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Research paper

Delayed affective recovery to daily-life stressors signals a risk for depression

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ABSTRACT

Objective: The aim of this study is to investigate the time to affective recovery from daily-life stressors between healthy controls (HC) and two groups with an increased risk for developing depression: individuals with sub-clinical symptoms of depression (SSD), and individuals remitted from a depressive episode with residual symptoms of depression (RRS).

Method: The experience sampling method (ESM) was used to measure affective recovery to daily-life stressors. Affective recovery was defined as the moment that negative affect (NA) returned to baseline level following the first stressful event of the day. We assessed two different operationalizations of the baseline: NA at the moment before the stressful event (t_{-1}), and mean-person NA. The effect of stress intensity, and cumulative stress were also assessed.

Results: Survival analyses showed significantly longer recovery times for the at risk groups in comparison to healthy individuals, albeit no significant difference was found between the two at risk groups (i.e. SSD and RRS). There was also an effect of cumulative stress, but not stress intensity on time to recovery in that cumulative stress resulted in significantly longer recovery times for all three groups.

Limitations: The present study is limited by the ESM sampling design, assessments take place post-stress and therefore do not capture peak stress. Additionally, we are only able to assess patterns at the group level. Finally, there is a significant age difference between groups.

Conclusion: Individuals at risk for depression display a delayed recovery to daily-life stressors when compared to healthy controls, which is not explained by differences in stress intensity or cumulative stress. Understanding what is driving this delay may help combat the development of depression.

1. Introduction

Stress is thought to be an adaptive process essential to promote homeostasis in the short term, but detrimental to health when recurrent or prolonged (McEwen, 2017). The diathesis-stress model posits that a genetic predisposition interacts with exposure to stressors, putting individuals at risk for mental illness (Ingram and Luxton, 2012). More concretely, sensitization of the stress system occurs through repeated or prolonged exposure to stressors, leading to exaggerated responses to smaller stressors, or daily hassles, and in turn facilitating the development of mental illness (Collip et al., 2008; Harkness et al., 2015). Sensitization to daily hassles has been consistently demonstrated in studies using experience sampling methodology (ESM). This is a diary method where participants report their momentary affect, context, and appraisals a number of times a day for multiple days (Myin-Germeys et al., 2009; Myin-Germeys et al., 2018). These studies have shown that

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individuals at a greater risk of mental illness display larger affective reactivity (higher levels of negative affect) to daily-life stressors compared to healthy individuals (Husky et al., 2009; Lataster et al., 2013; Myin-Germeys et al., 2003; Myin-Germeys et al., 2018; Reininghaus et al., 2016; Palmier-Claus et al., 2012; Pruessner et al., 2017; van der Steen et al., 2017; van der Stouwe et al., 2019; van Winkel et al., 2015). However, less is known about the period following this initial reactivity: the recovery aspect of the stress response during which affect returns to baseline level. Individuals who take longer to recover from minor daily stressors are exposed to the harmful effects of stress for longer periods of time, thereby enhancing its detrimental effects on health. It follows that in the context of mental health, stress recovery may in fact be more relevant than initial reactivity. Existing studies do point to a link between delayed affective recovery from daily-life stress and a risk for mental illness. For instance, recovery appears especially slow for individuals in the early stages of psychosis over individuals at an already chronic stage (Vaessen et al., 2019). More generally, individuals at a subclinical level of any mental illness who showed an increase in symptoms at a one-year follow-up took, on average, an additional 90 min to recover from a daily stressful situation compared to those whose symptoms remained stable (Kuranova et al., 2020), highlighting the transdiagnostic nature of this process. Delayed stress recovery may therefore already signal a risk for mental illness before it develops.

Depression may be a disorder in which delayed recovery plays a particularly important role. Depression has been conceptualized as a disorder of emotion dysregulation in which individuals engage in dysfunctional emotion regulation strategies (Ehring et al., 2008), and is marked by cognitive and affective inflexibility, leading individuals to become "stuck" in negative affective states (Koval et al., 2012). Findings from an ESM study provide an initial indication that recovery to daily stressors may also be delayed in depression, with affect from prior negative events persisting in individuals with depression (Peeters et al., 2003). Laboratory studies mirror this finding, with prolonged levels of negative affect (NA) following a psychosocial stressor (Schiweck et al., 2019). It may be that, like in psychosis (Vaessen et al., 2019), recovery to stress may already be delayed in the early stages of the illness, in individuals at risk for depression, indicating that it may be a meaningful vulnerability factor for depression.

Individuals with depression report negative events as more unpleasant, important, and stressful than healthy individuals (Peeters et al., 2003), resulting in a more salient affective reactivity (Myin-Germeys et al., 2003; Wichers et al., 2007) that may consequentially result in longer recovery periods. Similarly, experiencing multiple successive stressors has been shown to lead to an additive effect of NA (Schilling and Diehl, 2014) that may likewise delay recovery. Although individuals with depression appear to encounter an equivalent number of stressful events during the day as healthy individuals (Peeters et al., 2003), a differential effect of cumulative stress (i.e., exposure to multiple stressors before complete recovery) between groups might influence recovery. The influence of stress intensity and cumulative stress on affective recovery thus has to be taken into account when interpreting potential differences in the time to recovery between groups.

