it is known that stress sensitivity influences risk for affective symptoms (8). To ensure that possible protective effects of high reward experience were not an artefact of low stress sensitivity, all analyses were repeated whilst correcting for stress sensitivity. Second, all analyses were repeated excluding the 20 participants who were depressed at baseline, to verify that the retrospective measures of childhood adversity and recent SLE were not contaminated by current depression status. Third, all analyses were re-run whilst additionally correcting for the number of positive events experienced by each participant and for the average PA per participant.

Results

Participant characteristics

Of the total sample of 621 participants, 610 participated in the ESM procedure. Thirty-one participants were excluded because they had fewer than 17 valid ESM self-reports. Another 81 participants were excluded due to missing data on either i) affective symptoms at all follow-ups, ii) baseline reward experience or iii) baseline affective symptoms. This resulted in a dataset of 498 participants that were part of 246 different twin pairs. Of the 498 participants, 286 were monozygotic twins, 170 were dizygotic twins, two were twins of unknown zygosity, and 40 were non-twin sisters of twins. For the analysis on genetic risk, non-twin sisters and twin-pairs of unknown zygosity were excluded. Of the remaining twins, 13 had missing data on co-twin affective symptoms, leaving 443 participants (280 monozygotic and 163 dizygotic twins) in the analyses involving genetic risk. Five of the 498 participants had missing data on childhood adversity, leaving 493 participants in the analyses involving childhood adversity. Mean age of the 498 participants was 28 years (SD: 7.9 years, range: 18–61). Sixty-five per cent had a college or university degree, 33% completed secondary education and 1% had

primary education only. The majority was currently employed (60% employed, 31% student, 2% unemployed, 3% homemaker and 1% sick leave). Ninety-seven per cent participated in at least two, 91% in at least three and 79% in all four follow-ups.

Multilevel analyses showed no significant association between reward experience on the one hand and genetic risk [$\beta = -0.15$, $\chi^2(1) = 2.84$, $P = 0.11$], childhood adversity ($\beta = 0.08$, $P = 0.09$) and recent SLE [$\beta = 0.03$, $\chi^2(1) = 0.50$, $P = 0.48$] on the other. Childhood adversity was significantly associated with later experience of SLE [$\beta = 0.21$, $\chi^2(1) = 40.38$, $P = 0.00$]. Therefore, the independence of their interaction terms was tested (see *Independence of moderation* later on). Other risk factors were not associated with each other. Mean score of affective symptoms was 1.47 (SD = 0.50) at baseline and 1.42 (SD = 0.48) at follow-up assessments. Mean score of childhood adversity was 1.64 (SD = 0.55), and mean score of recent SLE was 2.1 (SD = 2.3).

Associations between reward experience and future affective symptoms

Main effect. There was no overall association between reward experience and later affective symptoms [$\beta = 0.02$, $\chi^2(1) = 0.49$, $P = 0.49$].

Moderation by childhood adversity. The two-way interaction between childhood adversity and reward experience in the model of affective symptoms was significant [$\beta = -0.07$, $\chi^2(1) = 6.05$, $P = 0.01$]: after childhood adversity, high reward experience was associated with less future affective symptoms. A dose-response association was apparent in that the effect of reward experience on future affective symptoms became stronger with higher levels of childhood adversity [average compared to low childhood adversity: $\beta = -0.06$, $\chi^2(1) = 0.85$, $P = 0.36$; high compared to low childhood adversity: $\beta = -0.15$, $\chi^2(1) = 4.65$, $P = 0.03$]. For effect sizes of baseline daily reward experience on follow-up affective symptoms, stratified by degree of childhood adversity, see Fig. 2. Descriptive information on the average levels of affective symptoms for high and low levels of reward experience per tertile of childhood adversity is presented in Table 1.

