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Emotional Inertia and External Events: The Roles of Exposure, Reactivity, and Recovery

Peter Koval[1,2], Annette Brose[1,3], Madeline L. Pe[1], Marlies Houben[1], Yasemin Erbas[1], Dominique Champagne[1], and Peter Kuppens[1]

[1]KU Leuven – University of Leuven

[2] Australian Catholic University

[3] Humboldt University Berlin

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Please address correspondence to Peter Koval, School of Psychology, Australian Catholic University, Fitzroy, VIC 3056, Australia. Tel: +61 420 620 364; E-mail: peter.koval@acu.edu.au

Abstract

Increased moment-to-moment predictability of negative affect, or *inertia*, has been identified as an important dynamic marker of psychological maladjustment, and increased vulnerability to depression in particular. However, little is known about the processes underlying emotional inertia. The current paper examines how the emotional context, and people's responses to it, are related to emotional inertia. We investigated how individual differences in the inertia of negative affect (NA) are related to individual differences in exposure, reactivity, and recovery from emotional events, in daily life (assessed using experience sampling) as well as in the lab (assessed using an emotional film-clip task), among 200 participants commencing their first year of tertiary education. This dual-method approach allowed us to assess affective responding on different timescales, and in response to standardized as well as idiographic emotional stimuli. Our most consistent finding, across both methods, was that heightened NA inertia is related to decreased NA recovery following negative stimuli, suggesting that higher levels of inertia may be mostly driven by impairments in affect repair following negative events.

Keywords: emotional inertia, affect dynamics, exposure, reactivity, recovery, experience sampling

A primary function of feelings is to signal changes in the environment that have the potential to impact a person's well-being (Frijda, 2007; Russell, 2003). Consequently, the patterns of temporal fluctuation in people's feelings should be strongly related to the events people encounter, as well as how they respond to and recover from such events. However, research on *emotional inertia*–the tendency for affective states to be resistant to change over time–suggests that this may not hold equally for different individuals. Rather, people differ in the extent to which their affect dynamics are contingent upon external events, with downstream consequences for their well-being (Bolger & Zuckerman, 1995; Kuppens, Allen, & Sheeber, 2010). Yet, very little is known about how emotional inertia is shaped by contextual factors. In the current study, we investigated how individual differences in emotional inertia are related to the nature and frequency of events people encounter (i.e., *exposure*), their initial affective response to those events (i.e., *reactivity*), and the extent to which their affect returns to its baseline level or equilibrium following such events (i.e., *recovery*).

Emotional inertia

A fundamental feature of emotion dynamics is the degree to which the current level of an emotion can be predicted by the previous level of emotion, as captured by an autocorrelation or autoregressive slope, (Jahng, Wood, & Trull, 2008; Wang, Hamaker, & Bergeman, 2012). This measure has been labeled *emotional inertia* because higher autocorrelations reflect greater resistance to affective change (Gottman, Murray, Swanson, Tyson & Swanson, 2005; Kuppens, Allen, et al., 2010; Suls, Green, & Hillis, 1998). To illustrate, someone with relatively strong degree of negative affect (NA) inertia will be more likely to experience a high level of NA at a given moment if their level of NA was also high at the previous moment, whereas their current level of NA is likely to be low if it was also

previously low. In contrast, for a person with less NA inertia, their current level of NA is relatively independent from their previous level of NA.

Higher levels of NA inertia have consistently been linked with reduced well-being (Houben, Van Den Noortgate, & Kuppens, 2014). In particular, NA inertia appears to be a vulnerability factor for depression (Koval, Kuppens, Allen, & Sheeber, 2012; Koval, Pe, Meers, & Kuppens, 2013; Koval & Kuppens, 2012; Kuppens et al., 2012 Kuppens, Allen, et al., 2010; Wenze, Gunthert, Forand, & Laurenceau, 2009; van de Leemput et al., 2014), Given that inertia appears to be a dynamical signature of affective dysfunction, it is important to understand the processes underlying it. We contribute to this aim in the current study by examining three possible determinants of inertia; exposure, reactivity, and recovery from emotional events. We limited the current investigation to NA inertia because it has been more strongly associated with reduced well-being than inertia of positive affect (Houben et al., 2014),

Determinants of emotional inertia

Emotional inertia is thought to reflect ineffective emotion regulation as well as altered responding to the external environment. Yet, previous research has mainly focused on the former, linking higher NA inertia with greater use of rumination (Koval et al., 2012) and expressive suppression (Koval, Butler, Hollenstein, Lanteigne, & Kuppens, 2014). The current study aims to redress this imbalance by specifically examining how emotional inertia is associated with external events, and people's responses to them. Drawing on the work of Bolger and Zuckerman (1995) and Davidson (1998; 2000), we distinguish between three processes through which external events may influence emotional inertia: (a) exposure, consisting of the frequency and intensity of encountered events; (b) reactivity, which refers to the initial affective response to an event; and (c) recovery, or the degree to which affect returns to baseline in the post-event period. To be clear, we certainly do not consider

exposure, reactivity, or recovery to be independent of emotion regulation (Davidson, 1998; Gross & Barrett, 2011; Koole, 2009). Yet, by focusing on these three aspects, we aim to directly examine how the context impinges on people's level of inertia from the perspective of how people deal with events.

Exposure. People differ substantially in terms of the emotional events they encounter, either due to factors outside their control, or because they actively select certain situations and avoid others (Almeida, 2005; Frederickx & Van Mechelen, 2012; Gross & Thompson, 2007). From one perspective, one could argue that if emotional inertia reflects a resistance to affective change, it may be related to the tendency to report fewer emotional events in general. Alternatively, individuals exposed to more negative events and/or fewer positive events may have higher NA inertia because their emotional context evokes NA on a more continuous basis and offers fewer opportunities to alleviate NA. Indirect support for the latter comes from research linking more frequent exposure to negative events in daily life with neuroticism (e.g., Bolger & Zuckerman, 1995) and depression (e.g., Thompson et al., 2012), both of which have also been related to higher levels of NA inertia (e.g., Suls et al., 1998; Koval et al., 2012). While these findings raise the possibility that exposure may play a role in heightened NA inertia, no studies have investigated this to date.

Apart from the sheer number of events they come across, people may also differ in the intensity of events they encounter. Although event intensity is to some extent objective (e.g., being involved in a car crash is a more intense negative event than temporarily misplacing one's pen), it is ultimately people's subjective appraisals of event intensity that determine their affective responses (e.g., Lazarus, 1991). More intense negative events are likely to evoke more intense negative emotions, which tend to last longer (Verduyn, Delvaux, Van Collie, Tuerlinckx, & Van Mechelen, 2009), possibly contributing to greater predictability of NA over time (i.e., higher NA inertia). Conversely, less intense positive events may be

unlikely to interrupt the cycle of persistent NA, and may thus also contribute to higher NA inertia.

