Developmental Cognitive Neuroscience 47 (2021) 100900

Contents lists available at ScienceDirect

Developmental Cognitive Neuroscience

journal homepage: www.elsevier.com/locate/dcn

A person-centered examination of emotion dysregulation, sensitivity to threat, and impulsivity among children and adolescents: An ERP study

Taylor Heffer *, Teena Willoughby

Department of Psychology, Brock University, 1812 Sir Isaac Brock Way, St. Catharines, ON, L2S 3A1, Canada

ARTICLE INFO

Keywords:
Adolescent
Children
Error-related negativity
Sensitivity to threat
Impulsivity
Dysregulation

ABSTRACT

Objectives: Adolescence often is characterized by the onset of social anxiety and risk taking; yet, not all youth are anxious and/or risk takers. There are several factors that may help differentiate youth with anxiety (e.g., threat sensitivity and emotion dysregulation) and youth who take risks (e.g., impulsivity and emotion dysregulation). We conducted a latent class analysis to identify groups of youth who differ in these processes, and then investigated group differences on the error-related negativity, an ERP that has been differentially associated with threat sensitivity and impulsivity.

Method: Youth (N = 1313, M_{age} = 11, range = 8–15 years) completed a survey assessing their emotion dysregulation, sensitivity to threat, and impulsivity. A subsample (N = 424) also completed a go/no-go task while EEG was recorded.

Results and conclusions: Four groups were identified with differential levels of emotion dysregulation, sensitivity to threat, and impulsivity. Adolescents had greater odds than children of being in the High_Dysregulation/ThreatSensitivity or ModerateDysregulation/HighImpulsivity Groups in comparison to two other groups with lower scores. The High_Dysregulation/ThreatSensitivity Group had the largest ERN, while the ModerateDysregulation/HighImpulsivity Group had the smallest ERN. The ERN may be a potential biomarker to help distinguish between different profiles of adolescents who may be at risk for either anxiety or risk taking.

1. Introduction

Adolescence often has been suggested to be a sensitive period of development, characterized by the onset of both internalizing problems (e.g., social anxiety; Beesdo et al., 2009) and externalizing problems (e.g., risk taking; Casey and Caudle, 2013; Casey et al., 2008; Dahl, 2004; Ernst, 2014). Yet, not all youth are socially anxious and/or engaging in extreme risks. Identifying factors that are differentially associated with these outcomes is critical in order to gain a better understanding of adolescent development. There are several important constructs that may help differentiate youth who may be more likely to develop anxiety versus risk-taking problems. Previous research has found that heightened *sensitivity to threat* (heightened responsiveness to threat) is associated with anxiety (Balle et al., 2013; Bar-Haim et al., 2007; Johnson et al., 2003; Katz et al., 2020; Pérez-Edgar et al., 2010, 2011), while *impulsivity* (non-reflective stimulus-driven response; Nigg, 2017) has been found to be associated with risk taking (Khurana et al., 2018; Romer et al., 2009). At the same time, both risk taking (Leith and Baumeister, 1996; Tull et al., 2012; Weiss et al., 2015) and anxiety (Cisler and Olatunji, 2012; Hannesdottir and Ollendick, 2007; Jazaieri et al., 2015; Mennin et al., 2009; Neumann et al., 2010; Suveg and Zeman, 2004; Tortella-Feliu et al., 2010) have been linked to *emotion dysregulation* (poor control over emotions). The current study used latent class analysis to investigate whether there are youth with different profiles of sensitivity to threat, impulsivity, and emotion dysregulation and whether these profiles are associated with a variety of factors (risk taking, social anxiety, age, pubertal status, sex, and parental education). A critical component of our study also was to investigate whether groups differ on the error-related negativity (ERN: An ERP elicited when making mistakes on an inhibitory control task), given that this neural indicator has been associated with both threat sensitivity and impulsivity (e.g., Boksem et al., 2008; Checa et al., 2014; Hajcak et al., 2003; Meyer, 2017; Ruchsow et al., 2005). Thus, the ERN may be an important way to distinguish between different profiles of individuals who may be at risk for the development of anxiety and/or risk-taking problems.

Neurodevelopmental imbalance models can help explain why adolescence may be a sensitive period for development. Specifically, asynchrony in the maturation of neural connections within and between

* Corresponding author.
E-mail address: th10ww@brocku.ca (T. Heffer).

https://doi.org/10.1016/j.dcn.2020.100900
Received 19 August 2020; Received in revised form 29 October 2020; Accepted 8 December 2020
Available online 24 December 2020

the prefrontal executive system (associated with self-control and potential suppression of socioemotional impulses) and the subcortical limbic-striatal system (associated with socioemotional processing) is thought to contribute to adolescents being more sensitive to emotionally salient events than children (Casey, 2015; Somerville et al., 2010; Steinberg, 2008). According to these models, circuitry within the subcortical limbic-striatal system matures early in adolescence (likely due to puberty), but interconnections to the prefrontal executive system mature later in adolescence. This asynchrony in maturity is thought to lead to heightened activation of the limbic-striatal region during a time when neural connections to the prefrontal cortex that might dampen the activation (if appropriate) are not fully mature. As a result, adolescents may be more susceptible than children to impulsive or emotionally driven responses (e.g., heightened sensitivity to threat), during a time when their ability to regulate their emotions is not yet mature.

Some researchers have investigated the imbalance model by assessing associations between neural activity in the prefrontal executive system and subcortical regions (e.g., amygdala, striatum). For example, Galvan et al. (2006) found that adolescents, compared to children and adults, had exaggerated activity in the accumbens relative to prefrontal activation during and fMRI task. Long-range neural connections between frontal regions and subcortical regions also have been found to increase from childhood to adolescence (Hwang et al., 2010). Hare et al. (2008) used an emotional go/no-go task and found that adolescents, compared to children, have exaggerated amygdala activity to fearful faces. They also found that stronger connectivity between the ventral medial prefrontal cortex and the amygdala was associated with habituation of the amygdala activity across trials. Dreyfuss et al. (2014) found that adolescents and adults showed greater activation in prefrontal regions than children, but they did not find significant age differences in the striatum. Mills et al. (2014) found that there was a structural mismatch in developmental timing between the amygdala and prefrontal cortex; however, they did not find clear evidence for the developmental mismatch between the nucleus accumbens and the prefrontal cortex. Overall, while there are some inconsistencies depending on what regions are investigated, there is some support for the imbalance model, suggesting that adolescence may be a sensitive period of development.

Puberty is thought to play an important role in why adolescence may be a sensitive period of development (Casey, 2015; Somerville et al., 2010; Steinberg, 2008). Previous research, however, often uses age rather than puberty as a key measure to investigate developmental differences between children and adolescents. While puberty and age are of course associated, puberty is marked by important changes in hormone levels that can impact adolescent brain development (Blakemore et al., 2010; Goddings et al., 2014; Sisk and Zehr, 2005; Vijayakumar et al., 2018). Indeed, there is considerable variability in the age at which different features of puberty develop (see Berenbaum et al., 2015 for an overview of the timing and measurement of pubertal development). Puberty is not a single event; thus, it is important to differentially measure a variety of physical signs associated with gonadal and adrenal hormonal development [e.g., body hair, breast development and menarche (in females), voice change and facial hair (in males)], especially when using self-reported measures of pubertal development which rely on youth self-identifying these features (Shirtcliff et al., 2009). Previous research has found that *self-reported* pubertal development (as measured by the Pubertal Development Scale) is associated with biological pubertal development (Schmitz et al., 2004; Shirtcliff et al., 2009). Taken together, it is important to investigate pubertal status (in addition to assessing age) in order to gain a better understanding of adolescent brain development.

In line with research on adolescent brain development, adolescents also may self-report greater sensitivity to threats, impulsivity, and emotion dysregulation, compared to children. For example, researchers have found that adolescents experience heightened sensitivity to threat compared to children (e.g., Heffer and Willoughby, 2020; O'Brien and Bierman, 1988; Vervoort et al., 2010; Westenberg et al., 2004). Research on age-related differences in impulsivity has been mixed. While some researchers have found that adolescents are more impulsive than children (Collado et al., 2014; Dreyfuss et al., 2014; Figner et al., 2009; Kasen et al., 2011; Khurana et al., 2018), others have found that impulsivity decreases from childhood to adolescence (Harden and Tucker-Drob, 2011; Quinn and Harden, 2013; Steinberg et al., 2008). Further, some researchers have found that dysregulation decreases throughout adolescence (Ahmed et al., 2015; Gee et al., 2013), but adolescents in particular may have difficulties with emotion regulation during 'hot' situations (i.e., when they are stressed or emotionally-aroused; e.g., Prencipe et al., 2011; Zelazo et al., 2010).

