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The affect stabilization function of nonsuicidal self injury in Borderline Personality Disorder: An Ecological Momentary Assessment study

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ABSTRACT

Nonsuicidal Self-Injury (NSSI) is prominent in individuals with Borderline Personality Disorder (BPD), and there is abundant evidence that affect regulation plays an important role in NSSI in the majority of patients. Affective variability is a core feature of BPD, and thus, we hypothesize that NSSI has an affect stabilization function in BPD. Affect stabilization is a process through which individuals attempt to make their affect more stable by reducing affective variability. We tested this hypothesis in 32 participants with BPD who reported on their NSSI and affect –using a displeasure-pleasure (valence) and activation-deactivation (activation) dimension– in an experience sampling study with 10 random signals scheduled per day for 8 days. Results indicated that individuals who engaged in NSSI show more Within Subject (WS) variance in valence and activation than individuals who did not engage in NSSI. However, within the NSSI patients, individuals who engaged more frequently in NSSI during the study showed less WS variance in valence and activation than patients who engaged less frequently in NSSI. This suggests that NSSI may be reinforced by its affect stabilization function. In the discussion, we explore alternative explanations for the relation between NSSI and affective variability, and consider the clinical implications.

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1. Introduction

Nonsuicidal Self-Injury (NSSI), the deliberate and direct destruction of body tissue in the absence of lethal intent (Claes & Vandereycken, 2007; Nock & Favazza, 1986; Nock, 2010; Nock, Wedig, Janis, & Deliberto, 2008), is prominent in a wide range of disorders in general (Nock, Joiner, Gordon, Lloyd-Richardson, & Prinstein, 2006) and in patients with Borderline Personality Disorder (BPD) in particular. NSSI is estimated to occur in between 50 and 80% of patients with BPD (Snir, Rafaeli, Gadassi, Berenson, & Downey, 2015; Soloff, Lis, Kelly, Cornelius, & Ulrich, 1994; Zanarini et al., 2007). Moreover, NSSI is listed as one of the diagnostic criteria of BPD and is even a separate category in Section III of the Diagnostic Manual of Mental Disorders, 5th edition (DSM-5, American Psychiatric Association, 2013). Given the fact that individuals who engage in NSSI are at increased risk for lasting physical injury (Stanley, Gameroff, Michalsen, & Mann, 2001; Stellrecht et al., 2006) and suicide (Brown, Comtois, & Linehan, 2002; Joiner, 2005; Nock et al., 2006), and given the enormous associated public health costs of NSSI (see Linehan, 1993) including increased emergency room utilization (Olfson, Gameroff, Marcus, Greenberg, & Shaffer, 2005), there is no doubt that it is important to gain more insight into the function of this behaviour.

1.1. Affect optimization and stabilization

Several studies have examined the reasons underlying NSSI. While NSSI has multiple functions (Brain, Haines, & Williams, 1998,

Abbreviations: NSSI, Nonsuicidal Self-injury; BPD, Borderline Personality Disorder; EMA, Ecological Momentary Assessment; FGA, First Generation Antipsychotics; SGA, Second Generation Antipsychotics.

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2002; Brown et al., 2002; Gratz, 2003; Haines & Williams, 2003; Haines, Williams, Brain, & Wilson, 1995; Kerr & Muehlenkamp, 2010; Kleindienst et al., 2008; Klonsky, 2006; Nock & Prinstein, 2004; Paris, 2005), it has become clear that, in the majority of individuals, affect regulation plays an important role (Adrian, Zeman, Erdley, Lisa, & Sim, 2011; Armey, 2012; Crowell et al., 2012; Gratz & Chapman, 2007; Klonsky, 2006; Skodol et al., 2002; Zanarini et al., 2008). In this study, we define affect regulation as the ability to regulate and modulate the experience and expression of emotions (Stepp et al., 2014). Thus, we distinguish affect regulation from both affect itself, and affective instability. Affective instability is another defining feature of BPD, and refers to the tendency to experience emotions that fluctuate frequently and intensively over time (American Psychiatric Association, 2013; Nica & Links, 2009). It is important to note that affect regulation may occur in both maladaptive (e.g., NSSI) and adaptive forms (e.g., cognitive reappraisal).

In this paper, inspired by the work of Hedeker, Mermelstein, and Demirtas (2012), we aim to further elaborate the concept of affect regulation by making a distinction between two subtypes of affect regulation: affect optimization and affect stabilization. *Affect optimization* involves adjusting one's affect to an optimal level, for example by reducing negative affect. When examining this construct in the context of NSSI, one would consider the effect of NSSI on the subjects' affect level (e.g. how negative or positive affect is), and whether negative affect improves when a person engages in NSSI. Until now, theory on NSSI in general and in BPD in particular has focused mainly on this form of affect regulation. Indeed, both the biosocial theory (Linehan, 1993) and the emotional cascade model (Selby & Joiner, 2013; Selby, Anestis, Bender, & Joiner, 2009) hypothesize that NSSI in BPD is a maladaptive affect regulation strategy that provides an effective, short-term relief of the patient's large emotional burden. Moreover, according to these theories, NSSI is not only maintained by the negative reinforcement of aversive affective and physiological tension, but also interferes in the learning of adaptive affect regulation strategies such as cognitive reappraisal or behavioural distraction (Brown, 1998).

Affect stabilization involves attempting to make one's affect more stable, reducing affective variability. When examining this construct in the context of NSSI, one would consider the effect of NSSI on affective variability (e.g., how labile affect is), and whether affects stabilizes when a person engages in NSSI. Understanding variability is important, as research has shown that not only mean levels of variables, but also their variability can convey important information (Hertzog & Nesselroade, 2003). Affect stabilization may be particularly interesting in BPD, as affective instability is a defining characteristic of this disorder. In summary, affect optimization targets the *level* of affect whereas affect stabilization targets the *variability* of affect. We hypothesize that NSSI may serve an affect stabilization function in BPD patients.

1.2. Ecological Momentary Assessment

We use an Ecological Momentary Assessment (EMA) protocol to examine the affect stabilization function of NSSI. EMA has many names (e.g., diary studies, experience sampling, ambulant monitoring) and exists in many shapes (e.g., time contingent, event contingent), but at its core EMA consists of intensive repeated measurements of individuals in their natural circumstances at specific moments in time, using an electronic device like a smartphone. For an overview of diary methods, designs, and intensive longitudinal methods, see Bolger and Laurenceau (2013), and Mehl and Conner (2012).

