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The Role of Childhood Trauma in Affective Stress Recovery in Early Psychosis: An Experience Sampling Study

Leonie Ader[1,], Anita Schick[1,], Thomas Vaessen[2–4], GROUP investigators[14], Craig Morgan[5,6], Matthew J. Kempton[7], Lucia Valmaggia[8–10], Philip McGuire[7,11], Inez Myin-Germeys[2], Ginette Lafit[2,12], and Ulrich Reininghaus[*,1,6,13]

[1]Department of Public Mental Health, Central Institute of Mental Health, Medical Faculty Mannheim, Heidelberg University, Mannheim, Germany; [2]Department of Neurosciences, Center for Contextual Psychiatry, KU Leuven, Leuven, Belgium; [3]Department of Psychology, Health & Technology, Faculty of Behavioural, Management and Social Sciences (BMS), University of Twente, Enschede, The Netherlands; [4]Department of Neurosciences, Mind Body Research, KU Leuven, Leuven, Belgium; [5]Health Service and Population Research Department, Institute of Psychiatry, Psychology and Neuroscience, King's College London, London, UK; [6]ESRC Centre for Society and Mental Health, King's College London, London, UK; [7]Department of Psychosis Studies, Institute of Psychiatry, Psychology & Neuroscience, King's College London, London, UK; [8]Department of Psychology, Institute of Psychiatry, Psychology & Neuroscience, King's College London, London, UK; [9]South London and Maudsley NHS Foundation Trust, London, UK; [10]Department of Psychiatry, KU Leuven, Leuven, Belgium; [11]Department of Psychiatry, University of Oxford, Warneford Hospital, Oxford, UK; [12]Research Group of Quantitative Psychology and Individual Differences, Faculty of Psychology, KU Leuven, Leuven, Belgium; [13]Centre for Epidemiology and Public Health, Health Service and Population Research Department, Institute of Psychiatry, Psychology & Neuroscience, King's College London, London, UK

[14]See list of GROUP investigators before references.

[*]To whom correspondence should be addressed; Department of Public Mental Health, Central Institute of Mental Health, Medical Faculty Mannheim, Heidelberg University, J5, 68159 Mannheim, Germany; tel: +49-621-1703-1930, fax: +49-621-1703-801930, e-mail: ulrich.reininghaus@zi-mannheim.de

***Background and Hypotheses*: Affective recovery, operationalized as the time needed for affect to return to baseline levels after daily stressors, may be a putative momentary representation of resilience. This study aimed to investigate affective recovery in positive and negative affect across subclinical and clinical stages of psychosis and whether this is associated with exposure to childhood trauma (sexual, physical, and emotional abuse). *Study Design*: We used survival analysis to predict the time-to-recovery from a daily event-related stressor in a pooled sample of 3 previously conducted experience sampling studies including 113 individuals with first-episode psychosis, 162 at-risk individuals, and 94 controls. *Study Results*: Negative affective recovery (ie, return to baseline following an increase in negative affect) was longer in individuals with first-episode psychosis compared with controls (hazard ratio [HR] = 1.71, 95% confidence interval [CI; 1.03, 2.61], *P* = .04) and in at-risk individuals exposed to high vs low levels of emotional abuse (HR = 1.31, 95% CI [1.06, 1.62], *P* = .01). Positive affective recovery (ie, return to baseline following a decrease in positive affect) did not differ between groups and was not associated with childhood trauma. *Conclusions*: Our results give first indications that negative affective recovery may be a putative momentary representation of resilience across stages of psychosis and may be amplified in at-risk individuals with prior experiences of emotional abuse. Understanding how affective recovery contributes to the development of psychosis may help identify new targets for prevention and intervention to buffer risk or foster resilience in daily life.**

Key words: experience sampling methodology (ESM)/ecological momentary assessment (EMA)/clinical staging/at-risk mental state/stress reactivity/mental health

Introduction

Momentary psychological mechanisms in daily life can enhance our understanding and inform treatment of mental disorders.[1–3] Especially in psychosis, the response to daily stressors,[4–8] but also the recovery from a stressor,[9,10] may vary across clinical stages.

Behavioral sensitization is a concept that links the response to major stressors occurring over the life course (eg, childhood trauma) to the response to minor stressors in daily life (eg, missing the bus).[11] Accordingly, exposure to severe or repeated adversity over the life course, ie, on the macro-level, is posited to gradually increase the amplitude of the stress response. Consequently, the response

to even minor daily stressors, ie, on the micro-level is amplified, which in turn, is deemed to increase the risk of transitioning to mental disorder.[11] In addition, this putative mechanism may extend to the time-to-recovery following the initial response to a stressor.

Following the clinical staging model that emerged from the early intervention field, individuals are assumed to experience a risk state before meeting diagnostic criteria for full-threshold disorder.[12–16] Experience sampling studies have used stress reactivity, operationalized as the association between momentary stress and affect, as a marker for behavioral sensitization to observe the process across stages of psychosis. Stress reactivity has been found to be elevated in patients with psychosis[4,6] and individuals at risk for psychosis compared with controls,[6,7] suggesting the interplay between stress and affect may be particularly relevant to early psychosis phenotypes. Less attention has been payed to the temporal trajectory of affect after the initial stress response compared across stages of psychosis.

To investigate stress reactivity as a marker for behavioral sensitization further, the role of childhood trauma as a major stressor in life associated with risk for developing mental disorders,[17,18] including psychosis,[19–23] has been taken into account in experience sampling studies. Indeed, stress reactivity has been found to be higher in patients and at-risk individuals with high compared with low overall scores of a Childhood Trauma Questionnaire (CTQ).[2,24–26] This may suggest that having experienced childhood trauma may continue to impact risk, course, and outcome via its association with the momentary stress response. However, in controls, differences in stress reactivity between individuals with high and low levels of childhood trauma were less pronounced, suggesting these individuals may be more resilient.[2,25] In fact, focusing not only on risk, but also on protective factors that make individuals more resilient against developing psychosis has become increasingly relevant, not least to identify targets for enhancing prevention and early treatment intervention.[2,25]

Across different models, resilience is defined as positive outcome or adaptation in the face of risk or adversity.[27–29] In process-based definitions, positive adaptation can be defined as individual regaining of a prior level of functioning.[29] In addition, there have been efforts to integrate the stress and coping approach and the emotion and emotion-regulation approach to resilience by acknowledging that both coping and emotion regulation are types of affect regulation.[30] In line with this, positive affect has been suggested to contribute to psychological resilience by helping to overcome adversity.[28,31] We have recently started to examine whether this conceptualization can be transferred to the realm of daily life by investigating trajectories of positive affect in response to daily stressors, ie, positive affective recovery.[10] In a transdiagnostic sample, we found indications that individuals with a psychotic or depressive disorder and individuals at risk for those disorders may take longer to recover from a daily stressor than controls. Another study suggests that individuals in early stages of psychosis require more time than controls and than individuals with a chronic course of psychosis to recover to baseline values of negative affect.[9] Early stages of psychosis included at-risk status and first-episode psychosis,[9] but group differences between these groups were not investigated. Using survival analysis, another study found that recovery to baseline levels of negative affect after stressful events was slower in individuals with increased risk for depression.[32] Taken together, these findings showing differences in affective recovery across clinical stages, may suggest that it may, indeed, be a momentary micro-level representation of resilience, which has been so far primarily investigated in relation to significant adversity, at the macro-level. As childhood trauma reflects an established risk for developing psychosis and other disorders, it may also be important to understand its association with affective recovery as a mechanism through which it may continue to impact risk, course, and outcome.