There also has been some research investigating the associations between impulsivity, sensitivity to threat and emotion dysregulation. For example, among adult samples, higher levels of emotion dysregulation has been associated with greater threat sensitivity (Schreiber et al., 2012; Slessareva and Muraven, 2004) and impulsivity (Jakubczyk et al., 2018; Schreiber et al., 2012). Khurana et al. (2018) used a latent growth curve analysis and identified two different groups of impulsive adolescents: high-increasing and low-stable. They found that adolescents in the high-increasing impulsive group had lower top-down control than those in the low-stable group. Overall, this work highlights that impulsivity and sensitivity to threat have both been separately associated with poor emotion regulation.

Of course, there likely are important individual differences among adolescents in the amount of sensitivity to threat, impulsivity, and dysregulation that they experience (Crone et al., 2016; Somerville et al., 2010). For example, although Hare et al. (2008) found that adolescents had exaggerated amygdala activity in an emotional-processing task compared to children and adults, there also was a great deal of variability in activity among the adolescents. van Duijvenvoorde et al. (2015) found that some adolescents were more sensitive to threat (e.g., avoiding risks), while others showed more impulsive tendencies. To address these individual differences and provide a more holistic understanding of adolescent's sensitivity to threat, impulsivity, and emotion dysregulation, a person-centered approach is needed.

A *person-centered* approach can be used to explore whether there are distinct subgroups of individuals who have different combinations of dysregulation, threat sensitivity, and impulsivity within the larger sample. For example, there may be a group within the population that has high dysregulation, high sensitivity to threat, but is less impulsive (this group may be at risk for the development of anxiety), whereas a separate group of youth may have high dysregulation, high impulsivity, but is less sensitive to threats (this group may be more likely to engage in risk taking). Both groups may be characterized by high dysregulation (a measure associated with both risk taking and anxiety) but would have differential levels of impulsivity and sensitivity to threat. Identifying subgroups of individuals who vary on these measures— as opposed to investigating associations among variables— is of key importance to gain a better understanding of adolescent development and to identify those who are risk for the development of anxiety and/or risk taking. Indeed, a person-centered analysis can capture important individual differences that may otherwise be missed in a variable-centered approach (Howard and Hoffman, 2018).

1.1. The error-related negativity

It is critical also to investigate whether groups of youth are distinguishable based on neural indicators. Neural indicators could potentially help predict later development of internalizing or externalizing problems (e.g., social anxiety and/or risk taking) at younger ages (e.g., before children are able to self-report issues with anxiety). The ERN (error-related negativity) may be a potential biomarker used to help distinguish between the different profiles of adolescents. The ERN is thought to be associated with performance monitoring, specifically the motivational significance of errors; whereby a larger ERN is associated with greater motivation to avoid errors (e.g., Hajcak and Foti, 2008;

Meyer et al., 2017). Previous research has found that impulsive individuals tend to have smaller ERN amplitudes than those who are less impulsive (Checa et al., 2014; Pailing et al., 2002; Ruchsow et al., 2005; Stahl and Gibbons, 2007; Taylor et al., 2018); perhaps as a result of reduced behavioral monitoring. In contrast, individuals with greater threat sensitivity or anxiety tend to have larger ERNs when making errors than those with lower threat sensitivity or anxiety (Boksem et al., 2008; Chong and Meyer, 2019; Hajcak et al., 2003; Ladouceur et al., 2006; Meyer, 2017; Meyer and Hajcak, 2019; Weinberg et al., 2010). No research, however, has taken into consideration whether different patterns of dysregulation, sensitivity to threat, and impulsivity are differentially associated with the ERN. Thus, it remains unclear as to whether the ERN may be a biomarker that can help to distinguish between different profiles of adolescents (e.g., those who may be more likely to engage in risk taking compared to those who may be more likely to develop anxiety).

1.2. Current study

The current study seeks to address three questions: (1) Using a person-centered latent-class approach, are there distinct groups of individuals who vary in levels of emotion dysregulation, sensitivity to threat, and impulsivity? (2) If there are distinct groups, what factors (risk taking, social anxiety, age, pubertal status, sex, and parental education) predict group membership? (3) Do groups show different neural activation on the ERN during an inhibitory control task?

2. Method

2.1. Participants

Participants ($N = 1314$, age range = 8–15, 49.96 % female) were drawn from several elementary and high schools in southern Ontario, Canada and were part of a larger study examining the associations between wellbeing and youth health-risk behaviors. Most participants were between the ages of 9 and 14 and the sample was fairly even distribution among these ages. Parent report indicated that 84.20 % of the children and adolescents were White, 1.70 % were Black, 2.12 % were Asian, 2.76 % were Hispanic, 0.85 % Indigenous, and 7.53 % were Mixed (a further 0.85 % of parents indicated that they preferred not to answer the question). Data on socioeconomic status indicated that mean levels of education for mothers and fathers was, on average, “completed an associate degree and/or technical diploma”.

2.2. Procedure

Students were invited to participate in the study through visits to schools. Surveys were completed in classrooms during school hours and all participants received gifts (e.g., backpacks) as compensation. The survey was split into two sections to reduce fatigue, with both sections completed within a 1-month period sometime between January and April. Starting in year 2 of the study, a subsample ($N = 468$) of participants also completed a Mobile Lab component in which EEG data was recorded. Parents were asked to identify if their child had any illnesses or disabilities (either physical or mental). Two participants were excluded because of a diagnosis of autism, one participant was excluded because they are prone to seizures, and one participant was excluded because of a diagnosis of cerebral palsy. There were 14 participants who had equipment issues during the task (e.g., the event markers did not show up) and three participants did not complete the task. There also were 16 participants who were not included because their EEG data was not usable (e.g., contained a larger number of muscle/movement artifacts). Seven participants did not follow the instructions (e.g., they were off task). Thus, the final sample included 424 participants. The sample of participants who had useable EEG data did not differ on any of the study variables compared to the sample of participants who were excluded (p’s $> .05$). The University Ethics Board approved this study and participants provided informed assent and their parents provided informed consent. Of note, there also were no significant differences between the full sample and the mobile lab sample, with one exception. The age of participants in the mobile lab ($M = 11.45$, $SD = 1.78$) on average, was younger than the age of participants in the full sample ($M = 11.77$, $SD = 1.72$), $p = .003$.

2.3. Missing data analysis

Missing data occurred because some participants did not finish the questionnaire (average missing data for the first section of the survey = 1.764 %; average missing data for the second section of the survey = 4.788 %) and because some participants were absent during the time of the survey. The percentage of students who completed the survey was 82 % for the first section and 81 % for the second section. Missing data was primarily due to absenteeism but also occasionally due to time conflicts, students declining to participate in one part of the survey, RA mistakes (e.g., not inviting a child to complete the survey), or students moving to another school district with no contact information. Missing data were imputed using the expectation-maximization algorithm (EM). EM retains cases that are missing survey waves and thus avoids the biased parameter estimates that can occur with pairwise or listwise deletion (Schafer and Graham, 2002).

2.4. Measures

2.4.1. Demographics

Pubertal status, age, sex, and parental education (one item per parent, averaged together) using a scale of 1= *did not finish high school* to 6 = *professional degree*) were collected. Pubertal status was assessed using the Puberty Development Scale (PDS; Petersen et al., 1988). The PDS is a self-report measure that assesses body hair, facial hair, and voice development in boys, and body hair, menarche, and breast development in girls. All items were rated on a 4-point scale from 1 (*not yet started changing*) to 4 (*change seems complete*). The PDS scale exhibits good reliability and validity (Carskadon and Acebo, 1993; Petersen et al., 1988). In our sample, Cronbach alpha was .81 for boys and .80 for girls.

