NEURAL AND SELF-REPORTED REWARD RESPONSIVENESS ARE ASSOCIATED WITH DISPOSITIONAL AFFECTIVITY AND EMOTION DYSREGULATION IN ADOLESCENTS WITH EVIDENCE FOR CONVERGENT AND INCREMENTAL VALIDITY

Evelin A. Zubovics[1], Richárd Fiáth[2,3], Alexandra Rádosi[1,4], Bea Pászthy[5], János M. Réthelyi[6], István Ulbert[2,3], & Nóra Bunford[1]

[1] 'Lendület' Developmental and Translational Neuroscience Research Group, Institute of Cognitive Neuroscience and Psychology, Research Centre for Natural Sciences, 1117 Budapest, Magyar Tudósok körútja 2, Hungary

[2] Comparative Psychophysiology Research Group, Institute of Cognitive Neuroscience and Psychology, Research Centre for Natural Sciences, 1117 Budapest, Magyar Tudósok körútja 2, Hungary

[3] Faculty of Information Technology and Bionics, Pázmány Péter Catholic University, 1083 Budapest, Práter utca 50/A, Hungary

[4] Semmelweis University, Doctoral School of Mental Health Sciences, 1083 Budapest, Balassa u. 6, Hungary

[5] Semmelweis University, 1st Department of Paediatrics, 1083 Budapest, Bókay János u. 53-54, Hungary

[6] Semmelweis University, Department of Psychiatry and Psychotherapy, 1083 Budapest, Balassa u. 6, Hungary.

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Correspondence concerning this article should be addressed to Nóra Bunford at bunford.nora@ttk.hu.

Abstract

Adolescence is a developmental period characterized by heightened reward sensitivity which, in turn, confers risk for pertinent negative outcomes, underscoring need to better understand biological bases and behavioral correlates of reward responsiveness during this developmental phase. Our goals in the current study were to examine, in a sample of 43 typically developing adolescents (M_{age}=15.67 years; *SD*=1.01; 32.6% boys), (1) evidence of convergent validity between neural and self-report reward responsiveness, (2) associations between neural reward responsiveness and self-report dispositional affectivity and emotion dysregulation (ED) and (3) evidence of incremental validity of self-report beyond neural reward responsiveness in predicting affectivity and ED. During electroencephalography (EEG), adolescents completed two experimental paradigms probing event-related potential (ERP) indices of reward anticipation and initial responsiveness to reward attainment. Following EEG, they completed self-report measures of reward responsiveness, affectivity, and ED. Findings indicated some evidence of convergent validity between enhanced ERP indices of reward anticipation and initial response to reward and greater reinforcement sensitivity; that ERP indices of both reward responsiveness aspects predicted lower negative affectivity and less ED; and evidence of incremental validity of self-report beyond neural reward responsiveness in predicting outcomes. Results underscore utility of a multi-method framework in assessing adolescent reward responsiveness and support the relevance of reward responsiveness in explaining individual differences in dispositional affectivity and ED.

Keywords: adolescent, reward responsiveness, ERP, affectivity, emotion dysregulation

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The functioning and underlying reactivity of an architecture of attention- and motivation-regulating systems (described in the most recent formulation of the Reinforcement Sensitivity Theory (RST); McNaughton & Corr, 2004; McNaughton & Gray, 2000) is conceptualized as contributing to individual differences in temperament (Derryberry & Rothbart, 1997). As part of these attention- and motivation-regulating systems, the behavioral activation system (BAS), activated by the potential for or receipt of reward, is conceptualized to regulate reward processing (Dillon et al., 2014) (*Note.* reward-driven processes are only one part of motivation and motivated behavior, and motivated behaviors are only one part of reward processing). The BAS is often contrasted with the behavioral inhibition system (BIS), activated by approach-avoidance conflict, and hypothesized to regulate response to punishment and threat (Bunford, Roberts, Kennedy, & Klumpp, 2017).

Differences in BAS sensitivity – drive, reward responsiveness, and reward sensitivity – are reflected in Research Domain Criteria (RDoC) Positive Valence Systems (PVS) characteristics (Olino et al., 2018). Drive can be conceptualized as a marker of effort valuation (i.e., processes by which *value of a reinforcer is computed* as a function of its magnitude and perceived costs of the effort required to obtain it; PVS Work Group, 2011); reward responsiveness is a marker of reward responsiveness/*initial response* to reward (i.e., neural or behavioral processes evoked by initial presentation of a positive reinforcer; PVS Work Group, 2011); and reward sensitivity is a marker of reward valuation (i.e., processes by which the *probability and benefits of a prospective outcome are computed*; PVS Work Group, 2011; Olino et al., 2018). Greater BAS (Smillie, 2013) and reward (Lucas et al., 2000; Olino et al., 2005) sensitivity are primarily paired with dispositional positive affectivity (PA), i.e., the stable, trait-level tendency to experience positive emotions (Hamilton et al., 2017). Both BAS and BIS are implicated in dispositional negative affectivity (NA), i.e., the stable, trait-level tendency to experience negative emotions (Hamilton et al., 2017), with low BAS sensitivity linked to depression-related symptomology and high BIS sensitivity to anxiety-related

symptomology (Bijttebier et al., 2009). (Of note, these definitions of PA and NA, adopted in this manuscript, exclude momentary or state-based affectivity as well as transient mood states.) Theoretically, individual differences in reinforcement sensitivity also have implications for development of emotion regulation (Depue & Iacono, 1989), insofar as such differences affect the way in which individuals regulate and respond to their emotions. As noted, with greater BAS/BIS sensitivity may come enhanced emotional responding (Bijttebier et al., 2009) which may in turn create greater opportunity, i.e., an emotional context, for maladaptive emotion regulation that is, emotion dysregulation (ED); difficulties with or inability to modulate the behavioral, experiential, or physiological escalation, intensity, or de-escalation of emotions in service of adaptive functioning (Bunford, Evans, & Wymbs, 2015). Regarding the BAS, dispositional PA, and ED, it stands to reason that attenuated reward responsiveness, by affecting an individual's ability to anticipate or detect rewards (Henriques & Davidson, 2000) may subsequently impair ability to regulate emotional reactivity, manifesting as a tendency to experience intense emotional arousal (Karrass et al., 2006). Further, greater BAS sensitivity is associated with impulsivity, including impulsive action without regard for long-term goals and outcomes in response to distress (Tull et al., 2010), characterizations that are part of some conceptualizations of ED (Gratz & Roemer, 2004). Even more directly, mediated by the BAS (Corr, 2002), frustrative nonreward (i.e., prevention/ withdrawal of an expected reward or inability to obtain an expected reward following repeated or sustained effort), typically elicits anger and an increase in arousal (Dixon et al., 2013) and has been shown to elicit behaviors consistent with ED (Binder et al., 2020).

Others have argued that BIS-related higher levels of dispositional NA might facilitate ED (Fox et al., 2005; Hundt et al., 2013), with greater levels of emotional arousal necessitating greater levels of regulatory effort. High childhood reactivity has also been conceptualized as impeding use of adaptive emotion regulation strategies, thereby contributing to ED (Suveg et al., 2009).

Of note, despite richness of theoretical literature on reinforcement sensitivity and affectivity, there is relatively less empirical research on the association between neural processes linked to reinforcement sensitivity and in particular reward processing – as defined in the RDoC framework (NIMH, 2011a, 2011b) – and dispositional affectivity (Kennis et al., 2013; Kujawa et al., 2015) and ED. For example, there is a

paucity of research on the association between neural reward responsiveness and dispositional PA, though there are event-related potential (ERP) and functional magnetic resonance imaging (fMRI) studies on the relation between neural reward responsiveness and characteristics *relevant to* dispositional PA, such as extraversion (e.g., M. X. Cohen et al., 2005; Cooper et al., 2014; Smillie et al., 2011; Speed et al., 2018), state- or transient positive affect or mood (Forbes et al., 2009, 2010), or experimentally-induced positive affect (e.g., Young & Nusslock, 2016) (but see, for exception, e.g., Kujawa et al., 2015 for an ERP and dispositional PA study and Sutton & Davidson, 1997; Tomarken et al., 1992 for resting state electroencephalogram (EEG) and dispositional PA studies). Regarding the association between neural reward responsiveness and dispositional NA, available findings with adults, as detailed later, are meager and mixed (Santesso et al., 2012), though indirect evidence on relations between blunted reward responsiveness and depression (Cléry-Melin et al., 2011; Dillon et al., 2014; Pizzagalli et al., 2008; Sherdell et al., 2012; Treadway et al., 2012) underscore the relevance of altered reward responsiveness to dispositional NA. Further, the majority of pertinent studies on the association between neural processes linked to reinforcement and in particular reward processing and dispositional affectivity and ED, have been conducted with children and adults, despite adolescence being a developmentally sensitive period with regard to changes in reward processing (Ernst, 2014; Ernst & Spear, 2009; Shulman et al., 2016) and dispositional affectivity (Silk et al., 2003) and to development of emotion regulation skills (Bunford, 2019). Each of these characteristics, in turn, are relevant to a host of functional outcomes, including risk-taking (Bunford, 2019; Steinberg, 2004, 2005), affective and substance use difficulties (Bunford, Wymbs, Dawson, & Shorey, 2017; Casey et al., 2008; Steinberg, 2004), and social problems (Bunford, Evans, Becker, & Langberg, 2015; Bunford, Evans, & Langberg, 2018), underscoring the conceptual and public health relevance of better understanding neural underpinnings and behavioral correlates of adolescent reward responsiveness. In this regard, research in typical youth is critical for elucidating the etiology and pathophysiology of reward-related negative outcomes in clinical populations (Forbes et al., 2010).

Relevant gaps in knowledge pertain to complex relations between adolescent brain response to experimentally-induced (i.e., state) and self-report (i.e., trait) reward responsiveness and trait-level affective

processing (Forbes et al., 2010). Accordingly, our goals in this research were to assess, in adolescents, (1) evidence of convergent validity between neural and self-report indices of reward responsiveness, (2) the association between neural reward responsiveness and dispositional affectivity and ED, and (3) evidence of incremental validity of neural and self-report indices of reward responsiveness in predicting dispositional affectivity and ED.

Adolescence as a developmental phase of interest

Adolescence is a developmental phase of interest both with regard to changes in reward processing and to changes in affectivity and to ED. Adolescents, relative to children and adults, experience heightened reward sensitivity (Ernst, 2014; Ernst & Spear, 2009; Shulman et al., 2016), and more intense and labile emotions (Silk et al., 2003). Certain brain regions undergo striking developmental changes during this period and some of these – primarily the amygdala, striatum, and prefrontal cortex (Ernst, 2014; Ernst & Spear, 2009) – are sites for dopamine neurons activated by reward and implicated in triggering appetitive behavior as well as generating and regulating affect (Galván, 2013; Spear, 2013, 2018). As the striatum and other subcortical regions mature earlier and the prefrontal cortex matures later (Ernst & Spear, 2009; Kringelbach, 2005), there is an imbalance between drive and regulatory functions (Casey et al., 2008; Galvan, 2010; Shulman et al., 2016). This asynchrony contributes to difficulties with self-regulation and occurs in a context of decreased environmental support and structure (e.g., parents play increasingly less of a role in aiding child self-regulation) but increased environmental and social demands for adult-like regulation (Bunford, 2019), creating prime opportunity for difficulties with self-regulation.

Of note, in adolescence, as in any developmental phase, there will be individual differences in reward sensitivity (with some individuals showing greater whereas others showing lower e.g., sensitivity to reward) as well as with regard to dispositional affectivity (e.g., with some being higher on negative affectivity but average on positive affectivity, some being lower on both negative and positive affectivity) and trait emotion regulation (e.g., with some individuals having greater difficulties with returning to emotional baseline, others exhibiting no difficulty with trait emotion regulation, and yet others experiencing difficulties with engaging in goal-directed behavior when experiencing strong emotions). Accordingly, both

vertical (i.e., developmental) and horizontal (i.e., individual differences) considerations are applicable to adolescent reward responsiveness and affective processing, with the former likely more relevant to adolescence than during other developmental periods and the latter as relevant to adolescence as any other developmental period.

Our focus in this research is on reward responsiveness (as opposed to reward learning, or reward valuation) (NIMH, 2011b) and – as our interest is in developmentally-relevant associations between individual differences in reward responsiveness and outcomes – more specifically, focus is on initial responsiveness to reward attainment and on reward anticipation (of the reward responsiveness subconstructs, available tasks for probing these are sensitive to within-person change; National Institute of Mental Health, 2016). Validated, pertinent experimental paradigms recommended in the RDoC framework are guessing tasks; the Card Guessing task developed by Delgado and colleagues (Delgado et al., 2000), adapted and used in the Forbes (Forbes et al., 2009, 2010) studies reviewed here and the Doors task (Dunning & Hajcak, 2007; Foti & Hajcak, 2009; Kujawa et al., 2013, 2014, 2018) used in the current study, to probe initial responsiveness to reward attainment and a monetary incentive delay (MID) task (Knutson, Fong, Adams, Varner, & Hommer, 2001; Knutson, Fong, Bennett, Adams, & Hommer, 2003; Knutson, Fong, & Hommer, 2001) to probe reward anticipation (and initial responsiveness to reward attainment).