Therefore, to disentangle further at the micro-level, whether affective recovery from minor stressors reflects momentary resilience in early psychosis it may be important to consider the role of childhood trauma and how this may modify this putative protective mechanism. Specifically, exposure to childhood trauma may prolong momentary stress recovery in at-risk individuals or patients, whereas controls exposed to childhood trauma may be more resilient and recover more readily from minor stressors, but this needs to be directly tested in intensive longitudinal experience sampling research.

Therefore, the aim of this study was (1) to investigate group differences in affective recovery from minor event-related stressors in daily life along the continuum of mental ill-health in patients with first-episode psychosis, individuals at psychometric risk for psychosis, and controls without a personal or family history of psychosis and (2) whether previous research into effect modification of stress reactivity by childhood trauma[2,24–26] extends to time-to-recovery. Specifically, we aimed to test the following hypotheses:

H1: The time-to-recovery, ie, return to baseline level negative or positive affect following an event-related stressor, will be (1) longer in at-risk individuals than in controls, (2) longer in patients than in controls, and (3) longer in patients than in at-risk individuals.

H2: Within the at-risk and patient group, the time-to-recovery from event-related stress (as a putative protective mechanism) will be associated with childhood trauma, ie, (1) will be longer in at-risk individuals exposed to high vs low levels of childhood trauma, (2) will be longer in patients exposed to high vs low levels of childhood trauma, but (3) will not differ in controls exposed to high vs low levels of childhood trauma representing resilience.

H3: The difference in the time-to-recovery in individuals exposed to high vs low levels of childhood trauma varies across groups, ie, will be (1) smaller in controls than in patients, (2) smaller in controls than in at-risk individuals, and (3) smaller in at-risk individuals than in patients.

Methods

Participants

Data from 3 previously conducted studies were pooled to form 1 dataset. A detailed description of the samples and procedures can be found in Multimedia Appendix, table 1. Participants from 3 groups were included, ie, patients with first-episode psychosis, individuals at psychometric risk for developing psychosis, and controls without a personal or family history of psychosis. All studies received approval by their respective medical ethics committees. The current analysis was preregistered on the Open Science Framework (osf.io/jszf5).

Experience Sampling Method

Participants took part in a 6-day study using the experience sampling method (ESM), for which feasibility and validity has been established in individuals with psychotic disorder.[33] Participants received a study smartphone or a PsyMate device. All studies used a blocked-random sampling scheme with 10 assessments per day scheduled at semi-random time points within set blocks of time between 7:30 AM and 10:30 PM. Thus, time points were approximately 90 minutes apart. Experience sampling data collection included ESM measures of negative and positive affect and event stress. A detailed description of the ESM measures is provided in table 1.

Childhood Trauma

Data collection included 3 subscales of the CTQ in all studies to assess experiences of sexual, physical, and emotional abuse. In total, the CTQ consists of 28 self-report items divided into 5 subscales (sexual, physical, and emotional abuse, physical and emotional neglect). All items are answered on a 5-point Likert scale (1 = never true; 5 = very often true). Psychometric properties have been demonstrated to be sufficient.[35,36] Mean scores of the 5 items per subscale were calculated and used in the analysis.

Statistical Analysis

R version 4.1.2 was used for data preparation and statistical analysis.[37] Descriptive statistics were calculated for all variables. Group differences of time-invariant variables were investigated using chi-square tests, ANOVAs and pairwise comparisons as appropriate, while group differences of time-varying variables were investigated using mixed models to account for the multilevel structure of the ESM data (observations nested within individuals).

To prepare the data for survival analysis, the intervals between the occurrence of a stressor (t_0) and recovery were determined. We identified for each day per individual

Table 1. ESM Measures and Operationalizations

Domain	ESM Measure
Negative affect	Momentary negative affect was operationalized as the mean score of 4 items (I feel insecure/lonely/anxious/down) answered on a 7-point Likert scale (1 = not at all; 7 = very much). Person-mean item scores provided an internal consistency of Cronbach's α = 0.93 (between-person reliability), while person-mean centered items provided an internal consistency of Cronbach's α = 0.70 (within-person reliability).[a]
Positive affect	At each ESM assessment, positive affect was operationalized as the mean score of 3 items (I feel cheerful/relaxed/ satisfied) answered on a 7-point Likert scale (1 = not at all; 7 = very much). Person-mean item scores provided an internal consistency of Cronbach's α = 0.89 (between-person reliability), while person-mean centered items provided an internal consistency of Cronbach's α = 0.67 (within-person reliability).[a]
Baseline	Baseline affect (PA_{-1} and NA_{-1}) was operationalized as the level of negative and positive affect at the time point before a stressful event was reported (t_{-1}).[9,32]
Recovery	Recovery was defined as the moment, at which negative or positive affect reached at least the baseline level, ie, $NA \leq NA_{-1}$ and $PA \geq PA_{-1}$.
Stress intensity	Momentary event-related stress was measured by asking about the most important event for the participant that happened since the last assessment, followed by the question how pleasant this event was on a bipolar scale (−3 = very unpleasant; 3 = very pleasant), which has been found to be a common operationalization.[34] Positive values (ie, pleasant event) were set to zero and negative values were reverse coded to represent event stress. Therefore, stress intensity had values between 0 and 3 with higher values representing more intense stress. For the recovery analysis, the time point of the first stressful event of a day (stress intensity >0) that was not the first prompt of the day, as there is no baseline measure available, was defined as the stress prompt t_0.
Cumulative stress	Cumulative stress was computed as any additional stressor reported on the same day during the recovery period, ie, between t_1 and recovery, with binary coding for absence (= 0) or presence (= 1) of subsequent stressors.

Note: ESM, experience sampling method.
[a]For internal consistency separated by group, see Multimedia Appendix, table 2.

the time point at which a stressful event occurred. Days without stress or without a baseline measurement (ie, time point before a stressful event was reported t_{-1}), and days with all assessments missing after t_0 were excluded. Recovery was determined as the moment at which affect had at least reached the level of t_{-1} following an increase in negative affect or a decrease in positive affect. Data were treated as right-censored when recovery was not observed. More details on data preparation and operationalizations can be found in table 1 and in a previous study.[32] Both trajectories of negative and positive affect in relation to a stressor were used to allow for comparisons with previous findings on negative affective recovery[32] in a different population and between negative and positive affective recovery within one population.