Moderation by recent stressful life events. As hypothesized, the two-way interaction between recent SLE and reward experience was significant [$\beta = -0.08$, $\chi^2(1) = 4.65$, $P = 0.01$]. High reward experience was associated with less future affective

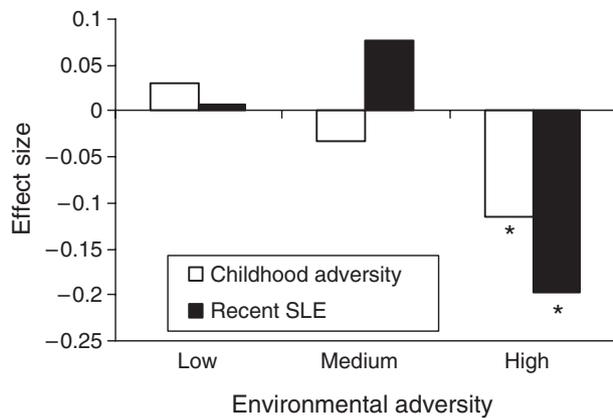


Fig. 2. Effect of reward experience on follow-up affective symptoms, stratified by level of environmental adversity (childhood adversity and recent SLE respectively). A negative effect size indicates a protective effect. Note: * represents a statistically significant difference compared to the low risk group.

Table 1. Means (SD) of affective symptoms by tertiles of childhood adversity and recent stressful life events, and high vs. low reward experience

	Affective symptoms	
	Low reward experience	High reward experience
Childhood adversity		
Low	1.25 (0.26)	1.30 (0.30)
Medium	1.44 (0.40)	1.36 (0.33)
High	1.62 (0.55)	1.54 (0.49)
Recent stressful life events		
Low	1.35 (0.34)	1.35 (0.36)
Medium	1.36 (0.34)	1.50 (0.47)
High	1.60 (0.57)	1.43 (0.37)

symptoms after recent SLE. There was no linear dose–response relationship: reward experience was only associated with future affective symptoms in participants with a high amount of recent SLE, compared to those with a low amount [$\beta = -0.21$, $\chi^2(1) = 7.99$, $P = 0.01$]. A moderate amount of recent SLE was not associated with an increase in the effect of reward experience on follow-up affective symptoms [$\beta = 0.07$, $\chi^2(1) = 0.63$, $P = 0.43$]. For effect sizes of baseline reward experience on follow-up affective symptoms, stratified by level of recent SLE, see Fig. 2. For descriptive information on the average levels of affective symptoms for high and low levels of reward experience per tertile of recent SLE, see Table 1.

Moderation by genetic risk. The three-way interaction between affective symptoms in the co-twin, zygosity and reward experience in the model of follow-up affective symptoms in the participant was not statistically significant [$\beta = 0.14$,

$\chi^2(1) = 0.90$, $P = 0.34$]. However, the two-way interaction between reward experience and co-twin affective symptoms on follow-up affective symptoms was larger in monozygotic twins ($\beta = -0.20$, $P = 0.03$) than in dizygotic twins (dizygotic twins: $\beta = -0.06$, $P = 0.61$). This means that the relationship between high reward experience and less future affective symptoms appears to be more pronounced in monozygotic twins with a symptomatic co-twin than in dizygotic twins with a symptomatic co-twin, suggesting genetic moderation. The difference between mono- and dizygotic twins, however, is not significant.

Independence of moderation by childhood adversity and recent stressful life events. To examine to what extent the significantly associated risk factors childhood adversity and recent SLE were independent, affective symptoms were regressed on both two-way interactions (childhood adversity \times reward experience, recent SLE \times reward experience) simultaneously. The effect size of childhood adversity \times reward experience was reduced by 24%, and the effect size of recent SLE \times reward experience was reduced by 38%. Both interaction terms were no longer significant.

Results of all analyses remained similar when additionally controlled for daily life stress sensitivity or when participants who were depressed at baseline ($n = 20$) were excluded from the analyses. Correcting for the average number of positive events per participant and for participants' average PA did not influence our results, either.