A promising assessment method of neural reward responsiveness is measurement of electrocortical changes, via EEG, including of ERPs linked to specific events and reflecting synchronous activity of populations of neurons (Bunford, Kujawa, Swain, et al., 2017; Hajcak et al., 2010)). Consistent with choice of experimental paradigms and corresponding past research, of interest were the following ERP components: The reward positivity (RewP) is a positivity in the ERP waveform following feedback that reflects neural activity associated with reward processing and typically has a relatively larger amplitude for positive than negative or neutral outcomes (Foti et al., 2011). The Cue P3 reflects attention allocation to cue, modulated by reward value and variations in the emotional significance of stimuli (Chronaki et al., 2017). The Target P3, similar to the Cue P3, reflects motivated and task-relevant attention following appearance of a target and is further considered a neural marker of stimulus evaluation and categorization

processes (Broyd et al., 2012; Groom et al., 2010). The stimulus preceding negativity (SPN) is a slow cortical potential that can be measured as a growing negativity reaching its maximum prior to the onset of a relevant stimulus, indexing anticipatory processes, that may specifically reflect anticipatory attention or anticipation of the affective valence of a(n informative) feedback (Foti & Hajcak, 2012)**.**

Convergent, predictive, and incremental validity of neural and self-report indices of reward responsiveness

Although associated with important outcomes, surprisingly little is known about (1) convergent validity between neural and self-report indices of reward responsiveness, (2) the association between neural reward responsiveness and dispositional affectivity and ED, or (3) about incremental validity of neural and self-report indices of reward responsiveness in predicting dispositional affectivity and ED; virtually no research has been conducted on the association between *adolescent* reward responsiveness and these outcomes using multiple, e.g., biological combined with self-report, methods (but see for exceptions, two fMRI studies evincing that striatal reward reactivity in depressed and in typically developing adolescents is correlated with self-reported average state PA (Forbes et al., 2009, 2010)).

Regarding (1), assessment of individual differences in reward responsiveness as a multisystemic phenomenon, calls for multi-method measurement (Eid & Diener, 2006), such as combination of biological/ physiological and rating scale measures (De Los Reyes et al., 2015). If use of ERP and self-report indices of reward responsiveness are to be useful elements of a multi-method assessment framework, then there should be evidence of (a) their convergent validity (i.e., that they correlate) as such evidence would be indication that they are measuring the same general phenomenon and of (2) their divergent validity (i.e., that they correlate but not to an extent that would indicate isomorphism or redundancy) as such evidence would be indication that they measure unique aspects of the phenomenon.

The available literature on convergent validity between neural and self-report indices of reward responsiveness is comprised of seven studies that we could identify; of these, only one was conducted with children (Kujawa et al., 2019), the rest with adults, and none with adolescents. Initial responsiveness to reward attainment was probed in three studies via the Doors task (Bress & Hajcak, 2013; Kujawa et al.,

2019; Van den Berg et al., 2011), initial responsiveness to reward attainment – though to (predicted and unpredicted) reward attainment (and omission) – was probed in another study via a passive gambling task (Salim et al., 2015) and reward anticipation probed in only one study (via the MID task; Oumeziane et al., 2019). In the remaining investigations, reward learning (and response to reward during learning) was probed in a double choice Go/No-Go task (De Pascalis et al., 2010) and reward expectation mismatch was probed in a choice task (Lange et al., 2012). Consistent with the aspect of reward responsiveness probed by these tasks, ERP indices of reward processing included the P2 (attention selection and salience detection), P3 (attention reallocation when task demands change or an update of task representations is needed), and FN/ FRN/ RewP, but, except for (Oumeziane et al., 2019), none assessed the Cue P3 or SPN and none assessed the Target P3. Regarding self-report measures, across available studies, measures were either based on outdated conceptualizations of the RST-P (De Pascalis et al., 2010; Lange et al., 2012; Van den Berg et al., 2011) or assessed reward responsiveness narrowly but not reinforcement sensitivity broadly (Bress & Hajcak, 2013; Kujawa et al., 2019; Oumeziane et al., 2019; Salim et al., 2015). Accordingly, assessment of convergent validity between ERP components reflecting initial responsiveness to reward attainment and reward anticipation and self-report indices of reinforcement sensitivity (in keeping with most recent theoretical conceptualizations of) *in adolescents* – fills an important gap in the literature.

Regarding (2), and more specifically neural reward responsiveness – as defined in the RDoC framework (NIMH, 2011b) and dispositional PA, there is a paucity of research on this association, with the majority of relevant studies involving resting-state EEG and fMRI methods. Greater self-reported BAS sensitivity is linked with greater left prefrontal activation in resting-state EEG studies with adults (Sutton & Davidson, 1997; Tomarken et al., 1992) and enhanced orbitofrontal and ventral striatal response to rewards in fMRI studies with adolescents and adults (M. X. Cohen et al., 2005; Forbes et al., 2010; Kennis et al., 2013; Simon et al., 2010).

Still regarding (2) but for neural reward responsiveness and dispositional NA, although it is primarily the BAS that is hypothesized to regulate reward processing (Dillon et al., 2014) and thus generate PA, prior data raise questions about whether the link between reinforcement sensitivity and dispositional

affectivity is specific to reward reactivity and approach-related dispositional affect, i.e., dispositional PA (Kujawa et al., 2015) or is relevant more generally to reinforcement sensitivity and dispositional affectivity. For example, early behavioral inhibition has been linked to greater striatal activation to cues of both potential reward *and loss* (Bar-Haim et al., 2009; Guyer et al., 2012) and available findings with adults are mixed, with some indicating enhanced neural response to negative performance feedback is associated with greater dispositional NE (Santesso et al., 2012) but others suggesting such an enhanced response when reward feedback was changed to non-reward feedback in adults high on BAS sensitivity but a reduced response in adults high on BIS sensitivity (Lange et al., 2012).

Another line of reasoning that conceptually evinces relevance of reward reactivity to dispositional NA can be extrapolated from findings on the association between reward processing and depression, e.g., there is a positive association between how much a cartoon is enjoyed and how much effort is expended to obtain it in healthy adults but this association is absent in depressed adults (Sherdell et al., 2012), who make fewer high-effort/high-reward choices, with the number of such choices negatively associated with the length of the current major depressive episode (Treadway et al., 2012). The prospect of increased monetary rewards elicits greater handgrip effort in healthy but not in depressed adults (Cléry-Melin et al., 2011) and evidence indicates an association between deficient reward learning and depression symptoms (Dillon et al., 2014; Pizzagalli et al., 2008). With heightened NA a hallmark characteristic of depression, drawing on such findings is informative for hypothesizing an association between reward responsiveness and dispositional NA (Dillon et al., 2014). The putative mechanism linking reward processing to depression, is such that typical motivation to work harder for rewards is sapped by depression, potentially reflecting excessively conservative calculations on likelihood of benefit relative to cost, or failure of biological mechanisms to translate incentive motivation into action (Dillon et al., 2014). Of note, regarding neural reward responsiveness and dispositional PA, blunted PA is also a hallmark characteristic of depression (Deldin et al., 2001), in fact, relative to heightened NA, it may be a more prominent marker thereof (Kasch et al., 2002; Kujawa et al., 2014).

Still related to (2) but for neural reward responsiveness and ED, despite reason to believe that

reinforcement sensitivity in general and reward responsiveness specifically are linked to emotion regulation, the available empirical evidence is far from equivocal and gaps in knowledge remain. Regarding research with rating scale data, first, much of the available literature is comprised of studies assessing proxies of ED (e.g., experiential avoidance, e.g., (Pickett et al., 2012)). In studies where *bona fide* emotion regulation (as opposed to redolent or related characteristics) is assessed, focus was on overall as opposed to specific aspects of ED and in studies where specific aspects of ED are assessed, measures of reward responsiveness are based on outdated conceptualizations of the RST (Izadpanah et al., 2016; Tull et al., 2010) or are exclusive to the association between punishment sensitivity and rumination (Leen-Feldner et al., 2004; Manfredi et al., 2011). Second, although evidence is fairly consistent on the nature of the relation between BIS sensitivity and ED (e.g., greater BIS sensitivity is associated with greater ED (Markarian et al., 2013; Pickett et al., 2012; Tull et al., 2010)), the same cannot be said about the relation between BAS sensitivity and ED (e.g., lower BAS sensitivity is associated with greater ED (Markarian et al., 2013), with specific BAS dimensions negatively (Reward), some positively (Fun-seeking), and some not (Drive) associated with this outcome (Tull et al., 2010)). Third and finally, there is limited research with adolescents (but see, e.g., (Izadpanah et al., 2016) for a study with adolescents) and a paucity of studies on neural reward responsiveness and ED. Regarding research with neuroimaging data, available data suggest neural reward response can be regulated via cognitive control over emotional responses (Martin & Delgado, 2011) and striatal reward response can be modulated by emotion regulation strategies during expectation of drug (Kober et al., 2010; Volkow et al., 2010) and monetary (Delgado et al., 2008; Staudinger et al., 2009) rewards.

Regarding (3), the advantage of multi-method measurement is that it capitalizes on advantages of different measurement modalities (e.g., biological measures provide relatively objective insight into biological underpinnings of behavior and have better signal-to-noise ratio whereas e.g., rating scales have higher ecological validity), with combination of such measurements conceptually better capturing the complexity of assessed phenomena. The pertinent empirical question, then, is whether or not in combination ERP and self-report indices of reward responsiveness provide a more comprehensive picture of (i.e., account for more of the variance in) adolescent affectivity and ED than either on its own.

Current study

With the aim of beginning to address gaps in knowledge about the relationship between reward responsiveness and affective processing in adolescence, adhering to a multi-method framework, our goals in the current study were to assess (1) evidence of convergent validity between neural reward responsiveness measured during two reward processing experimental paradigms and self-reported reinforcement sensitivity following the conceptual framework of the Reinforcement Sensitivity Theory (Corr & Cooper, 2016), (2) associations between neural reward responsiveness and self-report indices of dispositional affectivity and ED, and (3) evidence of incremental validity of self-report beyond neural reward responsiveness in predicting dispositional affectivity and ED.

We hypothesized that (1) neural and self-report indices of reward responsiveness will be associated, supporting evidence of convergent validity; (2) neural reward responsiveness will predict self-report dispositional affectivity and ED; and (3) self-report reward responsiveness will predict dispositional affectivity and ED beyond the effect of neural indices, supporting evidence of incremental validity.

As our hypothesis for (1) is based on mixed prior findings (Bress & Hajcak, 2013; Kujawa et al., 2019; Lange et al., 2012; Oumeziane et al., 2019; Salim et al., 2015; Van den Berg et al., 2011), we expected that some but not overwhelming evidence would be obtained in support of convergent validity, including, for example, in terms of a positive association between P3 to both monetary gain and loss and self-report reward responsiveness (Van den Berg et al., 2011) and a positive association between RewP to gain and self-report reward responsiveness (Bress & Hajcak, 2013) and a negative association between RewP to gain and BIS sensitivity (Lange et al., 2012). Regarding (2), we specifically expected that neural reward responsiveness would predict self-report dispositional PA but in the absence of sufficient prior data, we did not have specific hypotheses about the relation between neural reward responsiveness and dispositional NA or between neural reward responsiveness and specific indices of ED. Regarding (3), also in the absence of sufficient prior data, we did not have strong hypotheses about the relation between self-report reward responsiveness and dispositional affectivity and ED beyond neural responsiveness, though given the largely

internal and subjective nature of the experience and regulation of emotions (Bunford, Evans, & Wymbs, 2015), we expected that there would be some evidence of incremental validity of self-report.

Method

Procedures

Data were collected in the context of a larger (BLINDED) project. Adolescents between the ages of 14-18 years were recruited from public middle- and high schools in Budapest, Hungary. This research was approved by the National Institute of Pharmacy and Nutrition (OGYÉI/17089-8/2019) and has been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. Participants' parents provided informed consent and participants provided assent, followed by participants' completion of experimental tasks and questionnaires. Following informed consent procedures, the EEG cap was applied and experimental tasks were administered, the Doors task followed by the MID. After participants completed the EEG tasks, they completed self-reported questionnaires.

Participants

Participants were 43 adolescents (M_{age}=15.67 years; *SD*=1.01; 32.6% boys). All identified as Caucasian. Exclusionary criteria were (a) self-reported past or present diagnosis of any psychiatric or neurological disorder, such as pervasive developmental disorder, bipolar disorder, psychosis, substance abuse/dependence or epilepsy; (b) having visual impairment as defined by impaired vision <50 cm, unless corrected by glasses or contact lenses.

Measures

Experimental Paradigms. As noted, the Doors task was used to probe initial responsiveness to reward attainment and a validated monetary incentive delay (MID) task was used to probe reward anticipation and initial responsiveness to reward attainment.