In an attempt to untangle the various effects on delayed affective recovery to daily stress, we will employ survival analysis to model the time to recover from a stressor. To our knowledge, this method has yet to be used in the context of ESM; rather it is primarily used in clinical trials to model the time to a specific event, typically illness recovery or relapse (Clark et al., 2003). In the current context, this event denotes affective recovery from a stressor, or the moment that affect returns to the baseline value. Since the exact moment of recovery happens between assessments, survival analysis is especially relevant as it can account for censoring, that is either when the event does not happen or when the exact moment that it happens is not known.

In sum, we will apply survival analysis to investigate the patterns of affective stress recovery between healthy controls (HC) and two groups with an increased risk for developing depression: individuals with subclinical symptoms of depression (SSD), and individuals remitted from a depressive episode with residual symptoms of depression (RRS). Aligned with existing literature, we investigated: (1) if affective recovery from an unpleasant event is longer for both SSD and RRS groups in comparison to HC, (2) if stress intensity, and cumulative stress result in longer recovery periods, and (3) if potential group differences in recovery can be explained by stress intensity and/or cumulative stress.

2. Methods

2.1. Participants

Data collected in three previous studies (i.e. SMARTSCAN: van Aubel et al., 2020, MindMaastricht: Geschwind et al., 2011, and Genetic Risk and Outcome of Psychosis (GROUP): Collip et al., 2011; GROUP, 2011) were combined to form a total sample of 349 participants between the ages of 16 and 65 years with varying risk for depression. Data collection for all three studies was carried-out in the Dutch language, and extracted to create three groups: HC (GROUP, and SMARTSCAN studies), SSD (SMARTSCAN), and RRS (MindMaastricht study). All studies obtained ethical permission from their local ethical committee and were carried-out in agreement with the world medical association declaration of Helsinki.

2.1.1. GROUP study

The GROUP study (Collip et al., 2011; GROUP, 2011) is a large study assessing patients with non-affective psychotic disorder, siblings of patients with a non-affective psychotic disorder, and healthy controls between 16 and 50 years. Individuals with a first-degree relative with a psychotic disorder were excluded from the healthy control group. We used the data from the 84 healthy controls as part of our HC group.

2.1.2. SMARTSCAN study

The SMARTSCAN study (van Aubel et al., 2020) is a randomized controlled trial in a sample of individuals with subclinical symptoms of depression and healthy controls between the ages of 16 and 25 years. Participants took part in ESM prior to randomization and allocation to the experimental condition. Individuals who were undergoing any form of treatment for mental health concerns, reported using psychotropic drugs, required significant care or were not indicated for MRI were all excluded from the study. The ESM data from 48 healthy controls were combined with data from the GROUP study to form the HC group. In addition, 88 individuals with subclinical symptoms of depression were used to form the SSD group.

2.1.3. MindMaastricht study

The MindMaastricht study (Geschwind et al., 2011) was conducted in the Netherlands in adults with a life-time history of depression and current residual depressive symptoms. This is a randomized controlled trial testing the effectiveness of a mindfulness-based cognitive therapy in increasing positive emotions. Individuals with a current or recent (past four weeks) depressive, or psychotic episode were excluded from the study, as were those who had recently changed their treatment (i.e. psycho- or pharmacological). Participants between the ages of 19 and 65 years all took part in six consecutive days of ESM prior to being randomly assigned to a condition. Data from 129 participants were used to form the RRS group.

2.2. Experience sampling method

In all three samples, participants took part in a six-day ESM study (Myin-Germeys et al., 2009; Myin-Germeys et al., 2018; Wichers et al., 2008). In case participants continued ESM for more than six days, only the first six consecutive days were used in our study. Participants received 10 semi-random beeps a day, for six days, on a digital

wristwatch. Each beep signalled the participant to fill a paper and pencil diary assessing their current mood, thoughts, and context. Assessments took place between 07:30 h to 22:30 h, with an average of a 90-minute interval between assessments.

2.3. Measures

2.3.1. Demographic items

Participants all completed a baseline questionnaire that included several demographic items such as age and gender. Additionally, the inventory of depressive symptomatology-self report (IDS-SR: Rush et al., 2000) was used to measure depressive symptoms. The IDS-SR is a thirty-item questionnaire assessing mood and behaviour in the past seven days. Each item is scored from 0 to 3 and results in a total score ranging from 0 to 84.

2.3.2. Negative affect

The measure of NA is a composite measure of mood items. NA is the mean score per participant per day on the items “down”, “insecure”, “lonely”, “anxious”, and “guilty”. These items were the same across all three studies and have been used in other studies to compute NA as well. All items are measured on a 7-point Likert scale from *1. not at all* to *7. very much* (Myin-Germeys et al., 2018). Person mean-centered items provided a moderately high internal consistency with a Cronbach alpha of 0.72 and 0.91 for within- and between-person respectively.