2.4.2. Emotion dysregulation

Emotion dysregulation was measured using three items from the Difficulties with Emotion Regulation Scale (DERS; Gratz and Roemer, 2004). Participants reported the extent to which they agreed with items (“When I’m upset or stressed, I have difficulty concentrating”, “When I’m upset or stressed, I have difficulty thinking about anything else”, “When I’m upset or stressed, I start to feel bad about myself”) on a scale from 1 (*almost never*) to 4 (*almost always*). Higher scores indicate higher levels of emotion dysregulation. The Cronbach alpha in the present study was 0.81. Of note, the original DERS contains 36 items with six different subscales. Given that this study is part of a larger study investigating a wide range of health-risk behaviors among youth, it was not feasible to include all items. Previous research has investigated the DERS as a unitary construct and found that using a shortened scale with a subset of items is related to expected adjustment indicators (e.g., sleep, non-suicidal self-injury; Heffer and Willoughby, 2018; Semplonius et al., 2015; Tavernier and Willoughby, 2015). Regarding the current study, we ran an exploratory factor analysis with our DERS items and found that the items formed one factor (all factor loadings > 0.77).

2.4.3. Sensitivity to threat

Participants reported the extent to which they agreed with three items specifically examining sensitivity to threat from the Behavioral Inhibition Scale (Carver and White, 1994; “Criticism hurts me quite a bit”, “I feel worried when I think I have done poorly at something”, “I feel pretty worried or upset when I think or know somebody is angry at me") on a scale ranging from 1 (*strongly disagree*) to 4 (*strongly agree*). Higher scores indicate higher levels of threat sensitivity. Cronbach's alpha was 0.82.

2.4.4. Impulsivity

Impulsivity was measured using 4 items ("I do not consider the consequences before I act", "I say things without thinking", "I often act on the spur of the moment", "I do things without thinking"; Baars et al., 2015; Barratt, 1959; Patton et al., 1995; Van der Elst et al., 2012). Items were assessed on a 4-point scale from 1 (*almost never*) to 4 (*almost always*). Higher scores indicate higher impulsivity. Cronbach's alpha for this scale was .83.

2.4.5. Risk taking

Risk taking was assessed by asking students the extent to which they engaged in 21 risky behaviors in the past year (e.g., rode a bike without a helmet, cheated on a test, skipped school without permission, etc.). The list of risky behaviors was adapted from the Risk Involvement and Perception Scale (Shapiro et al., 1998) and overlaps with behaviors generated from other studies (e.g., Gonzalez et al., 1994; Gullone et al., 2000). Response options ranged from 0 (*0 times*) to 4 (*10 or more times*). We calculated the average for each student's risk-taking engagement; higher scores reflect higher risk taking.

2.4.6. Social anxiety

Four items from the Social Anxiety Scale for Children – Revised (SASC-R; La Greca and Stone, 1993) were used to assess symptoms of social anxiety. These items (e.g., "I am afraid other students my age will not like me", "I am quiet when I am with a group of other students my age") were measured on a 4-point Likert scale ranging from 1 (*almost never)* to 4 (*almost always*). Higher scores indicated higher levels of social anxiety. Cronbach's alpha for this scale was 0.71. Typically, the SASC-R contains 18 items, with three subscales. Given the nature of our study, we were unable to include all items and subscales. Previous research, however, has used this shortened 1-factor version of the SASC-R (Daly and Willoughby, 2020).

2.4.7. Go/No-go task

Participants completed the go/no-go task (DuPuis et al., 2015) while EEG was recorded. Participants were instructed to continuously push a button every time a stimulus appeared (a Go trial) unless the newly presented stimulus matched the previously presented stimulus (i.e., the same stimulus appeared twice in a row), in which case the participant needed to refrain from pushing the button on that trial (a No-go Trial). Stimuli were presented 1000 ms apart and there were a total of 225 trials. On average, participants committed 20 errors (sd = 9.78). The average reaction time to a no-go trial was 363 ms (sd =53.72 ms).

2.5. Electrophysiological recording

Electroencephalography (EEG) was recorded continuously from a BioSemi ActiveTwo system using a 96-channel montage and 7 face sensors. The data were digitized at a sampling rate of 512 Hz. Pre-processing was conducted to identify (1) channels/components that were unreliable within a given time-period, (2) time-periods that were unreliable, (3) and channels/components that were unreliable throughout the recording.

2.5.1. Pre-processing (Channels)

Pre-processing was automated (using MATLAB 2012b) using EEGLAB (Delorme & Makeig, 2004) version 13.6.5b, executed using Octave on Compute Canada's high performance computer cluster (Cedar: see Desjardins and Segalowitz, 2013; Desjardins et al., 2020; van Noordt et al., 2017, 2015 for more details). EEG Integrated Platform Lossless (EEG-IP-L) pre-processing pipeline has been shown to retain more data (trials and subjects) than other standardized pipelines [e.g., The Maryland Analysis of Developmental EEG (MADE)] without negatively impacting known ERP effects (Desjardins et al., 2020).

The data were first separated into 1 s non-overlapping time windows. For each time window, the voltage variance across each channel was calculated (a 20 % trimmed mean was used). Channels were flagged as unreliable if they had a z-score six times greater than the voltage variance across all channels. Time-periods (i.e., the 1 s time windows) were considered unreliable if more than 10 % of the channels were identified as having extreme voltage variances. Finally, any channels that were flagged in more than 20 % of the time-periods were considered unreliable throughout the recording.

The data were re-referenced to an interpolated average of 19 sites, excluding flagged channels. The data were filtered with a 1 Hz high pass and 30 Hz low pass filter given that cortical activity would not be expected to exceed 30 Hz. After this step, the data were again checked for the same issues reported above: (1) channels that are unreliable within a given time-period, (2) time-periods that are unreliable, (3) and channels that are unreliable throughout the recording. Specifically, any channels that were unlike its neighbouring channels (e.g., had a low correlation with channels around it), were flagged. A channel was flagged as unreliable if it had a z-score that was 2.326 times greater than the mean of the 20 % trimmed distribution of correlation coefficients. Time-periods were considered unreliable if more than 10 % of the channels within the window were flagged as unreliable. Any individual channels that were flagged in more than 10 % of time-periods were considered unreliable across the entire recording. Bridged channels (i.e., channels that are highly correlated with invariable signal) were identified after dividing the average maximum correlation by the standard deviation of the distribution of correlation coefficients. Channels that had a positive z-score that was eight times greater than the 40 % trimmed distribution of coefficients were flagged as bridged channels.

2.5.2. Pre-processing (components)

After pre-processing the channel data, all data that had not been flagged as unreliable was concatenated back into continuous data. These data were then submitted to an initial Adaptive Mixture of Independent Component Analysis (AMICA) to identify different components of the EEG data (e.g., heart rate components, eye blink components, cortical components etc.). This process helps to separate brain activity (neural components) from non-neural activity (e.g., muscle movement).

During this procedure, the data were windowed into 1 s time epochs. Unreliable components were detected by comparing each individual component to the variance among all components. Components were flagged if they had a z-score that was 2.326 times greater than the trimmed mean. Time-periods that had more than 10 % of its components flagged were considered unreliable. The data were then concatenated into the continuous time course and submitted to three simultaneous AMICA decompositions to assess whether components were replicable (i.e., is muscle movement consistently being classified as muscle movement when the process is repeated multiple times). The procedure above for identifying unreliable components (within 1 s epochs) was completed again using the continuous time series data. Next, a dipole (which identifies the position and orientation for the distribution of positive and negative voltages) was fit using the dipfit plugin in Matlab (Oostenveld et al., 2011). Components with a dipole fit residual variance greater than 15 % were flagged. Finally, components were classified using the ICMARC plugin. This process assesses each component against a crowd-sourced database to identify activation consistent with five different categories: eye blinks, neural, heart, lateral eye movements, muscle contamination, and mixed signal.

After pre-processing, a manual quality control review was completed to ensure that the decisions made during pre-processing were appropriate. This procedure was completed by one trained research assistant who assessed the accuracy of the independent component classifications. For example, the research assistant would identify whether cortical components were correctly distinguished from non-cortical components (e.g., muscle, eye blinks, etc.) based on topographical projection, continuous activation, dipole fit and power spectrum profile. Thus, the quality control review involved using the independent components to help with artifact correction.