To test the hypotheses, parametric survival models (ie, Weibull regression models for time interval-censored data) were computed.[38] We used these models to predict the time-to-recovery. Time-to-recovery was modeled as the probability of non-recovery, which technically reflects "survival" (ie, the event "recovery" did not occur). Therefore, hazard ratios (HRs) >1 and positive *b*-coefficients reflect an increase and a higher probability of *not* returning to baseline levels of affect at any time point within the same day, meaning that it takes longer to recover. To test H1, time-to-recovery was predicted by group status (patient vs control and at-risk vs control). To test H2, for each group separately, each childhood trauma variable (sexual, physical, and emotional abuse) was used as a predictor for time-to-recovery in a separate model (resulting in 9 models). Lastly, for each childhood trauma variable, interaction terms between group and childhood trauma (childhood trauma × patient vs childhood trauma × control and childhood trauma × at-risk vs childhood trauma × control) were included into between-group analysis to test H3 (resulting in 3 models). All models were adjusted for age, gender (0 = male, 1 = female), ethnicity (0 = white; 1 = other ethnicity), stress intensity, and cumulative stress (see table 1).

In response to a unique pattern of deviance residuals (ie, a diagnostic measure of goodness-of-fit) that was observed in diagnostic plots for participants above the age of 40 in the model predicting time-to-recovery by group status in both negative and positive affect (see Multimedia Appendix, figures 1 and 2), we decided to exclude participants above the age of 40 (n = 79) from the analytic sample. Basic sample characteristics and all models using the full sample as specified in the preregistration are reported in Multimedia Appendix, tables 3–6.

Results

Descriptive Statistics

The analytic sample comprised 369 participants aged 14–40 years. This includes 113 patients with first-episode psychosis, 162 individuals at risk for psychosis, and 94 controls. As can be seen in table 2, the groups differed significantly in gender and ethnicity. Furthermore, the at-risk group was the youngest group, while the control group was the oldest (F(2, 115.03) = 1847.50, P < .001). The at-risk group had the highest momentary negative affect overall (F(2, 363.98) = 52.49, P < .001), at baseline (F(2, 235.56) = 26.39, P < .001), and at the stress prompt t_0 (F(2, 235.59) = 32.19, P < .001), while controls had the lowest. Moreover, controls had the highest momentary positive affect overall (F(2, 362.81) = 43.74, P < .001), at baseline (F(2, 202.26) = 22.13, P < .001), and at the stress prompt (F(2, 209.11) = 24.16, P < .001), while the at-risk group had the lowest (see table 2). Patients had the highest exposure to all types of abuse (all P < .001), while controls had the lowest.

Group Differences in Time-to-Recovery (H1)

When looking at trajectories of negative affect, being in the patient group, but not in the at-risk group, had an effect on the probability not to recover at any time point (HR = 1.71, 95% confidence interval [CI; 1.03, 2.61], P = .04) reflecting "survival" in the model, ie, the event "recovery" did not occur (see table 3). This indicated that the patient group required more time to recover compared with the control group. Participants in this group were 1.71 times more likely not to have recovered at any time point than participants in the control group. Therefore, we found evidence supporting H1.1. Additionally, stress intensity (HR = 1.22, 95% CI [1.03, 1.49], P = .03) and cumulative stress (HR = 8.68, 95% CI [6.49, 12.81], P < .001) were associated with longer negative affective recovery (see table 3). This showed that a more pronounced unpleasantness of the reported event increased the time-to-recovery. It also required more time to recover when individuals experienced at least one more stressor during the recovery period compared with only the initial stressor. Estimates in minutes for 10th, 50th, and 90th percentiles of the time-to-recovery by group can be found in Multimedia Appendix, table 7.

There was no evidence for group differences in time-to-recovery in trajectories of positive affect (see table 3). Again, stress intensity (HR = 1.17, 95% CI [1.05, 1.45], P = .01) and cumulative stress (HR = 5.59, 95% CI [4.06, 7.61], P < .001) were predictors for longer positive affective recovery.

Association Between Childhood Trauma and Time-to-Recovery Within Each Group (Patients, At-Risk Individuals, Controls) (H2)

In trajectories of negative affect, emotional abuse had an effect on time-to-recovery within the at-risk group (HR = 1.31, 95% CI [1.06, 1.62], P = .01) (see table 4), supporting H2.1. There was some evidence for physical abuse being associated with time-to-recovery in the

Table 2. Basic Sample Characteristics and Group Differences in the Analytic Sample Including Only Participants Younger Than 40 Years of Age Due to a Unique Pattern of Deviance Residuals

Measure	Patients (Pat) (n = 113)	At-Risk Individuals (Atr) (n = 162)	Controls (Con) (n = 94)	Total (n = 369)	Test Statistic	P Value	Significant Contrasts
Age, M (SD)	25.19 (5.14)	23.48 (4.92)	28.16 (5.47)	25.20 (5.46)	$F(2, 115.03) = 1847.50$	<.001	Pat vs Con; Atr vs Con; Pat vs Atr
Gender, N					$\chi^2(2) = 9.11$	.01	
Female	49 (43%)	91 (56%)	60 (64%)	200 (54%)			
Ethnicity, N					$\chi^2(2) = 7.12$	.03	
White	67 (59%)	114 (70%)	71 (76%)	252 (68%)			
Other ethnicity	46 (41%)	48 (30%)	23 (24%)	117 (32%)			
Childhood trauma, M (SD)							
Physical abuse	1.46 (0.71)	1.55 (0.78)	1.16 (0.45)	1.42 (0.71)	$F(2, 12.70) = 846.70$	<.001	Pat vs Con; Atr vs Con; Pat vs Atr
Emotional abuse	2.04 (1.80)	2.46 (1.07)	1.50 (0.66)	2.09 (1.03)	$F(2, 9.12) = 1629.20$	<.001	Pat vs Con; Atr vs Con; Pat vs Atr
Sexual abuse	1.35 (0.78)	1.43 (0.91)	1.21 (0.62)	1.35 (0.81)	$F(2, 1762.80) = 131.93$	<.001	Pat vs Con; Atr vs Con; Pat vs Atr
Experience sampling compliance, M (SD)	0.40 (0.25)	0.49 (0.23)	0.66 (0.20)	0.51 (0.25)	$F(2, 366) = 33.68$	<.001	Pat vs Con; Atr vs Con; Pat vs Atr
Momentary affect overall							
Negative affect (NA), M (SD)	2.58 (1.19)	2.98 (1.11)	1.65 (0.63)	2.52 (1.17)	$F(2, 363.98) = 52.49$	<.001	Pat vs Con; Atr vs Con; Pat vs Atr
Positive affect (PA), M (SD)	4.17 (1.42)	3.71 (1.38)	4.69 (1.15)	4.14 (1.39)	$F(2, 362.81) = 43.74$	<.001	Pat vs Con; Atr vs Con; Pat vs Atr
Observations with stress, N	110	215	158	483			
Measures used in survival models[a]							
NA at baseline t_{-1}	2.80 (1.56)	2.87 (1.40)	1.78 (0.92)	2.49 (1.40)	$F(2, 235.56) = 26.39$	<.001	Atr vs Con; Pat vs Atr
PA at baseline t_{-1}	4.05 (1.37)	3.60 (1.17)	4.58 (1.17)	4.03 (1.29)	$F(2, 202.26) = 22.13$	<.001	Pat vs Con; Atr vs Con; Pat vs Atr
NA at stress t_0	3.22 (1.67)	3.29 (1.49)	1.99 (1.01)	2.48 (1.52)	$F(2, 235.59) = 32.19$	<.001	Atr vs Con; Pat vs Atr
PA at stress t_0	3.38 (1.41)	3.14 (1.23)	4.17 (1.19)	3.63 (1.34)	$F(2, 209.11) = 24.16$	<.001	Atr vs Con; Pat vs Atr
Stress intensity	1.80 (0.82)	1.75 (0.80)	1.57 (0.73)	1.70 (0.79)	$F(2, 226.76) = 2.21$	.11	
Cumulative stress for PA recovery[b]	0.20 (0.40)	0.28 (0.45)	0.22 (0.41)	0.24 (0.43)	$F = 1.71$		None
Cumulative stress for NA recovery[c]	0.22 (0.41=	0.24 (0.43)	0.16 (0.37)	0.21 (0.41)	$F = 1.93$		None