Discussion

Findings

We hypothesized that high daily life reward experience increases resilience against follow-up affective symptoms, and that this association is stronger in the presence of environmental adversity or genetic liability. In disagreement with the original hypothesis, there was no main protective effect of reward experience on follow-up affective symptoms. However, in line with our hypothesis, high reward experience was indeed associated with low follow-up affective symptoms in participants reporting childhood adversity or recent SLE. For childhood adversity, there was a dose–response association: with increasing levels of childhood adversity, the protective effect of high reward experience became more pronounced. For recent SLE, the protective effect of high reward experience was apparent only in the group reporting the

highest number of recent SLE. Reward experience thus becomes more important in the context of increased vulnerability. The fact that people who were not or minimally exposed to environmental adversity do not need extra protection against the development of affective symptoms makes sense, since they are not likely to develop symptoms in the first place.

The experience of childhood adversity increased the likelihood of recent SLE, which is a phenomenon known from other studies (31, 32). Although both childhood adversity and recent SLE did not contribute significantly after correcting one for the other, the effect of childhood adversity remained more stable than that of adult SLE. This suggests that early experiences may create an enduring liability that can be counteracted by resilience factors.

The three-way interaction reflecting our overall measure of genetic risk (*co-twin affective symptoms* × *zygosity* × *reward experience*) was not significant. This may reflect low statistical power, given the fact that three-way interactions are power consuming. Effect sizes of reward experience, however, were associated in a dose–response fashion with level of genetic risk: high reward experience protected stronger against follow-up affective symptoms in mono- than in dizygotic twins with high-symptomatic co-twins. This is in accordance with the finding above that especially those individuals at increased risk of developing symptoms benefit from the ability to effectively generate PA in daily life. Nevertheless, new studies with more statistical power are needed to assess whether high reward experience can indeed increase resilience for people with high genetic risk for low mood.

Additional analyses showed that the protective effects of high reward experience were independent of daily life stress sensitivity (negative affective reactivity to unpleasant daily life events), indicating that these represent two separate emotional pathways. Thus, reward experience, as shown previously (6) as well as in the current study, appears to be involved mainly in the development of resilience whereas stress sensitivity may play a role in the development of vulnerability (8, 16).

Furthermore, the results were unchanged by the exclusion of participants who were depressed at baseline, indicating that their depression status did not distort the results.

Results also remained similar to the original analyses when correcting for the amount of pleasant events per participant and average PA per participant, indicating that PA boosts from pleasant daily life events contribute to resilience independently from experiencing PA in general or from

encountering pleasant events. In other words, the increase in PA after pleasant events appears to be important apart from overall PA and apart from the mere frequency of pleasant events.

Taken together, our findings indicate that the ability to generate PA boosts from pleasant daily life events increases resilience against affective symptoms in people with a history of early or late environmental adversity.

Implications

Individual variation in the ability to generate PA boosts from pleasant daily events may explain significant variation in resilience against stress-related affective disorders. Our findings are in line with a recent study of Cohn and colleagues (33), who found that high levels of daily PA predicted increases in trait resilience. The study of PA is therefore worthy of greater attention, complementing the main focus on NA. Focussing on PA should lead to novel treatment approaches and urgently needed prevention strategies against increasing numbers of stress-related disorders like depression. A recent study showed that increases in reward experience predicted recovery from depression (34). This implies that the ability to generate PA boosts from pleasant daily life events can change. With regard to the timely question of which groups should be targeted for prevention (35), our study suggests that efforts to prevent affective disorders should be aimed at people exposed to environmental risk factors like childhood adversity or recent SLE.

Regarding our concept of reward experience, a number of open questions remain: First of all, because of a delay of, on average, 90 min between consecutive beeps, there can be relatively long time delays between the recorded pleasant event and participants' report of PA. Therefore, high reward experience in our study may also have been a question of being more capable of maintaining PA, once activated, rather than letting it dissipate to a minimal, base level. More research is needed in order to find out exactly what constitutes the protective component in reward experience – the ability to generate PA efficiently, or the ability to maintain it for longer. Furthermore, because current PA was rated at the same time as 'pleasantness of the most important event since the last beep', beep-moment mood may have influenced ratings of event pleasantness, rather than the pleasantness of the most important event influencing mood. Future research should try to disentangle these effects, maybe by separating those questions into different beeps. A third question is whether – and how – the

ability to experience reward from everyday events can be enhanced. Both pharmacological as well as non-pharmacological approaches are possible.