Since operationalization of baseline NA differs in the literature, we identified baseline levels of NA in two ways to compare results. First, baseline was defined as the level of NA before onset of the stressful event (i.e., t_{-1}) (NA_{-1}) (Vaessen et al., 2019). Second, baseline affect was defined as the mean-person NA (NA_m) throughout the entire ESM period (Kuranova et al., 2020).

2.3.3. Stressful events

To identify stressful events, participants were asked at every assessment-point to think about the most important event since the last assessment and rate how pleasant it was from *−3. very unpleasant* to *3. very pleasant*. Positive scores (i.e., non-stressful events) were converted to 0 and the remaining scores reversed so that a higher score reflected a higher stress intensity. Stress intensity was thus scored from *0* to *3* with 0 reflecting no stressful event and the higher the score, the more stressful the event. Only the first stressful event of a day was used for our recovery estimation to prevent influences by potential incomplete recovery from previous stressors. The first stressful event of a day was designated as t_0 and was used to assess recovery; any following stressors on the same day were used to compute the cumulative stress measure. Cumulative stress was computed and coded as 1 if any additional stressful events were experienced and coded as 0 if no additional stressor was experienced prior to full recovery.

2.4. Statistical analysis plan

2.4.1. Data preparation

To reliably investigate the recovery of a stressor separate from the experience of additional stressors, data had to be carefully pre-processed and restructured. This process was performed for each day per individual and resulted in several observations being discarded. The first stage was to identify for every participant and for every day if a stressful event occurred. Days when no stressful event had occurred, or when the data after the stressful event at t_0 are missing were discarded. For the model with NA_{-1} as baseline, days were discarded if the stressful event occurred on the first assessment of the day or if NA_{-1} was missing, as there was no information on baseline NA.

The second stage was to delineate the event interval, i.e. the time between stressor (t_0) and the moment that negative affect had recovered to baseline values. The event, in this case the moment of affective recovery, happened between assessments meaning that the exact minute of recovery was unknown, but the time interval in which recovery took place could be derived. The moment of stress at t_0 marked the start of the interval which ended at the moment of recovery, when $NA \leq NA_b$ (see Fig. 1.a and b). Recovery was understood to have already happened by t_0 in cases where NA at t_0 (NA_0) was lower or equal to NA at baseline (NA_b), meaning that NA_0 marked the end of the interval of recovery (see Fig. 1.c and d)

In case the participant had not yet recovered by end of day (see Fig. 1.e) or the data is missing before recovery happened (see Fig. 1.f), data were treated as right censored, meaning that this observation was computed as showing no recovery by the time of the last assessment of the day.

2.4.2. Data analyses

First, we calculated descriptive statistics (i.e. mean, sd, range, %) for all variables, and performed ANOVA tests to assess differences in participants' averages between groups. When the ANOVA test was significant, the Tukey's honest significant difference test served to perform multiple pairwise-comparisons between the three groups.

Second, to test the effect of depression group on time to affective recovery (hypothesis 1.), we estimated a Weibull regression model for time interval-censored data (Bellamy et al., 2004). We included depression group (i.e., HC, SSD, RRS) as a predictor and the interval in which recovery took place as the dependent variable. Stress intensity, cumulative stress, age, and gender (i.e. male = 0, and female = 1) were included as covariates in all models. Finally, the model was run with baseline NA_{-1} and baseline NA_m seperately.

Data analysis was conducted in R version 4.0.3 (R Core Team, 2020) using the survival package (Therneau and Grambsch, 2000; Therneau et al., 2020) that includes a function to estimate Weibull regression models. We estimate the model using Generalized estimating equation with adjusted covariance matrix that supports interval-censored data and accounts for the multilevel nature of the data, with intervals nested within participants (Liang and Zeger, 1986). We report hazard ratios, indicating how much higher (if larger than 1) or lower (if below 1) the hazard rate of non-recovery is in the SSD and RRS groups relative to the HC group. A hazard ratio larger than 1 indicates a longer time to recover while a ratio below 1 reflects a shorter time to recover compared to the HC group (Barraclough et al., 2011).

Finally, to assess whether there was a significant difference in time to affective recovery between SSD and RRS, we performed a linear hypothesis test. Code is available at the OFS page: https://osf.io/u92t3/?view_only=f73d706c68c745b38349300a51fc7427.

3. Results

3.1. Descriptive statistics

A total of 349 participants were included: HC (N = 132), SSD (N = 88), and RRS (N = 129). The majority (77 %) of participants was female. Age and percentage of completed questionnaires are significantly different across all three groups with participants in the RRS group being the oldest and most compliant and vice versa for participants in the SSD group. Moreover, the depression score (i.e., IDS-SR) and all NA averages (i.e., NA, NA_{-1}, NA_m, NA_0) were significantly lower for HC in comparison to the two at risk groups. Likewise, the RRS group reported on average significantly higher stress intensity than the other two groups. See Table 1 for details on the reliability of NA across assessments. Finally, the number of stressful events differed between models: the model with baseline NA_{-1} included 684 stressful events, while baseline NA_m included 1102 events. Further details on the frequency of the distribution of stressful events per individual can be found in the Supplementary material in Fig. 2. By contrast, there were no significant group differences in gender distribution and cumulative stress (see Table 2).