We opted for this method primarily because Snir et al. (2015) argue that not all functions of NSSI may be consciously accessible; instead, the affect optimization and stabilization function of NSSI may operate outside conscious awareness. Therefore, they argue that these functions may not be amenable to self-report, and should be examined using indirect methods. We address this concern using EMA methods, since these methods allow us to estimate the affective variability of a participant without asking the participant to report directly on his or her affective variability. In addition, EMA techniques minimize the impact of retrospective recall bias, and are better suited to examine dynamic change processes, including NSSI (Ebner-Priemer & Trull, 2009).

1.3. Literature examining the affect optimization and stabilization function of NSSI

To date, a number of EMA studies have examined the affect optimization function of NSSI. In particular, these studies (Armey, 2012; Houben et al., 2017; Muehlenkamp et al., 2009; Snir et al., 2015) have examined in detail the affective experiences surrounding a NSSI episode. A common finding across these studies is that negative affect increased prior to a NSSI episode. In addition, the studies including positive affect found that positive affect decreased prior to the NSSI event (Muehlenkamp et al., 2009) or in the time interval when the NSSI occurred (Houben et al., 2017).

However, the results of these studies regarding affect following the NSSI episode are more mixed. The only study providing support for the affect optimization function of NSSI was conducted by Armey (2012), using a sample of college students. The researchers found that negative affect peaked during the NSSI episode and faded gradually in the hours following the episode, with affective change surrounding the NSSI episode roughly approximating a quadratic curve. These patterns of affective change were detected only at times in which individuals engaged in NSSI, and were absent for individuals who did not report NSSI over the course of the study.

In contrast with this study, other EMA studies involving individuals with eating and personality disorders found no decrease in negative affect following a NSSI episode. For example, Muehlenkamp et al. (2009) found that patients with bulimia nervosa reported an increase in positive affect following a NSSI episode while negative affect remained unchanged. In another study, Snir et al. (2015) found no evidence that general negative affect showed a quadratic relationship —a curve with one inflexion point— surrounding NSSI acts in individuals with BPD and avoidant personality disorders. On the contrary, while general negative affect indeed increased before the NSSI urge, it showed a further increase after the NSSI urge, and then faded following a complex function with multiple turning points (or vertices). Finally, in a recent study in patients with high levels of BPD symptoms, Houben et al. (2017) found that high levels of negative emotion predicted a higher probability of subsequent NSSI. They also found that the NSSI event itself was associated with an increase in negative emotion and a decrease in positive emotion in the same time interval, but contrary to expectations, negative emotions showed a further increase in the next time interval.

In summary, the hypothesis that NSSI is negatively reinforced by a subsequent reduction in negative affect was supported in a sample of college students (Armey, 2012), but not in (pathological) groups with a disorder (Houben et al., 2017; Muehlenkamp et al., 2009; Snir et al., 2015). This suggests that the mechanisms involved in NSSI may be different in groups of individuals with a disorder. Moreover, the fact that two studies (Houben et al., 2017; Snir et al., 2015) in patients with BPD found that negative affect increased rather than decreased following NSSI prompted us to further reflect on reinforcing mechanisms for NSSI that are specific to BPD patients. Given the fact that affective instability is a defining feature of BPD, we hypothesized that in this group, NSSI may have an affect stabilization function.

To our knowledge, very few studies have investigated whether NSSI results in the stabilization of affect. In a mobile phone based study, Palmier-Claus et al. (2013) found that in individuals with psychosis, the instability of depression predicted greater concurrent and subsequent levels of thoughts about self-injury, even after controlling for level of depression. Conversely, self-injurious thoughts predicted more stable depression. These relations were not found for guilt, anxiety, or hostility. Note that in this study, the researchers studied self-injurious thoughts rather than actual NSSI behaviour. In another study, Selby and Joiner (2013) found that rumination instability, negative affect instability, and their interaction predicted NSSI in daily life in a sample of undergraduate students and individuals from the community. They found that unstable (in contrast with stable) rumination predicted NSSI irrespective of the level of affective instability, and stable rumination only predicted NSSI in the presence of unstable affect. Finally, in a study on eating disorders, Vansteelandt et al. (2013a) found that patients characterized by greater variability in the activation dimension of core affect had a higher probability of engaging in lifetime NSSI. However, whereas these studies used affective variability as a predictor for NSSI, in this study, we will focus on modelling affective variability as a function of predictors like NSSI.

1.4. Current study

Given the fact that there is little research on this topic in general, and BPD in particular, the main goal of this study was to examine the affect stabilization function of NSSI in patients with BPD. First, in line with previous research (Selby & Joiner, 2013; Vansteelandt et al., 2013a), we hypothesized that patients with BPD who engaged in NSSI would be characterized by more within-subject affective variability than patients who did not engage in NSSI. Second, as we hypothesized that NSSI would be negatively reinforced by its affect stabilization function, we expected that patients who engaged more frequently in NSSI would be characterized by less within-subject variability in affect than patients who engaged less frequently in NSSI.

These hypotheses will be tested using heterogeneous mixed models developed by Hedeker et al. (2012)(see also the Method section). These models allow us to examine the effect of covariates (e.g., NSSI) on the mean, the between subject (BS) variance, and the within subject (WS) variance. BS variance pertains to individual differences in mean affect (e.g. valence); that is, how a subject's mean affect (averaged over signals) deviates from the overall population's mean affect. WS variance pertains to differences in affect within subjects; that is, how a participant's affect varies over time and deviates at a particular moment in time from his or her own mean affect. In this context, it is worth noting that while these models allow us to examine whether engaging (and the relative frequency of engaging) in NSSI predicts WS variance, these models examine associations, and do not allow causal conclusions.

Finally, it is important to note that we examined the affect stabilization function of NSSI in BPD adjusting for the (sedative) effects of psychopharmacological agents. Although there are no indications that a pharmacological treatment improves NSSI per se, between 80 and 90% of patients with BPD are prescribed psychotropic medications, either for comorbid conditions or “by default” (Ingenhoven, 2015). We controlled for these agents because they may have an impact on affective experience and affect regulation. In previous research on eating disorders (Vansteelandt et al., 2013a), we found that Selective Serotonin Reuptake Inhibitors (SSRI) predicted the presence of lifetime NSSI in a sample of patients with eating disorders. This suggested that subjects with a history of NSSI were characterized by more affective tension and affective instability, for which SSRIs were prescribed. For this reason, we adjusted the analyses on the affect stabilization function of NSSI for the use of such medication. To test our hypotheses, we will use data that were also reported in Houben et al. (2017), however testing different, non-overlapping hypotheses about the relation between affect and NSSI, and consequently, making use of a very different (statistical) modelling approach.