Reactivity. Reactivity refers to the magnitude of a person's affective response to a given stimulus or event (Davidson, 1998). Given that emotional inertia is understood to reflect resistance to affective change, Kuppens, Allen, et al. (2010) proposed that it should be related to reduced reactivity to external events. According to this view, higher NA inertia should be related to blunted NA reactivity to both positive and negative events, paralleling the emotional context insensitivity (ECI) view of depression (Rottenberg, 2005). ECI is thought to involve disengagement from the external environment, resulting in predictable and inflexible affective responding (i.e., increased emotional inertia). Supporting this view, depression has been related to both increased NA inertia and blunted emotional reactivity (for meta-analyses, see Houben et al., 2014; Bylsma, Morris & Rottenberg, 2008).

However, there is also reason to postulate a positive association between NA reactivity and inertia: more intense NA reactions may be more difficult to down-regulate, leading to increased duration of NA and therefore increased moment-to-moment predictability (i.e., inertia). For instance, neuroticism has consistently been linked with greater NA reactivity (e.g., Bolger & Zuckerman, 1995; Gross, Sutton, & Keterlaar, 1998; Mroczek & Almeida, 2004), yet also with heightened NA inertia: Suls et al. (1998) reported that individuals scoring high on neuroticism showed both increased NA reactivity, and higher levels of NA inertia in daily life. These findings suggest that NA inertia and NA reactivity may be positively related, although Suls et al. (1998) did not test this association directly.

To our knowledge, the only study that has directly investigated how NA inertia relates to NA reactivity found no association (Thompson et al. 2012). However, this study only measured reactivity to events in daily life using experience sampling, which suffers from two important limitations: First, since people may differ substantially in terms of the kinds of

events they encounter very in their daily lives (see above), individual differences in reactivity assessed using experience sampling may be confounded with differences in exposure. Second, the temporal resolution of experience sampling studies (affect and events are typically reported every 1-2 hours) may be suboptimal for capturing reactivity. An increase in NA two hours after a negative event is unlikely to be a pure measure of reactivity, and may also capture affective recovery. Given the equivocal evidence reviewed above, further research using both naturalistic and laboratory methods is needed to clarify how NA inertia relates to NA reactivity.

Recovery. Following initial reactivity to an event–facilitating the mobilization of resources to prepare the person to respond appropriately–the proper functioning of the affective system relies on a recovery process whereby affect returns to its baseline level (Kuppens, Oravecz, & Tuerlinckx, 2010; Taylor, 1991). Nevertheless, the affective recovery process differs substantially between persons, and these individual differences predict well-being (Davidson, 1998; Hemenover, Augustine, Shulman, Tran, & Barlett, 2008). Given that emotional inertia reflects resistance to affective change, it may (also) be partly driven by impaired recovery from previous events. Put otherwise, after an event has elicited an affective reaction, some individuals may be less able to recover from the event, resulting in the tendency for affect to spill-over into the subsequent time period. This was the mechanism proposed by Suls and colleagues to explain the increased NA inertia they observed among more neurotic individuals (Suls et al., 1998; Suls & Martin, 2005).

At an empirical level, neuroticism has been associated with impaired recovery following negative events, both in terms of subjective experiences of NA (Hemenover, 2003), and slower decay of amygdala activation (Schuyler et al., 2012). Similarly, increased vulnerability to depression has been linked with impairments in neural circuitry associated with NA recovery (Holtzheimer & Mayberg, 2011; Pezawas et al., 2005). Such impairments

in recovery from negative events may be partly driven by habitual use of emotion regulation strategies that are ineffective in down-regulating negative feelings, such as rumination and expressive suppression (see Webb, Miles & Sheehan, 2012). As reviewed above, these regulation styles have also been related to increased NA inertia, raising the possibility that impaired NA recovery from negative events may play a role in NA inertia.

The current study

Our aim in the current study was to investigate how emotional inertia is related to exposure, reactivity, and recovery from emotional events, focusing specifically on the dynamics of NA. We assessed participants' subjective experiences of NA and the occurrence of negative and positive events as they naturally occurred in daily life using the experience sampling method (ESM). In addition, we measured participants' subjective emotional responses to a series of standardized film-clips in the lab. This hybrid approach capitalizes on the strengths of each method (e.g., the high ecological validity of ESM and control over situational variables in the lab), and allowed us to obtain measures of NA inertia, reactivity, and recovery on two distinct timescales and in response to both standardized and idiographic emotional events. Exposure was measured exclusively using the ESM as the frequency and intensity of events were held constant across participants in the film-task. Based on the previous empirical research and theory reviewed above, we predicted that NA inertia would be related to more frequent exposure to negative and less frequent positive events, and/or exposure to more intense negative and less intense positive events. Regarding reactivity, we predicted that NA inertia would either be associated with blunted reactivity to both negative and positive stimuli (following from the ECI hypothesis; see Kuppens, Allen, et al., 2010), or increased reactivity specifically to negative stimuli (see Suls et al. 1998; Suls & Martin, 2005). Finally, we expected that NA inertia would be related to impaired recovery from negative stimuli.

Method

Participants and Pre-Screening

We aimed to recruit 200 students commencing their first year of tertiary education in the Leuven area. To ensure our sample included participants with a broad range of psychological well-being levels, we screened a large number of eligible students using the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977). We advertised widely at secondary schools and university/college orientation sessions and an initial pool of 686 students (65.7% female) completed the online CES-D pre-screening. Using a stratified sampling approach (Ingram & Siegle, 2009), we recruited 180 participants with a broad range of CES-D pre-screening scores (*Range* = 0–39), including 69 participants scoring at or above the clinical cutoff score of 16 proposed by Radloff (1977), and 18 participants scoring at or above the more conservative clinical cutoff score of 27 proposed by Gotlib, Lewinson, & Seeley (1995). An additional 22 participants were recruited after the study had already begun and therefore did not complete the CES-D pre-screening.[1]

Two participants were excluded due to poor compliance with the ESM protocol (i.e., < 50%), leaving a final sample of 200 participants (110 female) ranging in age from 17 to 24 years (M = 18.32; SD = 0.96). Some participants were excluded from certain analyses due to missing data on measures of reactivity and recovery (see below).

Materials and Procedure[2]

[1] The CES-D was administered again during the study, and participants who did not complete the CES-D pre-screening did not differ from those who did in their CES-D scores (p = .70).

[2] The data presented here were collected during the first wave of a three-wave longitudinal study on emotional functioning and well-being. The broader study included the following additional measures, not reported here: a) structured clinical interviews; b) self-report questionnaires assessing well-being, personality, stress, and emotional functioning; and c) lab tasks assessing executive functioning and emotion differentiation. No other measures or manipulations were administered. The initial sample size of 200 was determined to ensure sufficient power to detect small-to-medium effects ($r \approx .20$) allowing for 25% drop-out over the 1-year study. Participants received 60€ for completing all lab tasks plus at least 80% of all ESM surveys in each wave, and a 60€ bonus for completing all three waves.