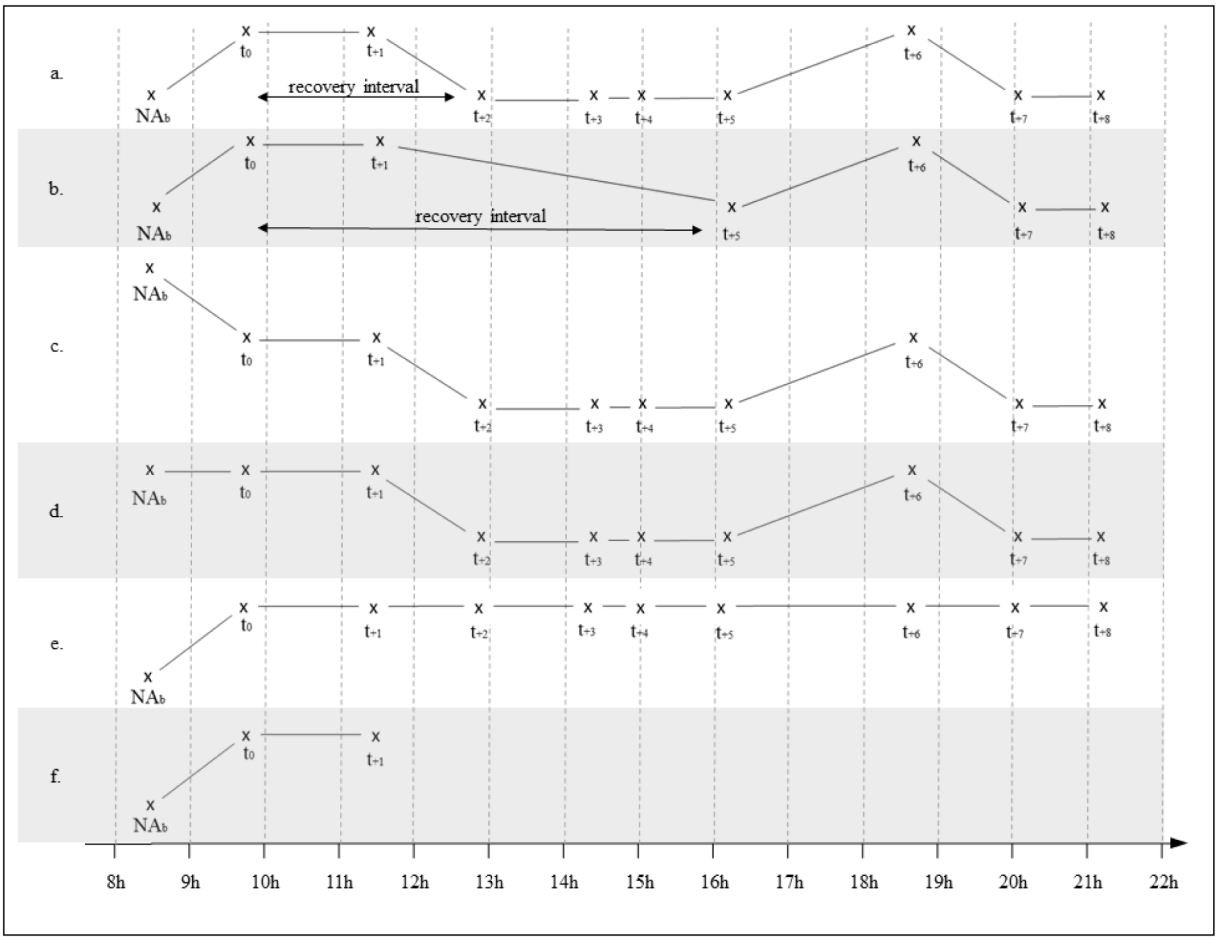

Fig. 1. Illustrative cases with NA at baseline (NA_b), at the moment of stress (t_0) and following assessments (t_{+1}, t_{+2}, t_{+3}, t_{+4}, t_{+5}, t_{+6}, t_{+7}, t_{+8}).

3.2. Effect of depression group on recovery

3.2.1. Model with baseline NA_{-1}

Depression group had a significant effect on the time to recovery, with both at risk groups (SSD and RRS) taking significantly longer than HC to recover. More specifically, SSD and RRS had a hazard ratio of non-recovery of 1.53 and 1.49 respectively, meaning that participants in these group were approximately 53 % and 49 % less likely to have recovered at any given time point in comparison to HC (see Table 3).