2. Method

2.1. Participants

The participants is this study were 32 patients[1] diagnosed with BPD according to the Diagnostic and Statistical Manual of Mental Disorders 5th edition (DSM-V). 23 patients were recruited at the University Psychiatric Center KU Leuven, and 9 patients at the Psychiatric Hospital in Duffel in Belgium. Participants were 5 (16%) men and 27 (84%) women with a mean age of 28 years ($SD = 9$). Ethical approval for this study was given by the ethical review board of the Faculty of Psychology and Educational Sciences of KU Leuven (*s*54563), the medical Ethics Committees of UZ Leuven and KU Leuven (ML8517 and ML5967), and the Local Ethical Committees of the involved hospitals. All patients gave written informed consent.

2.2. Materials

2.1.1. Diagnosis

Assessment of DSM-IV Personality Disorders (ADP-IV). The presence of the diagnosis of BPD was assessed using the BPD scale of the Assessment of DSM-IV Personality disorders (Schotte, De Doncker, Vankerckhoven, Vertommen, & Cosyns, 1998), a self-report instrument of the DSM-IV Axis II personality disorders (which are identical to DSM-*V*). This scale consisted of 10 items, and each item was assessed on a 7-point trait scale and on a 3-point distress scale. The presence of BPD is assigned when participants score both higher than 4 on the trait scale *and* higher than 1 on the distress scale on at least 5 items. The ADP-IV shows good convergent validity (Schotte et al., 2004) with the Structured Clinical Interview of DSM-IV Axis II Personality Disorders (SCID-II; First et al., 1995).

2.2.2. EMA questionnaire

Affect. Research on core affect (Feldman Barrett & Russell, 1998; Russell, 1980, 2003; Russell & Barrett, 1999) has revealed that a large part of the information conveyed in people's daily momentary affect can be captured by two orthogonal dimensions: a valence or pleasure-displeasure dimension, and an activation-deactivation dimension. To assess core affect, one core affect word in each of the four quadrants of the core affect space was selected: stressed (negative valence, high activation), depressed (negative valence, low activation), cheerful (positive valence, high activation), and relaxed (positive valence, low activation). Each core affect word was preceded by the sentence: ‘At this moment, I feel *core affect word*’ (e.g., At this moment, I feel stressed). At each signal, items were presented in random order, and participants had to indicate the

[1] There were 30 participants reported in research by Houben et al. (2017), which uses the same data reported here. However, both studies were conducted relatively independently from one another and this difference is due to the fact that a somewhat different inclusion criterion was used. Houben et al. (2017) included participants with a study compliance rate (number of responded signals in daily life) of 25% and above, but in this research, we included participants with 20% and above. We also performed all reported analyses using only the 30 participants reported by Houben et al. (2017): these analyses resulted in similar results and identical conclusions with respect to the affect stabilization function of NSSI.

extent to which the questions were applicable to them at the moment of the signal using a slider (varying from 0 to 100).

To obtain valence and activation scores for each participant at each signal, the following calculations were performed: Valence was calculated as score for 'cheerful' (pa) + score for 'relaxed' (pd) − score for 'stressed' (na) − score for 'depressed' (nd), and activation was calculated as score for 'cheerful' (pa) + score for 'stressed' (na) − score for 'relaxed' (pd) − score for 'depressed' (nd). When using this subtraction method, all scores have a natural (neutral) midpoint of 0 and minimum and maximum scores of −200 and 200 respectively. This subtraction method to assess valence and activation has been used and validated in different types of studies using both healthy control and patient populations (e.g., Kuppens, Tuerlinckx, Russell, & Barrett, 2013; Kuppens, Van Mechelen, Nezlek, Dossche, & Timmermans, 2007; Vansteelandt & Verbeke, 2016; Vansteelandt et al., 2013a; Vansteelandt, Probst, & Pieters, 2013).

NSSI. NSSI was measured in the EMA questionnaire with the following question: 'Since the previous signal, I have engaged in automutilation' with two answer categories ('yes' vs.'no'). This question was used to construct the following variables: (a) engaging in NSSI (0 versus 1), which was a time-invariant dichotomous variable that indicates whether a patient engaged in NSSI during the study, and (b) the relative frequency of engaging in NSSI, which was the proportion calculated by dividing the number of signals at which the participant answered 'yes' by the total number of signals at which the participant responded during the study.

2.2.3. Medication

The use of First Generation Antipsychotics (FGA), Second Generation Antipsychotics (SGA), benzodiazepines, antidepressants, mood stabilizers, and stimulantia during the course of the study was coded using six dummy variables with the following categories: use (1) versus non-use (0). The majority of patients (94%) were taking psychotropic medication, with antidepressants being taken by most patients (72%), followed by SGA (53%), benzodiazepines (41%), and FGA (34%). Finally, two patients were taking mood stabilizers (6%), and one patient was taking a stimulant (3%).

2.3. Procedure

Participants were recruited after the intake process when they received a BPD diagnosis by an experienced senior psychiatrist, and this diagnosis was confirmed by the ADP-IV (ADP-IV- Borderline scale, Schotte et al., 1998; see also the Materials section). Next, patients were informed about the study and invited to participate. If they agreed to participate, they signed an informed consent and then completed a number of self-report questionnaires. Next, they received a handheld computer and instructions about how to complete questionnaires on it. In addition, participants were asked if all questions were clear, and it was assured that they completely understood how to complete all questions following the signal. Next, participants carried the handheld computer and were prompted by signals scheduled 10 times a day for 8 days during waking hours (yielding 8 days x 10 signals = 80 possible signals per participant). At each signal, participants were asked to complete the EMA questionnaire. The signals were programmed according to a stratified random interval time-series; this means that a day (e.g., 8:30 a.m.–9:30 p.m.) was subdivided into 10 successive intervals of equal length with one signal being randomly generated in each interval. It was emphasized that we hoped they would respond to as many signals as possible, but that it was reasonable they would miss some signals because they would not be able to report (e.g., when in the shower) or simply because they did not hear the signal.