Participants attended the lab in small groups (2-8 people per session) where they completed a number of self-report questionnaires and computer tasks (not relevant to the current report). At the end of the lab session, participants received a Motorola Defy Plus smartphone running custom-built ESM software and were given detailed instructions for using the phone and completing the ESM questionnaire. Special emphasis was put on motivating participants to complete the ESM questionnaires as conscientiously as possible. Participants practiced completing the ESM questionnaire in the presence of an experimenter and could ask clarification questions before leaving the lab. For the next week, participants carried the smartphone with them during their daily activities and were prompted to respond to the ESM questionnaire 10 times per day. After one week, participants returned to the lab to give back their smartphones and complete additional lab tasks, including the film-task (see below).

ESM protocol. Smartphones were programmed to signal ('beep') participants 10 times each day for 7 days between 10 a.m. and 10 p.m. according to a stratified random interval scheme. On average, a beep occurred every 71.7 min (*SD* = 29.2 min). Participants completed 55-100% of all beeps (*M* = 87.27%, *SD* = 9.05%), reflecting very good compliance. At each beep, participants indicated how angry, sad, anxious, and depressed they felt at that moment using a visual slider scale from 0 (*not at all*) to 100 (*very much*). These four items were averaged to form a measure of NA. Following Nezlek (2012), we estimated the multilevel equivalent of Cronbach's alpha for the NA scale to be .71. Two items assessed the occurrence of positive and negative events: "think about the most [negative/positive] event that has occurred since the last beep. How intense was this event?" Responses to the event items were on continuous slider scales from 0 (*there was no event*) to 100 (*very negative*/*positive*). The ESM questionnaire also contained several items assessing the use of emotion regulation strategies. Each emotion regulation item began with "since the last beep,

have you" and was followed by "viewed the cause of your feelings from a different perspective?" (*cognitive reappraisal*), "suppressed the expression of your feelings" (*expressive suppression*), "distracted your attention away from your feelings" (*distraction*), "talked about your feelings with others" (*social sharing*), "brooded about something in the past" and "brooded about something in the future" (*rumination*). Responses to the emotion regulation items ranged from 0 (*not at all*) to 100 (*almost all the time*). The two rumination items were significantly positively correlated (within-persons r=.35; between-persons r=.68, ps<.001) and were averaged into a single rumination score. The ESM questionnaire comprised 24 items in total (including several items not relevant to the current study) and took approximately 1-2 min to complete.

Film-task. This task was adapted from Koval et al. (2013). Participants watched 10 emotional film-clips in a fixed order[3] and rated their subjective feelings following each film. Film-clips were selected from a validated database of emotional film stimuli (Schaefer, Nils, Sanchez, & Philippot, 2010). We made two modifications to the original task: First, we added an additional 20 second 'rest period' following each film-clip, during which participants were asked to keep their attention on the screen, and after which they again rated their affect. The addition of this rest period allowed us to calculate separate measures of emotional reactivity and recovery for each film-clip. Second, to keep the length of the task comparable with the original version, the time limit for responding to each affect item was reduced from 7 to 5 seconds. Participants reported their negative feelings using the same four items as in the ESM rated on a scale from 0 (*not at all*) to 6 (*very much*). These were averaged to form an NA scale, which we estimated to have an occassion-level reliability of .79 and a person-level reliability of .88.

[3] Four negative, four positive and two neutral films were shown in the following fixed order: negative, negative, neutral, positive, neutral, negative, positive, positive, negative, positive (see Koval et al., 2013 for the specific film-clips that were used).

Data Pre-Processing

Events in the ESM. We derived separate measures of *event occurrence* and *event intensity* from responses to the ESM event items (see above) following Schimmack and Diener (1997). For event occurrence, we created dummy variables where 1 indicated that an event had occurred since the previous beep, and 0 indicated no event. Because it may have been difficult for participants to indicate precisely a value of 0 on the smartphone touchscreen, we recoded all ratings ≤ 5 into 0s. This aimed to reduce the influence of measurement error and exclude very low intensity events. Ratings ≥ 6 were considered events and recoded into 1s. For the event intensity variables, we again recoded all scores ≤ 5 on the original event items to 0s, and subtracted 5 from all other ratings. Thus, a rating of 6 on the original item was recoded into a 1, and 100 was recoded into a 95, etc.

Exposure. Using the event occurrence and intensity variables, we calculated the following measures of exposure separately for each participant and separately for positive and negative events: the *frequency of events*, calculated as the number of events reported divided by the total number of completed ESM observations (i.e., the proportion), and the *mean intensity of events*, calculated as the average intensity rating across all ESM observations.

Reactivity. We operationalized NA reactivity using simple difference scores, such that positive reactivity scores indicate increases in NA whereas negative scores indicate decreases in NA in response to an event/film. For the film-task, we subtracted NA ratings completed before each film ($NA_{pre\text{-}film}$) from NA ratings completed immediately after each film (NA_{film}) and computed each participant's mean NA reactivity (mean NA_{film} – mean $NA_{pre\text{-}film}$) separately for negative and positive films.[4] For the ESM, recall that events were reported as occurring "since the previous beep." Thus, when an event was reported at time *t*, we calculated NA reactivity as $NA_t - NA_{t\text{-}1}$. We then computed each participant's mean NA

[4] For consistency with the ESM (where only positive & negative events were reported), we do not report data for neutral films. However, the results for neutral films were very similar to those found for positive films.

reactivity separately for negative and positive events. However, since participants could report events at both time *t* and time *t*−1, leading to a possible confound in the measure of reactivity, we calculated NA reactivity after excluding beeps on which two events of the same valence were reported on consecutive occasions (e.g., reactivity to negative events at *t* was calculated after excluding occasions on which a negative event was also reported at *t*−1).[5]

Recovery. Similarly, we operationalized recovery from events using difference scores, such that negative recovery scores indicated decreases in NA, whereas positive recovery scores indicated increases in NA in the period following an event/film. For the film-task, we subtracted NA ratings completed immediately after watching each film (NA_{film}) from NA ratings completed after the subsequent rest period ($NA_{post\text{-}film}$) and computed each participant's mean NA recovery score ($NA_{post\text{-}film}$) separately for negative and positive films. For the ESM, when an event was reported at time *t* (as having occurred since the previous beep) we calculated recovery as $NA_{t+1} - NA_t$. Each participant's mean NA recovery was calculated separately for negative and positive events. Following the same rationale as for reactivity, we calculated recovery in the ESM excluding data for which events of the same valence were reported on two consecutive occasions (e.g., to calculate recovery from negative events at *t*, we excluded occasions with negative events at *t*+1).[6]

Data Analyses

We used multilevel modeling for our main analyses to account for the hierarchical structure of the ESM and film-task data, in which occasions/beeps (Level-1) were nested within participants (Level-2). At Level-1, we modeled NA inertia using a first-order autoregressive model (Butler, 2011; Gottmann et al., 2005; Kuppens, Allen, et al., 2010; Suls

[5] Excluding observations with events at *t*−1 resulted in a reduced sample size: reactivity to negative events could be calculated for 188 participants with an average of 7.01 negative events each (*SD* = 4.16); reactivity to positive events could be calculated for 168 participants, with an average of 6.11 positive events each (*SD*=3.54).