The median estimate values provide the distribution of the survival time, in this case that refers to the probability of not recovering. Here the median estimates show that there is a 50 % chance of participants in the HC group not recovering after 56 min or below. On the other hand, individuals in the SSD and the RRS groups show a 50 % probability of not recovering by 89 min, and 78 min respectively when no additional stressor occurs prior to full recovery (see Table 4, and Fig. 3).

A linear hypothesis test was performed to assess whether the difference between the SSD and the RRS group is significant. Results showed no significant difference in recovery between the two groups $X^2(1) = 0.02$, $p = .89$.

In addition, the main effects of cumulative stress and stress intensity (see Table 3) suggest that there was a significant effect of the former but not the latter on the time to recovery. Namely, cumulative stress was related to a significantly longer time to recovery for all groups (see Table 3, and Fig. 3). For example, when no cumulative stress is experienced, HC have a 50 % probability of not recovering by 56 min following the stressor, compared to 301 min if they experience an additional stressor prior to affective recovery (see Table 4, and Fig. 3).

3.2.2. Model with baseline NA_m

Similar to the model with baseline NA_{-1}, the depression group showed a significant effect on the time to recovery, with SSD and RRS taking significantly longer than HC to recover. More specifically, the SSD and RRS group have a hazard ratio of non-recovery of 1.29 and 1.27 respectively, meaning that participants in these group were approximately 29 % and 27 % less likely than HC to have recovered at any given time point (see Table 3).

The median estimate values showed that individuals in the HC, SSD, and RRS groups have a 50 % probability of not recovering by 63, 84, and 75 min respectively following the stressor if no additional stressor is experienced during the window of recovery (see Table 4, and Fig. 3).

A linear hypothesis test was performed to assess whether the difference between the SSD and RRS group was significant. Results show no significant difference in time to recovery between the two groups $X^2(1) = 0.004$, $p = .95$. That is, while SSD and RRS took significantly longer to recover from a daily-life stressor in comparison to HC, differences between the SSD and the RRS group were not significant.

Finally, the main effect of cumulative stress and stress intensity on time to recovery show that there was a significant effect of cumulative stress but not stress intensity on the time to recovery (see Table 3, and Fig. 3). That is, on average, when no cumulative stress is experienced, HC have a 50 % probability of not recovering by 63 min following the stressor, compared to 160 min if they experience cumulative stress (see Table 4, and Fig. 3).

4. Discussion

Our results show that individuals at risk of depression display a

Table 2
Descriptive statistics by group: healthy controls (HC), subclinical symptoms of depression (SSD), and remitted with residual symptoms (RRS).

	HC	SSD	RRS	Total
Demographics				
N	132	88	129	349
Age (mean (sd), range)[†‡ §]	28.38 (10.41), 16–58	20.66 (2.37), 16–25	43.79 (9.55), 19–65	32.13 (12.84), 16–65
Female (N (%))	100 (76)	70 (80)	98 (76)	268 (77)
IDS-SR[†‡] (mean (sd), range)	5.96 (4.16), 0–20	22.37 (10.35), 4–46	23.08 (9.97), 2–52	17.75 (11.80), 0–52
Compliance[†‡§] (N (%))	5497 (69)	3279 (62)	6168 (80)	14, 994 (72)
NA[†‡] (mean (sd), range)	1.32 (0.55), 1–7	2.17 (1.04), 1–7	2.15 (1.11), 1–7	1.85 (1.01), 1–7
Baseline NA_{-1}				
Days with stress[§] (N (%))	227 (4.1)	148 (4.5)	309 (5)	684 (4.6)
NA_{-1}[†‡] (mean (sd), range)	1.42 (0.67), 1–7	2.30 (1.11), 1–6	2.27 (1.16), 1–7	2 (1.09), 1–7
NA_0[†‡] (mean (sd), range)	1.58 (0.76), 1–4.5	2.66 (1.12), 1–5.5	2.67 (1.20), 1–7	2.30 (1.17), 1–7
stress intensity[‡§] (mean (sd), range)	1.74 (0.81), 1–3	1.60 (0.78), 1–3	1.97 (0.82), 1–3	1.81 (0.82), 1–3
cumulative stress (mean (sd), range)	0.15 (0.36), 0–1	0.26 (0.44), 0–1	0.29 (0.46), 0–1	0.24 (0.43), 0–1
Baseline NA_m				
Days with stress[‡§] (N (%))	336 (6.1)	257 (7.8)	509 (8.3)	1102 (7.4)
NA_m[†‡] (mean (sd), range)	1.39 (0.36), 1–3	2.31 (0.69), 1–4	2.21 (0.74), 1–4	1.98 (0.75), 1–4
NA_0[†‡] (mean (sd), range)	1.59 (0.73), 1–4.5	2.68 (1.12), 1–7	2.65 (1.24), 1–7	2.33 (1.19), 1–7
stress intensity [‡§] (mean (sd), range)	1.81 (0.83), 1–3	1.68 (0.80), 1–3	2.04 (0.83), 1–3	1.89 (0.84), 1–3
cumulative stress (mean (sd), range)	0.28 (0.45), 0–1	0.41 (0.49), 0–1	0.37 (0.48), 0–1	0.35 (0.48), 0–1