Doors task (Dunning & Hajcak, 2007; Foti & Hajcak, 2009; Kujawa et al., 2013, 2014, 2018). The Doors task was used to probe initial responsiveness to reward attainment and consisted of a total of 120 trials, presented in 2 blocks of 30 trials/condition. Participants were told that on each trial, they could either gain 100 Hungarian Forints (HUF) or lose 50 HUF. At the beginning of each trial, a fixation mark (+)

appeared for 900 ms. Then, participants were presented with an image of two doors for 3000 ms and asked to choose one door by clicking the number 7 or 8 on the keypad (for the left or the right door, respectively). Finally, after a short delay (1100 ms with a jitter of ± 50 ms), feedback was presented on the screen for 1500 ms. A gain was indicated by a green "↑", and a loss was indicated by a red "↓". The duration of the intertrial interval was 2000 ms with a jitter of ± 250 ms. In a single block, 30 gain and 30 loss trials were presented in random order.

Monetary Incentive Delay task (MID; Knutson, Fong, Adams, Varner, & Hommer, 2001; Knutson, Fong, Bennett, Adams, & Hommer, 2003; Knutson, Fong, & Hommer, 2001). The MID task was used to probe reward anticipation and initial responsiveness to reward attainment and consisted of a total of 192 trials, presented in four blocks of 12 trials/condition. In the task, participants responded to a sequence of geometric shapes indicating whether money (1000 HUF) can be gained (e.g., full circle), loss of money can be avoided (full square), or that it is a neutral trial (e.g., empty circle and square), with no monetary consequence. Following each cue (2000 ms duration), there was an anticipatory phase (with a duration between 2000 and 2500 ms) during which participants waited for and were briefly presented with a target stimulus, to which participants had to respond quickly with a button press to gain or avoid losing money. Success or failure was indicated on the computer screen (2000 ms feedback duration), and the cumulative total of money won was shown also on the computer screen. The duration of the intertrial interval was between 1000 and 2000 ms. The duration of the target stimulus was determined before the first block using a shorter training block. The target duration was set to a winning chance of 66%. Trials corresponding to different conditions were presented in a random order.

Of note, there is some debate as to whether the MID task is appropriate, beyond probing reward anticipation, for probing initial responsiveness to reward attainment, with the RDoC proceedings recommending against the latter, noting that although outcomes from the MID task that measure reward anticipation are independent and can be isolated, outcomes that measure response to reward cannot be dissociated from each other (National Institute of Mental Health et al., 2016). Yet, empirical findings suggest that this task is ideal for differentiating between these two aspects of reward responsiveness (Knutson, Fong,

Adams, et al., 2001). As the issue remains open, we chose to interpret results with ERP components preceding reward receipt (Cue P3, Target P3, SPN) as reflecting reward anticipation and results with ERP components following reward receipt (RewP) as reflecting initial responsiveness to reward attainment.

To maximize effectiveness of both paradigms, participants were told that the virtual money they accumulated during each task can be exchanged for fruits and snacks (candy, chips, etc.), with more virtual money exchangeable for more desirable fruit and snack options (as ranked by the participant prior to the tasks).

EEG Data Acquisition and Processing

Continuous EEG was recorded using a 64-channel BrainAmp DC system with actiCAP active electrodes (Brain Products GmbH, Gilching, Germany), using the following electrodes: Fp1, Fp2, AF3, AF7, AF4, AF8, F1, F3, F5, F7, F2, F4, F6, F8, Fz, FC1, FC3, FC5, FT7, FT9, FC2, FC4, FC6, FT8, FT10, FCz, C3, C5, T7, C4, C6, T8, Cz, CP1, CP3, CP5, TP7, TP9, CP2, CP4, CP6, TP8, TP10, CPz, P1, P3, P5, P7, P2, P4, P6, P8, Pz, PO3, PO7, PO9, PO4, PO8, PO10, POz, O1, Oz, O2. The FCz electrode served as an online reference. Two electrodes were used to record the electrooculogram (EOG): one electrode was placed below the left eye and the other was applied lateral to the outer canthus of the right eye. During EEG recordings, electrode impedances were kept below 15 kΩ.

Data were digitized at 16-bit resolution and a sampling rate of 1000 Hz. The recorded EEG was low-pass filtered online at 250 Hz.

For offline data processing, the FieldTrip open source Matlab toolbox (Oostenveld et al., 2011) and custom Matlab analysis scripts (R2017a, The MathWorks, Inc, Natick, MA, USA) were used. All filters applied during EEG signal processing were Hamming-windowed sinc finite impulse response (FIR) filters (passband deviation: 0.0022 (0.22%); stopband attenuation: -53 dB) implemented as one-pass zero-phase forward filters with delay compensation (built-in "firws" filter type in FieldTrip). Half-amplitude (-6 dB) cutoff frequencies are reported. Pre-processing of continuous EEG data involved the following steps: (1) individual channels with poor signal quality (e.g., due to a high amount of drift or jumps, saturation) were removed (*M*±*SD*: .46±.95 channels, range: 0-4). In each individual EEG recording, a custom-made

algorithm detected and marked bad channels: when a channel contained more than 20% of saturated samples (or high/low amplitude samples caused by large drifts and sudden voltage changes), it was selected for possible removal. Prior to actual removal, a final visual inspection of the data on the selected channel was conducted to determine whether it needs to be removed or can be kept. (2) The continuous EEG was high-pass filtered at 1 Hz (order: 1650; transition width: 2 Hz) to aid independent component analysis (ICA)-based artifact rejection (Winkler et al., 2015). (3) On filtered data, muscle artifacts were detected using the automatic artifact rejection function of FieldTrip and marked for later removal. (4) ICA was carried out using the logistic infomax ICA algorithm (Bell & Sejnowski, 1995). ICA components reflecting blinks and eye-movements were identified via visual inspection of the topographical distribution and the time course of the components. In certain cases, to enhance signal quality, other components representing transient or persistent noise artifacts were also selected. (5) Identified ICA components were removed from the original, unfiltered EEG (*M*±*SD*: 3.4±1.26 components, range: 1-7). (6) ICA-cleaned EEG data were then high-pass filtered at 0.1 Hz (order: 16500; transition width: 0.2 Hz). (7) The previously removed bad channels were interpolated by a weighted average of all neighboring channels of the same participant. Weights were calculated based on the distances between the bad electrode and the surrounding electrodes. (8) Finally, the processed EEG was re-referenced to the average of the electrodes located at the left and right mastoids (TP9 and TP10, respectively), and the online reference electrode (FCz) was added to the group of active electrodes.

Calculation of ERP averages involved the following steps: (1) pre-processed continuous EEG data (i.e., final output of the pre-processing workflow) was segmented into stimulus-locked epochs from 200 ms before (or, in case of the SPN, from 1200 ms before) to 1000 ms after stimuli (feedback, cue or target). (2) Epochs were low-pass filtered at 45 Hz (order: 294; transition width: 11.3 Hz) to ensure proper operation of our automatic artifact rejection algorithm. (3) Epochs with high muscle activity (previously detected during the pre-processing) were rejected. (4) Using an automatic artifact rejection method, the following criteria were used to remove additional trials with artifacts: (i) a voltage step of more than 50 μV between data points, (ii) a voltage difference of 300 μV within a trial, and (iii) a voltage difference of less than .50

μV within 100 ms intervals (Bunford, Kujawa, Fitzgerald, et al., 2017; Bunford, Kujawa, Swain, et al., 2017; Kujawa et al., 2015, 2016). (5) Final visual inspection was carried out to identify and remove trials with remaining artifacts (e.g. artifacts not exceeding the thresholds of the artefact rejection algorithms but still affecting the activity on a high number of channels; or trials containing a strong linear trend, for instance, due to cable movement). (6) Trials were then baseline corrected using the pre-stimulus 200 ms intervals (for the SPN, the interval from -1200 ms to -1000 ms before the stimulus was used as baseline). (7) ERP averages were calculated for each participant and for each condition, then the averages were low-pass filtered at 30 Hz (order: 442; transition width: 7.5 Hz). Finally, from individual ERP averages, grand average ERP waveforms were computed for each component. As such, based on chosen electrodes and time windows, one ERP value per condition was calculated for each participant.

Following artifact rejection, for each condition, participants had an average of 53±5 trials (88.6%±8.5%, 4571/5160 trials in total) in case of the Doors task (range: 37-60) and 43±4 trials (89.5%±7.4%, 29390/32832 trials in total) in case of the MID task (range: 22-48, one participant had one less block, due to technical issues). Our exclusionary criterion for excessively noisy EEG data was <60% artifact-free trials (averaged across all ERP components and all conditions for each paradigm). No data were excluded for this reason.

For information on each ERP component of interest, e.g., the electrodes, time windows, and conditions for which they were calculated, see Table 1 and Figures 1-5.

Self-reported questionnaires. Participants completed self-report rating scales of reward responsiveness, dispositional affectivity, and emotion regulation.

Reinforcement Sensitivity Theory of Personality Questionnaire (RST-PQ, Corr & Cooper, 2016). The RST-PQ is a 79-item self-report measure of revised Reinforcement Sensitivity Theory (rRST) personality dimensions, comprised of three subscales: Flight-Fight-Freeze system (FFFS; 10 items, e.g., *I would be frozen to the spot by the sight of a snake or spider, There are some things that I simply cannot go near*), Behavioral Activation System (BAS; 32 items), and Behavioral Inhibition System (BIS; 23 items, e.g., *When nervous, I find it hard to say the right words, I take a long time to make decisions*), and two

additional subscales developed to complement the core RST-PQ: Defensive Fight (8 items, e.g., *I can be an aggressive person when I need to be*) and Panic (6 items, e.g., *I sometimes wake up in a state of terror*). Prior findings suggest Defensive Fight, as it loads highly on BAS and Panic, as it loads highly on both FFFS and BIS, should nevertheless be considered separately from the three main subscales (Corr & Cooper, 2016). Of interest to the current study, the BAS subscale consists of four further subscales: Reward Interest (7 items, e.g., *I regularly try new activities just to see if I enjoy them*), Goal-Drive Persistence (7 items, e.g., *I often overcome hurdles to achieve my ambitions*), Reward Reactivity (10 items, e.g., *Sometimes even little things in life can give me great pleasure*) and Impulsivity (8 items, e.g., *I find myself doing things on the spur of the moment*). Respondents rate how accurately each item describe them on a four-point Likert-type response format scale (1 – 'not at all' to 4 – 'highly'). Higher scores indicate higher sensitivity to reinforcement. Prior findings indicate that RST-PQ demonstrated good internal consistency and adequate convergent and discriminant validity with other personality measures (e.g., Eysenck Personality Questionnaire-Revised [EPQ-R], BIS/BAS, State-Trait Anxiety Inventory [STAI]) (Corr & Cooper, 2016; Eriksson et al., 2019; Pugnaghi et al., 2018).

For purposes of the current study, the English version of the RST-PQ was translated into Hungarian following evidence-based guidelines: (1) the English version was translated into Hungarian by three independent translators; (2) these three translations were combined into a single "summary translated" measure by a fourth independent translator, reconciling all discrepancies across the three translations/ors; (3) the "summary" was back-translated into English by two additional independent translators and (4) the two back-translations were combined into a single "summary back-translated" measure by members of the research team, reconciling all discrepancies in a manner that the "summary back-translation" measure best matches the Hungarian "summary translated" measure. This "summary back-translated" questionnaire was sent to the original author(s) who provided the research team with feedback and ultimately approved the translated measure (P. Corr, personal communication, May 29, 2019). In the current sample, the FFFS subscale exhibited acceptable (α=.754) and the BAS (α=.843) and BIS (α=.873) subscales exhibited good internal consistency. In the current sample, the BAS Goal-Drive Persistence subscale exhibited good

(α=.801), Reward Interest (α=.704) and Reward Reactivity (α=.775) subscales exhibited acceptable and Impulse (α=.671) exhibited questionable-acceptable internal consistency.

Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). The PANAS is a 20-item self-report measure of state and trait positive and negative affect, comprised of two subscales, the positive affect (PA) subscale, reflecting the extent to which a person feels enthusiastic, active and alert, and a negative affect (NA) subscale, reflecting a general dimension of subjective distress and a variety of aversive mood states such as anger, contempt, disgust, fear, guilt, and nervousness. Respondents rate the extent to which they are experiencing each mood state "right now" (i.e., state version) or "during the past two weeks" (i.e., trait version) on a five-point Likert-type response format scale (1 – 'very slightly or not at all' to 5 – 'very much'). Higher scores on the PA and NA subscales indicate greater positive and negative affect, respectively. Prior findings indicate that PANAS scales have good internal consistency (αs ranging from .86 to .90 for PA and from .84 to .87 for NA) and good convergent and discriminant associations with distress and psychopathology measures of the underlying affectivity factors (e.g., Beck Depression Inventory [BDI], Hopkins Symptom Checklist [HSCL], STAI) (Watson, Clark, & Tellegan, 1988). The Hungarian translation also demonstrated acceptable psychometric properties, including good internal consistency (PA α=.82, NA α=.83 [alpha values are provided only to the second decimal in the source article]) (Gyollai et al., 2011). In the current study, the PANAS-trait was administered and in this sample, the PA subscale exhibited questionable-acceptable internal consistency (α=.664) and the NA subscale exhibited good internal consistency (α=.791).

Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). The DERS is a 36 item self-report measure of ED, comprised of six subscales, Nonacceptance of Emotional Responses (Nonacceptance, e.g., *When I'm upset, I become angry with myself for feeling that way*), Difficulties Engaging in Goal-Directed Behavior (Goals, e.g., *When I'm upset, I have difficulty concentrating*), Impulse Control Difficulties (Impulse, e.g., *When I'm upset, I become out of control*), Lack of Emotional Awareness (Awareness, e.g., *When I'm upset, I acknowledge my emotions*), Limited Access to Emotion Regulation Strategies (Strategies, e.g., *When I'm upset, I believe that wallowing in it is all I can do*), and Lack of

Emotional Clarity (Clarity, e.g., *I have difficulty making sense out of my feelings*). Items are rated on a five-point Likert-type response format scale (1 – 'Almost Never' to 5 – 'Almost Always'), with higher scores indicating greater difficulty with emotion regulation. Prior findings indicate the DERS has acceptable psychometric properties, including good internal consistency, good test–retest reliability, and adequate construct and predictive validity in multiple adolescent samples (Adrian et al., 2009; Bunford et al., 2018; Bunford, Evans, Becker, et al., 2015; Vasilev et al., 2009; Weinberg & Klonsky, 2009). In addition, the DERS exhibited robust correlations with psychological problems reflecting ED (Weinberg & Klonsky, 2009) and physiological measures of ED (Vasilev et al., 2009). The Hungarian translation also demonstrated acceptable psychometric properties, including good internal consistency (all αs>.70) as well as construct and convergent validity with the Zung Self-rated Depression Scale (Kökönyei et al., 2014).

In the current study, we used the subscale scores but not also the total score to index ED for two reasons. First, as already noted, in pertinent studies, focus was on overall as opposed to specific aspects of ED. Thus, using subscale scores is a step towards advancing the literature in this regard. Second, to limit the number of analyses. In the current sample, the Nonacceptance subscale exhibited good (α=.849), Goals exhibited questionable (α=.619), Impulse exhibited acceptable (α=.740), Awareness exhibited good (α=.813), and the Strategies subscale exhibited acceptable (α=.751) internal consistency. As the Clarity subscale exhibited poor (α=.448) internal consistency, we did not include it in further analyses.

Analytic Plan

Given our exploratory approach, in line with relevant precedents (e.g., Bunford et al., 2017), data were analyzed through a two-step process that involved bivariate correlation and linear regression analyses.

First, to determine which, if any of our variables of interest, were related to each other, we conducted tests of bivariate correlations among all variables of interest, that is, among RewP, Cue P3, Target P3, SPN, and MID RewP following monetary gain and loss, RSTP-Q subscales, PANAS subscales, and DERS subscales. The purpose of these analyses was to identify a parsimonious set of variables to enter into regression models. Following Cumming (Cumming, 2013) and Field (Field, 2013), we relied not only on *p* values, but also on the magnitude of the correlation coefficients as indices of effects size to determine which

variables were meaningfully related. In addition, following Cumming (Cumming, 2013) and Field (Field, 2013), we employed bootstrapping with 1000 resamples and calculated 95% confidence intervals (CIs) around the obtained *r* values so as to account for multiple comparisons. We relied on established convention (J. Cohen, 1992) to determine the meaningfulness of the magnitude of the observed bivariate effects and thus considered an $r \geq .3$ (medium) (or a $p < .05$) as meaningful.

Second, to address aims 1 and 2, variables that emerged in correlation analyses as exhibiting a meaningful relationship were entered into multivariate linear regression models. Independent variables were RewP, Cue P3, Target P3, SPN, and MID RewP following monetary gain and loss, and dependent variables were self-reported reward responsiveness, affectivity, and ED. We conceptualized the ERP variables as the independent variables and the self-reported variables as the dependent variables in light of the reasoning that individual variability in relatively homogenous characteristics (such as neural reactivity) precede the development of individual variability in relatively heterogeneous characteristics, such as behaviorally observable reward responsiveness and especially in phenomena as complex as dispositional affectivity and emotion regulation (see [Bunford et al., 2017; Merwood et al., 2014] for pertinent precedent). We relied not only on *p* values, but also on the magnitude of the $\eta^2_{partial}$ values as indices of effects size to determine which regression results to interpret as reflecting meaningful relationships. Thus, we considered $\eta^2_{partial} > .13$ (medium) (or a $p < .05$) as meaningful.

Third, to address aim 3, variables that emerged in correlation analyses as exhibiting a meaningful relationship were entered into hierarchical linear regression models. Independent variables entered in step one were RewP, Cue P3, Target P3, SPN, and MID RewP following monetary gain and loss, independent variables entered in step two were self-reported reward responsiveness, and dependent variables were dispositional affectivity and ED. We relied not only on *p* values, but also on the magnitude of the Cohen's f^2 values as indices of effects size to determine which regression results to interpret as reflecting meaningful relationships and thus to report. Thus, we considered Cohen's $f^2 > .15$ (medium) (or a $p < .05$) as meaningful.

Results

Correlation Analyses

For descriptive statistics on self-report indices of reward responsiveness, dispositional affectivity, and ED, see Table 2. Correlation analyses between neural and self-report indices of reward responsiveness indicated, based on effect size (r>.3) and/or statistical significance (p<.05), for *Doors* initial responsiveness to reward attainment, RewP to monetary loss was positively associated with RSTP-Q FFFS. For *MID* reward anticipation, Target P3 to monetary gain and to loss were positively associated with RSTP-Q Reward Reactivity and for *MID* initial responsiveness to reward attainment, RewP to gain and to loss were positively associated with Reward Reactivity and RewP to gain was positively associated with RSTP-Q FFFS (Table 3).

Correlation analyses between neural reward responsiveness and self-report dispositional affectivity and ED, for *Doors* initial responsiveness to reward attainment, RewP to gain was negatively associated with PANAS NA and DERS Awareness. RewP to loss was negatively associated with PANAS NA and DERS Strategies (Table 3). For *MID* reward anticipation, Cue P3 to both gain and loss was negatively associated with DERS Strategies. SPN to loss was negatively associated with DERS Impulse (Table 3).

Although not a primary goal, we also examined associations between self-reported indices of reward responsiveness and dispositional affectivity and emotion regulation. Based on effect size (r>.3) and/or statistical significance (p<.05), PANAS PA was positively associated with most RSTP-Q reward responsiveness subscales (Table 4) and PANAS NA was positively associated with RSTP-Q BIS and Panic. Most DERS subscales were also associated with RSTP-Q BIS (Goals, Impulse, Nonacceptance, Strategies) and Panic (Impulse, Nonacceptance, Strategies). None of the dispositional affectivity or emotion regulation variables were associated with RSTP-Q Impulse, FFFS, or Defensive Fight (Table 4).

Regression Analyses

Aims 1 and 2

Multivariate regression analyses indicated, based on effect size ($\eta^2_{partial}$>.13) and/or statistical significance (p<.05), that (all reported p values are Benjamini-Hochberg-corrected for FDR) for *Doors* initial responsiveness to reward attainment, the RewP to gain model was supported, $F(2, 40)=5.383$, $p=.039$, $\eta^2_{partial}=.212$ (medium effect), with a negative association between RewP and PANAS NA ($\beta=-.491$, $p=.046$,

$\eta^2_{partial}$=.144 (medium effect)) and DERS Awareness (β=-.374, p=.046, $\eta^2_{partial}$=.112 (small effect)). The RewP to loss model was supported, F(3, 39)=4.627, p=.039, $\eta^2_{partial}$=.263 (large effect), with a negative association between RewP and PANAS NA (β=-.437, p=.046, $\eta^2_{partial}$=.129 (medium effect)), and DERS Strategies (β=-.360, p=.046, $\eta^2_{partial}$=.093 (small effect)) and a positive association between RewP and RSTP-Q FFFS (β=.341, p=.046, $\eta^2_{partial}$=.097 (small effect)) (Table 5).

For *MID* reward anticipation the Cue P3 to gain model was supported, F(1, 41)=5.014, p=.043, $\eta^2_{partial}$=.109 (small effect), with a negative association between Cue P3 and DERS Strategies (β=-.382, p=.046). The Cue P3 to loss model was also supported, F(1, 41)=4.288, p=.045, $\eta^2_{partial}$=.095 (small effect), with a negative association between Cue P3 and DERS Strategies (β=-.330, p=.046). The Target P3 to gain model was supported, F(1, 41)=5.179, p=.042, $\eta^2_{partial}$=.112 (small effect), with a positive association between Target P3 and RSTP-Q Reward Reactivity (β=.350, p=.046). The SPN to loss model was supported, F(1, 41)=4.440, p=.045, $\eta^2_{partial}$=.098 (small effect), with a negative association between SPN and DERS Impulse (β=-.308, p=.046).

For *MID* initial responsiveness to reward attainment, the RewP to gain model was supported, F(2, 40)=4.261, p=.040, $\eta^2_{partial}$=.176 (medium effect), with a positive association between RewP and RSTP-Q Reward Reactivity (β=.417, p=.046, $\eta^2_{partial}$=.100 (small effect)) and FFFS (β=.562, p=.046, $\eta^2_{partial}$=.147 (medium effect)) (Table 5). The RewP to loss model was supported, F(1, 41)=5.285, p=.041, $\eta^2_{partial}$=.114 (small effect), with a positive association between RewP and RSTP-Q Reward Reactivity (β=.470, p=.046) (Table 5).

Aim 3

Multivariate regression analyses indicated, based on effect size (Cohen's f^2 values of >.02 - <.15 as small, >.15 - <.35 as medium and ≥.35 as corresponding to a large effect) and/or statistical significance (p<.05), that for *Doors* initial responsiveness to reward attainment, at step one, RewP to gain contributed to the regression model, F(1, 41)=6.903, p=.017 and accounted for 14.4% of the variance in PANAS NA. Self-reported RST-PQ Panic scores explained an additional 19% of the variance in the outcome, ΔR^2=.190; ΔF(1,40)=11.433, p=.008, f^2=.285 (medium effect).

For *MID* reward anticipation, with DERS Strategies as the outcome, at step one, Cue P3 to gain contributed to the regression model, $F(1,41)=5.014$, $p=.035$ and accounted for 10.9% of the variance in the outcome. Self-reported RST-PQ BIS scores explained an additional 29.8% of variance in DERS Strategies, $\Delta R^2=.298$; $\Delta F(1,40)=20.085$, $p<.001$, $f^2=.502$ (large effect). With DERS Impulse as the outcome, at step one, SPN to loss contributed to the regression model, $F(1,41)=4.440$, $p=.041$ and accounted for 9.8% of the variance in the outcome. Self-reported RST-PQ BIS scores explained an additional 18.2% of variance in DERS Impulse, $\Delta R^2=.182$; $\Delta F(1,40)=10.118$, $p=.008$, $f^2=.253$ (medium effect).

Discussion

Generally, findings indicate some evidence of convergent validity between electrophysiological and self-report indices of reward responsiveness; evince that in adolescents, individual differences in neural reward responsiveness are associated with differences in dispositional affectivity and ED; and suggest evidence of incremental validity of self-report beyond neural reward responsiveness in predicting dispositional affectivity and ED.

Specifically, regarding convergent validity of electrophysiological and self-report reward responsiveness, when considered in the context of bivariate correlations, an enhanced motivated/ task-relevant attention following appearance of the target (Target P3) in gain and loss trials was associated with greater reward reactivity (with items reflecting reward sensitivity accompanied by a stronger experiential and physiological, hedonic response even to smaller rewards). Further, enhanced neural initial responsiveness to reward (i.e., motivationally-salient feedback as indicated by this association emerging to both gain and loss) across the two tasks was associated with greater FFFS (with items reflecting responsiveness to aversive-stimuli) and enhanced neural initial responsiveness to reward in the monetary incentive delay paradigm was associated with greater reward reactivity.