The mean time interval between consecutive signals was 80 min ($SD = 35$ min). This sample scheme was based on findings of Ebner-Priemer and Kuo et al., (2007); Ebner-Priemer and Welch et al., (2007) who have suggested to study time intervals under two hours when examining affective changes in BPD. Participants filled-out an average of 50 (63% of 80 generated signals per person) repeated assessments per person (SD = 17, min = 15, max = 75), which is in line with similar EMA protocols used in patients with BPD (e.g., Chapman, Rosenthal, Dixon-Gordon, Turner, & Kuppens, 2016 (62.5%); Links et al., 2007 (58%); Snir et al., 2015 (70%)). Two participants were removed from the analysis because they responded to less than 20% of the scheduled signals.

2.4. Analysis

To examine the affect stabilization function of NSSI, we used a heterogeneous mixed model developed by Hedeker et al. (2012) for the joint modelling of the mean, or location, and the variability, or (square of the) scale, of a variable (e.g. valence). Consider the following linear mixed model (LMM) (see also Verbeke & Molenberghs, 2000) for the affect measurement $\boldsymbol{Y}$, either valence or activation, of Subject i ($i = 1, \ldots, N$ subjects) on occasion j ($j = 1, \ldots, Oi$):

$$Y_{ij} = \boldsymbol{x}'_{ij}\boldsymbol{\beta} + u_i + \varepsilon_{ij} \quad (1)$$

where $\boldsymbol{x}'_{ij}$ is the p x 1 vector of predictors including a constant for the intercept, and the variables of interest including (a) engaging in NSSI, (b) relative frequency of engaging in NSSI, and (c) other covariates like medication use. $\boldsymbol{\beta}$ is the corresponding p x 1 vector of regression coefficients. The random effects u_i are subject-specific random effects that are assumed to be normally distributed with a mean of zero and a variance of $\sigma^2{}_u$. In addition, the residuals ε_{ij} were assumed to be normally distributed with mean of zero and a variance of $\sigma^2{}_\varepsilon$, and independent of the random effects. This is a linear mixed model with random intercepts, which has the following characteristics: (a) the coefficients $\boldsymbol{\beta}$ indicate the effect of the covariates $\boldsymbol{x}'_{ij}$ on the mean affect, (b) $\sigma^2{}_u$ represents BS variation in mean affect, and (c) $\sigma^2{}_\varepsilon$ reflects WS variation in affect. To allow covariates (i.e., engaging in NSSI) to influence the Between Subject (BS) and Within Subject (WS) variances, Hedeker and Mermelstein (2007) extended this model using a log-linear representation for the variances:

$$\sigma_{ui}{}^2 = exp\left(\boldsymbol{v}_i{}'\boldsymbol{\alpha}\right) \quad (2)$$

$$\sigma^2{}_{\varepsilon ij} = exp\left(\boldsymbol{w}_{ij}{}'\boldsymbol{\tau}\right) \quad (3)$$

It may be noted that these variances have subscripts i and ij respectively to indicate that their values change depending on the covariates $\boldsymbol{v}'_i$ and $\boldsymbol{w}'_{ij}$ which are time-invariant for the BS variance and may be time-invariant or time-varying for the WS variance. Both $\boldsymbol{v}'_i$ and $\boldsymbol{w}'_{ij}$ typically include a column of ones for the reference BS and WS variance (α_0 and τ_0 resp.). As a result, the linear mixed model described above is a special case where $\alpha = \tau = 0$ for all covariates in $\boldsymbol{v}'_i$ and $\boldsymbol{w}'_{ij}$ excluding the columns of ones. In this particular case, $\sigma^2_u = exp(\alpha_0)$ and $\sigma^2{}_\varepsilon = exp(\tau_0)$. However, both the BS and WS variance may vary as a function of the covariates in $\boldsymbol{v}'_i$ and $\boldsymbol{w}'_{ij}$ respectively.

In studying the affect stabilization of NSSI, we are especially interested in the effects of (a) engaging in NSSI, and (b) the frequency of engaging in NSSI on the WS variability in affect. Therefore, we included these predictors in the vector $\boldsymbol{w}'_{ij}$. To control for the effects of medication use, dummy variables indicating medication use can also be included in this vector. In

addition, we examined the effect of all these variables on mean affect and the BS variability in affect by including these variables in the vectors $\boldsymbol{x}'_{ij}$ and $\boldsymbol{v}'_i$ respectively. However, to avoid overly complex models, we estimated preliminary heterogeneous mixed effects models (without effects of NSSI) to examine the effects of medication on the mean, the BS variance and the WS variance of valence (and activation). In the final models *with the effects of NSSI*, only effects of medication that were significant in these preliminary models were retained. All these models were estimated using the NLMIXED procedure of SAS 9.4 (SAS Institute Inc.,2011;see also Hedeker et al., 2012).

3. Results

3.1. Between subject and within subject variance in affect

First, we fit models for valence and activation without any covariates. Results indicated that patients with BPD disorder had a mean valence and activation score of −33.08 and 3.55 respectively. This mean valence score was significantly below the neutral midpoint of the scale, $t(31) = -2.61$, $p = 0.01$, indicating that patients with BPD experience, on average, affect with a negative valence. In contrast, the mean activation score was not significantly different from zero, $t(31) = 0.94$, $p = 0.35$, indicating that patients' affective experience is, on average, neutral with respect to activation.

In addition, there was considerable heterogeneity in mean valence and activation between subjects, as these estimates largely exceeded their standard errors (valence: $\alpha_0 = 8.51$, *standard error* $= 0.26$, and activation: $\alpha_0 = 6.03$, *standard error* $= 0.28$). This indicates that there were substantial individual differences among patients in mean valence and activation. The BS parameters can be re-expressed in terms of their original scale as follows: $\sigma_u^2 = exp(\alpha_0) = exp(8.51) = 4981.57$ for valence and $\sigma_u^2 = exp(\alpha_0) = exp(6.03) = 417.13$ for activation. There was also substantial variation within subjects in valence and activation, as these estimates largely exceeded their standard errors (valence: $\tau_0 = 8.71$, *standard error* $= 0.04$, *and activation:* $\tau_0 = 7.29$, *standard error* $= 0.04$). This indicates that individuals showed substantial variation in valence and activation during the day. These parameters can re-expressed as $\sigma^2{}_\varepsilon = exp(\tau_0) = 6090.59$ for valence and $\sigma^2{}_\varepsilon = exp(\tau_0) = 1464.84$ for activation.