NA = negative affect, NA_{-1} = negative affect at t-1, NA_m = mean person negative affect, NA_0 = negative affect at the moment of stress (t_0), N = number, % = percentage, sd = standard deviation, compliance = number of filled-in questionnaires, and † signifies a significant difference (<0.05) between HC and SSD, ‡ a difference between HC and RRS, and § a difference between SSD and RRS.
Note: IDS-SR for HC is computed only on the SMARTSCAN study data.

delayed affective recovery to stress from an unpleasant event in daily-life in comparison to healthy individuals. Occurrence of additional stressors significantly increased the time to recovery in all groups, but does not explain group differences. Finally, findings were consistent across both operationalisations of baseline, albeit with baseline NA_{-1} presenting a larger effect than NA_m.

4.1. Affective recovery in individuals at risk for depression

Previous laboratory (Sanchez et al., 2013; Schiweck et al., 2019) and daily-life research (Peeters et al., 2003) pointed to a potential delayed stress recovery in depression, mirroring findings in early psychosis (Vaessen et al., 2019), and general psychopathology (Kuranova et al., 2020). The current study adds further support for this association with a significant effect of depression risk on the time to recovery. More specifically, individuals in the SSD or the RRS groups are significantly less likely to have recovered from a daily stressor in comparison to HC at any given time point. Ergo, as is the case for psychosis (Vaessen et al., 2019), delayed affective recovery to daily stressors also seems relevant in the groups at risk for depression.

Differences in recovery could not be explained by group differences in stress intensity or cumulative stress. The consistent finding that more intense stressors elicit a larger immediate increase in NA (Lataster et al., 2013; Myin-Germeys et al., 2018; Pruessner et al., 2017; van Winkel et al., 2015) led us to hypothesize that higher stress intensity may predict longer recovery times and hence that prolonged recovery in individuals at risk for depression could be a result of them experiencing more intense stressors than HC (as was the case with our RRS individuals). Our findings do not support this theory, but add support for exploratory findings showing that greater NA reactivity was associated with a larger drop in NA on the next assessment than lower NA reactivity (Almeida et al., 2020).

In contrast to stress intensity, experiencing additional stressors did result in significantly longer recovery times. This is in line with existing findings on cumulative stress where cumulative and concurrent stressors lead to a greater increase in NA (Schilling and Diehl, 2014) and aversive health outcomes (Grzywacz and Almeida, 2008). However, cumulative stress did not differ between our groups and did not explain the longer recovery times of SSD and RRS individuals.

4.2. Stress recovery and emotion regulation

Instead of stress intensity or cumulative stress, cognitive and behavioural processes may be responsible for the delayed recovery in individuals at risk for depression. For example, individuals with SSD and RRS struggle to regulate their emotions appropriately, and are more likely to engage in ruminative behaviour in comparison to healthy individuals (Berking et al., 2014; Ehring et al., 2008; Papadakis et al., 2006). Consequently, individuals at risk for depression may require more resources to cope with the stressor and therefore take longer to recover. Likewise, an inability to disengage from negative stimuli has also been shown to slow affective recovery in depression (Sanchez et al.,

Table 3
Weibull survival model for time to affective recovery by depression group: healthy controls (HC), subclinical symptoms of depression (SSD), and remitted with residual symptoms (RRS), with baseline NA_{-1} and NA_m.

Covariates	Baseline NA_{-1}				Baseline NA_m			
	B	z	*p*	HR	*B*	z	*p*	HR
Intercept	4.63	18.45	≤0.01		4.76	22.59	≤0.01	
Gender	−0.07	−0.56	0.57	0.93	−0.03	−0.33	0.74	0.97
Age	<−0.01	−1.06	0.29	0.99	≤−0.01	−0.83	0.40	1
Stress intensity	≤0.01	0.16	0.87	1.01	−0.03	−0.68	0.50	0.97
Cumulative stress	1.68	13.62	≤0.01	5.35	0.93	0.09	≤0.01	2.53
Group								
HC[a]								
SSD	0.43	2.44	0.01	1.53	0.25	2.05	0.04	1.29
RRS	0.40	3.17	≤0.01	1.49	0.24	2.05	0.04	1.27
Log (scale)	0.08	1.62	0.11		0.12	0.03	≤0.01	
AIC	1813.138				3060.516			

[a] Stands for control group, other groups are compared against it, HR = the hazard ratio. NA_{-1} = negative affect at t_{-1}, NA_m = person-mean negative affect.

Table 4
Estimate in minutes (E) and confidence interval (CI) for 10, 50, and 90th percentiles of the survival times (i.e. not recovery) by depression group: healthy controls (HC), subclinical symptoms of depression (SSD), and remitted with residual symptoms (RRS), with baseline NA_{-1} and NA_m for model with and without cumulative stress.