When considered in the context of regressions, enhanced neural initial responsiveness to motivationally-salient feedback (gain and loss) across the two paradigms predicted greater FFFS, with the magnitude of corresponding effects being small-medium. Further, enhanced motivated/ task-relevant attention following appearance of the target (Target P3) in gain trials of the monetary incentive delay

paradigm and enhanced neural initial responsiveness to motivationally-salient feedback (gain and loss) also in the monetary incentive delay paradigm predicted greater reward reactivity, with the magnitude of corresponding effects being small. These findings are consistent with earlier results indicating a positive association between P3 to both monetary gain and loss and self-report reward responsiveness (Van den Berg et al., 2011) and positive associations between FRN to initial response to reward and self-report reward responsiveness (Bress & Hajcak, 2013) and FRN to reward expectation mismatch and BAS sensitivity (Lange et al., 2012). These findings add to the available literature in extending the neural and self-report reward responsiveness association beyond self-reported responsiveness to rewarding to responsiveness to aversive stimuli (i.e., FFFS sensitivity). As hypothesized, our data are some evidence of convergent validity between electrophysiological and self-report reward responsiveness and thereby underscore the importance of- and utility in multi-method measurement (De Los Reyes et al., 2015; Dirks et al., 2012) of biopsychological characteristics; although there is correspondence between certain electrophysiological and certain self-report indices of reward responsiveness, this correspondence is small-medium and is not applicable across aspects of reinforcement sensitivity (e.g., there was no association between other electrophysiological indices, such as the Cue P3 or SPN and other self-report indices, such as goal-drive persistence, reward interest, or BIS). One explanation for these results is that while ERP measures index acute state-based responses to motivationally-salient feedback, self-report indices reflect a trait-based temperament and, in combination with differences with regard to measurement method (i.e., physiology vs. self-report), similarities in underlying mechanisms accessed (i.e., reinforcement sensitivity) and differences in sampling-frame (i.e., state vs. trait) might explain why there is some evidence of correspondence but also some indication of lack thereof. Indeed, despite utility of clinical and demographic measures, on their own, they tend to be weak predictors of psychiatric heterogeneity and of treatment response but neural measures and combination of clinical and neural measures are promising in this regard (Gabrieli et al., 2015); e.g., in pediatric anxiety, anxiety severity did not, but enhanced neural index of sustained attention to affectively and motivationally salient stimuli did predict heterogeneity in anxiety presentation with regard to externalizing behavior and social problems (Bunford, Kujawa, Swain, et al., 2017) and to response to

pharmaco- and psychotherapy treatment (Bunford, Kujawa, Fitzgerald, et al., 2017).

Taken together, electrophysiological measures of reward responsiveness probed by validated experimental paradigms and self-report measures of reward responsiveness index some of the same aspects of the phenomenon but are also sources of data on unique aspects thereof. Along similar lines, relative to the correspondence between neural reward responsiveness and self-report affectivity and emotion regulation, there were a greater number of associations observed between self-report reward responsiveness and self-report affectivity and emotion regulation. Certainly, these differences are partly explainable by shared method variance between the various self-reports, though these differences may also reflect that different measures of the same phenomenon index not only different aspects of that phenomenon but also different manifestations of its association with other, relevant characteristics.

Regarding relations between neural reward responsiveness and dispositional affectivity and ED, associations between each of five indices of neural reward responsiveness and several dispositional affectivity (NA) and ED variables (difficulty with attention to- and conscious processing of emotions (DERS Awareness); perception of not having access to emotion regulation strategies when experiencing negative emotions (DERS Strategies); and behavioral dyscontrol when experiencing negative emotions (DERS Impulse)) were observed. As bivariate correlations served the purpose of identifying variables to enter into regression models, we do not further elaborate on these results.

When considered in the context of regressions, enhanced neural reward anticipation, specifically attention allocation (Cue P3) to motivationally-salient feedback (again, we refer to this as motivationally-salient feedback as this relationship was observed in case of both gain and loss) predicted *less difficulty with* perception of not having access to emotion regulation strategies when experiencing negative emotions (DERS Strategies). Similarly, enhanced neural reward anticipation, specifically anticipatory attention or anticipation of the affective valence of feedback (SPN) to loss predicted *less difficulty with* behavioral dyscontrol when experiencing negative emotions (DERS Impulse).

Further, enhanced neural initial responsiveness to reward (RewP) to motivationally-salient feedback (both gain and loss) in the simple guessing task predicted *lower* dispositional NA. Enhanced RewP to gain

in the simple guessing task predicted *less difficulty with* attention to- and conscious processing of emotions (DERS Awareness). Enhanced neural initial responsiveness to reward (RewP) in loss trials in the simple guessing task predicted *less difficulty with* perceived access to emotion regulation strategies (DERS Strategies). That enhanced neural initial responsiveness to reward predicted lower dispositional NA is consistent with prior findings but also extend those to greater reward responsiveness to loss and ED. Similarly, that enhanced neural initial responsiveness to reward predicted *less difficulty with* attention to- and conscious processing of emotions, is consistent with prior findings suggesting blunted reward responsiveness is associated with difficulties with emotion regulation (Laakso et al., 2003; Sauder et al., 2015), though compared to earlier studies, our current data are more direct evidence of this association. Individuals with greater sensitivity to rewarding stimuli may be better able to rely on such stimuli in (cognitive) emotion regulation strategies involving flexibly changing attention to emotions, e.g., cognitive restructuring or refocusing.

Even more novel are the findings that enhanced neural reward anticipation and initial reward response not only to reward but also to loss, i.e., to motivationally-salient feedback in general, predict lower dispositional NA and less difficulty with perceived access to emotion regulation strategies and that enhanced neural initial reward response to loss also predicts less difficulty with perceived access to emotion regulation strategies. It stands to reason that responsiveness not only to rewarding stimuli specifically but responsiveness to motivationally-informative stimuli more generally is related to individual differences in affective processing. Greater openness to feedback in general may correspond to a greater likelihood of obtaining rewards, which in turn brings about lower trait-level NA, which then supports use of adaptive emotion regulation strategies, thereby contributing to less dysregulation (Suveg et al., 2009).

Of note, as we discuss earlier, there is surprisingly little research on correspondence between neuroimaging predictors of reward responsiveness and dispositional PA. One exception where correspondence between ERP indices of reward responsiveness and PA was assessed is the study by Kujawa et al. (2015), where enhanced ΔFN (the relative response to winning vs. losing money) at age 9 years was associated with greater observed PA at age 3 years and greater self-report dispositional PA at age 9 years,

though these associations were modest. In the current study, ERP indices of reward responsiveness were also associated with dispositional PA and the direction and magnitude of these associations is more consistent than those for dispositional NA, albeit correlations were small in magnitude and did not reach statistical significance (e.g., correlation between RewP to gain and dispositional PA was r=.26 and between Target P3 to gain and dispositional PA was also .26). Indeed, it is generally true, beyond neural reward responsiveness and dispositional PA that in the current study, the magnitude of the observed effects between neural reward responsiveness and dispositional affectivity and ED ranged from small to medium. This is consistent with evidence from large samples (which generate more reliable/less variable effect sizes) indicating that the association between neural/ physiological measures and various characteristics relevant to psychopathology is likely quite modest (Kujawa & Burkhouse, 2017; Yancey et al., 2016). Accordingly, there are likely other extrinsic and intrinsic factors that modulate the links between neural reward responsiveness and subjective affective processing. These considerations underscore the importance of constructing and construing multi-method models, as we have done here, to improve prediction of outcomes and understanding of relationships across levels of analysis.

Our findings are evidence of incremental validity of self-report beyond neural reward responsiveness in predicting dispositional affectivity and ED. Interestingly, unlike in case of self-report subscales implicated in convergence between neural and self-report reward responsiveness, i.e., BAS reward reactivity and FFFS subscales, in case of self-report subscales explaining additional variance in affective processing beyond neural reward responsiveness, it was almost exclusively the BIS subscale that was relevant. (The only exception was self-reported RST-PQ Panic explaining variance in dispositional NA beyond RewP). Specifically, across two DERS subscales (Impulse, and Strategies), self-reported RST-PQ BIS scores explained an additional 18-30% of variance beyond reward anticipation ERP components. These results make sense when considering that with variables that correspond (i.e., that show greater convergent validity), there is less area of non-overlap in terms of ability to explain outcomes but in case of variables that do not correspond, there is more area of non-overlap and thus incremental utility in explaining outcomes. These explanatory hypotheses support the argument that all aspects of reinforcement sensitivity

– and not just BAS or BIS sensitivity in isolation – are relevant to understanding the link between these attention- and motivation regulating systems and affective processing.

Interestingly in this sample, as evident from the relevant Figures, there does not appear to be a difference in relative RewP amplitudes to monetary gain and loss. As further evident from the preceding discussion, across analyses, self-report indices of reward responsiveness and dispositional affectivity and ED are associated with neural indices of reward responsiveness, including the RewP, to both monetary gain and loss. These data are not inconsistent with the literature, perhaps with the exception of the RewP – an ERP component argued to reflect reward responsiveness with a positivity in the ERP waveform increased for reward compared to loss. Earlier, the RewP was referred to as feedback negativity (FN), feedback-related negativity (FRN), medial frontal negativity (MFN), and feedback error-related negativity (FERN), reflecting that it is associated with error processing (Foti & Hajcak, 2009). However, as RewP amplitude was often observed to be larger for positive/rewarding compared to negative or neutral feedback, it is currently argued that it is associated with reward processing. Yet, our findings indicate, at least insofar as the relationships between the current variables are considered, that the RewP is sensitive to individual differences in initial responsiveness not only to reward but to motivationally-salient feedback.

Limitations and future directions

All questionnaires employed have been previously translated into and validated in Hungarian, with the exception of the RSTP-Q. Although this warrants some caution regarding the results obtained with this measure, our highly rigorous translation procedures as well the herein observed preliminary evidence of internal consistency and convergent validity of the scale increase confidence in the pertinent results.

Similarly, although our sample size with regard to number of participants was not large, a noteworthy aspect of this research is the sample size with regard to number of ERP trials, that was considerably greater than is typical in the literature (in case of the Doors task we had a total of 120 trials (60 per condition) compared to the typical 60 trials (30 per condition) (Kujawa et al., 2013, 2014, 2015) and in case of the MID task we had a total of 192 trials (48 per condition) compared to the greatest number for this task previously being a total of 180 trials (60 per condition) (Broyd et al., 2012)).

It is important that our results are interpreted in the context of us having defined adolescence based on chronological age and not on biological age and assessing additional indices of developmental status (e.g., pubertal status) will be important as pubertal hormonal changes have been shown to affect reward responsiveness (Casey et al., 2008; Galvan, 2010; Shulman et al., 2016).

Conclusions

The current research represents the first examination of correspondence between neural indices of reward responsiveness as probed by a simple guessing task (Doors) (initial responsiveness to reward attainment) and a monetary incentive delay paradigm (initial responsiveness to reward attainment and reward anticipation) and self-report indices of reward responsiveness in adolescents. This is also the first multi-method examination of the relationship between individual differences in reward responsiveness and dispositional affectivity and emotion regulation in this developmental phase. Our findings evince convergent validity between the neural and self-report measures of reward responsiveness and also underscore importance of multi-method measurement as the neural and self-report measures exhibited areas of correspondence and non-correspondence, indicating they may also allow for access into different aspects of the phenomenon. Our results further indicate that individual differences in responsiveness not only to reward specifically but to motivationally-salient information generally correspond to dispositional affectivity and emotion regulation, indicating prevention efforts focused on reward-related negative outcomes may also affect a cascade of subsequent negative outcomes related to low positive/high negative dispositional affectivity or emotion dysregulation.

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Figure legends

Figure 1. (A) Scalp distributions depicting the RewP to gain and loss trials during the Doors task in the 175–275 ms time window. (B) ERPs (*negative up*) across central and centroparietal electrode sites for the entire sample.

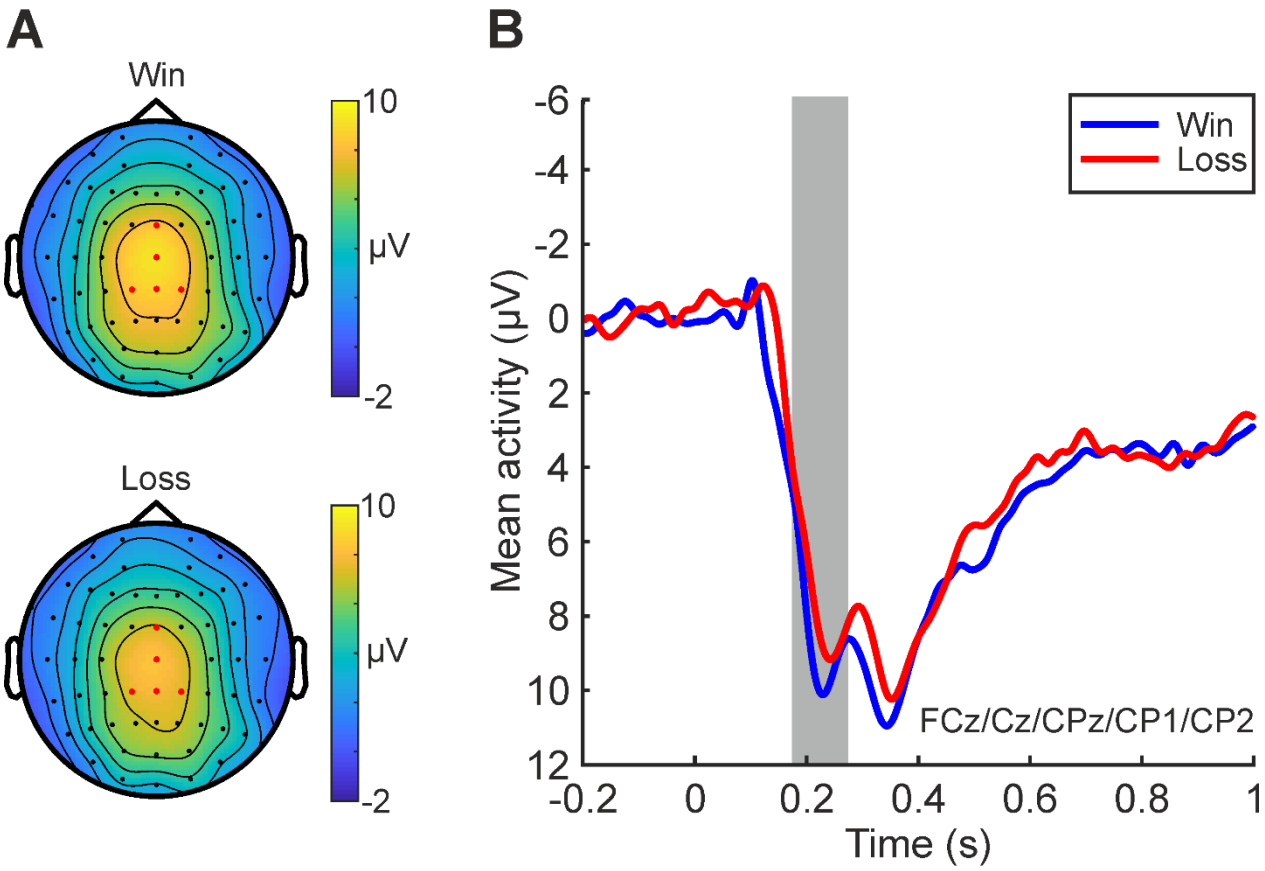
Figure 2. (A) Scalp distributions depicting the MID Cue P3 to gain, loss and neutral trials during the MID task in the 450–650 ms time window. (B) ERPs (*negative up*) across centroparietal and centro-occipital electrode sites for the entire sample.