[6] Excluding observations with events at *t*+1 resulted in a reduced sample size: recovery from negative events could be calculated for 187 participants with an average of 7.56 negative events each (*SD*=4.21); recovery from positive events could be calculated for 170 participants, with an average of 6.48 positive events each (*SD*=3.92).

et al., 1998). Autoregressive slopes representing NA inertia were allowed to vary randomly across participants and their associations with person-level measures of exposure, reactivity, and recovery were modeled at Level-2. Model equations were as follows:

Level-1 (occassions):

$$NA_{tj} = \pi_{0j} + \pi_{1j} (NA_{t-1j}) + e_{tj}$$

Level-2 (persons):

$$\pi_{0j} = \beta_{00} + \beta_{01} (X_j) + r_{0j}$$

$$\pi_{1j} = \beta_{10} + \beta_{11} (X_j) + r_{1j}$$

At Level-1, the outcome NA_{tj} (person *j*'s level of NA at time *t*) was modeled as a function of an intercept (π_{0j}), and an autoregressive slope (π_{1j}) representing the effect of NA_{t-1j} (person j's level of NA at time $t-1$). Because the lagged predictor, NA_{t-1j}, was person-mean centered the intercept (π_{0j}) reflects person *j*'s mean level of NA. All person-level predictors, represented by the generic symbol X_j, were standardized before being entered at Level-2.[7] As a consequence (and similar to the effect of grand-mean centering), the Level-2 intercepts (β_{00}) and (β_{10}) reflect the mean level and inertia of NA, respectively, at the average value of the Level-2 predictor, X_j. The Level-2 slopes (β_{01}) and (β_{11}) are standardized regression weights representing associations between the person-level predictor, X_j, and the mean level and inertia of NA, respectively. We ran separate analyses modeling NA inertia in the ESM and film-task at Level-1, and separately examining each measure of exposure, reactivity, and recovery as predictors of NA inertia at Level-2.

[7] As can be expected for measures of affective responding, we observed large individual differences on all measures of affective responding (e.g., Davidson, 1998). To reduce the influence of outliers on estimated associations with NA inertia, we winsorized all person-level predictors by replacing scores more than 3 SDs above or below the sample mean with values equal to the mean +/- 3 *SD*s. In total, 207 out of a total of 3826 person-level scores (5.4%) were winsorized. Importantly, we repeated all analyses using the raw (unwinsorized) data and obtained very similar results, supporting identical conclusions. In fact, our main findings were stronger when analyses were conducted using raw scores. Results reported in Tables 2-4 are based on winsorized scores and may therefore slightly underestimate the true size of the associations.

Controlling for baseline NA. Although difference scores are intuitive and easily interpretable measures of emotional reactivity and recovery, they may be influenced by individual differences in baseline level of emotion (Nelson, Shankman, Olino, & Klein, 2011). For instance, individual differences in NA reactivity to an event may be influenced by baseline levels of NA prior to the event. To address this, we controlled for pre-event/film NA mean level in a second step of analyses examining associations between reactivity and inertia. Specifically, at 'Step 2' of reactivity models, we added the mean level of $NA_{t\text{-}1}$ (i.e., before an event/film) as a covariate, as shown below:

Level-2 Model for Reactivity at Step 2:

$$\pi_{0j} = \beta_{00} + \beta_{01}\,(\text{NA Reactivity}_j) + \beta_{02}\,(\text{Mean NA}_{t\text{-}1j}) + r_{0j}$$

$$\pi_{1j} = \beta_{10} + \beta_{11}\,(\text{NA Reactivity}_j) + \beta_{12}\,(\text{Mean NA}_{t\text{-}1j}) + r_{1j}$$

Similarly, in a second step of analyses examining associations between recovery and inertia, we controlled for the influence of NA level during events/films. As shown below, at 'Step 2' of recovery models, we added the mean level of NA_t (i.e., concurrent with events/films) as a covariate:

Level-2 Model for Recovery at Step 2:

$$\pi_{0j} = \beta_{00} + \beta_{01}\,(\text{NA Recovery}_j) + \beta_{02}\,(\text{Mean NA}_{tj}) + r_{0j}$$

$$\pi_{1j} = \beta_{10} + \beta_{11}\,(\text{NA Recovery}_j) + \beta_{12}\,(\text{Mean NA}_{tj}) + r_{1j}$$

Controlling for emotion regulation. Given that exposure, reactivity, and recovery may be influenced, to some extent, by emotion regulation processes, we additionally controlled for individual differences in the use of emotion regulation strategies in daily life. Specifically, we calculated mean scores for each emotion regulation strategy across all ESM

occasions and included these as additional, grand-mean centered, Level-2 covariates in a final step of all analyses.

Results

Descriptive Statistics and Correlations

Table 1 displays means and standard deviations of the exposure, reactivity, and recovery measures (raw and winsorized), as well as correlations between all winsorized measures. Correlations between lab and ESM measures were very weak (non-significant), whereas associations between different measures within each paradigm (e.g., between reactivity and recovery assessed in the lab) were considerably stronger. We estimated mean levels and standard deviations of NA inertia across the sample using preliminary multilevel models similar to those described above, but without Level-2 predictors. The mean level of NA inertia in the ESM was 0.30, *SD* = 0.20, 95% CI [0.26, 0.34]. In the film-task, the mean level of NA inertia was 0.26, *SD* = 0.11. 95% CI [0.22, 0.29]. The two measures of NA inertia correlated at r=.24, p=.001.[8]

Associations between NA Inertia and Exposure, Reactivity, and Recovery

Tables 2–4 display results of multilevel models testing associations between exposure (Table 2), reactivity (Table 3), and recovery (Table 4) with NA inertia. Associations with NA inertia in daily life (i.e., ESM) are shown in the top half of each table, and associations with NA inertia in the lab (i.e., Film-Task) are shown in the bottom half of each table. Because exposure, reactivity and recovery scores were standardized, regression weights in Tables 2–4 can be interpreted as follows: a regression weight of β=0.05 indicates that an individual

[8] Because there were significant linear time trends in both the ESM and film-task data, we repeated all analyses including time at Level-1 (with a random effect at Level-2). After adjusting for the linear time trend, mean levels of NA inertia were lower in both the ESM (β_{10} = 0.27, *SD* = 0.20, 95% CI [0.24, 0.31]) and film-task (β_{10} = 0.10, *SD* = 0.11, 95% CI [0.07, 0.14]). However, these adjusted NA inertia estimates (controlling for time) correlated very strongly with the original (unadjusted) NA inertia estimates (ESM: r=.99, p<.001; film-task: r=.91, p<.001) Importantly, the results of our main analyses (reported below, see also Tables 2-4) were highly similar and supported identical conclusions when controlling for the linear effect of time. For simplicity, results reported in Tables 2-4 are based on models not including time.

scoring one *SD* above the sample average on a given predcitor (e.g., reactivity) is predicted to have an NA autocorrelation .05 higher than the average NA autocorrelation (see above), indicating greater NA inertia. In contrast, an individual scoring one *SD* below the sample average on a given predictor (e.g., reactivity) is predicted to have an NA autocorrelation .05 lower than average, reflecting lesser NA inertia.