2.5.3. EEG post-processing

EEG data were then segmented into single trials and time-locked to the onset of No-go responses from the Go/No-go task. A final quality check was completed to identify (and remove) channels that had extreme voltage fluctuations (+/−50 mV). Channels that were removed during pre-processing were interpolated (i.e., rebuilt using the remaining channel data) to the full montage of 103 channels (96 scalp, 7 exogenous) using spherical spline. The current study used fronto-central midline sites (FCz: electrodes A8 and B8 on our montage) to identify the ERN. Response-locked epochs were baseline corrected at -600 to -400. Participants with less than six error trials were removed from the analysis (Olvet and Hajcak, 2009).

2.6. Plan of analysis

Latent class analysis (LCA) was conducted using Mplus 7 (Muthén and Muthén, 2012). We used *MplusAutomation* (Hallquist and Wiley, 2018), a package in R (R Core Team, 2019), to automate the LCA and extract the model parameters from Mplus. The three dysregulation, three sensitivity to threat, and four impulsivity items were used as latent class indicators in order to explore whether different groups of individuals could be identified based on their responses to these items. In order to determine the number of groups that were best represented by the data, four criteria were considered: 1) interpretability of the classes, 2) Bayesian information criterion (BIC), such that smaller values of BIC indicate a better fit model, 3) significance of the Lo-Mendell-Rubin (LMR) significance value— once non-significance is reached, the number of classes prior to non-significance is defined as the appropriate number, and 4) average latent class conditional probabilities are close to 1.00 (Nylund et al., 2007). Entropy (an index of confidence that individuals belong to the correct class and that adequate separation between latent classes exist) also was examined; scores >.80 are good but there is no set cut-off criterion for entropy (Jung and Wickrama, 2008).

Once groups were identified, we investigated what factors (risk behaviors, social anxiety, age, pubertal status, sex, and parental education) predict group membership (see Table 1 for means, standard deviations, and correlations among study variables). Specifically, we ran a multinomial logistic regression with group status as the dependent variable and all of the factors were entered simultaneously as the independent variables. We also tested what factors (risk behaviors, social anxiety, age, pubertal status, sex, and parental education) predict the conditional probability of group membership (e.g., does risk taking predict whether individuals have a greater probability of being in a group with higher levels of impulsivity and higher levels of dysregulation?).

In order to investigate class differences on the ERN, we used STATSLAB, an open-source toolbox that implements robust statistics for analysis of EEG data (Campopiano et al., 2018). This software allows for testing using percentile bootstrap and trimmed means, a technique that is robust to distribution characteristics such as skew, outliers, uneven tails, and various model assumption violations (see Wilcox, 2017).

In STATSLAB, single trial data for our channels were extracted and averaged together. For each subject, the single trial data were resampled, with replacement, to generate a surrogate sampling distribution. The 20 % trimmed mean was taken across trials, at each time point (i.e., removing the most extreme voltages at each time point), to generate a robust bootstrapped ERP. This process was repeated for each group and the difference between groups taken. Iterating this process of resampling, trimming, and scoring the difference wave was performed 1,000 times to generate a distribution of differences between conditions (see Campopiano et al., 2018 for details). The 95 % confidence interval was obtained to test significant differences between ERP waveforms for each group.

3. Results

3.1. Q1: using latent class analysis, are there distinct groups of individuals who vary in emotion dysregulation, sensitivity to threat, and impulsivity?

The LCA was conducted for 1–6 classes. Four classes was chosen as the best solution (see Table 2). There was a decrease in BIC from 3 classes to 4 classes. Further, the 4-class solution had an entropy value above .80 and average latent class posterior probabilities were close to 1. The LMR was significant at 5 classes, but no longer significant for 6 classes. The 5th class, however, only contained 48 participants (3.7 % of the sample) and added little value to the interpretability of the groups. Therefore, a 4-class solution was chosen (see Fig. 1). The classes were labeled as follows: (1) HighDysregulation/HighThreatSensitivity/LowModImpulsivity (14.6 % of the sample) – hereafter labeled the *High_Dysregulation/ThreatSensitivity* group, given that the high levels of dysregulation and threat sensitivity indicators clearly distinguish this group from the other groups, (2) ModDysregulation/ModThreatSensitivity/HighImpulsivity (11.4 % of the sample) – labeled *ModDysregulation/HighImpulsivity* group, as this group is the only group with moderate dysregulation and high levels of impulsivity, (3) LowModDsyregulation_ModThreatSensitivity _LowModImpulsivity (57.6 %

Table 2
Latent class analysis (LCA) fit indices.

Number of Classes	BIC	Entropy	Conditional Probabilities	LMR *p*-value
2 Classes	29943.77	0.771	0.918−0.945	0.0000
3 Classes	29064.62	0.829	0.898−0.937	0.0097
4 Classes	28477.85	0.851	0.847−0.945	0.0033
5 Classes	28121.68	0.850	0.855−0.935	0.0038
6 Classes	27842.06	0.843	0.827−0.926	0.2636

Note. BIC = Bayesian information criterion. LMR = Lo-Mendell-Rubin.

Table 1
Means, standard deviations, and correlations with confidence intervals.

Variable	*M*	*SD*	1	2	3	4	5	6	7	8
1. ST	2.58	0.71								
2. DYS	2.19	0.75	.50**							
3. Imp	1.93	0.61	.17**	.36**						
4. Risk Taking	1.43	0.46	−.05	.09**	.25**					
5. Anxiety	1.95	0.66	.39**	.44**	.17**	.03				
6. Age	11.65	1.75	.17**	.13**	.19**	.15**	.08**			
7. Pubertal Status	2.07	0.87	.26**	.21**	.17**	.14**	.15**	.73**		
8. Sex	1.50	0.50	.25**	.06*	−.16**	−.12**	.17**	.06*	.28**	
9. Parental Education	4.12	0.75	.02	−.11**	−.14**	−.01	−.14**	.01	−.03	−.03

Note. M and *SD* are used to represent mean and standard deviation, respectively. ST = Sensitivity to threat, DYS = emotion dysregulation, IMP = impulsivity. * indicates $p < .05$. ** indicates $p < .01$.

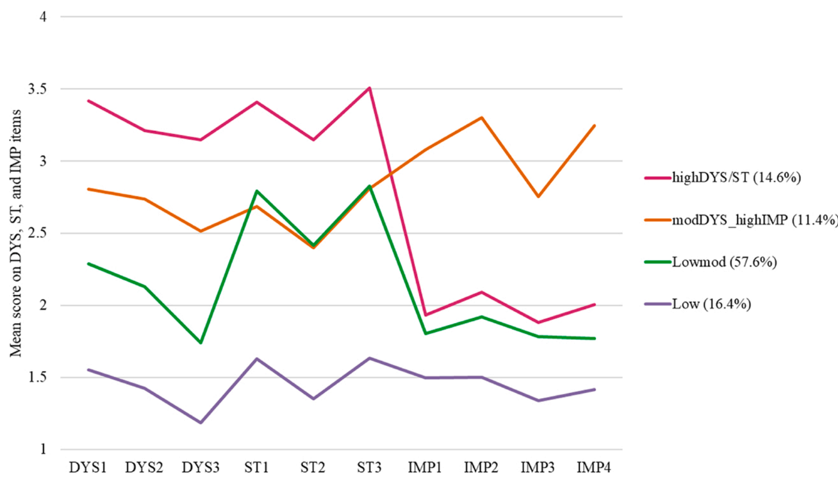

Fig. 1. Results of latent class analysis (LCA). DYS = Emotion dysregulation item, ST = Sensitivity to threat item, IMP = Impulsivity Item. highDYS/ST = High_Dysregulation/ ThreatSensitivity group; modDYS_highIMP = ModDysregulation/HighImpulsivity group. Percentages in brackets indicate the percent of the sample in each group.

of the sample) – labelled *lowmod* (normative) group, given that over 50 % of the sample is in this group, and (4) Low-Dysregulation_LowThreatSensitivity _LowImpulsivity (16.4 % of the sample) – labeled the *low* group, given their low scores on all indicators (see Table 3 for group differences on the indicators).