Figure 3. (A) Scalp distributions depicting the MID Target P3 to gain, loss, and neutral trials during the MID task in the 200–375 ms time window. (B) ERPs (*negative up*) across parietal and centroparietal electrode sites for the entire sample.

Figure 4. (A) Scalp distributions depicting the MID SPN to gain, loss, and neutral trials during the MID task in the -200 – 0 ms time window (before the feed). (B) ERPs (*negative up*) across central and centroparietal electrode sites for the entire sample.

Figure 5. (A) Scalp distributions depicting the RewP to gain and loss trials during the MID task in the 175–275 ms time window. (B) ERPs (*negative up*) across centrofrontal and centroparietal electrode sites for the entire sample.

Figure 1.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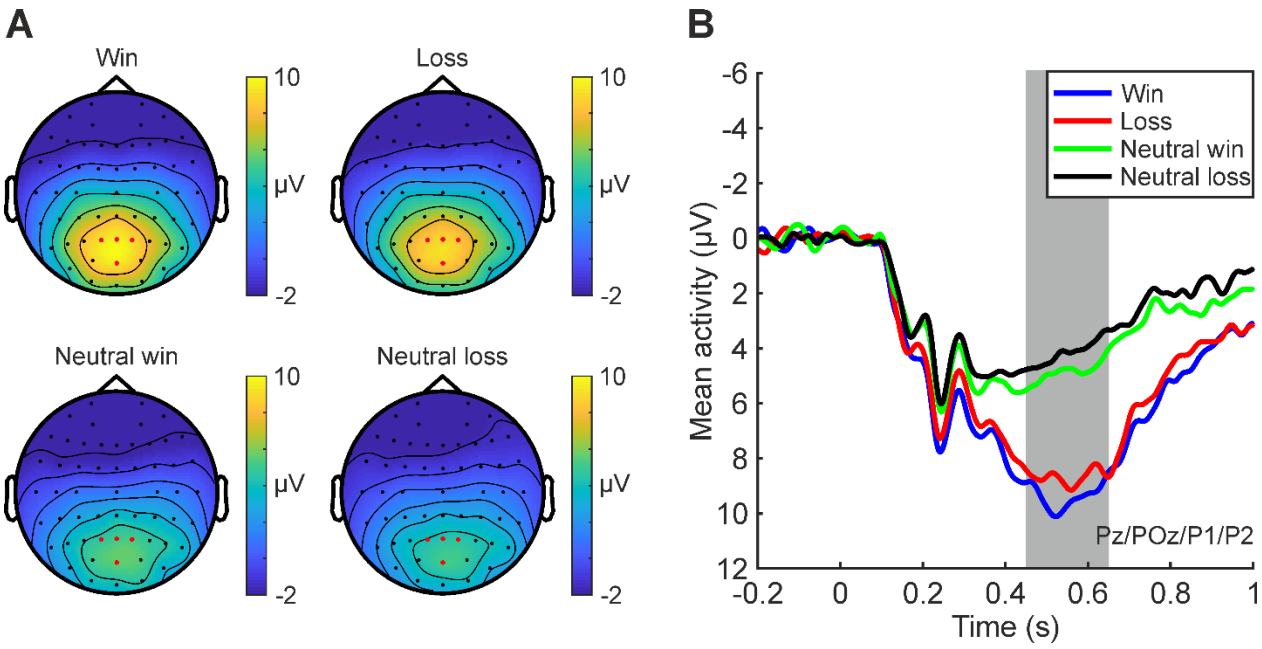
Win
10
μV
-2
Loss
10
μV
-2
B
-6
-4
-2
0
2
4
6
8
10
12
Mean activity (μV)
Win
Loss
FCz/Cz/CPz/CP1/CP2
-0.2
0
0.2
0.4
0.6
0.8
1
Time (s)

Figure 2.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Loss
Neutral win
Neutral loss
10
μV
-2
B
Win
Loss
Neutral win
Neutral loss
Mean activity (μV)
Time (s)
Pz/POz/P1/P2
-6
-4
-2
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2
4
6
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10
12
-0.2
0
0.2
0.4
0.6
0.8
1

Figure 3.

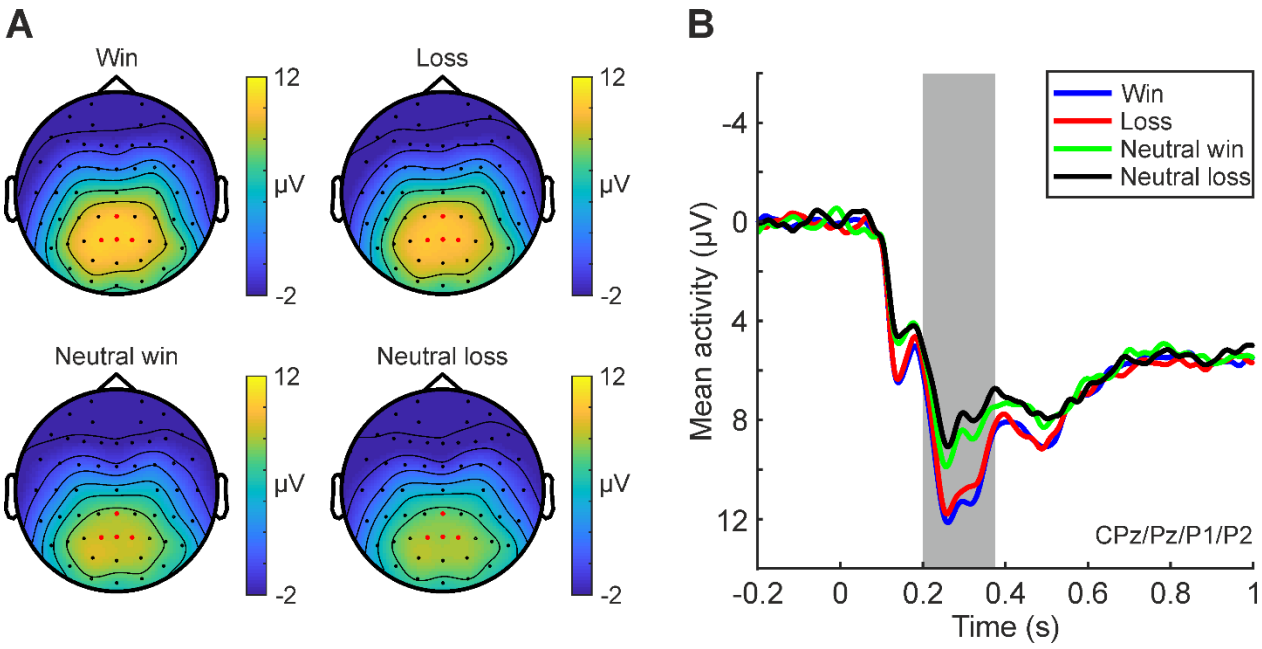
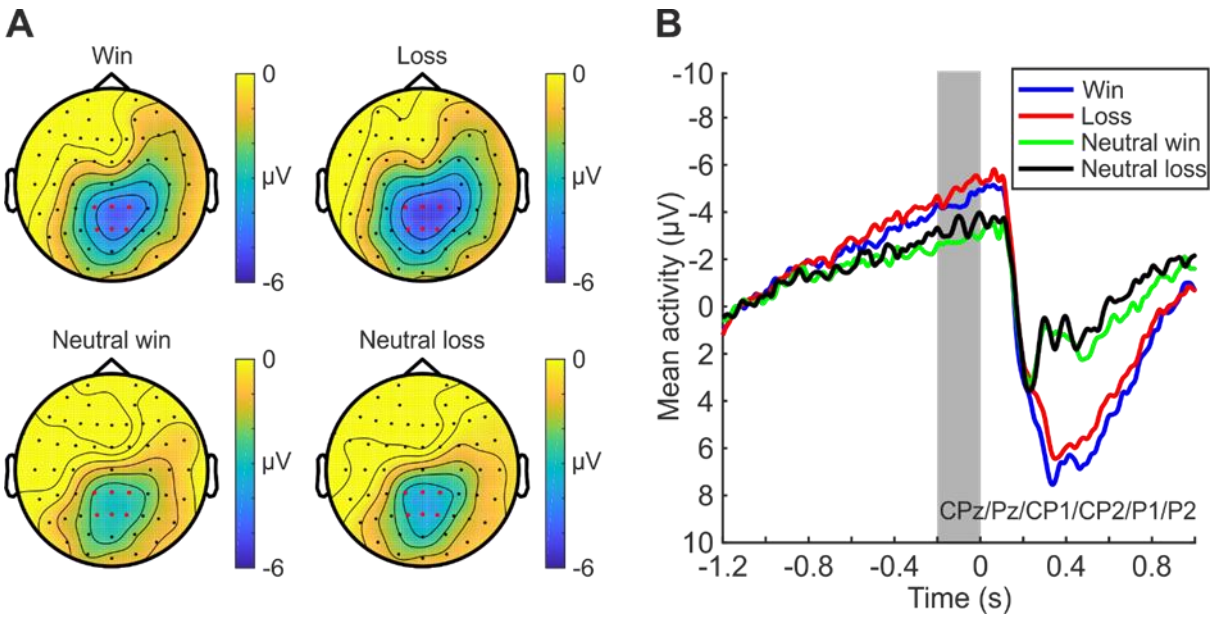

A
Win
Loss
Neutral win
Neutral loss
12
μV
-2
B
Win
Loss
Neutral win
Neutral loss
Mean activity (μV)
-4
0
4
8
12
CPz/Pz/P1/P2
-0.2
0
0.2
0.4
0.6
0.8
1
Time (s)

Figure 4.


A
Win
Loss
Neutral win
Neutral loss
0
μV
-6
B
Win
Loss
Neutral win
Neutral loss
Mean activity (μV)
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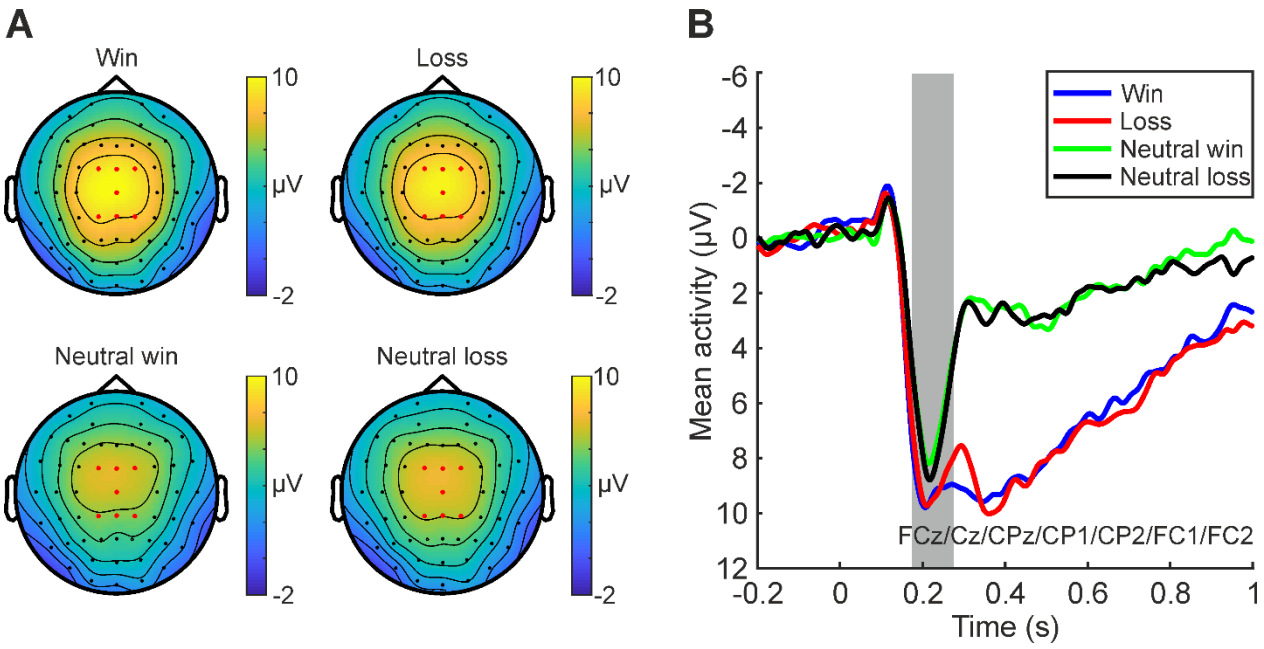
-8
-6
-4
-2
0
2
4
6
8
10
-1.2
-0.8
-0.4
0
0.4
0.8
Time (s)
CPz/Pz/CP1/CP2/P1/P2

Figure 5.