Exposure. As shown in Table 2, we found no statistically significant associations between frequency of events and NA inertia in the ESM or film-task. However, mean intensity of negative events was significantly positively associated with NA inertia in the ESM, indicating that participants who reported more intense negative events tended to have higher NA inertia in daily life. This association was independent of mean use of emotion regulation in daily life (see Step 2). Mean intensity of negative events was not related to NA inertia in the film-task. In contrast, mean intensity of positive events was significantly negatively related to NA inertia in the film-task, although this effect became marginally significant after controlling for mean use of emotion regulation (see Step 2). Finally, mean intensity of positive events was unrelated to NA inertia in daily life.

Reactivity.

Reactivity to events in daily life. As shown in Table 3, none of the simple associations between reactivity to events in daily life and NA inertia in the ESM or film-task were statistically significant (see Step 1). However, at Step 2 (controlling for pre-event NA level) there was a marginally significant (p = .055) positive association between reactivity to positive events and NA inertia in the ESM, which became statistically significant (p = .007) after additionally controlling for mean use of emotion regulation (see Step 3).

Reactivity to films in the lab. We found a marginally significant negative association between reactivity to positive films and ESM inertia. Note that this association was in the opposite direction to the finding for reactivity to positive events in daily life, reported above.

Importantly, this effect was no longer evident after controlling for pre-film NA level (see Step 2) and mean use of emotion regulation strategies in daily life (see Step 3). We found no significant associations between reactivity to negative films and NA inertia in the ESM.

Regarding associations between reactivity to films and NA inertia in the film-task, we found a significant negative association between reactivity to negative films and NA inertia in the lab (see Step 1), which was independent of pre-film NA level (see Step 2) and use of emotion regulation in daily life (see Step 3). This indicates that smaller increases in NA in response to negative films (i.e., blunted reactivity) were associated with higher levels of NA inertia in the lab, as hypothesized by Kuppens and colleagues (Kuppens, Allen, et al., 2010; Kuppens et al., 2012). Reactivity to positive films was also negatively related to NA inertia in the film-task (see Step 1), even after controlling for pre-film NA level (see Step 2), as well as the use of emotion regulation strategies in daily life (see Step 3). Note that because, on average, NA decreased in response to positive films (see Table 1), this finding implies that stronger reactivity (i.e., larger decreases in NA in response to positive films) was related to higher NA inertia in the film-task, which is opposite to the finding for reactivity to negative films (discussed further below).

Recovery.

Recovery from events in daily life. As shown in Table 4, we found a marginally significant positive association (p = .071) between recovery from negative events and NA inertia in the ESM (see Step 1), which became statistically significant (p = .016) after controlling for NA mean level concurrent with negative events (see Step 2) and remained significant (p = .022) after controlling for mean use of emotion regulation strategies in daily life (see Step 3). Note that although NA decreased by approximately 3 scale points in the period following negative events, on average, individuals differed widely in their degree of NA recovery from negative events, with some participants showing no change or even

increases in NA (see Table 1). Thus, the positive association between NA recovery and NA inertia in daily life indicates that individuals who experienced smaller decreases/larger increases in NA in the period following negative events (i.e., impaired recovery from negative events) showed higher levels of NA inertia in daily life. Recovery from positive events was unrelated to NA inertia in daily life.

Regarding associations between recovery from events in daily life and NA inertia in the film-task, we found a significant positive association between recovery from negative events and NA inertia in the film-task, but only after statistically controlling for NA level concurrent with negative events (see Step 2). This association remained significant after controlling for mean use of emotion regulation in daily life (see Step 3). This finding indicates that individuals displaying smaller decreases/larger increases in NA (i.e., impaired recovery) following negative events also tended to have higher levels of NA inertia in the lab, consistent with our findings for NA inertia in daily life. Recovery from positive events was not related to NA inertia in the film-task.

Recovery from films in the lab. We found a marginally significant (p = .08) positive association between recovery from negative films and NA inertia in daily life after controlling for NA level during negative films (see Step 2). This effect remained marginally significant at Step 3 after additionally controlling for mean use of emotion regulation in daily life (p = .097). This result is consistent with the associations between recovery from negative events and NA inertia reported above. Recovery from positive films was not associated with NA inertia in the ESM. Recovery from positive films was not related to NA inertia in daily life.

Regarding NA inertia in the lab, recovery from negative films was positively related to NA inertia in the film-task (see Step 1), independent of differences in NA level during negative films (see Step 2) and independent of mean use of emotion regulation strategies in daily life (see Step 3). Consistent with our other findings for recovery from negative

events/films, this indicates that individuals showing a pattern indicative of impaired NA recovery (i.e., smaller decreases or larger increases in NA) after negative films also tended to have higher NA inertia in the lab. Recovery from positive films was also positively related to NA inertia in the film-task, even after controlling for NA level during positive films (see Step 2) and mean emotion regulation use (see Step 3). Note that in the context of positive films, greater "recovery" implies a stronger increase in NA following the offset of positive stimuli.[9]

Follow-up analyses: Combined models. Given the associations between exposure, reactivity, and recovery (particularly within each paradigm; see Table 1), we sought to identify the unique roles of each process in relation to NA inertia. We therefore ran two additional multilevel models including all predictors that showed (marginally) significant associations with NA inertia in the separate analyses, reported above. First, we examined unique associations with NA inertia in daily life by entering the mean intensity of negative events, reactivity to positive events and films, and recovery from negative events and films, as simultaneous predictors of NA inertia in the ESM.[10] NA inertia in daily life was significantly independently associated with mean intensity of negative events ($\beta = 0.05$, $SE = 0.02$, $p < .015$), and marginally with recovery from negative events ($\beta = 0.03$, $SE = 0.02$, $p = .086$). No other predictors showed independent effects (ps > .58). Second, we examined the unique predictors of NA inertia in the lab by entering mean intensity of positive events, reactivity to positive and negative films, recovery from negative events, and recovery from positive and

[9] The analyses reported above were limited to linear relationships. However, there may also be non-linear (e.g., quadratic) associations between NA inertia and exposure, reactivity, and recovery (e.g., Thompson et al., 2012). For instance, one might postulate an inverse U-shaped relationship between NA inertia and reactivity to negative events, such that individuals whose experience no change in NA in response to negative events have the highest NA inertia, whereas individuals displaying either an increase or decrease in NA have lower levels of NA inertia. To explore this possibility, we repeated all analyses additionally including quadratic effects for all predictors. We found only three statistically significant quadratic effects, two of which reflected very subtle deviations from the linear associations reported above. The third was an inverse U-shaped association between NA recovery from positive events and NA inertia in the ESM.