The BS and WS variances for valence were 45% and 55% of the total variance respectively, whereas the BW and WS variances for activation were 22% and 78% of the total variance respectively. For both variables, the WS variance is higher than the BS variance, and the total variance of valence is much larger than the total variance of activation, implying that subjects show more variability in valence than activation.

3.2. Effects of engaging in NSSI on mean affect and the between and within variance in affect

Next, we examined the effect of the presence of NSSI on mean valence and activation, as well as on the BS and WS variance of valence and activation. Table 1 demonstrates that patients who engaged in NSSI experienced greater negative affect; when compared to patients who did not engage in NSSI, their valence score dropped by an average of 85 points on the displeasure-pleasure valence dimension. This indicated that patients who engaged in NSSI had a less optimal valence level than patients who did not engage in NSSI. The variability in subject-specific mean valence among patients who engaged in NSSI was similar to the variability among patients who did not engage in NSSI. However, in line with our hypothesis, patients who engaged in NSSI were characterized by significantly more WS variance in valence than patients who did not engage in NSSI.

Table 1 also shows that BPD patients who engage in NSSI experienced, on average, the same mean levels of activation as patients who did not engage in NSSI. In addition, there was no difference in the BS variance around this mean between those who engaged in and those who did not engage in NSSI. However, patients who engaged in NSSI were characterized by significantly more WS variance in activation ($\tau_1 = 0.48$, $p < 0.0001$). In summary, subjects who engaged in NSSI showed significantly more negative affect on the valence dimension, and were characterized by more WS variance in both valence and activation.

3.3. The affect stabilization function of NSSI

So far, we only distinguished between patients who engaged versus patients who did not engage in NSSI. To further examine the effects of NSSI on affect stabilization, we next took the (relative) frequency of engaging in NSSI into account (expressed as the proportion of signals at which the subject reported engaging in NSSI). The results (see Table 2) indicated that patients who engaged in NSSI had a significantly lower mean score on the pleasure-displeasure dimension than patients who did not engage in NSSI. The patients who engaged in NSSI scored 81 points lower than those who did not. Moreover, the frequency of engaging in NSSI did not add extra predictive value to this observation. In addition, there was no effect of either engaging in NSSI (α_1), or of the frequency of engaging in NSSI (α_2), on the BS variance around the mean valence.

However, engaging in NSSI (τ_1) and the frequency of engaging in NSSI (τ_2) both had a significant effect on the WS variance of valence. The regression coefficient for engaging in NSSI was positive, indicating that individuals who engaged in NSSI were characterized by more WS variance in valence. Further, the (relative) frequency of engaging in NSSI showed a quadratic relationship with the WS

Table 1
Effect of engaging in NSSI on the mean, between-subject and within-subject variance: Valence and Activation.

Parameters	Valence		*p*-value	Activation		*p*-value
	Est.(SE)	*t*-test (df)		Est. (SE)	*t*-test (df)	
Mean						
Intercept (β_0)	17.21(19.74)	0.87(31)	0.39	7.86(5.13)	1.53(31)	0.14
Eng. in NSSI(β_1)	−84.80(22.54)	−3.76(31)	0.0007	−7.18(7.28)	−0.99(31)	0.33
BS Variance						
Intercept (α_0)	8.50(0.40)	21.01(31)	<0.0001	5.74(0.45)	12.81(31)	<0.0001
Eng. in NSSI (α_1)	−0.85(0.53)	−1.60(31)	0.12	0.41(0.57)	0.72(31)	0.48
WS Variance						
Intercept (τ_0)	8.41(0.06)	137.06(31)	<0.0001	6.95(0.06)	113.05(31)	<0.0001
Eng. in NSSI (τ_1)	0.43(0.08)	5.67(31)	<0.0001	0.48(0.08)	6.40(31)	<0.0001

Note. NSSI=Non-suicidal Self-Injury, Eng. = Engaging, Est. = Estimate, df = degrees of freedom, BS=Between-subject, WS=Within-subject, SE = standard error.

Table 2
Effect of Engaging and Relative Frequency of engaging in NSSI on the Mean, Between-subject and Within-subject Variance: Valence and Activation.

Parameters	Valence		*p*-value	Activation		*p*-value
	Est.(SE)	*t*-test (df)		Est. (SE)	*t*-test (df)	
Mean						
Intercept (β_0)	17.07(19.74)	0.86(31)	0.39	7.86(5.13)	1.53(31)	0.14
Eng. in NSSI(β_1)	−81.21(28.92)	−2.81(31)	0.0085	−9.46(9.18)	−1.03(31)	0.31
RFE NSSI(β_2)	−46.76(255.58)	−0.18(31)	0.8560	8.92(89.32)	0.10(31)	0.92
BS Variance						
Intercept (α_0)	8.50(0.40)	21.03(31)	<0.0001	5.74(0.45)	12.81(31)	<0.0001
Eng. in NSSI (α_1)	−1.20(0.90)	−1.33(31)	0.19	−0.21(0.68)	−0.31(31)	0.76
RFE NSSI(α_2)	3.50(7.24)	0.48(31)	0.63	4.48(3.67)	1.22(31)	0.23
WS Variance						
Intercept (τ_0)	8.41(0.06)	137.06(31)	<0.0001	6.94(0.06)	113.05(31)	<0.0001
Eng. in NSSI (τ_1)	0.71(0.12)	5.17(31)	<0.0001	0.49(0.11)	7.02(31)	<0.0001
RFE NSSI (lin) (τ_2)	−4.63(1.53)	−3.03(31)	0.0050	−5.95(1.44)	−4.12(31)	0.0003
(RFE NSSI)(quad) (τ_3)	5.96(2.71)	−2.20(31)	0.0354	10.99(2.59)	4.25(31)	0.0002

Note. NSSI=Non-suicidal Self-Injury, Eng. = Engaging, RFE = Relative Frequency of Engaging in NSSI, Est. = Estimate, df = degrees of freedom, BS=Between-subject, WS=Within-subject, SE = standard error, Lin = Linear, quad = quadratic.

variance in valence, with negative and positive signs for the linear and quadratic terms respectively. The fact that the first parameter is negative, and the second is positive suggests that a greater relative frequency of engaging in NSSI resulted in a decrease in the WS variance in valence, but that this decrease levelled off when the frequency of engaging in NSSI became larger.