Pharmacological approaches to enhancing reward experience. Knowledge about the biological mechanisms of reward experience is important for discovering new ways to enhance resilience. Studies suggest that dopamine projections to the prefrontal cortex are involved in the conscious experience of reward (36–39). The COMT enzyme, which breaks down dopamine, is closely involved in dopamine regulation in the prefrontal cortex (40, 41). Lower levels of COMT lead to higher prefrontal dopamine levels, and should therefore be associated with a higher ability to experience reward.

Indeed, a recent experience sampling study found that daily life reward experience increased proportionally with the number of Met alleles on the COMT Val158Met polymorphism (29). The Met allele is associated with lower COMT activity, and, as a result, higher levels of prefrontal dopamine than the Val allele (42). Participants with the Met/Met genotype generated comparable levels of reward experience from events rated as ‘a bit pleasant’ as participants with the Val/Val genotype generated from events rated as ‘very pleasant’ (29). Similarly, participants with the Met/Met genotype had the highest prefrontal responses to anticipated and delivered rewards in a recent neuroimaging study (43), again indicating that individuals with Met allele experience more reward than individuals with the Val allele.

In sum, the available evidence suggests that biological agents acting on dopamine pathways might be successful in enhancing the ability to experience reward. Although many antidepressants target the dopaminergic system (44), it is still unclear to what extent antidepressants improve reward experience. One recent study using imipramine (34) suggests that response to antidepressants is mediated at least in part by changes in reward experience. However, further research is necessary to find out to what extent pharmacological interventions can improve reward experience.

Non-pharmacological approaches to enhancing reward experience. The fact that biological factors partially underlie reward experience does not exclude the possibility that non-pharmacological interventions are effective in modifying reward experience. For example, Goldapple and colleagues demonstrated that cognitive-behavioural therapy led to changes in cortical-limbic pathways in depressed patients (45), and Davidson and colleagues (46) found that an 8-week mindfulness meditation

program resulted in increases in activation in the left anterior hemisphere, which is associated with the experience of PA (47, 48). Furthermore, many patients prefer non-pharmacological treatment over taking medication (49).

Research suggests that meditation-based techniques, such as mindfulness-based cognitive therapy, might be an alternative way to enhance daily life reward experience. Participation in a 7-week meditation workshop led to significant increases in PA in a non-clinical sample (4). Furthermore, more advanced meditators experienced more PA than less advanced meditators (50), and participants reported more PA when in a mindful compared to a non-mindful state (51).

During mindfulness training sessions, participants are trained towards increased moment-to-moment awareness of experience, resulting in increased openness or receptiveness (52). An enhanced receptiveness might improve the ability to make use of natural daily life rewards. However, these suggestions remain speculative. Discovering to what extent – and how – daily life reward experience can be enhanced therefore remains a challenging task for future research.

Strengths

Strengths of the study include the use of the ESM to establish a measure of reward experience in daily life. In contrast to conventional questionnaires or interviews, ESM measurements are prospective, repeated and cover only a short and recent time span, thereby decreasing memory bias (22). Furthermore, ESM provides sensitive information on the dynamic relationship between the emotional responses of an individual and his or her daily life context.

A second strength is the study’s longitudinal design, with affective symptoms being measured at baseline and at four follow-up time points.

Finally, genetic risk was estimated sensitively by combining zygosity with a continuous measure of affective symptoms in the co-twin.

Limitations

First, it has been suggested that problems may arise in the ESM procedure, as it depends on participants’ compliance (53). In particular fixed time sampling protocols may be problematic and can bias results. However, this report did not use a fixed-time sampling frame, and our ESM procedure was validated in a previous report that used electronic monitoring devices. Results showed that compliance was high (over 90%) and inclusion of

the inaccurately timed reports did not distort the data (54).