Group	Percentile	Baseline NA_{-1}		Baseline NA_m	
		Cumulative stress = 0	Cumulative stress = 1	Cumulative stress = 0	Cumulative stress = 1
HC	0.1	7 (5–10)	39 (26–52)	8 (5–10)	19 (14–24)
	0.5	56 (43–70)	301 (226–376)	63 (51–75)	160 (128–191)
	0.9	205 (165–245)	1100 (807–1393)	246 (206–286)	621 (492–750)
SSD	0.1	12 (7–16)	62 (43–82)	10 (7–13)	25 (19–31)
	0.5	89 (62–116)	477 (345–609)	84 (67–100)	211 (174–249)
	0.9	326 (230–421)	1744 (1194–2294)	326 (264–288)	822 (653–991)
RRS	0.1	10 (7–14)	54 (39–69)	9 (7–11)	23 (17–28)
	0.5	78 (63–92)	416 (331–501)	75 (64–87)	190 (161–219)
	0.9	284 (241–326)	1519 (1155–1883)	293 (256–329)	738 (613–863)

E (CI: 95 %) for the model without (i.e. cumulative stress = 0) and with the effect of cumulative stress (i.e cumulative stress = 1), NA_{-1} = negative affect at t-1, NA_m = person-mean negative affect.

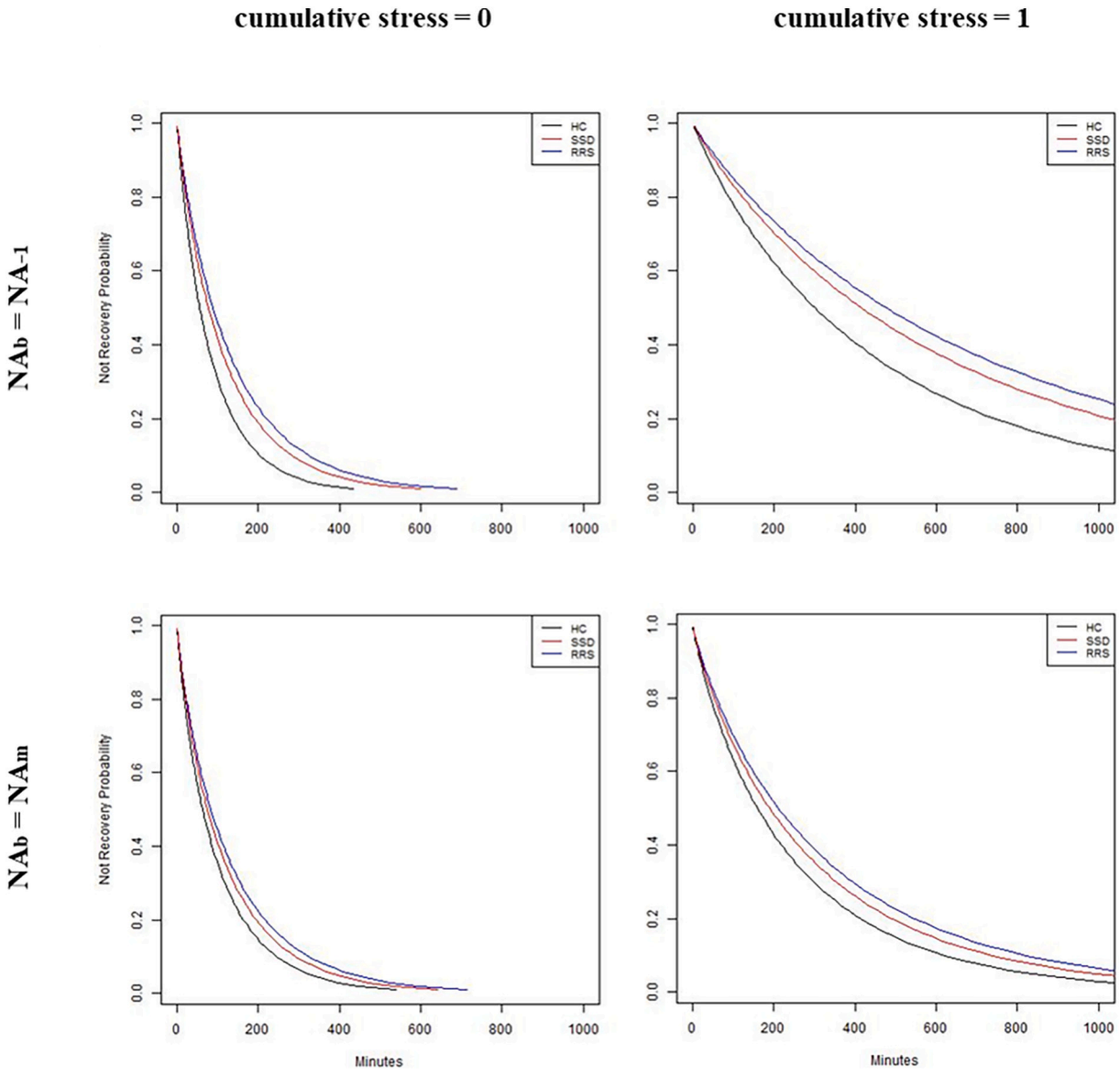

Fig. 3. The survival curve (i.e. Not recovery probability) of each depression group (i.e. HC, SSD, and RRS) for models with baselines NA_{-1} and NA_m, without (i.e. 0) and with (i.e. 1) the effect of cumulative stress. HC = healthy controls, SSD = individuals with subclinical symptoms of depression, RRS = individuals remitted from a depressive episode with residual symptoms of depression, NA_b = negative affect at baseline, NA_{-1} = negative affect at t_{-1}, i.e. assessment before stressor = negative affect NA_m = person-mean negative affect.