3.2. Q2a: what factors predict group membership?

In order to investigate what factors (risk taking, social anxiety, age, pubertal status, sex, and parental education) predict group membership, we ran three multinomial logistic regressions where we changed the reference category each time (the low group was the reference group in the first analysis, the lowmod (normative) group was the reference category for the second analysis, and the ModDysregulation/High-Impulsivity group was the reference category for the third analysis; see Tables 4–6 for complete model results).

The overall model was significant $\chi 2(18) = 361.231$, $p < .001$. Risk taking [$\chi 2(3)= 26.98$, $p < .001$], social anxiety [$\chi 2(3)= 178.919$, $p < .001$], pubertal status [$\chi 2(3)= 27.490$, $p < .001$], sex [$\chi 2(3)= 17.170$, $p < .001$], and parental education [$\chi 2(3)= 15.686$, $p = .001$] significantly differentiated among the classes. Age [$\chi 2(3)= 2.591$, $p = .459$] was not a significant predictor of class membership.

3.2.1. Risk taking

The ModDysregulation/HighImpulsivity group had greater odds of engaging in risk behaviors than the low group (OR = 2.776, $p < .001$), the lowmod (normative) group (OR = 2.403, $p < .001$), and the High_Dysregulation/ThreatSensitivity group (OR = 2.040, $p = .001$).

Table 3
Group means on latent class indicators.

	Low	Lowmod	ModDysregulation/ HighImpulsivity	High_Dysregulation/ ThreatSensitivity
DYS1	1.554_d	2.288_c	2.805_b	3.417_a
DYS2	1.422_d	2.131_c	2.739_b	3.211_a
DYS3	1.183_d	1.742_c	2.516_b	3.149_a
ST1	1.629_c	2.791_b	2.688_b	3.407_a
ST2	1.350_c	2.417_b	2.397_b	3.146_a
ST3	1.632_c	2.825_b	2.810_b	3.506_a
IMP1	1.499_c	1.804_b	3.080_a	1.934_b
IMP2	1.502_d	1.921_c	3.299_a	2.093_b
IMP3	1.340_c	1.784_b	2.752_a	1.881_b
IMP4	1.415_d	1.772_c	3.248_a	2.007_b

Note. DYS= Emotion dysregulation item, ST = Sensitivity to threat item, IMP = Impulsivity Item. Significant differences across groups are represented by letter subscripts that do not match (across rows), non-significant differences are represented by matching letter subscripts.

3.2.2. Social anxiety

Compared to the low group, individuals with higher social anxiety had greater odds of being in the lowmod (normative) group (OR = 2.882, $p < .001$), the ModDysregulation/HighImpulsivity group (OR = 5.401, $p < .001$), and the High_Dysregulation/ThreatSensitivity group (OR = 9.993, $p < .001$). Those with greater social anxiety also had greater odds of being in the ModDysregulation/HighImpulsivity group (OR = 1.874, $p < .001$) and the High_Dysregulation/ThreatSensitivity group (OR = 3.467, $p < .001$) compared to the lowmod (normative) group. Higher social anxiety was also associated with greater odds of being in the High_Dysregulation/ThreatSensitivity group compared to the ModDysregulation/HighImpulsivity group (OR = 1.850, $p < .001$).

3.2.3. Pubertal development

Participants with greater pubertal development had higher odds of being in the lowmod (OR = 1.554, $p = .005$), ModDysregulation/ HighImpulsivity (OR = 2.384, $p < .001$), and High_Dysregulation/ ThreatSensitivity (OR = 2.600, $p < .001$) groups compared to the low group. Participants with greater pubertal development also had greater odds of being in the ModDysregulation/HighImpulsivity (OR = 1.534, $p = .012$) and High_Dysregulation/ThreatSensitivity (OR = 1.673, $p < .001$) compared to the lowmod (normative) group.

3.2.4. Sex

Females had greater odds than males of being in the low (OR = 1.673, $p = .044$), lowmod (normative group; OR = 2.216, $p < .001$) and High_Dysregulation/ThreatSensitivity (OR = 2.370, $p < .001$) groups compared to the ModDysregulation/HighImpulsivity group.

3.2.5. Parental education

Individuals with higher parental education had greater odds of being in the low group (OR = 1.499, $p = .008$), lowmod (normative; OR = 1.637, $p < .001$), and High_Dysregulation/ThreatSensitivity (OR = 1.496, $p = .008$), compared to the ModDysregulation/HighImpulsivity group.

3.3. Q2b: what factors are associated with the conditional probabilities of group membership?

We also ran a follow up analysis to assess whether our study variables predict the *probability* of group membership (a continuous measure). To

Table 4
Multinomial Logistic Regression (Comparison Group: low).

Group	Variable	*B*	*SE*	OR	p.value	conf.low	conf.high
Lowmod	Risk_Taking	0.14	0.20	1.16	0.48	0.78	1.72
	Anxiety	1.06	0.17	2.88	0.00***	2.06	4.04
	Age	0.10	0.07	1.11	0.15	0.96	1.27
	Pubertal_Status	0.44	0.16	1.55	0.01**	1.14	2.12
	Sex	0.28	0.17	1.32	0.10	0.95	1.85
	Parent_Educ.	0.09	0.11	1.09	0.42	0.88	1.35
ModDysregulation/HighImpulsivity	Risk_Taking	1.02	0.24	2.78	0.00***	1.73	4.46
	Anxiety	1.69	0.21	5.40	0.00***	3.58	8.16
	Age	0.06	0.10	1.06	0.53	0.88	1.29
	Pubertal_Status	0.87	0.22	2.38	0.00***	1.56	3.64
	Sex	−0.52	0.26	0.60	0.04*	0.36	0.99
	Parent_Educ.	−0.40	0.15	0.67	0.01**	0.49	0.90
High_Dysregulation/ThreatSensitivity	Risk_Taking	0.31	0.26	1.36	0.24	0.82	2.26
	Anxiety	2.30	0.21	9.99	0.00***	6.68	14.95
	Age	0.04	0.09	1.04	0.68	0.86	1.25
	Pubertal_Status	0.96	0.21	2.60	0.00***	1.74	3.89
	Sex	0.35	0.24	1.42	0.15	0.88	2.28
	Parent_Educ.	−0.00	0.15	1.00	0.99	0.74	1.34

Note. Parent_Educ.= Parental education. SE = Standard error; OR = Odds Ratio; conf.low = lower bound confidence interval; conf.high = higher bound confidence interval.

Table 5
Multinomial Logistic Regression (Comparison Group: lowmod).

Group	Variable	*B*	*SE*	OR	p.value	conf.low	conf.high
low	Risk_Taking	−0.14	0.20	0.87	0.48	0.58	1.29
	Anxiety	−1.06	0.17	0.35	0.00***	0.25	0.49
	Age	−0.10	0.07	0.90	0.15	0.79	1.04
	Pubertal_Status	−0.44	0.16	0.64	0.01**	0.47	0.88
	Sex	−0.28	0.17	0.76	0.10	0.54	1.06
	Parental_Educ.	−0.09	0.11	0.92	0.42	0.74	1.13
ModDysregulation/HighImpulsivity	Risk_Taking	0.88	0.18	2.40	0.00***	1.70	3.39
	Anxiety	0.63	0.14	1.87	0.00***	1.42	2.48
	Age	−0.04	0.08	0.96	0.61	0.82	1.12
	Pubertal_Status	0.43	0.17	1.53	0.01*	1.10	2.14
	Sex	−0.80	0.21	0.45	0.00***	0.30	0.69
	Parental_Educ.	−0.49	0.13	0.61	0.00***	0.48	0.78
High_Dysregulation/ThreatSensitivity	Risk_Taking	0.16	0.19	1.18	0.40	0.81	1.72
	Anxiety	1.24	0.13	3.47	0.00***	2.69	4.48
	Age	−0.06	0.07	0.94	0.39	0.81	1.08
	Pubertal_Status	0.51	0.15	1.67	0.00***	1.24	2.26
	Sex	0.07	0.20	1.07	0.73	0.73	1.57
	Parental_Educ.	−0.09	0.12	0.91	0.45	0.72	1.16

Note. Parent_Educ.= Parental education. SE = Standard error; OR = Odds Ratio; conf.low = lower bound confidence interval; conf.high = higher bound confidence interval.

Table 6
Multinomial Logistic Regression (Comparison Group: ModDysregulation/HighImpulsivity).