[a]Descriptive statistics computed after data preparation for survival analysis, therefore cases were missing ($n_{patients}$ = 54, $n_{at\text{-}risk}$ = 56, $n_{controls}$ = 15).
[b]To test for group differences, a generalized linear mixed-effects model was used. All pairwise comparisons were nonsignificant (Ps > .28).
[c]To test for group differences, a generalized linear mixed-effects model was used. All pairwise comparisons were nonsignificant (Ps > .13).

at-risk group (HR = 1.35, 95% CI [0.98, 1.86], P = .07). In the patient group and in the control group, there was no significant effect of any type of childhood trauma on the time-to-recovery (see table 4). The effect of cumulative stress was significant in all models (all HRs ≥ 7.60, Ps < .001), while stress intensity was associated with time-to-recovery in the control group (all HRs ≥ 1.38, Ps < .05) (see Multimedia Appendix, table 6).

In trajectories of positive affect, there was no effect of childhood trauma on time-to-recovery in any model using emotional, physical, and sexual abuse as a predictor within each group. Therefore, for positive affect, there was no evidence that childhood trauma modified the time-to-recovery in any group. Cumulative stress was a predictor in each model (all HRs ≥ 4.59, Ps < 0.001). In the patient group, the effect of stress intensity was significant in the model testing emotional abuse (HR = 1.42, 95% CI [1.02, 1.99], P = .04) (see Multimedia Appendix, table 7).

Childhood Trauma and Time-to-Recovery by Group (Patients, At-Risk Individuals, Controls) (H3)

There were no interaction effects for childhood trauma × group on time-to-recovery in trajectories of negative and positive affect (see table 5). Therefore, there was no evidence that the difference in the time-to-recovery

Table 3. Weibull Survival Models for Group Differences in Time-to-Recovery in the Analytic Sample

	B (95% CI)	SE	*z*	*P* Value	Hazard Ratios (95% CI)
Recovery in trajectories of negative affect					
Intercept	3.59 (2.59; 4.60)	0.51	7.02	<.001	
Age	0.01 (−0.02; 0.04)	0.02	0.77	.44	0.99 (0.98; 1.04)
Gender	0.15 (−0.17; 0.46)	0.16	0.93	.36	1.21 (0.84; 1.58)
Ethnicity	0.10 (−0.25; 0.46)	0.18	0.58	.56	1.07 (0.78; 1.58)
Stress intensity	0.21 (0.03; 0.40)	0.10	2.23	.03	1.22 (1.03; 1.49)
Cumulative stress	2.21 (1.87; 2.55)	0.17	12.70	<.001	8.68 (6.49; 12.81)
Group status					
At-risk	0.29 (−0.08; 0.67)	0.19	1.54	.12	1.31 (0.92; 1.95)
Patient	0.49 (0.03; 0.96)	0.24	2.08	.04	1.71 (1.03; 2.61)
Log (scale)	0.21	0.05	4.48	<.001	
Recovery in trajectories of positive affect					
Intercept	4.04 (3.19; 4.89)	0.44	9.30	<.001	
Age	0.02 (−0.01; 0.05)	0.02	1.53	.13	1.00 (0.99; 1.05)
Gender	−0.03 (−0.30; 0.25)	0.14	−0.20	.85	1.03 (0.74; 1.28)
Ethnicity	−0.08 (−0.37; 0.21)	0.15	−0.56	.58	0.89 (0.69; 1.23)
Stress intensity	0.21 (0.05; 0.37)	0.08	2.53	.01	1.17 (1.05; 1.45)
Cumulative stress	1.72 (1.40; 2.03)	0.16	10.73	<.001	5.59 (4.06; 7.61)
Group status					
At-risk	−0.13 (−0.42; 0.17)	0.15	−0.84	.40	0.84 (0.66; 1.19)
Patient	−0.32 (−0.69; 0.05)	0.19	−1.68	.09	0.75 (0.50; 1.05)
Log (scale)	0.13	0.05	2.53	.01	

Note: Effects of female gender, non-white ethnicity, and group effects vs the control group are depicted. CI, confidence interval.

Table 4. Effects of Childhood Trauma on Time-to-Recovery Within Each Group in the Analytic Sample

	B (95% CI)	SE	*z*	*P* Value	AIC	Hazard Ratios (95% CI)
Recovery in trajectories of negative affect						
Patients						
Emotional abuse	0.31 (−0.23; 0.85)	0.28	1.14	.26	275.24	1.37 (0.79; 2.34)
Physical abuse	0.44 (−0.23; 1.12)	0.34	1.30	.20	275.22	1.56 (0.79; 3.06)
Sexual abuse	0.33 (−0.47; 1.12)	0.40	0.80	.42	276.67	1.38 (0.63; 3.06)
At-risk individuals						
Emotional abuse	0.27 (0.06; 0.48)	0.11	2.54	.01	533.40	1.31 (1.06; 1.62)
Physical abuse	0.30 (−0.02; 0.62)	0.17	1.82	.07	537.09	1.35 (0.98; 1.86)
Sexual abuse	−0.07 (−0.27; 0.13)	0.10	−0.67	.50	540.45	0.93 (0.76; 1.14)
Controls						
Emotional abuse	0.33 (−0.17; 0.84)	0.26	1.29	.20	319.80	1.39 (0.84; 2.32)
Physical abuse	0.44 (−0.50; 1.38)	0.48	0.92	.36	320.44	1.55 (0.61; 3.97)
Sexual abuse	−0.06 (−0.35; 0.22)	0.15	0.17	.67	312.17	0.94 (0.70; 1.25)
Recovery in trajectories of positive affect						
Patients						
Emotional abuse	−0.23 (−0.57; 0.10)	0.17	−1.37	.17	259.49	0.79 (0.57; 1.11)
Physical abuse	−0.10 (−0.50; 0.30)	0.21	−0.49	.63	261.33	0.91 (0.61; 1.35)
Sexual abuse	−0.17 (−0.60; 0.26)	0.22	−0.76	.45	261.03	0.85 (0.55; 1.30)
At-risk individuals						
Emotional abuse	0.10 (−0.07; 0.27)	0.09	1.18	.24	550.49	1.12 (0.93; 1.31)
Physical abuse	0.08 (−0.17; 0.34)	0.13	0.64	.52	551.28	1.09 (0.84; 1.40)
Sexual abuse	0.09 (−0.17; 0.35)	0.13	0.67	.50	550.73	0.91 (0.84; 1.42)
Controls						
Emotional abuse	−0.13 (−0.66; 0.40)	0.27	−0.47	.64	437.90	1.14 (0.52; 1.49)
Physical abuse	−0.17 (−0.76; 0.42)	0.30	−0.55	.58	437.89	0.85 (0.47; 1.52)
Sexual abuse	0.22 (−0.09; 0.52)	0.16	1.38	.17	416.93	1.24 (0.91; 1.68)

Note: CI, confidence interval; AIC, Akaike information criterion.

between individuals exposed to high vs low levels of childhood trauma varied across groups, leaving H3 unsupported. The effects of cumulative stress (all HRs ≥ 5.56, *P*s < .001) and stress intensity (all HRs ≥ 1.22, *P*s < .05) were significant in all models (see Multimedia Appendix, table 8).