Second, reward experience was assessed prospectively but contained a cross-sectional element as the relationship between PA and pleasantness of most important event (since the last beep) was assessed at each ESM moment. Therefore, it is possible that participants matched their recall of the event to their mood at the moment of the beep, rather than, pleasantness of the most important event influencing mood. However, either explanation bears clinical relevance, and the interpretation that pleasant events contribute to PA undoubtedly has face validity.

Third, childhood adversity and recent negative life events were measured retrospectively and through self-report. It is possible that those self-report measures were influenced by current mood state. However, all analyses were controlled for baseline affective symptoms, making it unlikely that results of the current study are the consequence of confounding by mood state. Moreover, the childhood trauma questionnaire has been found to have high test-retest reliability as well as good convergence with other (i.e. interview) forms of administration (27), and the interview for recent life events was designed specifically to collect datable occurrences involving changes in the external social environment rather than internal occurrences (28).

A fourth limitation is that the sample was female only. Consequently, our findings cannot be generalized to the male population. Furthermore, participants were mainly of middle age and, on average, higher educated than the general Belgium population.

Acknowledgements

This research was supported by the Netherlands Organisation for Scientific Research (VENI grant nr 916.76.147 to Dr Wichers) and by the Fund for Scientific Research, Flanders and Twins, a Belgian non-profit association for scientific research in multiple births (grant to the East Flanders Prospective Survey). We thank all twins for their cooperation.

Declaration of interests

None.

References

- DANNER DD, SNOWDON DA, FRIESEN WV. Positive emotions in early life and longevity: findings from the nun study. *J Pers Soc Psychol* 2001;**80**:804–813.
- FREDRICKSON BL. The role of positive emotions in positive psychology – the broaden-and-build theory of positive emotions. *Am Psychol* 2001;**56**:218–226.
- TUGADE MM, FREDRICKSON BL. Resilient individuals use positive emotions to bounce back from negative emotional experiences. *J Pers Soc Psychol* 2004;**86**:320–333.
- FREDRICKSON BL, COHN MA, COFFEY KA, PEK J, FINKEL SM. Open hearts build lives: positive emotions, induced through loving-kindness meditation, build consequential personal resources. *J Pers Soc Psychol* 2008;**95**:1045–1062.
- ZAUTRA AJ, JOHNSON LM, DAVIS MC. Positive affect as a source of resilience for women in chronic pain. *J Consult Clin Psychol* 2005;**73**:212–220.
- WICHERS MC, MYIN-GERMEYS I, JACOBS N et al. Evidence that moment-to-moment variation in positive emotions buffer genetic risk for depression: a momentary assessment twin study. *Acta Psychiatr Scand* 2007;**115**:451–457.
- FREDRICKSON BL, TUGADE MM, WAUGH CE, LARKIN GR. What good are positive emotions in crises? A prospective study of resilience and emotions following the terrorist attacks on the United States on September 11th, 2001. *J Pers Soc Psychol* 2003;**84**:365–376.
- WICHERS M, MYIN-GERMEYS I, JACOBS N et al. Genetic risk of depression and stress-induced negative affect in daily life. *Br J Psychiatry* 2007;**191**:218–223.
- WICHERS M, KENIS G, JACOBS N et al. The psychology of psychiatric genetics: evidence that positive emotions in females moderate genetic sensitivity to social stress associated with the BDNF Val(66)Met polymorphism. *J Abnorm Psychol* 2008;**117**:699–704.
- KENDLER KS, KUHN JW, PRESCOTT CA. Childhood sexual abuse, stressful life events and risk for major depression in women. *Psychol Med* 2004;**34**:1475–1482.
- AFIFI TO, BROWNRIDGE DA, COX BJ, SAREEN J. Physical punishment, childhood abuse and psychiatric disorders. *Child Abuse Negl* 2006;**30**:1093–1103.
- KESSLER RC. The effects of stressful life events on depression. *Annu Rev Psychol* 1997;**48**:191–214.
- HAMMEN C. Stress and depression. *Annu Rev Clin Psychol* 2005;**1**:293–319.
- VAN PRAAG HM, DE KLOET R, VAN OS J. Life events and depression: is there a causal connection. In: VAN PRAAG HM, DE KLOET R, VAN OS J, eds. *Stress, the brain, and depression*. Cambridge: Cambridge University Press, 2004:38–58.
- GLASER J-P, VAN OS J, PORTEGIJS PJM, MYIN-GERMEYS I. Childhood trauma and emotional reactivity to daily life stress in adult frequent attenders of general practitioners. *J Psychosom Res* 2006;**61**:229–236.
- WICHERS M, SCHRIJVERS D, GESCHWIND N et al. The mechanism of gene-environment interactions in depression: evidence that genes potentiate multiple sources of adversity. *Psychol Med* 2008;**6**:1–10.
- DEROM CA, VLIETINCK RF, THIERY EW, LEROY FOG, FRYNS JP, DEROM RM. The East Flanders Prospective Twin Survey (EFPTS). *Twin Res Hum Genet* 2006;**9**:733–738.
- LOOS R, DEROM C, VLIETINCK R, DEROM R. The East Flanders Prospective Twin Survey (Belgium): a population-based register. *Twin Res* 1998;**1**:167–175.
- JACOBS N, KENIS G, PEETERS F, DEROM C, VLIETINCK R, VAN OS J. Stress-related negative affectivity and genetically altered serotonin transporter function: evidence of synergism in shaping risk of depression. *Arch Gen Psychiatry* 2006;**63**:989–996.
- CSIKSZENTMIHALYI M, LARSON R. Validity and reliability of the experience sampling method. *J Nerv Ment Dis* 1987;**175**:526–536.
- DE VRIES M, editor. *The experience of psychopathology: investigating mental disorders in their natural settings*. Cambridge: Cambridge University Press, 1992.