[10] $N = 166$ for this analysis, due to missing data for measures of reactivity and recovery in the ESM (at Level-2).

negative films, as simultaneous predictors of NA inertia in the film-task.[11] Recovery from negative films was significantly independently related to NA inertia in the film-task ($\beta = 0.11$, $SE = 0.02$, $p < .001$). No other predictors showed significant independent effects (ps > .10).

Discussion

Emotional inertia is increasingly coming to be seen as an indicator of affective dysfunction, and in particular of increased vulnerability to depression (van de Leemput et al., 2013; Kuppens et al., 2012; Koval et al., 2012). Despite its potential importance, few studies have investigated the processes underlying emotional inertia. To our knowledge, the current study was the first to systematically investigate the roles of exposure, reactivity, and recovery from events in relation to NA inertia. Below, we first recap and interpret our findings separately for exposure, reactivity, and recovery and then summarize our findings and discuss the general implications of the current study.

Exposure

We found only limited evidence for an association between NA inertia and exposure to events in daily life. Contrary to our predictions, the frequency with which people encountered positive and negative events was not related to their levels of NA inertia either in daily life or in the lab. In contrast, and as predicted, individuals who reported encountering more intense negative events displayed higher levels of NA inertia in the ESM (but not in the film-task), and this association was independent of their levels of reactivity and recovery (see results of combined models). In contrast, participants who reported encountering less intense positive events had higher levels of NA inertia in the film-task (but not in the ESM). However, this association was no longer statistically significant after controlling for other predictors of NA inertia in the film-task (see results of combined models). Thus, although the quantity of events people encounter in daily life was not related to NA inertia, self-reported

[11] $N = 187$ for this analysis, due to missing data for recovery from negative events in daily life (at Level-2).

intensity of events does appear to play an independent role in heightened NA inertia in daily life. Given the nature of self-reports, it is unclear whether individuals with higher NA inertia encounter objectively more intense negative events, or whether this effect is (partly) driven by cognitive appraisal biases (Joorman & Siemer, 2011).

Reactivity

The evidence for an association between reactivity and NA inertia was also limited. First, we found no support for an association between reactivity to negative stimuli (either daily events or films) and NA inertia in daily life. Second, our findings regarding reactivity to positive stimuli in relation to NA inertia in the ESM were inconsistent across methods (i.e., opposite findings for reactivity to events vs. films). Although some divergence between the film-task and ESM can be expected given the methodological differences between these paradigms (discussed below), these inconsistent findings make it difficult to draw clear conclusions regarding the association between reactivity and NA inertia in daily life.

We found stronger evidence for associations between reactivity to films and NA inertia in the lab. Specifically, higher NA inertia in the film-task was related to blunted reactivity to negative films and increased reactivity to positive films, independent of pre-film NA levels. Thus, rather than displaying a general insensitivity to external stimuli (cf. Kuppens, Allen, et al., 2010), individuals with higher NA inertia displayed blunted reactivity to negative stimuli but greater sensitivity to positive stimuli. However, there is an important caveat: after controlling for individual differences in exposure and recovery, reactivity was no longer associated with NA inertia in the ESM or film-task (see results of combined models). Thus, whatever role reactivity may play in heightened NA inertia does not appear to be independent of exposure and recovery. In sum, our findings do not clearly support the hypothesized roles of either increased or blunted reactivity in NA inertia, but rather line up with Thompson et al.'s (2012) finding that NA inertia and reactivity are independent.

Recovery

The most consistent correlate of NA inertia in the current study was recovery from negative stimuli. As predicted, the less a person's NA decreased in the period following negative events or films, indicating impaired NA recovery, the higher their level of NA inertia. This finding was most pronounced when examining the relationship between decreased recovery from negative films and NA inertia in the lab, although reduced recovery from negative events was also consistently related to higher NA inertia in daily life. Importantly, these effects were partly independent from individual differences in exposure and reactivity (see results of combined models). Associations across paradigms (i.e., recovery from negative events in daily life in relation to inertia in the lab, or vice versa) were markedly weaker and did not remain after controlling for other predictors of NA inertia. Nevertheless, these findings suggest that impaired recovery from negative emotional events may play an important role in heightened NA inertia (Suls et al., 1998; Suls & Martin, 2005). Given that NA inertia is predictive of future clinical depression (Kuppens et al., 2012; van de Leemput et al., 2014) the current findings are consistent with previous research that has linked impaired recovery from negative stimuli with increased vulnerability to affective disorders (Hemenover, 2003; Pezawas et al., 2005; Schuyler et al., 2012).

Impaired recovery from negative stimuli may result from cognitive biases towards negative information, such as those associated with depression (Mathews & MacLeod, 2005) and neuroticism (Chan, Goodwin, & Harmer, 2007). For example, an elaborative attentional bias towards negative stimuli was recently found to predict impaired NA recovery (Clasen, Wells, Ellis, & Beevers, 2013). Cognitive biases towards negative information are also thought to underlie rumination, a maladaptive response style that impedes NA recovery (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008), which has also been related to increased NA inertia (Koval et al., 2012; Brose, Kuppens, Koval, & Schmiedek, in press). Furthermore,

even when individuals attempt to use putatively more adaptive strategies to down-regulate NA (e.g., cognitive reappraisal), cognitive biases may decrease their effectiveness (Pe, Raes, Koval, Brans, Verduyn, & Kuppens, 2013). In sum, cognitive biases may impair NA recovery either by promoting ineffective responses to negative events or by interfering with the success of normatively successful strategies for the down-regulation of NA.

Regarding recovery from positive stimuli, we found no associations with NA inertia in the ESM. However, NA inertia in the film-task was related to greater NA recovery following positive films. It is important to note that, in this context, greater recovery implies a stronger rebound in NA following positive stimuli. This suggests that the mood brightening effects of positive stimuli dissipate more quickly among individuals with heightened NA inertia, which could be seen as maladaptive. For instance, neuroticism has been associated with larger increases in NA following the offset of positive stimuli (Hemenover, 2003).