Table 5. Interaction Effects Between Childhood Trauma and Group Status on Time-to-Recovery in Analytic Sample

	B (95% CI)	SE	*z*	*P* Value	AIC	Hazard Ratios (95% CI)
Recovery in negative affect						
Emotional abuse					1122.43	
Emotional abuse × at-risk	−0.03 (−0.62; 0.56)	0.30	−0.10	.92		0.97 (0.54; 1.75)
Emotional abuse × patient	−0.12 (−0.83; 0.59)	0.36	−0.33	.74		0.89 (0.44; 1.80)
Physical abuse					1127.43	
Physical abuse × at-risk	−0.15 (−1.21; 0.91)	0.54	−0.28	.78		0.86 (0.30; 2.48)
Physical abuse × patient	−0.12 (−1.29; 1.04)	0.60	−0.21	.84		0.89 (0.28; 2.83)
Sexual abuse					1120.19	
Sexual abuse × at-risk	−0.07 (−0.47; 0.34)	0.21	−0.33	.74		0.93 (0.63; 1.40)
Sexual abuse × patient	0.22 (−0.49; 0.93)	0.36	0.61	.54		1.25 (0.61; 2.53)
Recovery in positive affect						
Emotional abuse					1233.37	
Emotional abuse × at-risk status	0.20 (−0.28; 0.68)	0.25	0.81	.42		1.22 (0.76; 1.97)
Emotional abuse × patient	−0.02 (−0.57; 0.53)	0.28	−0.06	.95		0.98 (0.57; 1.70)
Physical abuse					1234.35	
Physical abuse × at-risk	0.30 (−0.27; 0.87)	0.29	1.03	.30		1.35 (0.76; 2.39)
Physical abuse × patient	0.14 (−0.50; 0.78)	0.32	0.44	.66		1.15 (0.61; 2.18)
Sexual abuse					1213.71	
Sexual abuse × at-risk	−0.10 (−0.50; 0.31)	0.21	−0.46	.64		0.91 (0.61; 1.36)
Sexual abuse × patient	−0.21 (−0.65; 0.22)	0.22	−0.97	.34		0.81 (0.52; 1.25)

Note: CI, confidence interval; AIC, Akaike information criterion.

Discussion

Main Findings

This study aimed to investigate affective recovery from event-related stress as a momentary micro-level representation of resilience in trajectories of negative and positive affect in daily life and whether this time-to-recovery is modified by childhood trauma representing a macro-level adversity in patients with first-episode psychosis, individuals at risk for psychosis, and controls. Patients, but not at-risk individuals, took longer to return to baseline level of negative affect after an event-stressor compared with controls (H1). There was evidence that negative affective recovery was modified by exposure to emotional abuse in the at-risk group (H2), but not in the patient group. We found no evidence that the difference in time-to-recovery between those exposed to high vs low levels of childhood trauma varied across groups (H3). When looking at trajectories of positive affect, there were neither group differences in time-to-recovery nor associations between time-to-recovery and childhood trauma.

Comparison With Previous Research

So far, research into affective recovery has mostly focused on trajectories of negative affect. Previous results that individuals at risk for psychosis[9] or depression[32] took longer to recover compared with controls were consistent with findings from the present analysis for the patient group with first-episode psychosis. This may suggest that there are differences in negative affective recovery across the continuum of mental health, such that prolonged recovery representing less resilient processing may be positively associated with clinical stage. This may be seen in line with the affect-regulation framework of resilience stating that affect regulation and the resulting affective experience is associated with resilience framed by adversity in a broader context.[30]

This study moves beyond previous research by taking into account the role of childhood trauma. While similar to the patient group in HR and effect size, at-risk status alone was not associated with a longer time-to-recovery. However, there was evidence that experiences of emotional abuse may prolong negative affective recovery in the at-risk group. As posited by the concept of behavioral sensitization and supported by research into effect modification of stress reactivity by childhood trauma,[2,24–26] this finding may add that some types of exposure to major adversity may sensitize at-risk individuals to the negative consequences of daily stressors beyond the amplitude of the initial stress response. Therefore, it may be the cumulated risk of major and minor adversity resulting in a longer time-to-recovery placing individuals closer to full-threshold disorder on the continuum of mental health. Furthermore, as individuals in the at-risk group may experience heterogenous comorbid symptoms of mood, anxiety, and other mental disorders and may never transition to psychosis,[13] this may also indicate that, parallel to childhood trauma as a risk factor on the macro-level, negative affective recovery may be nonspecific or transdiagnostic on the micro-level.

While our previous findings also pointed toward longer positive affective recovery in patients with a mental disorder and individuals at risk for mental disorder,[10] we found no group differences in positive affective recovery

across transdiagnostic stages[10] or in stages of psychosis in the present study. Levels of positive affect were, on average, lower in patients, which may impact how affective recovery as a momentary mechanism operates on individuals to exert its detrimental and/or protective effects. In other words, lower baseline levels of positive affect may have been more readily maintained or restored after exposure to daily stress in patients. As medication use was no exclusion criterion in our sample, we cannot rule out that this may have affected trajectories of affect in patients.

Our findings evolving from the direct comparison between processes of recovery observed in trajectories of negative and positive affect may signify that these processes are distinct but related. This is in line with research showing that the up- and downregulation of positive and negative emotions have different neural correlates.[39-42] From an evolutionary perspective, it can be argued that negative affect has a warning function to promote behaviors to avoid danger, whereas positive affect has the function to motivate exploration and curiosity.[43] Therefore, regulating negative affect may have higher urgency than regulating positive affect. While quick recovery and restoration of the original level of affect after minor adversity may be a momentary mechanism representing adaptive (ie, resilient) processing of negative affect,[27] this might be different for positive affect.

While positive affect has previously been suggested to contribute to resilience by helping to overcome adverse events,[28,31] it may be more important for resilient processing to establish and maintain a higher level of positive affect on average,[44] rather than following a specific time course related to stress.[45] This is in line with the broaden-and-build-theory's "build effect" of positive emotions, ie, daily experiences of positive emotions build psychological resources available for coping with stress.[46] Additionally, given the evidence from previous experience sampling research on moment-to-moment fluctuations,[47,48] positive affect may exert its protective effects in moments of risk exposure via its interplay with negative affect, eg, by facilitating (or hindering) negative affective recovery from stress at critical tipping points. While we investigated trajectories of negative and positive affect separately, previous studies have found the co-activation of positive and negative emotions to be play a role in psychotic disorders.[49,50] Taken together, it may be the complex, dynamic interplay of positive affect, negative affect, and stressors across clinical stages that needs further scrutiny to elucidate their role as putative risk and protective mechanisms more fully.