Group	Variable	*B*	*SE*	OR	p.value	conf.low	conf.high
Low	Risk_Taking	−1.02	0.24	0.36	0.00***	0.22	0.58
	Anxiety	−1.69	0.21	0.19	0.00***	0.12	0.28
	Age	−0.06	0.10	0.94	0.53	0.78	1.14
	Pubertal_Status	−0.87	0.22	0.42	0.00***	0.28	0.64
	Sex	0.52	0.26	1.67	0.04*	1.01	2.77
	Parental_Educ.	0.40	0.15	1.50	0.01**	1.11	2.02
Lowmod	Risk_Taking	−0.88	0.18	0.42	0.00***	0.29	0.59
	Anxiety	−0.63	0.14	0.53	0.00***	0.40	0.71
	Age	0.04	0.08	1.04	0.61	0.89	1.21
	Pubertal_Status	−0.43	0.17	0.65	0.01*	0.47	0.91
	Sex	0.80	0.21	2.22	0.00***	1.45	3.38
	Parental_Educ.	0.49	0.13	1.64	0.00***	1.28	2.09
High_Dysregulation/ThreatSensitivity	Risk_Taking	−0.71	0.22	0.49	0.00**	0.32	0.76
	Anxiety	0.62	0.16	1.85	0.00***	1.35	2.54
	Age	−0.02	0.09	0.98	0.81	0.81	1.18
	Pubertal_Status	0.09	0.20	1.09	0.67	0.73	1.62
	Sex	0.86	0.26	2.37	0.00***	1.43	3.93
	Parental_Educ.	0.40	0.15	1.50	0.01**	1.11	2.02

Note. Parent_Educ.= Parental education. SE = Standard error; OR = Odds Ratio; conf.low = lower bound confidence interval; conf.high = higher bound confidence interval.

do this, we ran four linear regressions (one for each group's conditional probabilities). The probability of group membership was included as the dependent variable and the study variables (risk behaviors, social anxiety, age, pubertal status, sex, and parental education) were entered as the independent variables.

3.3.1. Low group

The overall model was significant, F(6,1303) = 27.645, p < .001. Lower social anxiety (B = -.241, SE = .014, p < .001) and less advanced pubertal status (B = -.142, SE = .016, p < .001) predicted greater probability of being in the low group.

3.3.2. Lowmod group

The overall model was significant, F(6,1303) = 7.202, p < .001. Lower social anxiety (B = -.122, SE = .019, p < .001), lower risk taking (B = -.083, SE = .026, p = .003), higher parental education (B = .061, SE = .016, p = .026), and female status (B = .059, SE = .026, p = .047) predicted greater probability of being in the lowmod group.

3.3.3. ModDysregulation/HighImpulsivity group

The overall model was significant, F(6,1303) = 16.623, p < .001. Higher social anxiety (B = .098, SE = .012, p < .001), higher risk taking (B = .165, SE = .018, p < .001), lower parental education (B = -.111, SE = .011, p < .001), male status (B = -.111, SE = .017, p < .001), and more advanced pubertal status (B = .090, SE = .014, p = .031) predicted greater probability of being in the ModDysregulation/HighImpulsivity group.

3.3.4. High_Dysregulation/ThreatSensitivity group

The overall model was significant, F(6,1303) = 39.889, p < .001. Higher social anxiety (B = .337, SE = .013, p < .001) and more advanced pubertal status (B = -.149, SE = .015, p < .001) predicted greater probability of being in the High_Dysregulation/ThreatSensitivity group.

3.4. Q3: do groups show different neural activation on the ERN during an inhibitory control task?

We investigated group differences on the ERN during an inhibitory control task. Results are presented in Fig. 2. The High_Dysregulation/ThreatSensitivity group had the largest ERN, while the ModDysregulation/HighImpulsivity group had the smallest ERN; the low and lowmod groups did not differ on their ERN (see Fig. 2). Of note, the groups did not differ on reaction time, F(3, 431) = 2.413, p = .066, or on the number of errors they committed, F(3, 431) = 1.907, p = .128, during the go/no-go task.

4. Discussion

Adolescence often has been suggested to be a sensitive period of development, characterized by the onset of both internalizing problems (e.g., social anxiety; Beesdo et al., 2009) and externalizing problems (risk taking; Casey et al., 2008; Casey and Caudle, 2013; Dahl, 2004; Ernst, 2014). Several factors may help to differentiate youth who are more likely to have anxiety problems (e.g., threat sensitivity and emotion dysregulation) and youth with risk-taking problems (e.g., impulsivity and emotion dysregulation). The current study examined whether there are distinct groups of individuals who vary on their levels of emotion dysregulation, impulsivity, and sensitivity to threat. At the same time, we were interested in differences between groups on both self-report measures (risk taking, social anxiety, age, pubertal status, parental education, and sex) and the error-related negativity.

We identified four groups with differential levels of emotion dysregulation, impulsivity and sensitivity to threat: (1) a group with high emotion dysregulation, high sensitivity to threat, and low/moderate impulsivity (labeled the *High_Dysregulation/ThreatSensitivity* group; 14.6 % of the sample) (2) a group with moderate emotion dysregulation, moderate sensitivity to threat, and high impulsivity (labeled *ModDysregulation/HighImpulsivity* group; 11.4 % of the sample) (3) a group with low/moderate emotion dysregulation, moderate sensitivity to threat, and low/moderate impulsivity (labeled the *lowmod group;* 57.6 % of the sample) and (4) a group with low emotion dysregulation, low

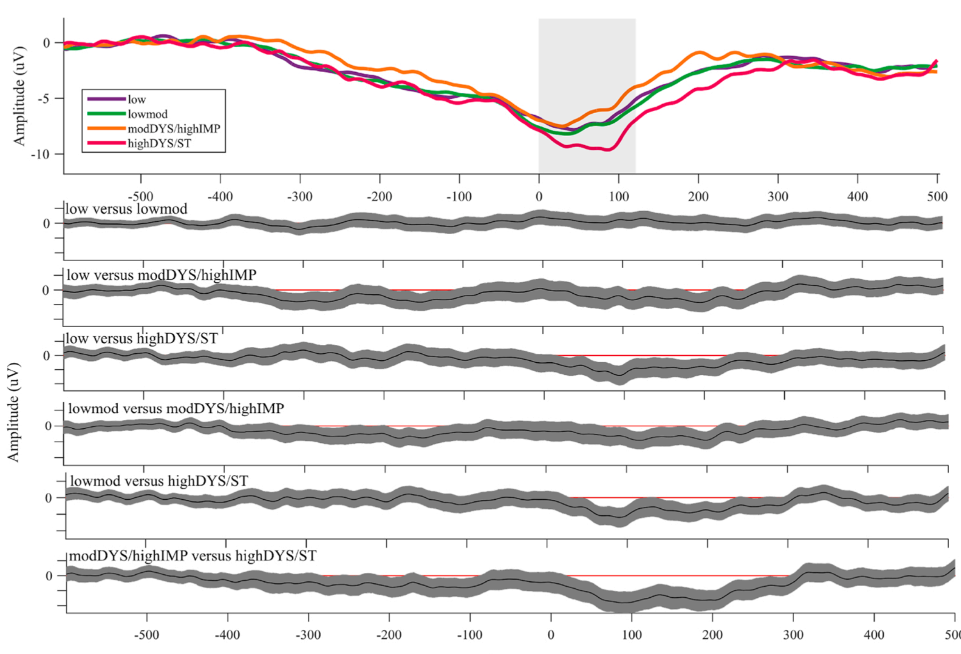

Fig. 2. Waveforms show the ERN for all groups. Bottom panels show the 95 % bootstrapped confidence intervals for the pairwise comparison for each group. Confidence intervals that do not overlap with the zero line (red) depict a significant difference at that time point. highDYS/ST = High_Dysregulation/ ThreatSensitivity group; modDYS_highIMP = ModDysregulation/ HighImpulsivity group.

sensitivity to threat, and low impulsivity (labeled the *low* group; 16.4 % of the sample). Given that over 50 % of the sample was part of the *lowmod* group, our results suggest that it is common for children and adolescents to experience low/moderate levels of both emotion dysregulation and impulsivity in combination with moderate sensitivity to threat.