Divergence between Findings in the Lab and in Daily Life

As mentioned above, our findings from the film-task were not entirely consistent with our findings from the ESM. More generally, measures of reactivity and recovery in the lab were largely unrelated to the same parameters assessed in daily life (see Table 1). Indeed, this is not the first study to report considerable differences between affective responding to standardized stimuli presented in the lab versus idiographic events encountered in daily life (e.g., van Eck, Nicolson, Berkhof, & Sulon, 1996; see also Bylsma & Rottenberg, 2011). Such inconsistencies may appear to undermine the validity of studying affective processes under “artificial” laboratory conditions. However, given the difficulties in reliably measuring affective functioning in daily life, controlled lab paradigms may still be of value, particularly when combined with naturalistic methods. Differences in timescale may contribute to the divergence between lab and ESM findings. Specifically, minute-to-minute changes in affect captured by the film-task may be driven by emotional processes, whereas affective

fluctuations on a scale of hours (as measured in the ESM) may reflect changes in mood (Rottenberg, 2005). Thus, measures of affect dynamics (e.g., autocorrelation) may reflect different processes when captured at different timescales (Hollenstein, Lichtwark-Aschoff, & Potworowski, 2013; Koval et al., 2013). In light of these substantial differences between paradigms, it is remarkable that we found a moderate positive correlation between NA inertia in the lab and in daily life, closely replicating Koval et al.'s (2013) findings. Also noteworthy is that, despite such differences, we found consistent evidence across both paradigms for an independent association between impaired recovery from negative stimuli and NA inertia.

Limitations and Future Directions

The current study represents an important first step towards examining the role of contextual factors in NA inertia. However, it is not without limitations. First, we collected little information about the specific nature of events in the ESM, restricting the kinds of distinctions that could be drawn between events. Events that differing on various appraisal dimensions (e.g., importance, agency, certainty, etc.) or contextual features (e.g., social context, interaction partners, etc.) may evoke distinct emotional responses, and may therefore play be differentially associated with how emotions fluctuate over time. For instance, individuals with higher inertia may react more strongly to certain kinds of negative events (e.g., interpersonal stressors) but show blunted reactivity to other kinds of negative events. This might account for the overall lack of a clear association between NA inertia and reactivity in the current study. Thus, a major challenge for future research on affect dynamics is to fully account for the complexity of real-world social contexts in which affective processes emerge (Butler, 2011; Boiger & Mesquita, 2013).

Second, it is unclear which factor(s) are responsible for our divergent findings in the film-task versus the ESM (discussed above). More research assessing emotional processes both in the lab and in daily life among the same participants is needed to better understand the

strengths and limitations of each and examine issues of generalizability of findings. Alternatively, it is possible to combine experimental and naturalistic methods by, for instance, manipulating an event in daily life and repeatedly assessing affect before and after the event (see e.g., Koval & Kuppens, 2012). This would ensure that all participants were exposed to the same event and yet maximize ecological validity by assessing emotional processes in daily life.

Third, our analyses controlling for emotion regulation suggest that the current findings are independent of individual differences in the mean use of five specific emotion regulation strategies. However, this does not indicate that emotion regulation processes do not play an important role in emotional inertia (see e.g., Koval et al., 2012; 2014). Future research should go beyond examining associations at the trait level and beyond merely examining how much people report using various emotion regulation strategies, and consider how effectively and flexibly people are able to implement emotion regulation across various contexts (Aldao, 2013).

Finally, our operationalization of reactivity and recovery (i.e., difference scores) relied on just two measurements of affect for each event or film. As a result, we may not have captured the full complexity of affective change that characterizes either reactivity or recovery. Indeed, emotional profiles may be more complicated than the linear increases or decreases captured by simple difference scores (e.g., Verduyn, Van Mechelen, Tuerlinckx, Meers, & Van Coillie, 2009). Similarly, emotional events may persist for prolonged periods or may be recurrent. A major challenge for future studies is to account for this temporal complexity without increasing participant-burden or measurement reactivity.

Conclusion

In sum, the findings of the current study suggest that NA inertia is a complex phenomenon, which may be driven by multiple processes depending on the context and the

timescale on which it observed. Thus, the findings of this study highlight the need to approach affect dynamics using multiple methods, study designs, and paradigms. Notwithstanding such complexity, we found consistent evidence, across different paradigms and analytic methods, for the role of impaired recovery from negative events in heightened NA inertia.

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Table 1

Pearson Correlations among Exposure, Reactivity, and Recovery Measures in the ESM and Film-Task

		Raw Data		Winsorized Data		Correlations										
Measure	*N*	*Range*	*M* (*SD*)	*Range*	*M* (*SD*)	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*
Negative Events (ESM)																
1. Mean intensity	200	1.09 to 54.31	14.16 (9.45)	1.09 to 42.51	14.10 (9.24)	1.00										
2. Frequency (proportion)	200	0.05 to 1.00	0.64 (0.28)	0.05 to 1.00	0.64 (0.28)	**.58**	1.00									
3. Reactivity (no event at *t*−1)	188	-16.75 to 23.38	2.76 (5.49)	-13.71 to 19.24	2.74 (5.29)	.02	**-.19**	1.00								
4. Recovery (no event at *t*+1)	187	-54.5 to 19.88	-3.11 (5.77)	-20.41 to 14.18	-2.96 (4.40)	-.12	.04	**-.28**	1.00							
Positive Events (ESM)																
5. Mean intensity	200	0.26 to 78.5	31.26 (16.08)	0.26 to 78.5	31.26 (16.08)	**.47**	**.27**	-.02	-.02	1.00						
6. Frequency (proportion)	200	0.04 to 1.00	0.78 (0.23)	0.09 to 1.00	0.78 (0.23)	**.50**	**.82**	-.17	.04	**.57**	1.00					
7. Reactivity (no event at *t*−1)	168	-90.75 to 42.25	-1.35 (9.94)	-31.17 to 28.47	-1.09 (7.01)	-.19	.01	-.04	.11	-.14	-.01	1.00				
8. Recovery (no event at *t*+1)	170	-24.5 to 96.75	1.27 (9.39)	-24.5 to 29.42	0.87 (6.22)	**.28**	-.01	**.27**	-.12	.06	-.03	**-.42**	1.00			
Negative Films (Film-Task)																
9. Reactivity	200	-0.25 to 4.13	1.33 (0.79)	-0.25 to 3.70	1.33 (0.78)	.05	.04	.07	.00	.15	.09	.04	-.03	1.00		
10. Recovery	200	-3.63 to 0.75	-0.88 (0.61)	-2.70 to 0.75	-0.87 (0.59)	-.10	-.04	-.08	.08	**-.19**	-.11	-.04	-.02	**-.73**	1.00	
Positive Films (Film-Task)																
11. Reactivity	200	-2.06 to 0.94	-0.18 (0.40)	-1.37 to 0.94	-0.17 (0.37)	-.12	-.10	.02	-.09	-.08	-.10	.09	-.01	**-.21**	**-.24**	1.00
12. Recovery	200	-0.75 to 1.56	-0.06 (0.25)	-0.75 to 0.70	-0.06 (0.22)	.10	.06	-.08	.06	-.03	.05	-.04	.03	**-.44**	**.33**	**-.36**

Note. *N*s differ due to missing data for participants who reported no ESM occasions on which a negative/positive event occurred at time *t* but no event of the same valence occurred at time *t*−1 (reactivity) or *t*+1 (recovery).
Correlations are based on winsorized data.
Correlations statistically significant at $p < .01$ are shown in **bold**.