Limitations

The following methodological considerations need to be taken into account when interpreting the present findings.

First, this study used a pooled dataset to reach a sufficient number of participants and observations for survival analysis (that excluded eg, observations on days without stress or without baseline measurement). The 3 previously conducted studies that contributed to the pooled dataset applied different inclusion and exclusion criteria (see Multimedia Appendix, table 1). This may have led to heterogeneity in our analytic sample, in that certain characteristics or symptoms were more likely to be represented in the patient and the at-risk group due to different screening instruments and unbalanced sample sizes across the original studies. Additionally, pooling datasets reduced the set of variables available in all primary studies. Therefore, potential confounding variables that may be relevant for secondary research questions, such as constructs associated with affect, affect regulation (eg, sleep abnormalities,[51] menstrual cycle phase[52]), reporting affective states and/or exposure to childhood trauma (eg, alexithymia[53]) or longitudinal information (eg, transition to psychosis) may not be accounted for. Nevertheless, issues arising by pooling samples are unlikely to affect group differences in time-to-recovery between clinical stages, but may reduce the proportion of explained variance or cause restrictions when comparing results with other studies.

Second, there might be a differential selection bias within the ESM protocol, such that participants might be less likely to answer assessments in moments of high stress, which may be more difficult to recover from than less intense stressors. Consequently, assessments might not be missing at random, as the trajectories of affect following moments of high stress that were not recorded might differ from those trajectories that were recorded. Nevertheless, as the survival models only include trajectories with relevant information regarding recovery (recovered or right-censored), they can be regarded as the closest observable approximation of the process. To account for bias caused by the intensity of the stressor, the measure was used as a covariate in all models. To increase the likelihood of prompts in moments of high stress to be answered, shorter ESM questionnaires with a higher sampling frequency could be used, as only longer questionnaires, but not higher sampling frequency have been found to be associated with increased burden and decreased compliance.[54]

Third, we operationalized the trajectories of affect in response to stress within 1 day. However, there is evidence that sleep deprivation[55] and stress[56] may have overnight effects on negative and positive affect and its regulation. This underlines the importance of investigating affective recovery within the context of several days to account for sleep abnormalities that especially affect individuals at-risk for or with first-episode psychosis compared with controls.[51]

Relatedly, it has been argued that the opportunity to recover between stressors may toughen individuals and, thereby, help them to cope with stressors more effectively.[57] Cumulative stress during the recovery period,

which had an effect on the time-to-recovery in the majority of our models, may, in turn, have the opposite effect. Mirroring previous findings,[9,10,32] cumulative stress may itself be a momentary factor contributing to risk and resilience and should further be investigated in the context of affective recovery. However, as there were no descriptive group differences in cumulative stress, the potential confounding effect is likely to be minimal.

Fourth, model fit for the prediction of time-to-recovery by group status was poorer in participants older than 40 suggesting that the prediction of affective recovery with our models was worse for those participants. This could imply that trajectories of affect after stress have to be modeled differently in older age groups (eg, late-onset psychosis[58–61]), which is why we excluded 79 participants who were older than 40 from the analysis sample. Age itself had no influence on recovery as a covariate in our analysis, whereas previous studies have shown that positive and negative emotional processing may be subject to age-related changes.[62] Additionally, as the median onset of psychosis is in the early 20s,[63] it is particularly important to focus on adolescents and young adults when investigating affective recovery as a putative protective mechanism in the development of psychosis in a larger sample.[64]

Conclusions

In this study, individuals at risk for psychosis exposed to emotional abuse and patients with first-episode psychosis irrespective of their exposure to childhood trauma took longer to recover to baseline negative affect from daily stressors. This suggests that delayed negative affective recovery may be a putative momentary psychological mechanism acting across the continuum of mental health as it may allow to distinguish individuals with full-threshold disorder from controls as well as individuals with combined childhood and adult risk (ie, individuals with psychometric risk exposed to childhood abuse) from those with less cumulated risk (ie, individuals with psychometric risk, but less exposure to childhood abuse). To address this, negative affective recovery may potentially be used in clinical assessment, treatment evaluation or as a target for interventions, eg, psychoeducation, affect-regulation strategies, mindfulness-based interventions, or principles of acceptance and commitment therapy, which may be presented as Ecological Momentary Interventions[65,66] by offering intervention components in moments of stress and thereafter to help the individual to recover. Disentangling how positive and negative affect may have a joint influence on the recovery from stress may help us further understand their role for risk and resilience in early psychosis on a momentary level. Additionally, investigating interventions targeted at enhancing positive affect as a psychological resource will provide further insight into ways of how positive affect may buffer risk or foster resilience in the development of psychosis and other mental disorders.

GROUP investigators are Behrooz Z. Alizadeh[a,b], Therese van Amelsvoort[f], Wiepke Cahn[c,i], Lieuwe de Haan[e,j], Frederike Schirmbeck[e,j], Claudia J.P. Simons[f,g], Jim van Os[d,h], Wim Veling[a].

[a]Department of Psychiatry, Rijksuniversiteit Groningen, University Medical Center Groningen, Groningen, Netherlands; [b]University Medical Center Groningen, Department of Epidemiology, Groningen, The Netherlands; [c]University Medical Center Utrecht, Department of Psychiatry, Brain Centre Rudolf Magnus, Utrecht University, Utrecht, The Netherlands; [d]University Medical Center Utrecht, Department of Translational Neuroscience, Brain Center Rudolf Magnus, Utrecht, The Netherlands; [e]Amsterdam UMC, University of Amsterdam, Department of Psychiatry, Amsterdam, The Netherlands; [f]Maastricht University Medical Center, Department of Psychiatry and Neuropsychology, School for Mental Health and Neuroscience, Maastricht, The Netherlands; [g]GGzE Institute for Mental Health Care, Eindhoven, The Netherlands; [h]King's College London, King's Health Partners, Department of Psychosis Studies, Institute of Psychiatry, London, United Kingdom; [i]Altrecht, General Mental Health Care, Utrecht, The Netherlands; [j]Arkin, Institute for Mental Health, Amsterdam, The Netherlands

Supplementary Material

Supplementary material is available at https://academic.oup.com/schizophreniabulletin/.

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Authors' Contributions

L.A. performed data analysis and drafted the manuscript. A.S., U.R., and G.L. supervised the study. G.L. provided advice on statistical analysis. L.A., A.S., and U.R. interpreted the findings. L.A., A.S., T.V., G.L., I.M.-G., and U.R. developed the conception of the study and contributed to the design of the study. M.J.K., L.V., P.M., GROUP investigators (B.A., T.v.A., W.C., L.d.H., F.S., C.S., J.v.O., and W.V.) designed and supervised the primary studies. All authors have read and approved the final version of the manuscript.

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Data Availability

This analysis has been preregistered prior to data analysis. Data pertain to the INTERACT, the EU-GEI, and the GROUP study and are available upon reasonable request by contacting the providers of the datasets (GROUP: via Joyce van Baaren, j.vanbaaren@umcutrecht.nl; INTERACT: by submitting a request via https://redcap.gbiomed.kuleuven.be/surveys/?s=WDYAFAHWK4; EU-GEI: via Bea Campforts, bea.campforts@maastrichtuniversity.nl).