2013). Nevertheless, daily-life studies investigating the relationship between emotion regulation and recovery to stress are necessary. Targeting these mechanisms may help promote efficient recovery and limit the risk of a depressive episode. Indeed, there is evidence that interventions geared towards emotion regulation and/or attentional bias are effective in reducing symptoms of depression, as well as stress (Jenaabadi, 2017), in some cases with a greater effect on individuals with mild symptoms over severe symptoms (Baert et al., 2010).

4.3. Baseline operationalization

An important challenge in the study of stress is how to operationalize the baseline. There is no consistent measure of baseline NA in the ESM literature. Both our operationalisations show a similar pattern in recovery per depression group, suggesting that baseline selection may not be a critical factor in the analysis. Baseline NA_m is more sensitive to extraneous factors such as stressor frequency and/or reactivity than NA_{-1}. On the other hand, it should be noted that baseline NA_{-1} required observations to be removed due to missingness of the n_{-1}th assessment, resulting in only 684 total observations as opposed to 1102 for mean-person NA (NA_m). Therefore, while NA_{-1} may be more reflective of a baseline mood state, more data is necessary to acquire sufficient statistical power.

4.4. Strenghts and limitations

The novel use of survival analyses to estimate the time to recover from the stress of an unpleasant event is an important strength of this study. This method proves to be suitable to more accurately estimate recovery, while accounting for the clustered nature of the data. We assumed a Weibull distribution after checking for model fit. However, it is not possible to ensure that this distribution is in fact accurate as we did not validate the performance of the model in out-of-sample data. Moreover, survival analysis using frailty models (Balan and Putter, 2020) could not be performed, as these models are not currently implemented in the survival package in R. As such the current models should be further validated in a separate dataset to ensure that findings replicate.

Survival analyses require large datasets to be able to effectively conduct this type of analyses. Only between four and 8 % of all events are stressful resulting in only a few relevant observations. Moreover, given that the time to recovery estimates are based on interval data, the more time-points are available the smaller the intervals will be, and consequently the more precise the estimates, and vice versa. These data demands mean that, for studies with only six days of data collection, these models are unable to provide reliable information at the individual level, but are nevertheless useful to inform on patterns at the group level.

Time-contingent ESM is currently the most effective way to track affect in daily-life. Still, assessment randomness means that it is unable to catch the stressor in the precise moment that it occurs. Rather, ESM captures the moment that follows the stressor. The time between stressor and assessment can vary between a mere few minutes to a total of 90 min, meaning that affect at t_0 is not peak affect. Nevertheless, the time between stressor and assessment is random and similar between groups therefore it should not bias the results.

It is important to note that the period between assessments provides a large window of time where unmeasured events can take place. It is therefore possible that recovery may take place between these assessments and an elevated NA reflect a reactivity to another event instead of an absence of recovery. Although it is expected that the measure of cumulative stress help minimize this risk, future studies could use a burst-design whereby a stressor triggers a cascade of assessments at shorter time intervals to more closely follow the recovery.

Finally, participants were not randomly assigned to each group instead, groups were drawn from three separate studies resulting in variability between groups that could not be accounted for. Namely, there is a significant age difference between groups.

5. Conclusion

This is the first study to utilize survival analyses to estimate the time to recovery from a daily stressor in individuals at risk for depression. This study has both theoretical and practical implications. Namely, delayed recovery to daily-life stressors is present in individuals who are at risk for depression, which is not explained by differences in stress intensity and cumulative stress. Consequently, to further our understanding of what is driving the delayed recovery, it is necessary to investigate its link with emotion regulation.

There are also practical points to consider when using survival analyses in daily-life. While in essence baseline selection does not seem to have an effect on our findings, low data quantity may cause important limitations. In fact, large datasets are necessary to properly conduct these types of analyses, which means that it's not possible to assess individuals' daily-life stress recovery. However, if available, survival analyses are useful in investigating group-level daily-life patterns.

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CRediT authorship contribution statement

All persons who meet authorship criteria are listed as authors, and all authors certify that they have participated sufficiently in the work to take public responsibility for the content, including participation in the concept, design, analysis, writing, or revision of the manuscript. Furthermore, each author certifies that this material or similar material has not been and will not be submitted to or published in any other publication.

Declaration of competing interest

Authors declare to have no known financial or personal interest, direct or indirect, that may have affected the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jad.2022.09.136.

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