We can depict the relationship between the relative frequency of engaging in NSSI and the WS variance in valence in a graph by putting the coefficients of the WS variance of Table 2 into Equation (3): $\sigma^2_{\varepsilon i} = \exp[8.41 + 0.71 \times \text{Engaging in NSSI}_i - 4.63 \times \text{Relative Frequency of Engaging in NSSI}_i\ (\text{RFE NSSI}_i) + 5.96 \times (\text{RFE NSSI}_i)^2\ ]$. Here, $\sigma^2_{\varepsilon i}$ is the Within-Subject (WS) variance of Subject i (i = 1 … i … N), engaging in NSSI_i is a dichotomous value representing whether Subject i engaged in NSSI during the study, and RFE NSSI_i is the relative frequency of engaging in NSSI of Subject i during the study. Note that this equation reduces to exp[8.41] for subjects who did not engage in NSSI during the study as Engaging in NSSI_i, the RFE NSSI_i, and $(\text{RFE NSSI})^2$ are all 0 for these individuals.

As demonstrated in Fig. 1a, this graph (with 95% Confidence Band) shows a piecewise relation between the relative frequency of engaging in NSSI and the WS variance of valence. The first (almost vertical) piece left in the figure −the effect for engaging in NSSI with positive sign− reflects the difference in the WS variance of valence between patients who did not engage in NSSI and patients who engaged rarely in NSSI (those with the lowest relative frequency of engaging in NSSI). The second piece −the effects of the relative frequency of engaging in NSSI with a quadratic relationship− reflects the effect of engaging more frequently in NSSI on the WS variance of valence *within* the subgroup of patients who engaged in NSSI. The results of this analysis confirmed our hypothesis. Patients who engaged in NSSI showed more WS variance in valence than patients who did not engage in NSSI. However, within the group of patients who engaged in NSSI, patients who engaged relatively more frequently in NSSI showed less WS variance in valence than patients who engaged relatively rarely in NSSI. However, this effect levelled off when the relative frequency of

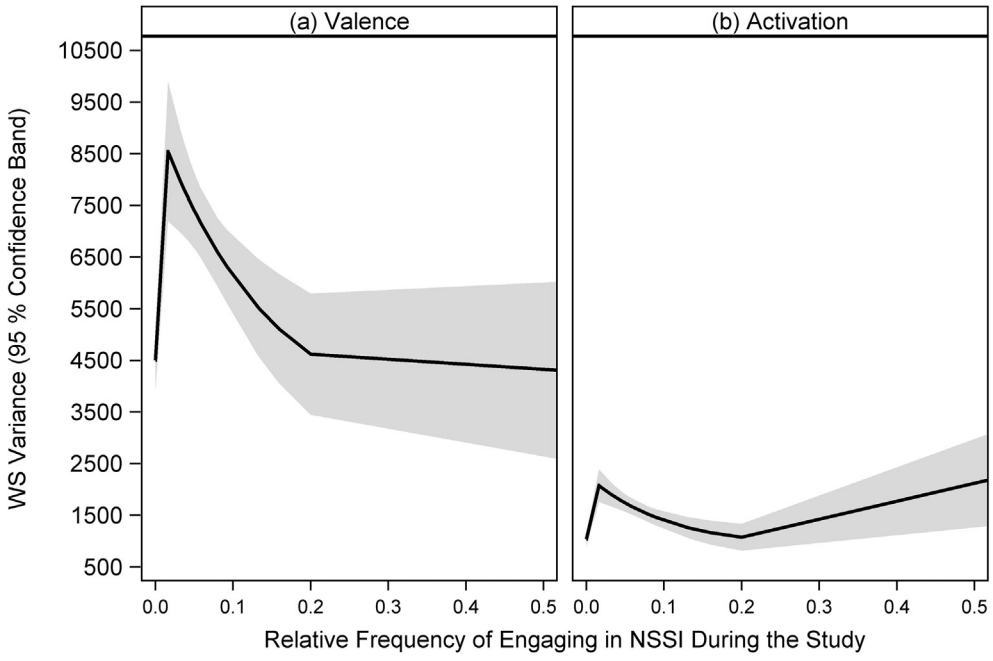

Fig. 1. Effect of the relative frequency of engaging in NSSI on the Within-Subject (WS) variance (with 95% Confidence Band): (a) Valence and (b) Activation.

engaging in NSSI became larger. However, caution must be used when interpreting the right side of Fig. 1a. This is because, as can be seen by the confidence bands that become wider, this part of the function is based on relatively sparse data, as few subjects engaged very frequently in NSSI during the study.

Finally, it is important to note that these results remain relatively unchanged after adjusting for the effects of medication use (see Table 3). The effect of engaging in NSSI is significant with positive sign, whereas the linear term for the relative frequency of engaging is NSSI is significant with negative sign, but the quadratic term is no longer significant. The latter finding may be because the results are less reliable for individuals who engaged very frequently in NSSI during the study (an indicated by the wider confidence band at the right side of Fig. 1a). This result indicates that patients who engaged relatively rarely in NSSI show more WS variance in valence than patients who did not engage in NSSI. Within the group of patients who engaged in NSSI, those who engaged more frequently in NSSI showed less WS variance in valence than those who engaged only rarely in NSSI. In this context, it is worth mentioning that medication use clearly has effects on the WS variance of valence, as patients who used SGA were characterized by more WS variance in valence, whereas patients who used benzodiazepines and/or FGA were characterized by less WS variance in valence.

With respect to activation, Table 2 demonstrates that engaging in NSSI and the relative frequency of engaging in NSSI have no effect on either mean activation or on the BS variability around this mean. However, in this table, we see effects of engaging in NSSI, and of the relative frequency of engaging in NSSI, on the WS variance of activation similar to the effects seen on valence. To show the relationship between (the relative frequency of) engaging in NSSI and the WS variance in activation graphically, we created Fig. 1b using a similar method to Fig. 1a by putting the coefficients of the WS variance in Table 2 into Equation (3).