References

1. Myin-Germeys I, Kasanova Z, Vaessen T, *et al.* Experience sampling methodology in mental health research: new insights and technical developments. *World Psychiatry.* 2018;17(2):123–132.
2. Reininghaus U, Gayer-Anderson C, Valmaggia L, *et al.* Psychological processes underlying the association between childhood trauma and psychosis in daily life: an experience sampling study. *Psychol Med.* 2016;46(13):2799–2813.
3. Rauschenberg C, van Os J, Goedhart M, Schieveld JN, Reininghaus U. Bullying victimization and stress sensitivity in help-seeking youth: findings from an experience sampling study. *Eur Child Adolesc Psychiatry.* 2021;30:591–605.
4. Reininghaus U, Kempton MJ, Valmaggia L, *et al.* Stress sensitivity, aberrant salience, and threat anticipation in early psychosis: an experience sampling study. *Schizophr Bull.* 2016;42(3):712–722.
5. Myin-Germeys I, Peeters F, Havermans R, *et al.* Emotional reactivity to daily life stress in psychosis and affective disorder: an experience sampling study. *Acta Psychiatr Scand.* 2003;107(2):124–131.
6. Myin-Germeys I, van Os J, Schwartz JE, Stone AA, Delespaul PA. Emotional reactivity to daily life stress in psychosis. *Arch Gen Psychiatry.* 2001;58(12):1137–1144.
7. Palmier-Claus JE, Dunn G, Lewis SW. Emotional and symptomatic reactivity to stress in individuals at ultra-high risk of developing psychosis. *Psychol Med.* 2012;42(5):1003–1012.
8. van der Steen Y, Gimpel-Drees J, Lataster T, *et al.* Clinical high risk for psychosis: the association between momentary stress, affective and psychotic symptoms. *Acta Psychiatr Scand.* 2017;136(1):63–73.
9. Vaessen T, Viechtbauer W, van der Steen Y, *et al.* Recovery from daily-life stressors in early and chronic psychosis. *Schizophr Res.* 2019;213:32–39.
10. Ader L, Schick A, Simons C, *et al.* Positive affective recovery in daily life as a momentary mechanism across subclinical and clinical stages of mental disorder: experience sampling study. *JMIR Mental Health.* 2022;9(11):e37394.
11. Collip D, Myin-Germeys I, Van Os J. Does the concept of "sensitization" provide a plausible mechanism for the putative link between the environment and schizophrenia? *Schizophr Bull.* 2008;34(2):220–225.
12. Hartmann JA, McGorry PD, Destree L, *et al.* Pluripotential risk and clinical staging: theoretical considerations and preliminary data from a transdiagnostic risk identification approach. *Front Psychiatry.* 2020;11:553–578.
13. Hartmann JA, Nelson B, Spooner R, *et al.* Broad clinical high-risk mental state (CHARMS): methodology of a cohort study validating criteria for pluripotent risk. *Early Interv Psychiatry.* 2019;13(3):379–386.
14. van Os J. The dynamics of subthreshold psychopathology: implications for diagnosis and treatment. *Am J Psychiatry.* 2013;170(7):695–698.
15. van Os J, Rutten BP, Poulton R. Gene-environment interactions in schizophrenia: review of epidemiological findings and future directions. *Schizophr Bull.* 2008;34(6):1066–1082.
16. Rucci P, Gherardi S, Tansella M, *et al.* Subthreshold psychiatric disorders in primary care: prevalence and associated characteristics. *J Affect Disord.* 2003;76(1–3):171–181.
17. McKay MT, Cannon M, Chambers D, *et al.* Childhood trauma and adult mental disorder: a systematic review and meta-analysis of longitudinal cohort studies. *Acta Psychiatr Scand.* 2021;143(3):189–205.
18. Larsson S, Andreassen OA, Aas M, *et al.* High prevalence of childhood trauma in patients with schizophrenia spectrum and affective disorder. *Compr Psychiatry.* 2013;54(2):123–127.
19. Janssen I, Krabbendam L, Bak M, *et al.* Childhood abuse as a risk factor for psychotic experiences. *Acta Psychiatr Scand.* 2004;109(1):38–45.
20. Heins M, Simons C, Lataster T, *et al.* Childhood trauma and psychosis: a case-control and case-sibling comparison across different levels of genetic liability, psychopathology, and type of trauma. *Am J Psychiatry.* 2011;168(12):1286–1294.
21. Varese F, Smeets F, Drukker M, *et al.* Childhood adversities increase the risk of psychosis: a meta-analysis of patient-control, prospective-and cross-sectional cohort studies. *Schizophr Bull.* 2012;38(4):661–671.
22. Fisher HL, Jones PB, Fearon P, *et al.* The varying impact of type, timing and frequency of exposure to childhood adversity on its association with adult psychotic disorder. *Psychol Med.* 2010;40(12):1967–1978.
23. Morgan C, Gayer-Anderson C. Childhood adversities and psychosis: evidence, challenges, implications. *World Psychiatry.* 2016;15(2):93–102.
24. Paetzold I, Myin-Germeys I, Schick A, *et al.*; EU-GEI High Risk Study. Stress reactivity as a putative mechanism linking childhood trauma with clinical outcomes in individuals at ultra-high-risk for psychosis: findings from the EU-GEI High Risk Study. *Epidemiol Psychiatr Sci.* 2021;30:e40,1–13.
25. Rauschenberg C, Van Os J, Cremers D, Goedhart M, Schieveld J, Reininghaus U. Stress sensitivity as a putative mechanism linking childhood trauma and psychopathology in youth's daily life. *Acta Psychiatr Scand.* 2017;136(4):373–388.
26. Lardinois M, Lataster T, Mengelers R, Van Os J, Myin-Germeys I. Childhood trauma and increased stress sensitivity in psychosis. *Acta Psychiatr Scand.* 2011;123(1):28–35.
27. Connor KM, Davidson JR. Development of a new resilience scale: the Connor-Davidson Resilience Scale (CD-RISC). *Depress Anxiety.* 2003;18(2):76–82.