Of interest, our results support neurodevelopmental imbalance models that suggest that adolescents may a sensitive period of development. Specifically, individuals with more advanced pubertal development had greater odds than those with lower pubertal development of being in the *High_Dysregulation/ThreatSensitivity* and *ModDysregulation/HighImpulsivity* groups. In other words, adolescents (those with greater pubertal development) were most likely to be part of the two groups with the highest dysregulation and high scores on either sensitivity to threat or impulsivity. We did not find, however, that age was a significant predictor of group membership. Thus, when pubertal status and age were included in the same model, age did not explain any additional variance that was not already captured by pubertal status. This result is in line with previous research and the imbalance model which highlights that pubertal status may be a more sensitive marker for adolescent sensitivity than age (e.g., Heffer and Willoughby, 2020; van den Bos et al., 2014).

We also found important individual differences that may help distinguish between the two groups that adolescents are most likely to be a part of – the *ModDysregulation/ HighImpulsivity* and the *High_Dysregulation/ThreatSensitivity groups*. For example, the *ModDysregulation/HighImpulsivity* group engaged in the most risk behaviors, were more likely to be male, and had lower parental education than the other groups. In contrast, the *High_Dysregulation/ThreatSensitivity* group had the greatest levels of social anxiety compared to all other groups. Our results remained consistent when we used conditional probabilities of group membership (a continuous measure of how likely an individual is to be part of each group)— notably, more advanced pubertal development and greater risk taking predicted higher probabilities of being in the *ModDysregulation/ HighImpulsivity group*, while more advanced pubertal development and greater social anxiety predicted higher probabilities of being in the *High_Dysregulation/ThreatSensitivity group*.

Our results suggest that adolescents have different profiles of impulsivity, sensitivity to threat, and emotion dysregulation that may contribute to whether they are more likely to display social anxiety or risk taking. Indeed, researchers interested in adolescent risk taking may need to target adolescents with moderate dysregulation and high impulsivity; males and individuals with lower parental education also may be particularly likely to be part of this group. In contrast, researchers interested in social anxiety may benefit from identifying adolescents who have high dysregulation in combination with high sensitivity to threat.

A critical component of our study was to identify neural differences between the groups. Specifically, we used the ERN, a neural measure of performance monitoring. Previous research has found that a larger ERN is associated with greater motivation to avoid errors (e.g., Hajcak and Foti, 2008; Meyer et al., 2017). We found that the *High_Dysregulation/ThreatSensitivity* group had the largest ERN, while the *ModDysregulation/HighImpulsivity* group had the smallest ERN. Thus, when individuals have high dysregulation and high sensitivity to threat, they may be particularly motivated to avoid making mistakes. In contrast, individuals who have moderate dysregulation, but high impulsivity may be less concerned with monitoring their performance. Indeed, one of the hallmarks of impulsivity is acting without thinking, which in combination with lower top-down control may contribute to this group having poorer performance monitoring. As a result, this group may be less bothered by (or take less notice of) making mistakes during the task, compared to groups with lower scores on impulsivity and dysregulation.

Our study has a number of strengths, including a large sample of children and adolescents, the use of a person-centered approach to isolate distinct groups, and the use of multiple methods (e.g., self-report and EEG), this study is not without limitations. First, we did not include the full scale for our core measures (emotion dysregulation, sensitivity to threat, and impulsivity). As the data were part of a larger study assessing a wide range of constructs, it was not feasible to include every item from each scale due to time constraints. Of note, however, the alpha for these measures were above .80, demonstrating good reliability (Cronbach, 1951). Second, causal inference cannot be concluded from our study. For example, we did not test whether having a profile of high dysregulation and high sensitivity to threat *causes* social anxiety, given the concurrent nature of this study. Finally, there are likely other factors that play a role in adolescents' sensitivity to emotion processing that were not included in this study (e.g., sensation seeking, peer presence).

Nonetheless, our study has important implications for adolescent development. Indeed, adolescents are more likely to be in the groups with greater dysregulation; at the same time there are differences in whether they have greater impulsivity or sensitivity to threat. Individual differences in emotion dysregulation, sensitivity to threat, and impulsivity are associated with differential outcomes. Specifically, high dysregulation in combination with high sensitivity to threat was associated with social anxiety, while moderate dysregulation combined with high impulsivity was associated with risk taking. It is imperative that researchers continue to investigate individual differences among adolescents. Our results highlight that not all adolescents are highly sensitive to threat, just as not all adolescents are highly impulsive. Therefore, sensitivity to emotional processing during adolescence may not be homogenous or display a universal profile. Finally, the ERN may be a potential biomarker to help distinguish between the different profiles of adolescents. Critically, this neural indicator could potentially help predict later development of internalizing or externalizing problems (e.g., social anxiety and/or risk taking) at younger ages (e.g., before children are able to self-report issues with anxiety).

Declaration of Competing Interest

The authors report no declarations of interest.

Acknowledgements

The second author acknowledges funding for this study received from Canadian Institutes of Health Research.

References

Ahmed, S.P., Bittencourt-Hewitt, A., Sebastian, C.L., 2015. Neurocognitive bases of emotion regulation development in adolescence. Dev. Cogn. Neurosci. 15, 11–25.
Baars, M.A.E., Nije Bijvank, M., Tonnaer, G.H., Jolles, J., 2015. Self-report measures of executive functioning are a determinant of academic performance in first-year students at a university of applied sciences. Front. Psychol. 6, 1131.
Balle, M., Tortella-Feliu, M., Bornas, X., 2013. Distinguishing youths at risk for anxiety disorders from self-reported BIS sensitivity and its psychophysiological concomitants. Int. J. Psychol. 48 (5), 964–977.
Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M.J., van IJzendoorn, M. H., 2007. Threat-related attentional bias in anxious and nonanxious individuals: a meta-analytic study. Psychol. Bull. 133 (1), 1–24.
Barratt, E.S., 1959. Anxiety and impulsiveness related to psychomotor efficiency. Percept. Mot. Skills 9 (3), 191–198.
Beesdo, K., Knappe, S., Pine, D.S., 2009. Anxiety and anxiety disorders in children and adolescents: developmental issues and implications for DSM-V. Psychiatr. Clin. North Am. 32 (3), 483–524.
Berenbaum, S.A., Beltz, A.M., Corley, R., 2015. Chapter two – the importance of puberty for adolescent development: conceptualization and measurement. In: Benson, J.B. (Ed.), Advances in Child Development and Behavior, Vol. 48. JAI, pp. 53–92.
Blakemore, S.-J., Burnett, S., Dahl, R.E., 2010. The role of puberty in the developing adolescent brain. Hum. Brain Mapp. 31 (6), 926–933.
Boksem, M.A.S., Tops, M., Kostermans, E., De Cremer, D., 2008. Sensitivity to punishment and reward omission: evidence from error-related ERP components. Biol. Psychol. 79 (2), 185–192.
Campopiano, A., van Noordt, S.J.R., Segalowitz, S.J., 2018. STATSLAB: an open-source EEG toolbox for computing single-subject effects using robust statistics. Behav. Brain Res. 347, 425–435.