As can be seen in Fig. 1b (with 95% Confidence bands), individuals who engaged rarely in NSSI showed more WS variability in activation than those who did not engage in NSSI (almost vertical line at the left of Fig. 1b). In addition, within the group of subjects who engaged in NSSI, subjects who engaged more frequently in NSSI showed less WS variability in activation, but this effect levelled off when the frequency of engaging in NSSI became larger. However, again, caution must be used when interpreting the right side of Fig. 1b, as this part of the graph is based on relatively sparse data, as can be seen by the wider confidence intervals. In addition, the quadratic function will necessarily increase by its mathematical nature, but this increase does not necessarily correspond to the real relationship (as it is based on fewer subjects).

Finally, it is important to note that these results remain unchanged after adjusting for the effects of medication use. In line with the results for valence, subjects who use SGA are characterized by more WS variance in activation, whereas patients who use benzodiazepines and/or FGA are characterized by less WS variance in activation (see Table 3).

4. Discussion

In an EMA study with BPD patients, we found that patients who engaged in NSSI scored lower on the pleasure-displeasure dimension of affect than those who did not engage in NSSI. Those who engaged in NSSI also showed more WS variability in valence and activation than subjects who did not engage in NSSI. However, within the subgroup of patients who engaged in NSSI, patients who engaged (relatively) more frequently in NSSI were characterized by less WS variability in valence and activation than patients who engaged less frequently in NSSI. This effect levelled off when the relative frequency of engaging in NSSI became larger. We believe that these results provide support for the affective stability function of NSSI. However, we refrain from making strong conclusions regarding the levelling off and eventual increase in the relationship between the (relative) frequency of engaging in NSSI and the WS variance in valence and activation for two reasons. First, this part of the relationship is based on rather sparse data, as a rather small number of patients engaged very frequently in NSSI (as indicated by the wider confidence bands in the right part of Fig. 1a–b). Second, the quadratic function will increase necessarily by its mathematical nature, but this does not necessarily correspond to the true relationship (as it is based on fewer subjects). However, in general, these results suggest that NSSI serves to stabilize affect, and that NSSI may be reinforced by its affect stabilization function. Indeed, there is abundant evidence that affective variability, especially in negative emotions, in associated with more pathology and lower psychological well-being (for a review and meta-analysis, see

Table 3
Effect of Engaging and Relative Frequency of engaging in NSSI on the Mean, Between-subject and Within-subject Variance after adjustment for medication: Valence and Activation.

Parameters	Valence		*p*-value	Activation		*p*-value
	Est.(SE)	*t*-test (df)		Est. (SE)	*t*-test (df)	
Mean						
Intercept (β_0)	21.67(18.14)	1.19(31)	0.24	5.37(5.08)	1.06(31)	0.30
Eng. in NSSI(β_1)	−68.05(26.86)	−2.53(31)	0.02	−12.70(9.72)	−1.31(31)	0.20
RFE NSSI(β_2)	−428.43(386.06)	−1.11(31)	0.28	70.96(82.97)	0.86(31)	0.40
BS Variance						
Intercept (α_0)	7.92(0.64)	12.39(31)	<0.0001	5.27(0.52)	10.16(31)	<0.0001
Eng. in NSSI (α_1)	−1.33(0.67)	−1.98(31)	0.06	−0.13(0.85)	−0.15(31)	0.88
RFE NSSI(α_2)	6.22(3.86)	1.61(31)	0.12	−1.57(7.49)	−0.21(31)	0.84
SGA (α_3)	0.69(0.67)	1.02(31)	0.32	0.99(0.58)	1.69(31)	0.10
WS Variance						
Intercept (τ_0)	8.68(0.09)	94.06(31)	<0.0001	7.15(0.08)	85.13(31)	<0.0001
Eng. in NSSI (τ_1)	0.58(0.14)	4.34(31)	0.0001	0.73(0.12)	6.04(31)	<0.0001
RFE NSSI (lin) (τ_2)	−3.57(1.70)	−2.10(31)	0.04	−6.08(1.57)	−3.87(31)	0.0005
(RFE NSSI)(quad) (τ_3)	1.77(3.13)	0.57(31)	0.58	10.46(2.96)	3.53(31)	0.0013
SGA (τ_4)	0.26(0.06)	4.28(31)	0.0002	0.05(0.07)	0.68(31)	0.50
Benzodiazepines (τ_5)	−0.27(0.07)	−4.05(31)	0.0003	−0.26(0.06)	−4.32(31)	0.0001
Antidepressants (τ_6)	−0.07(0.05)	−1.41(31)	0.17	0.02(0.05)	0.35(31)	0.73
FGA (τ_7)	−0.52(0.10)	−5.28(31)	<0.0001	−0.29(0.08)	−3.48(31)	0.0015

Note. NSSI=Non-suicidal Self-Injury, Eng. = Engaging, RFE = Relative Frequency of Engaging in NSSI, FGA=First Generation Antipsychotics, SGA=Second Generation Antipsychotics, Est. = Estimate, df = degrees of freedom, BS=Between-subject, WS=Within-subject, SE = standard error, Lin = Linear, quad = quadratic.

Houben, Van den Noortgate, & Kuppens, 2015). As a result, the reduction of affective variability may indeed be beneficial. Moreover, this may be especially true for patients with BPD, as affective variability is one of the core symptoms of BPD (American Psychiatric Association, 2013; Nica & Links, 2009).

In contrast with the effects of NSSI on WS variability, the relative frequency of engaging in NSSI had no effect on mean valence or activation. This suggests that the (relative) frequency of engaging in NSSI is especially relevant in stabilizing the WS variability of affect, rather than in reducing mean levels of affect. Given this fact, a clinical implication of this finding may be that psychotherapeutic interventions should focus not only on the reduction of negative affect, but also on the stabilization of affect (assuming that NSSI and affective variability are not caused by a third unknown factor, see further discussion).

These conclusions were unchanged after controlling for medication use. 94% of the patients included in our study received prescriptions for psychotropic medication, which is in line with clinical practice in other countries (Ingenhoven, 2015). The results show that medication like FGA, SGA, and benzodiazepines have an effect on the WS variability (but not the mean) of valence and activation, supporting the idea that these medications have a tranquilizing effect on patients by reducing affective turmoil. Until now, few studies (Ebner-Priemer & Kuo et al., 2007; Ebner-Priemer & Welch et al., 2007; Santangelo et al., 2014) on affect instability in BPD have taken the effect of medication use into account, and these findings may encourage researchers to include medication as a potentially important confounding variable in future research.