28. Tugade MM, Fredrickson BL. Resilient individuals use positive emotions to bounce back from negative emotional experiences. *J Pers Soc Psychol.* 2004;86(2):320–333.
29. Vella S-LC, Pai NB. A theoretical review of psychological resilience: defining resilience and resilience research over the decades. *Arch Med Health Sci.* 2019;7(2):233–239.
30. Troy AS, Willroth EC, Shallcross AJ, Giuliani NR, Gross JJ, Mauss IB. Psychological resilience: an affect-regulation framework. *Annu Rev Psychol.* 2023;74:547–576.
31. Tugade MM, Fredrickson BL, Barrett LF. Psychological resilience and positive emotional granularity: examining the benefits of positive emotions on coping and health. *J Pers.* 2004;72(6):1161–1190.
32. Velozo JDC, Lafit G, Viechtbauer W, *et al.* Delayed affective recovery to daily-life stressors signals a risk for depression. *J Affect Disord.* 2023;320:499–506.
33. Kimhy D, Delespaul P, Corcoran C, Ahn H, Yale S, Malaspina D. Computerized experience sampling method (ESMc): assessing feasibility and validity among individuals with schizophrenia. *J Psychiatr Res.* 2006;40(3):221–230.
34. Schick A, Rauschenberg C, Ader L, *et al.* Novel digital methods for gathering intensive time series data in mental health research: scoping review of a rapidly evolving field. *Psychol Med.* 2023;53:55–65.
35. Bernstein DP, Stein JA, Newcomb MD, *et al.* Development and validation of a brief screening version of the Childhood Trauma Questionnaire. *Child Abuse Negl.* 2003;27(2):169–190.
36. Thombs BD, Bernstein DP, Lobbestael J, Arntz A. A Validation Study of the Dutch Childhood Trauma Questionnaire-Short Form: Factor Structure, Reliability, and Known-Groups Validity. *Child Abuse Negl*. 2009;33(8):518–523.
37. R Core Team. *R: A Language and Environment for Statistical Computing*. Vienna, Austria: R Foundation for Statistical Computing. 2021. https://www.R-project.org/
38. Bellamy SL, Li Y, Ryan LM, Lipsitz S, Canner MJ, Wright R. Analysis of clustered and interval censored data from a community-based study in asthma. *Stat Med.* 2004;23(23):3607–3621.
39. Mak AK, Hu Z-G, Zhang JX, Xiao Z-W, Lee TM. Neural correlates of regulation of positive and negative emotions: an fMRI study. *Neurosci Lett.* 2009;457(2):101–106.
40. Kim SH, Hamann S. Neural correlates of positive and negative emotion regulation. *J Cogn Neurosci.* 2007;19(5):776–798.
41. Min J, Nashiro K, Yoo HJ, *et al.* Emotion downregulation targets interoceptive brain regions while emotion upregulation targets other affective brain regions. *J Neurosci.* 2022;42(14):2973–2985.
42. Díaz-García A, González-Robles A, García-Palacios A, *et al.* Negative and positive affect regulation in a transdiagnostic internet-based protocol for emotional disorders: randomized controlled trial. *J Med Internet Res.* 2021;23(2):e21335.
43. Norris CJ, Gollan J, Berntson GG, Cacioppo JT. The current status of research on the structure of evaluative space. *Biol Psychol.* 2010;84(3):422–436.
44. Garland EL, Fredrickson B, Kring AM, Johnson DP, Meyer PS, Penn DL. Upward spirals of positive emotions counter downward spirals of negativity: insights from the broaden-and-build theory and affective neuroscience on the treatment of emotion dysfunctions and deficits in psychopathology. *Clin Psychol Rev.* 2010;30(7):849–864.
45. McMakin DL, Santiago CD, Shirk SR. The time course of positive and negative emotion in dysphoria. *J Posit Psychol.* 2009;4(2):182–192.
46. 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(5):1045–1062.
47. Klippel A, Myin-Germeys I, Chavez-Baldini U, *et al.* Modeling the interplay between psychological processes and adverse, stressful contexts and experiences in pathways to psychosis: an experience sampling study. *Schizophr Bull.* 2017;43(2):302–315.
48. Klippel A, Schick A, Myin-Germeys I, Rauschenberg C, Vaessen T, Reininghaus U. Modelling the temporal interplay between stress and affective disturbances in pathways to psychosis: an experience sampling study. *Psychol Med.* 2022;52(13):2776–2785.
49. Cohen AS, Minor KS. Emotional experience in patients with schizophrenia revisited: meta-analysis of laboratory studies. *Schizophr Bull.* 2010;36(1):143–150.
50. Vakhrusheva J, Khan S, Chang R, *et al.* Lexical analysis of emotional responses to "real-world" experiences in individuals with schizophrenia. *Schizophr Res.* 2020;216:272–278.
51. Bagautdinova J, Mayeli A, Wilson JD, *et al.* Sleep abnormalities in different clinical stages of psychosis: a systematic review and meta-analysis. *JAMA Psychiatry.* 2023;80(3):202–210.
52. Reilly TJ, Sagnay de la Bastida VC, Joyce DW, Cullen AE, McGuire P. Exacerbation of psychosis during the perimenstrual phase of the menstrual cycle: systematic review and meta-analysis. *Schizophr Bull.* 2020;46(1):78–90.
53. Heshmati R, Jafari E, Hoseinifar J, Ahmadi M. Comparative study of alexithymia in patients with schizophrenia spectrum disorders, non-psychotic disorders and normal people. *Procedia Soc Behav Sci.* 2010;5:1084–1089.
54. Eisele G, Vachon H, Lafit G, *et al.* The effects of sampling frequency and questionnaire length on perceived burden, compliance, and careless responding in experience sampling data in a student population. *Assessment.* 2022;29(2):136–151.
55. Tomaso CC, Johnson AB, Nelson TD. The effect of sleep deprivation and restriction on mood, emotion, and emotion regulation: three meta-analyses in one. *Sleep.* 2021;44(6):zsaa289.
56. Chue AE, Gunthert KC, Kim RW, Alfano CA, Ruggiero AR. The role of sleep in adolescents' daily stress recovery: negative affect spillover and positive affect bounce-back effects. *J Adolesc.* 2018;66:101–111.
57. Seery MD. Resilience: a silver lining to experiencing adverse life events? *Curr Dir Psychol Sci.* 2011;20(6):390–394.
58. Suen Y, Wong SM, Hui CL, *et al.* Late-onset psychosis and very-late-onset-schizophrenia-like-psychosis: an updated systematic review. *Int Rev Psychiatry.* 2019;31(5–6):523–542.
59. Howard R, Rabins PV, Seeman MV, Jeste DV; Late-Onset I. Late-onset schizophrenia and very-late-onset schizophrenia-like psychosis: an international consensus. *Am J Psychiatry.* 2000;157(2):172–178.
60. Colijn MA, Nitta BH, Grossberg GT. Psychosis in later life: a review and update. *Harv Rev Psychiatry.* 2015;23(5):354–367.
61. Brunelle S, Cole MG, Elie M. Risk factors for the late-onset psychoses: a systematic review of cohort studies. *Int J Geriatr Psychiatry.* 2012;27(3):240–252.
62. Carstensen LL, DeLiema M. The positivity effect: a negativity bias in youth fades with age. *Curr Opin Behav Sci.* 2018;19:7–12.
63. Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE. Lifetime prevalence and age-of-onset distributions

of DSM-IV disorders in the National Comorbidity Survey Replication. *Arch Gen Psychiatry.* 2005;62(6):593–602.

64. Mesman E, Vreeker A, Hillegers M. Resilience and mental health in children and adolescents: an update of the recent literature and future directions. *Curr Opin Psychiatry.* 2021;34(6):586–592.
65. Heron KE, Smyth JM. Ecological momentary interventions: incorporating mobile technology into psychosocial and health behaviour treatments. *Br J Health Psychol.* 2010;15(1):1–39.
66. Myin-Germeys I, Klippel A, Steinhart H, Reininghaus U. Ecological momentary interventions in psychiatry. *Curr Opin Psychiatry.* 2016;29(4):258–263.