22. DELESPAUL PAEG. Assessing schizophrenia in daily life. The experience sampling method. Maastricht: Maastricht University Press, 1995.
23. LATASTER T, WICHERS M, JACOBS N et al. Does reactivity to stress cosegregate with subclinical psychosis? A general population twin study *Acta Psychiatr Scand* 2009;**119**:45–53.
24. BECH P. The impact of the SCL-90 on the validity of the DSM-IV neurotic or stress-related disorders. *Acta Psychiatr Scand* 2004;**110**:161–162.
25. DEROGATIS LR. SCL-90-R: symptom checklist-90-R. Administration, scoring and procedures manual. Minneapolis, MN: National Computer Systems, 1994.
26. BERNSTEIN DP, AHLUVALIA T, POGGE D, HANDELSMAN L. Validity of the Childhood Trauma Questionnaire in an adolescent psychiatric population. *J Am Acad Child Adolesc Psychiatry* 1997;**36**:340–348.
27. BERNSTEIN DP, FINK L, HANDELSMAN L et al. Initial reliability and validity of a new retrospective measure of child-abuse and neglect. *Am J Psychiatry* 1994;**151**:1132–1136.
28. PAYKEL ES. The interview for recent life events. *Psychol Med* 1997;**27**:301–310.
29. WICHERS M, AGUILERA M, KENIS G et al. The catechol-O-methyl transferase Val(158)Met polymorphism and experience of reward in the flow of daily life. *Neuropsychopharmacology* 2008;**33**:3030–3036.
30. SNUJERS T, BOSKER R. Multilevel analysis: an introduction to basis and advanced multilevel modeling. London: SAGE publications Ltd, 1999.
31. KOENEN KC, MOFFITT TE, POULTON R, MARTIN J, CASPI A. Early childhood factors associated with the development of post-traumatic stress disorder: results from a longitudinal birth cohort. *Psychol Med* 2007;**37**:181–192.
32. LESERMAN J, LI ZM, HU YMJB, DROSSMAN DA. How multiple types of stressors impact on health. *Psychosom Med* 1998;**60**:175–181.
33. COHN MA, FREDRICKSON BL, BROWN SL, MIKELS JA, CONWAY AM. Happiness unpacked: positive emotions increase life satisfaction by building resilience. *Emotion* 2009;**9**:361–368.
34. WICHERS MC, BARGE-SCHAAPVELD DQCM, NICOLSON NA et al. Reduced stress-sensitivity or increased reward experience: the psychological mechanism of response to antidepressant medication. *Neuropsychopharmacology* 2009;**34**:923–931.
35. CUIJPERS P, SMIT F. Has time come for broad-scale dissemination for prevention of depressive disorders? *Acta Psychiatr Scand* 2008;**118**:419–420.
36. SCHULTZ W. Getting formal with dopamine and reward. *Neuron* 2002;**36**:241–263.
37. KRINGELBACH ML. The human orbitofrontal cortex: linking reward to hedonic experience. *Nat Rev Neurosci* 2005;**6**:691–702.
38. GALVAN A, HARE TA, DAVIDSON M, SPICER J, GLOVER G, CASEY BJ. The role of ventral frontostriatal circuitry in reward-based learning in humans. *J Neurosci* 2005;**25**:8650–8656.