A
Win
Loss
Neutral win
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10
μV
-2
B
Win
Loss
Neutral win
Neutral loss
Mean activity (μV)
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-4
-2
0
2
4
6
8
10
12
FCz/Cz/CPz/CP1/CP2/FC1/FC2
-0.2
0
0.2
0.4
0.6
0.8
1
Time (s)

Table 1

ERP components of interest.

ERP component	Maximal	Time window	Electrodes (pooled)	Scoring
Doors RewP	central and centroparietal sites, consistent though somewhat more parietal compared to (Foti et al., 2011; Kujawa et al., 2018, 2019)	175–275 ms	FCz, Cz, CPz, CP1, CP2	gain and loss scores separately
MID Cue P3	centroparietal and centro-occipital sites, consistent with (Chronaki et al., 2017; Goldstein et al., 2008)	450–650 ms	Pz, POz, P1, P2	gain, loss and neutral scores separately
MID Target P3	parietal and centroparietal sites consistent with (Groom et al., 2010; Polich, 2007)	200–375 ms	CPz, Pz, P1, P2	gain, loss and neutral scores separately
MID SPN	central and centroparietal sites consistent though somewhat more parietal compared to (Wynn et al., 2010)	200 ms immediately before feedback onset	CPz, Pz, CP1, CP2, P1, P2	gain, loss and neutral scores separately
MID RewP	centrofrontal and centroparietal sites consistent with (Broyd et al., 2012)	175–275 ms	FCz, Cz, CPz, CP1, CP2, FC1, FC2	gain and loss scores separately

Note. RewP = Reward Positivity; MID = Monetary Incentive Delay task; SPN = Stimulus Preceding Negativity.

Table 2

Descriptive statistics on self-report measures.

	Range	*Minimum*	*Maximum*	*M (SD)*
PANAS PA	19	26	45	36.76 (4.38)
PANAS NA	31	11	42	20.86 (5.89)
DERS Nonacceptance	21	6	27	13.58 (5.29)
DERS Goals	13	8	21	15.51 (3.32)
DERS Impulse	20	6	26	13.93 (4.32)
DERS Awareness	21	9	30	19.76 (5.08)
DERS Strategies	24	10	34	20.13 (5.72)
RST-PQ Reward Interest	17	11	28	20.42 (3.48)
RST-PQ Goal	11	17	28	22.86 (3.27)
RST-PQ Impulse	17	11	28	20.65 (3.99)
RST-PQ BIS	47	37	84	60.74 (10.84)
RST-PQ FFFS	20	12	32	22.05 (5.32)
RST-PQ Panic	16	6	22	12.63 (4.04)
RST-PQ Reward Reactivity	19	19	38	28.79 (4.79)
RST-PQ BAS	50	66	116	92.72 (10.86)
RST-PQ Defensive Fight	15	16	31	24.02 (4.06)

Note. PANAS = Positive and Negative Affect Schedule; PA = Positive Affect; NA = Negative Affect; DERS = Difficulties in Emotion Regulation Scale; RST-PQ = Reinforcement Sensitivity Theory – Personality Questionnaire; BAS = Behavioral Activation System; BIS = Behavioral Inhibition System; FFFS = Fight-Flight-Freeze System.

Table 3

Bivariate correlations among neural indices of reward responsiveness, self-report indices of reward responsiveness, affectivity, and emotion regulation.

		Doors – initial responsiveness to reward attainment		MID – reward anticipation						MID – initial responsiveness to reward attainment	
		RewP gain	RewP loss	Cue P3 gain	Cue P3 loss	Target P3 gain	Target P3 loss	SPN gain	SPN loss	RewP gain	RewP loss
PANAS PA	*r* (*p*)	.259 (.093)	.286 (.063)	.164 (.293)	.106 (.499)	.257 (.097)	.273 (.077)	-.003 (.985)	-.101 (.519)	.130 (.406)	.231 (.137)
	Bias (SE)	-.005 (.160)	-.005 (.136)	-.002 (.143)	.005 (.147)	-.004 (.113)	-.004 (.127)	.009 (.161)	.010 (.142)	.004 (.133)	.002 (.108)
	95% CI	(-.046; .564)	(.016; .526)	(-.121; .446)	(-.171; .416)	(.030; .458)	(.016; .520)	(-.287; .338)	(-.354; .204)	(-.139; .391)	(.011; .440)
PANAS NA	*r* (*p*)	**-.380 (.012)**	**-.359 (.018)**	-.022 (.888)	-.019 (.904)	-.118 (.449)	-.046 (.770)	-.048 (.761)	.007 (.966)	-.141 (.367)	-.189 (.225)
	Bias (SE)	.025 (.162)	.023 (.166)	-.001 (.121)	.001 (.133)	-.001 (.123)	-.002 (.129)	.010 (.160)	.005 (.149)	.027 (.177)	.026 (.183)
	95% CI	(-.625; -.023)	(-.627; -.001)	(-.258; .207)	(-.272; .248)	(-.358; .112)	(-.320; .200)	(-.385; .270)	(-.269; .317)	(-.423; .269)	(-.483; .209)
DERS Nonacceptance	*r* (*p*)	-.109 (.488)	-.136 (.383)	-.013 (.934)	-.047 (.763)	-.186 (.234)	-.060 (.703)	-.064 (.684)	-.047 (.765)	.098 (.531)	-.034 (.827)
	Bias (SE)	-.005 (.172)	-.003 (.172)	-.002 (.155)	.003 (.137)	.003 (.123)	.001 (.135)	.008 (.154)	.009 (.150)	-.007 (.188)	-.012 (.215)
	95% CI	(-.443; .219)	(-.471;.199)	(-.307; .302)	(-.294; .243)	(-.418; .050)	(-.328; .209)	(-.377; .255)	(-.334; .246)	(-.275; .439)	(-.470; .362)
DERS Goals	*r* (*p*)	-.072 (.646)	-.101 (.517)	-.258 (.095)	-.129 (.408)	-.108 (.490)	-.079 (.613)	.270 (.080)	.269 (.081)	-.044 (.780)	-.167 (.285)
	Bias (SE)	.004 (.142)	.008 (.137)	.005 (.132)	.005 (.113)	.004 (.133)	.003 (.119)	-.005 (.136)	-.005 (.151)	.009 (.135)	.008 (.137)
	95% CI	(-.342; .211)	(-.355; .182)	(-.501; .012)	(-.333; .110)	(-.348; .182)	(-.306; .184)	(-.007; .516)	(-.033; .552)	(-.292; .237)	(-.414; .125)
DERS Impulse	*r* (*p*)	.090 (.567)	-.010 (.951)	.033 (.832)	.036 (.819)	.017 (.913)	.017 (.916)	-.175 (.262)	**-.313 (.041)**	.012 (.939)	-.163 (.296)
	Bias (SE)	.000 (.161)	.007 (.167)	-.005 (.157)	-.010 (.150)	-.007 (.159)	-.011 (.154)	-.005 (.131)	-.006 (.125)	.004 (.139)	.004 (.156)
	95% CI	(-.221; .406)	(-.308; .356)	(-.276; .357)	(-.267; .340)	(-.296; .314)	(-.326; .303)	(-.464; .079)	(-.561; -.061)	(-.252; .301)	(-.447; .167)
DERS Awareness	*r* (*p*)	**-.335 (.028)**	-.263 (.089)	.064 (.686)	.008 (.959)	-.031 (.844)	.026 (.867)	-.146 (.350)	-.019 (.902)	-.117 (.456)	-.162 (.299)
	Bias (SE)	.009 (.129)	.008 (.125)	.001 (.138)	-.003 (.146)	.010 (.151)	.003 (.155)	.001 (.127)	.005 (.143)	.004 (.138)	.005 (.135)
	95% CI	(-.551; -.043)	(-.470; .007)	(-.206; .326)	(-.280; .298)	(-.313; .293)	(-.283; .335)	(-.380; .120)	(-.280; .266)	(-.374; .171)	(-.415; .119)
DERS Strategies	*r* (*p*)	-.262 (.090)	**-.305 (.046)**	**-.330 (.031)**	**-.308 (.045)**	-.232 (.134)	-.201 (.195)	.043 (.783)	-.033 (.832)	-.221 (.155)	**-.333 (.029)**
	Bias (SE)	.009 (.116)	.014 (.128)	.005 (.127)	-.002 (.125)	.010 (.156)	.006 (.149)	-.007 (.129)	-.012 (.134)	.001 (.116)	.003 (.118)
	95% CI	(-.467; -.021)	(-.522; -.030)	(-.556; -.067)	(-.557; -.064)	-.502 (.102)	(-.470; .112)	(-.232; .293)	(-.316; .208)	(-.438; .019)	(-.544; -.086)
RSTPQ RI	*r* (*p*)	.235 (.129)	.131 (.403)	.014 (.932)	.000 (.998)	.205 (.188)	.151 (.333)	.058 (.713)	-.031 (.846)	.111 (.477)	.098 (.530)
	Bias (SE)	-.016 (.150)	-.011 (.146)	-.002 (.143)	.006 (.155)	.001 (.114)	.000 (.131)	-.003 (.140)	.003 (.138)	.004 (.124)	.004 (.139)
	95% CI	(-.092; 479)	(-.184; .376)	(-.261; .318)	(-.263; .342)	(-.014; .429)	(-.089; .425)	(-.235; .302)	(-.300; .238)	(-.132; .353)	(-.169; .368)

Table 3 continued.