Table 2
Standardized Regression Weights Reflecting Associations between Exposure and NA Inertia

	Step 1				Step 2 (control: Regulation)			
		95% CI				95% CI		
Model	β (*SE*)	*LL*	*UL*	*p*	β (*SE*)	*LL*	*UL*	*p*
NA Inertia in Daily Life (ESM)								
Frequency of Negative Events	0.02 (0.02)	-0.02	0.06	.382	0.01 (0.02)	-0.03	0.06	.564
Frequency of Positive Events	0.00 (0.02)	-0.04	0.04	.907	-0.01 (0.02)	-0.06	0.04	.704
Intensity of Negative Events	0.05 (0.02)	0.01	0.09	.009	0.05 (0.02)	0.01	0.09	.029
Intensity of Positive Events	-0.01 (0.02)	-0.05	0.03	.609	-0.02 (0.02)	-0.06	0.02	.382
NA Inertia in the Lab (Film-Task)								
Frequency of Negative Events	0.00 (0.02)	-0.04	0.04	.941	0.01 (0.02)	-0.03	0.06	.541
Frequency of Positive Events	-0.02 (0.02)	-0.06	0.02	.135	-0.02 (0.02)	-0.06	0.02	.368
Intensity of Negative Events	0.01 (0.01)	-0.01	0.03	.715	0.00 (0.02)	-0.03	0.03	.812
Intensity of Positive Events	-0.04 (0.01)	-0.06	-0.02	.008	-0.03 (0.02)	-0.06	0.00	.059

Note. $N = 200$ for all analyses
Step 1: simple associations between NA reactivity and NA inertia;
Step 2: associations between exposure and NA inertia controlling for mean use of Emotion Regulation strategies in daily life.

Table 3

Standardized Regression Weights Reflecting Associations between Reactivity and NA Inertia

		Step 1				Step 2 (control: NA_{t-1})				Step 3 (control: NA_{t-1} & Regulation)			
			95% CI				95% CI				95% CI		
Model	*N* (Level-2)	β (*SE*)	*LL*	*UL*	*p*	β (*SE*)	*LL*	*UL*	*p*	β (*SE*)	*LL*	*UL*	*p*
NA Inertia in Daily Life (ESM)													
Reactivity to Negative Events	188	-0.01 (0.02)	-0.05	0.03	.602	0.00 (0.02)	-0.04	0.04	.935	0.00 (0.02)	-0.04	0.04	.958
Reactivity to Positive Events	168	0.01 (0.02)	-0.03	0.05	.459	0.04 (0.02)	0.00	0.08	.055	0.05 (0.02)	0.01	0.09	.007
Reactivity to Negative Films	200	0.00 (0.02)	-0.04	0.04	.881	0.00 (0.02)	-0.04	0.04	.884	0.00 (0.02)	-0.04	0.03	.885
Reactivity to Positive Films	200	-0.04 (0.02)	-0.08	0.00	.056	-0.02 (0.02)	-0.06	0.02	.323	-0.03 (0.02)	-0.07	0.02	.207
NA Inertia in the Lab (Film-Task)													
Reactivity to Negative Events	188	0.01 (0.01)	-0.01	0.03	.328	0.02 (0.01)	0.00	0.04	.134	0.01 (0.02)	-0.02	0.04	.548
Reactivity to Positive Events	168	0.00 (0.02)	-0.04	0.04	.812	0.00 (0.02)	-0.04	0.04	.828	0.00 (0.02)	-0.03	0.04	.765
Reactivity to Negative Films	200	-0.05 (0.02)	-0.09	-0.01	.004	-0.05 (0.02)	-0.09	-0.01	.004	-0.05 (0.02)	-0.08	-0.01	.005
Reactivity to Positive Films	200	-0.06 (0.01)	-0.08	-0.04	< .001	-0.08 (0.02)	-0.12	-0.04	< .001	-0.07 (0.02)	-0.11	-0.04	< .001

Note. Level-2 *N*s differ due to missing data for participants with no ESM occasions on which they reported a negative/positive event at time *t* and no event of the same valence at time *t*−1.
Step 1: simple associations between reactivity and NA inertia;
Step 2: associations between reactivity and NA inertia controlling for mean level of NA at time *t*−1 (i.e., before events/films);
Step 3: associations between reactivity and NA inertia controlling for mean level of NA before films/events and mean use of Emotion Regulation strategies in daily life.

Table 4

Standardized Regression Weights Reflecting Associations between Recovery and NA Inertia

		Step 1				Step 2 (control: NA_t)				Step 3 (control: NA_t & Regulation)			
			95% CI				95% CI				95% CI		
Model	*N* (Level-2)	β (*SE*)	*LL*	*UL*	*p*	β (*SE*)	*LL*	*UL*	*p*	β (*SE*)	*LL*	*UL*	*p*
NA Inertia in Daily Life (ESM)													
Recovery from Negative Events	187	0.03 (0.02)	-0.01	0.07	.071	0.05 (0.02)	0.01	0.09	.016	0.05 (0.02)	0.01	0.09	.022
Recovery from Positive Events	170	0.00 (0.02)	-0.04	0.04	.992	0.00 (0.02)	-0.04	0.04	.835	0.01 (0.02)	-0.03	0.05	.596
Recovery from Negative Films	200	0.02 (0.02)	-0.02	0.06	.282	0.04 (0.02)	0.00	0.08	.080	0.04 (0.02)	-0.01	0.08	.097
Recovery from Positive Films	200	0.01 (0.02)	-0.03	0.05	.524	0.02 (0.02)	-0.02	0.06	.427	0.01 (0.02)	-0.03	0.05	.547
NA Inertia in the Lab (Film-Task)													
Recovery from Negative Events	187	0.02 (0.01)	0.00	0.04	.261	0.04 (0.02)	0.00	0.08	.015	0.04 (0.01)	0.01	0.07	.011
Recovery from Positive Events	170	0.00 (0.02)	-0.04	0.04	.787	0.01 (0.02)	-0.03	0.05	.748	0.00 (0.02)	-0.03	0.03	.963
Recovery from Negative Films	200	0.10 (0.01)	0.08	0.12	< .001	0.11 (0.02)	0.07	0.15	< .001	0.12 (0.02)	0.09	0.15	< .001
Recovery from Positive Films	200	0.05 (0.01)	0.03	0.07	< .001	0.05 (0.02)	0.01	0.09	.002	0.04 (0.02)	0.01	0.07	.017

Note. Level-2 *N*s differ due to missing data for participants with no ESM occasions on which they reported a negative/positive event at time *t* and no event of the same valence at time *t*+1.
Step 1: simple associations between recovery and NA inertia;
Step 2: associations between recovery and NA inertia controlling for mean level of NA at time *t* (i.e., concurrent with events/films);
Step 3: associations between recovery and NA inertia controlling for mean level of NA before films/events and mean use of Emotion Regulation strategies in daily life.