39. MALHI GS, BERK M. Does dopamine dysfunction drive depression? *Acta Psychiatr Scand* 2007;**115**:116–124.
40. BILDER RM, VOLAVKA J, LACHMAN HM, GRACE AA. The catechol-O-methyltransferase polymorphism: relations to the tonic-phasic dopamine hypothesis and neuropsychiatric phenotypes. *Neuropsychopharmacology* 2004;**29**:1943–1961.
41. CRADDOCK N, OWEN MJ, O'DONOVAN MC. The catechol-O-methyl transferase (COMT) gene as a candidate for psychiatric phenotypes: evidence and lessons. *Mol Psychiatry* 2006;**11**:446–458.
42. EGAN M, GOLDMAN D, WEINBERGER D. The human genome. *Am J Psychiatry* 2002;**159**:12.
43. DREHER JC, KOHN P, KOLACHANA B, WEINBERGER DR, BERMAN KF. Variation in dopamine genes influences responsivity of the human reward system. *Proc Natl Acad Sci USA* 2009;**106**:617–622.
44. MILLAN MJ. Dual- and triple-acting agents for treating core and co-morbid symptoms of major depression: novel concepts, new drugs. *Neurotherapeutics* 2009;**6**:53–77.
45. GOLDAPPLE K, SEGAL Z, GARSON C et al. Modulation of cortical-limbic pathways in major depression. Treatment-specific effects of cognitive behavior therapy. *Arch Gen Psychiatry* 2004;**61**:34–41.
46. DAVIDSON RJ, KABAT-ZINN J, SCHUMACHER J et al. Alterations in brain and immune function produced by mindfulness meditation. *Psychosom Med* 2003;**65**:564–570.
47. DAVIDSON RJ. Emotion and affective style: hemispheric substrates. *Psychol Sci* 1992;**3**:39–43.
48. DAVIDSON RJ, FOX NA. Asymmetrical brain activity discriminates between positive and negative affective stimuli in human infants. *Science* 1982;**218**:1235–1237.
49. VAN SCHAİK DJF, KLJN AFJ, VAN HOUT HPJ et al. Patients' preferences in the treatment of depressive disorder in primary care. *Gen Hosp Psychiatry* 2004;**26**:184–189.
50. EASTERLIN B, CARDENA E. Cognitive and emotional differences between short and long term Vipassana meditators. *Imagn Cogn Pers* 1999;**18**:69–81.
51. BROWN KW, RYAN RM. The benefits of being present: mindfulness and its role in psychological well-being. *J Pers Soc Psychol* 2003;**84**:822–848.
52. BAER RA. Mindfulness training as a clinical intervention: a conceptual and empirical review. *Clin Psychol Sci Pract* 2003;**10**:125–143.
53. KUDIELKA BM, BRODERICK JE, KIRSCHBAUM C. Compliance with saliva sampling protocols: electronic monitoring reveals invalid cortisol daytime profiles in noncompliant subjects. *Psychosom Med* 2003;**65**:313–319.
54. JACOBS N, NICOLSON NA, DEROM C, DELESPAUL P, VAN OS J, MYIN-GERMEYS I. Electronic monitoring of salivary cortisol sampling compliance in daily life. *Life Sci* 2005;**76**:2431–2443.