RSTPQ Goal	*r* (*p*)	.201 (.197)	.155 (.321)	.099 (.526)	.077 (.626)	.197 (.204)	.125 (.426)	.176 (.259)	-.066 (.674)	.091 (.560)	.090 (.566)
	Bias (SE)	-.005 (.145)	-.004 (.135)	-.002 (.122)	.003 (.137)	.000 (.136)	.002 (.135)	.003 (.127)	.003 (.141)	.000 (.134)	-.005 (.149)
	95% CI	(-.102; .470)	(-.103; .419)	(-.140; .335)	(-.167; .356)	(-.062; .463)	(-.125; .409)	(-.087; .427)	(-.343; .226)	(-.185; .353)	(-.230; .371)
RSTPQ RR	*r* (*p*)	.245 (.114)	.187 (.231)	.195 (.210)	.084 (.594)	**.335 (.028)**	.264 (.087)	-.080 (.608)	-.088 (.573)	**.316 (.039)**	**.338 (.027)**
	Bias (SE)	-.011 (.164)	-.009 (.152)	-.005 (.174)	.010 (.195)	-.001 (.134)	-.002 (.147)	.008 (.160)	.012 (.159)	-.001 (.155)	.000 (.156)
	95% CI	(-.115; .533)	(-.149; .468)	(-.158; .520)	(-.262; .476)	(.050; .568)	(-.047; .523)	(-.371; .271)	(-.381; .240)	(-.011; .573)	(.012; .605)
RSTPQ Impulse	*r* (*p*)	.035 (.821)	-.010 (.948)	.145 (.355)	.104 (.507)	.040 (.797)	-.015 (.925)	-.046 (.769)	-.021 (.894)	-.139 (.373)	-.075 (.633)
	Bias (SE)	-.002 (.180)	-.004 (.169)	-.013 (.171)	.002 (.165)	-.002 (.161)	.002 (.166)	-.008 (.163)	-.001 (.153)	.004 (.156)	.009 (.163)
	95% CI	(-.320; .387)	-.325 (.309)	(-.189; .450)	-.200 (.411)	(-.276; .352)	(-.341; .300)	(-.399; .246)	(-.322; .279)	(-.432; .196)	(-.366; -280)
RSTPQ BAS	*r* (*p*)	.257 (.096)	.167 (.284)	.173 (.267)	.098 (.532)	.288 (.061)	.197 (.205)	.019 (.903)	.076 (.627)	.152 (.332)	.180 (.248)
	Bias (SE)	-.013 (.151)	-.010 (.137)	-.009 (.180)	.007 (.205)	-.002 (.130)	-.001 (.145)	.000 (.136)	.006 (.138)	.003 (.127)	.001 (.133)
	95% CI	(-.080; .527)	(-.136; .430)	(-.208; .495)	(-.264; .496)	(.033; .527)	(-.085; .476)	(-.232;.306)	(-.326; .218)	(-.108; .383)	(-.095; .427)
RSTPQ BIS	*r* (*p*)	.029 (.854)	-.024 (.880)	-.162 (.299)	-.167 (.285)	-.179 (.252)	-.156 (.319)	-.002 (.991)	-.070 (.654)	.038 (.807)	-.101 (.519)
	Bias (SE)	.000 (.173)	.007 (.150)	-.003 (.146)	.006 (.136)	-.001 (.120)	.000 (.103)	.004 (.142)	.003 (.129)	.002 (.139)	.003 (.151)
	95% CI	(-.306; .351)	(-.295; .276)	(-.439; .126)	(-.400; .128)	(-.409; .055)	(-.368; .041)	(-.278; .293)	(-.313; .202)	(-.244; .300)	(-.391; .189)
RSTPQ FFFS	*r* (*p*)	.161 (.303)	**.311 (.042)**	.180 (.249)	.025 (.873)	.200 (.197)	.259 (.093)	-.012 (.937)	-.013 (.936)	**.384 (.011)**	*.296 (.054)*
	Bias (SE)	.001 (.128)	-.002 (.141)	.000 (.156)	.013 (.162)	-.003 (.182)	-.004 (.166)	.016 (.181)	.012 (.169)	.001 (.115)	-.001 (.131)
	95% CI	(-.098; .399)	(.017; .561)	(-.141; .477)	(-.269; .367)	(-.178; .525)	(-.082; .543)	(-.326; .346)	(-.326; .352)	(.157; .603)	(.021; .544)
RSTPQ Panic	*r* (*p*)	-.061 (.698)	-.019 (.903)	.069 (.660)	-.008 (.961)	-.013 (.934)	.096 (.542)	.033 (.831)	-.053 (.736)	.095 (.544)	-.042 (.788)
	Bias (SE)	.006 (.163)	.004 (.156)	-.002 (.157)	.010 (.162)	.001 (.147)	.002 (.145)	.012 (.143)	.004 (.129)	.004 (.151)	.002 (.150)
	95% CI	(-.354; .292)	(-.315; .298)	(-.227;.372)	(-.300; .335)	(-.302; .273)	(-.198; .380)	(-.219; .353)	(-.283; .213)	(-.197; .391)	(-.330; .260)
RSTPQ Def Fight	*r* (*p*)	.134 (.392)	.070 (.654)	.092 (.557)	.134 (.393)	-.001 (.996)	-.083 (.595)	-.032 (.839)	-.023 (.882)	-.109 (.488)	.018 (.909)
	Bias (SE)	.001 (.143)	.004 (.129)	-.008 (.127)	-.003 (.098)	.003 (.148)	.005 (.141)	-.007 (.160)	-.006 (.155)	-.003 (.117)	-.005 (.109)
	95% CI	(-.156; .410)	-.174 (.339)	(-.155; .328)	(-.049; .328)	(-.284; .308)	(-.341; .225)	(-.352; .246)	(-.355; .264)	(-.339; .113)	-.196 (.235)

Note. PANAS = Positive and Negative Affect Schedule; PA = Positive Affect; NA = Negative Affect; DERS = Difficulties in Emotion Regulation Scale; RST-PQ = Reinforcement Sensitivity Theory – Personality Questionnaire; RI = Reward Interest; RR = Reward Responsiveness; BAS = Behavioral Activation System; BIS = Behavioral Inhibition System; FFFS = Fight-Flight-Freeze System; Def Fight = Defensive Fight.
We considered an $r \geq .3$ (medium) (or a $p < .05$) as meaningful.

Table 4

Bivariate correlations among self-report indices of reward responsiveness, affectivity, and emotion regulation.

		RSTPQ RI	RSTPQ Goal	RSTPQ RR	RSTPQ Impulse	RSTPQ BAS	RSTPQ BIS	RSTPQ FFFS	RSTPQ Panic	RSTPQ Def Fight
PANAS PA	*r (p)*	**.576 (.000)**	**.489 (.001)**	**.421 (.005)**	.288 (.061)	**.623 (.000)**	.099 (.526)	.295 (.055)	**.321 (.036)**	.067 (.668)
	Bias (SE)	-.013 (.122)	-.001 (.133)	.001 (.151)	.000 (.141)	-.008 (.124)	-.008 (.191)	-.003 (.139)	.003 (.157)	.008 (.171)
	95% CI	(.281;.756)	(.198;.725)	(.098;.692)	-.030 (.533)	(.338;.818)	(-.294;.454)	(.003;.546)	-.009 (.621)	(-.247;.397)
PANAS NA	*r (p)*	.023 (.885)	-.185 (.235)	.029 (.852)	.188 (.226)	.034 (.831)	**.357 (.019)**	.082 (.600)	**.459 (.002)**	.185 (.234)
	Bias (SE)	.012 (.138)	.012 (.140)	.002 (.127)	-.026 (.184)	.002 (.111)	-.009 (.132)	-.004 (.143)	-.003 (.099)	-.005 (.145)
	95% CI	(-.198;.324)	(-.422;.126)	(-.211;.282)	(-.231;.489)	(-.178;.259)	.070 (.575)	(-.189;.370)	(.240;.632)	(-.135;.429)
DERS Nonacceptance	*r (p)*	.146 (.351)	-.103 (.513)	-.020 (.896)	.078 (.621)	.035 (.823)	**.481 (.001)**	.060 (.703)	.406 (.007)	-.129 (.408)
	Bias (SE)	-.008 (.148)	.001 (.141)	-.007 (.172)	-.003 (.165)	-.005 (.150)	-.012 (.114)	.004 (.167)	-.010 (.134)	-.004 (.136)
	95% CI	(-.164;.416)	(-.371;.178)	(-.398;.300)	(-.251;.386)	(-.269;.317)	(.228;.667)	(-.256;.405)	(.119;.649)	(-.390;.147)
DERS Goals	*r (p)*	.156 (.317)	.198 (.204)	.101 (.518)	.160 (.307)	.213 (.171)	**.310 (.043)**	.059 (.706)	.139 (.375)	.195 (.209)
	Bias (SE)	-.001 (.130)	.004 (.155)	.002 (.156)	.004 (.121)	.007 (.131)	.006 (.175)	.002 (.168)	.006 (.157)	.003 (.151)
	95% CI	(-.107;.400)	(-.106;.500)	(-.215;.394)	(-.077;.409)	(-.058;.480)	(-.058;.625)	(-.275;.383)	(-.186;.433)	(-.091;.489)
DERS Impulse	*r (p)*	**.342 (.025)**	.154 (.323)	.205 (.187)	.224 (.149)	**.329 (.031)**	**.448 (.003)**	-.011 (.943)	**.354 (.020)**	.258 (.095)
	Bias (SE)	-.015 (.144)	.000 (.140)	-.006 (.152)	-.008 (.152)	-.009 (.146)	-.012 (.138)	-.004 (.179)	.002 (.131)	.002 (.135)
	95% CI	(.024;.573)	-.131;.428)	(-.111;.481)	(-.103;.500)	(.012;.574)	(.136;.673)	(-.356;.350)	(.085;.613)	(-.030;.504)
DERS Awareness	*r (p)*	-.063 (.688)	.061 (.698)	.052 (.742)	-.114; (.465)	-.021 (.893)	-.018 (.907)	.102 (.513)	.172 (.271)	-.122 (.435)
	Bias (SE)	.001 (.143)	-.006 (.153)	.003 (.159)	.000 (.160)	.001 (.153)	-.008 (.149)	-.003 (.136)	-.017 (.157)	-.006 (.145)
	95% CI	(-.332;.230)	(-.255;.343)	(-.246;.368)	(-.426;.207)	(-.308;.284)	-.316 (.259)	(-.147;.365)	(-.165;.465)	(-.397;.166)
DERS Strategies	*r (p)*	.168 (.282)	.144 (.358)	.059 (.706)	.074 (.636)	.150 (.336)	**.592 (.000)**	-.034 (.829)	**.475 (.001)**	.179 (.250)
	Bias (SE)	-.008 (.143)	.009 (.139)	.003 (.132)	-.006 (.152)	.004 (.143)	-.010 (.106)	-.003 (.170)	-.005 (.127)	.002 (.137)
	95% CI	(-.132;.443)	(-.123;.416)	(-.186;.304)	(-.254;.358)	(-127;.425)	(.319;.750)	(-.353;.292)	(.193;.688)	(-.086;.455)

Note. PANAS = Positive and Negative Affect Schedule; PA = Positive Affect; NA = Negative Affect; DERS = Difficulties in Emotion Regulation Scale; RSTP-Q = Reinforcement Sensitivity Theory – Personal Questionnaire; RI = Reward Interest; RR = Reward Responsiveness; BAS = Behavioral Activation System; BIS = Behavioral Inhibition System; FFFS = Fight-Flight-Freeze System; Def Fight = Defensive Fight.
We considered an $r \geq .3$ (medium) (or a $p < .05$) as meaningful.

Table 5

Parameter estimates for individual dependent-independent variable pairings in each multivariate regression model.

DV	Parameter	*B*	*SE*	*t*	*P*[a]	95% CI	$\eta^2_{partial}$
Doors initial responsiveness to reward attainment – RewP to gain							
PANAS NA	Intercept	25.029	1.796	13.936	<.001	(21.402; 28.656)	.826
	RewP	**-.491**	**.187**	**-2.627**	**.046 (.012)**	**(-.868; -.114)**	**.144**
DERS Awareness	Intercept	22-943	1.578	14.542	<.001	(19.757; 26.129)	.838
	RewP	**-.374**	**.164**	**-2.278**	**.046 (.028)**	**(-.705; -.042)**	**.112**
Doors initial responsiveness to reward attainment – RewP to loss							
PANAS NA	Intercept	24.155	1.583	15.260	<.001	(20.958; 27.352)	.850
	RewP	**-.437**	**.177**	**-2.466**	**.046 (.018)**	**(-.794; -.079)**	**.129**
DERS Strategies	Intercept	22.858	1.568	14.578	<.001	(19.691; 26.024)	.838
	RewP	**-.360**	**.175**	**-2.054**	**.046 (.046)**	**(-.714; -.006)**	**.093**
RST-PQ FFFS	Intercept	19.471	1.456	13.375	<.001	(16.531; 22.411)	.814
	RewP	**.341**	**.163**	**2.096**	**.046 (.042)**	**(.012; .670)**	**.097**
MID initial responsiveness to reward attainment – RewP to gain							
RST-PQ Reward Reactivity	Intercept	24.984	1.916	13.041	<.001	(21.115; 28.853)	.806
	RewP	**.417**	**.195**	**2.135**	**.046 (.039)**	**(.023; .811)**	**.100**
RST-PQ FFFS	Intercept	16.912	2.073	8.159	<.001	(12.726; 21.098)	.619
	RewP	**.562**	**.211**	**2.662**	**.046 (.011)**	**(.136; .989)**	**.147**
MID initial responsiveness to reward attainment – RewP to loss							
RSTP-Q Reward Reactivity	Intercept	24.626	1.940	12.691	<.001	(20.707; 28.545)	.797
	RewP	**.470**	**.205**	**2.299**	**.046 (.027)**	**(.057; .883)**	**.114**
MID reward anticipation Cue P3 to gain							
DERS Strategies	Intercept	23.730	1.807	13.129	<.001	(20.080; 27.380)	.808
	Cue P3	**-.382**	**.171**	**-2.239**	**.046 (.031)**	**(-.727;-.037)**	**.109**
MID reward anticipation Cue P3 to loss							
DERS Strategies	Intercept	23.002	1.618	14.218	<.001	(19.735; 26.270)	.831
	Cue P3	**-.330**	**.160**	**-2.071**	**.046 (.045)**	**(-.652; -.008)**	**.095**
MID reward anticipation Target P3 to gain							
RST-PQ Reward Reactivity	Intercept	25.193	1.727	14.584	<.001	(21.705; 28.682)	.838
	Target P3	**.350**	**.154**	**2.276**	**.046 (.028)**	**(.039; .660)**	**.112**
MID reward anticipation SPN to loss							
DERS Impulse	Intercept	12.313	.995	12.373	<.001	(10.304; 14.323)	.789
	SPN	**-.308**	**.146**	**-2.107**	**.046 (.041)**	**(-.603; -.013)**	**.098**

Note. DV = dependent variable; PANAS = Positive and Negative Affect Schedule; NA = Negative Affect; PA = Positive Affect DERS = Difficulties in Emotion Regulation Scale; RST-PQ = Reinforcement Sensitivity Theory – Personality Questionnaire; FFFS = Fight-Flight-Freeze System; RewP = Reward Positivity. $\eta^2_{partial} \leq .02$ = small effect, $\leq .13$ = medium effect, $\leq .26$ = large effect. We considered $\eta^2_{partial} > .13$ (medium) (or a $p < .05$) as meaningful. [a] *p* values outside of the parentheses are Benjamini-Hochberg-corrected for FDR and *p* values inside the parentheses are uncorrected.