However, we also want to temper these conclusions for several reasons. First, it is important to note that causal conclusions cannot be drawn from this study. As mentioned in the introduction, the models we used only examine the association between NSSI and WS variability in valence and activation, and thus do not allow causal conclusions. We hypothesized that NSSI may be reinforced by its variance stabilization function, but we cannot determine whether this is the correct causal direction. A reverse causal order may be possible with, for example, decreased affective variability leading to chronic feelings of emptiness, which the patient aims to counteract by engaging in NSSI. Such a causal direction is within the bounds of possibility, as chronic feelings of emptiness are a feature of BPD. It may also be possible that other, unknown third factors caused both affective variability and NSSI.

Second, the affect stabilization function of NSSI is conceptually less appealing than the affect optimization function of NSSI. The affect *optimization* function is conceptually more appealing, as the relief from negative affect is hypothesized to follow immediately after NSSI, and the negatively reinforcing nature of NSSI is thus clear (Armey, 2012; Houben et al., 2017; Muehlenkamp et al., 2009; Snir et al., 2015). The negative reinforcing nature of the affect *stabilization* function of NSSI is less conceptually clear, as affect stabilization is not a discrete short-lived experience, but rather evolves more slowly over time. Nevertheless, our study adds evidence for the affect stabilization function of NSSI. In addition, in a sample of individuals with psychosis, Palmier-Claus et al. (2013) found evidence for the affect stabilization function of self-injury thoughts, with instability of depression predicting greater concurrent and subsequent levels of thoughts about self-injury, and, conversely, self-injurious thoughts predicting more stable depression, even after controlling for level of depression. In a sample of patients with eating disorders, Vansteelandt et al. (2013a) found evidence that those who were characterized by greater variability in the activation dimension of core affect had a higher probability to engage in lifetime NSSI. Taken together, our results, these existing studies, and the fact that affect instability is a BPD diagnostic criterion, provide preliminary evidence that individuals with BPD may use NSSI to temporarily reduce the affective turmoil they are experiencing in daily life. Future research, however, remains necessary to examine the affect stabilization function more in depth by testing, for example, whether affect is truly stabilizing in the period *following* NSSI. In such studies, stronger claims about the temporal order of NSSI and affect stabilization could be made.

Another striking result from this study is that the relationships we found between NSSI and affect are similar to the relationships found between smoking behaviour and affect. Indeed, Hedeker et al. (2012) found that smokers show, on average, more negative affect than non-smokers. However, similar to the results in our study, within the subgroup of smokers, those who smoked (relatively more) were characterized by less WS variability in negative affect than those who smoked less, suggesting an affect stabilization function of smoking. As a result, it appears that NSSI and smoking both have affect stabilization functions. It will be important for future research to determine the factors that contribute to selection of different mechanisms like smoking, NSSI, or other behaviours for affect stabilization.

In this study, we focused on the affect stabilization function of NSSI. However, as mentioned in the introduction, NSSI may also serve other functions. Nock and Prinstein (2004) proposed a model of NSSI functions defined by the intersection of two dimensions: the first distinguishing internally directed from interpersonally directed motives, and the second distinguishing positive from negative reinforcement motives. Internally directed functions of NSSI include emotion relief, which is negatively reinforcing, and the generation of feelings, for example to relieve dissociative states, which is positively reinforcing. Interpersonal functions of NSSI include the use of NSSI to avoid others or create social distance, which is negatively reinforcing, and the use of NSSI as a form of (maladaptive) communication to seek help or care, which is positively reinforcing. It is clear that the affect stabilization function proposed in this study is an internally and negative reinforcing motive of NSSI. However, we do not want to claim that this is the only function of NSSI in BPD, and the other functions of NSSI deserve further attention in future research without doubt.

Finally, this study is not without limitations. First, our sample was relatively small. As such, replication of these findings in larger datasets is necessary, especially datasets including more individuals who engage very frequently in NSSI. However, although the sample had rather few participants, the EMA protocol yields many repeated observations per subject, which may have resulted in rather reliable estimates of within-subject variance. In general, modern data collection methods like EMA provide a fair amount of both WS and BS data, and so allow the opportunity for modelling both WS and BS variances as a function of covariates. However, the models used in this study (Hedeker, Mermelstein, & Demirtas, 2008; Hedeker et al., 2012) are fairly new, and it remains an open question how much WS and BS data are necessary for variance modelling purposes. In this context, Hedeker et al. (2012) point to the fact that for the variation part of the model, there are currently no general guidelines about the degree of complexity that a given dataset could support (Hedeker et al., 2012).

Second, in this study, we used the broad dimensions of valence and activation to assess momentary core affect in daily life. In future research, it would be of interest to further examine the extent to which the conclusions concerning the affect stabilization function of NSSI in BPD hold for more discrete emotions, like anger and guilt, which are typical and highly relevant for both BPD and NSSI.

Third, the study sample consisted predominantly of women who were treated in a residential setting. Although the prevalence of NSSI is high in both inpatient and outpatients with BPD (Snir et al., 2015; Soloff et al., 1994; Turner et al., 2015; Zanarini et al., 2007), there are, to our knowledge, no studies that document whether (the

same) patients show different prevalence rates of NSSI when they are inside or outside the hospital. Although the patients in our sample certainly encountered intense emotional events and triggers, it is unclear the extent to which experiences outside the hospital (e.g., encounters with significant others) may affect the occurrence of NSSI, affect, and affective variability. For example, the frequency and severity of NSSI behaviour might be higher in patients not receiving treatment, or lower in outpatients, as they may be characterized by less severe psychopathology. It is unclear whether this would affect the mechanisms underlying NSSI, and therefore, the results of this study should be replicated in better gender balanced and outpatient samples before being generalized.

Finally, while the BPD diagnosis was made by an experienced psychiatrist, and verified by a validated self-report questionnaire (ADP-IV, Schotte et al., 2004) which shows good convergent validity with the SCID-II (First et al., 1995), our study could have benefitted from diagnoses being made by a validated structured interview like the SCID-II (First et al., 1995).

In conclusion, we have studied the affect stabilization function of NSSI. We hope that the results of this study will provide deeper insight into the function of NSSI, and motivate other researchers to examine this function in other groups of patients suffering from NSSI.

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