Carskadon, M.A., Acebo, C., 1993. A self-administered rating scale for pubertal development. J. Adolesc. Health 14 (3), 190–195.
Carver, C.S., White, T.L., 1994. Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: the BIS/BAS scales. J. Pers. Soc. Psychol. 67 (2), 319–333.
Casey, B.J., 2015. Beyond simple models of self-control to circuit-based accounts of adolescent behavior. Annu. Rev. Psychol. 66, 295–319.
Casey, B.J., Caudle, K., 2013. The teenage brain: self control. Curr. Dir. Psychol. Sci. 22 (2), 82–87.
Casey, B.J., Jones, R.M., Hare, T.A., 2008. The adolescent brain. Ann. N. Y. Acad. Sci. 1124, 111–126.
Checa, P., Castellanos, M.C., Abundis-Gutiérrez, A., Rosario Rueda, M., 2014. Development of neural mechanisms of conflict and error processing during childhood: implications for self-regulation. Front. Psychol. 5, 326.
Chong, L.J., Meyer, A., 2019. Understanding the link between anxiety and a neural marker of anxiety (the error-related negativity) in 5 to 7 year-old children. Dev. Neuropsychol. 44 (1), 71–87.
Cisler, J.M., Olatunji, B.O., 2012. Emotion regulation and anxiety disorders. Curr. Psychiatry Rep. 14 (3), 182–187.
Collado, A., Felton, J.W., MacPherson, L., Lejuez, C.W., 2014. Longitudinal trajectories of sensation seeking, risk taking propensity, and impulsivity across early to middle adolescence. Addict. Behav. 39 (11), 1580–1588.
Crone, E.A., van Duijvenvoorde, A.C.K., Peper, J.S., 2016. Annual Research Review: neural contributions to risk-taking in adolescence–developmental changes and individual differences. J. Child Psychol. Psychiatry Allied Disciplines 57 (3), 353–368.
Dahl, R.E., 2004. Adolescent brain development: a period of vulnerabilities and opportunities. Keynote address. Ann. N. Y. Acad. Sci. 1021, 1–22.
Daly, O., Willoughby, T., 2020. A longitudinal person-centered examination of affinity for aloneness among children and adolescents. Child Dev. 41, 341.
Desjardins, J.A., Segalowitz, S.J., 2013. Deconstructing the early visual electrocortical responses to face and house stimuli. J. Vis. 13 (5) https://doi.org/10.1167/13.5.22.
Desjardins, J.A., van Noordt, S., Huberty, S., Segalowitz, S.J., Elsabbagh, M., 2020. EEG Integrated Platform Lossless (EEG-IP-L) pre-processing pipeline for objective signal quality assessment incorporating data annotation and blind source separation. J. Neurosci. Methods 347, 108961.
Dreyfuss, M., Caudle, K., Drysdale, A.T., Johnston, N.E., Cohen, A.O., Somerville, L.H., et al., 2014. Teens impulsively react rather than retreat from threat. Dev. Neurosci. 36 (3–4), 220–227.
DuPuis, D., Ram, N., Willner, C.J., Karalunas, S., Segalowitz, S.J., Gatzke-Kopp, L.M., 2015. Implications of ongoing neural development for the measurement of the error-related negativity in childhood. Dev. Sci. 18 (3), 452–468.
Ernst, M., 2014. The triadic model perspective for the study of adolescent motivated behavior. Brain Cogn. 89, 104–111.
Figner, B., Mackinlay, R.J., Wilkening, F., Weber, E.U., 2009. Affective and deliberative processes in risky choice: age differences in risk taking in the Columbia Card Task. J. Exp. Psychol. Learn. Mem. Cogn. 35 (3), 709–730.
Galvan, A., Hare, T.A., Parra, C.E., Penn, J., Voss, H., Glover, G., Casey, B.J., 2006. Earlier development of the accumbens relative to orbitofrontal cortex might underlie risk-taking behavior in adolescents. J. Neurosci. 26 (25), 6885–6892.
Gee, D.G., Humphreys, K.L., Flannery, J., Goff, B., Telzer, E.H., Shapiro, M., et al., 2013. A developmental shift from positive to negative connectivity in human amygdala-prefrontal circuitry. J. Neurosci. 33 (10), 4584–4593.
Goddings, A.-L., Mills, K.L., Clasen, L.S., Giedd, J.N., Viner, R.M., Blakemore, S.-J., 2014. The influence of puberty on subcortical brain development. NeuroImage 88, 242–251.
Gonzalez, J., Field, T., Yando, R., Gonzalez, K., Lasko, D., Bendell, D., 1994. Adolescents' perceptions of their risk-taking behavior. Adolescence 29 (115), 701–709.
Gratz, K.L., Roemer, L., 2004. Multidimensional assessment of emotion regulation and dysregulation: development, factor structure, and initial validation of the difficulties in emotion regulation scale. J. Psychopathol. Behav. Assess. 26 (1).
Gullone, E., Moore, S., Moss, S., Boyd, C., 2000. The adolescent risk-taking questionnaire. J. Adolesc. Res. 15 (2), 231–250.
Hajcak, G., Foti, D., 2008. Errors are aversive: defensive motivation and the error-related negativity. Psychol. Sci. 19 (2), 103–108.
Hajcak, G., McDonald, N., Simons, R.F., 2003. Anxiety and error-related brain activity. Biol. Psychol. 64 (1–2), 77–90.
Hallquist, M.N., Wiley, J.F., 2018. MplusAutomation: an r package for facilitating large-scale latent variable analyses in Mplus. Struct. Equ. Model. A Multidiscip. J. 25 (4), 621–638.
Hannesdottir, D.K., Ollendick, T.H., 2007. The role of emotion regulation in the treatment of child anxiety disorders. Clin. Child Fam. Psychol. Rev. 10 (3), 275–293.
Harden, K.P., Tucker-Drob, E.M., 2011. Individual differences in the development of sensation seeking and impulsivity during adolescence: further evidence for a dual systems model. Dev. Psychol. 47 (3), 739–746.
Hare, T.A., Tottenham, N., Galvan, A., Voss, H.U., Glover, G.H., Casey, B.J., 2008. Biological substrates of emotional reactivity and regulation in adolescence during an emotional go-nogo task. Biol. Psychiatry 63 (10), 927–934.
Heffer, T., Willoughby, T., 2018. The role of emotion dysregulation: a longitudinal investigation of the interpersonal theory of suicide. Psychiatry Res. 260, 379–383.
Heffer, T., Willoughby, T., 2020. Sensitivity to negative feedback among children and adolescents: an ERP study comparing developmental differences between high-worriers and low-worriers. Cogn. Affect. Behav. Neurosci. 20, 624–635. https://doi.org/10.3758/s13415-020-00791-8.
Howard, M.C., Hoffman, M.E., 2018. Variable-centered, person-centered, and person-specific approaches: where theory meets the method. Organ. Res. Methods 21 (4), 846–876.
Hwang, K., Velanova, K., Luna, B., 2010. Strengthening of top-down frontal cognitive control networks underlying the development of inhibitory control: a functional magnetic resonance imaging effective connectivity study. J. Neurosci. 30 (46), 15535–15545.
Jakubczyk, A., Trucco, E.M., Kopera, M., Kobyliński, P., Suszek, H., Fudalej, S., et al., 2018. The association between impulsivity, emotion regulation, and symptoms of alcohol use disorder. J. Subst. Abuse Treat. 91, 49–56.
Jazaieri, H., Morrison, A.S., Goldin, P.R., Gross, J.J., 2015. The role of emotion and emotion regulation in social anxiety disorder. Curr. Psychiatry Rep. 17 (1), 531.
Johnson, S.L., Turner, R.J., Iwata, N., 2003. BIS/BAS levels and psychiatric disorder: an epidemiological study. J. Psychopathol. Behav. Assess. 25 (1), 25–36.
Jung, T., Wickrama, K.A.S., 2008. An introduction to latent class growth analysis and growth mixture modeling. Soc. Personal. Psychol. Compass 2 (1), 302–317.
Kasen, S., Cohen, P., Chen, H., 2011. Developmental course of impulsivity and capability from age 10 to age 25 as related to trajectory of suicide attempt in a community cohort. Suic. Life-Threat. Behav. 41 (2), 180–192.
Katz, B.A., Matanky, K., Aviram, G., Yovel, I., 2020. Reinforcement sensitivity, depression and anxiety: a meta-analysis and meta-analytic structural equation model. Clin. Psychol. Rev. 77, 101842.
Khurana, A., Romer, D., Betancourt, L.M., Hurt, H., 2018. Modeling trajectories of sensation seeking and impulsivity dimensions from early to late adolescence: universal trends or distinct sub-groups? J. Youth Adolesc. 47 (9), 1992–2005.
La Greca, A.M., Stone, W.L., 1993. Social anxiety scale for children-revised: factor structure and concurrent validity. J. Clin. Child Psychol. 22 (1), 17–27.
Ladouceur, C.D., Dahl, R.E., Birmaher, B., Axelson, D.A., Ryan, N.D., 2006. Increased error-related negativity (ERN) in childhood anxiety disorders: ERP and source localization. J. Child Psychol. Psychiatry Allied Disciplines 47 (10), 1073–1082.
Leith, K.P., Baumeister, R.F., 1996. Why do bad moods increase self-defeating behavior? Emotion, risk tasking, and self-regulation. J. Pers. Soc. Psychol. 71 (6), 1250–1267.
Mennin, D.S., McLaughlin, K.A., Flanagan, T.J., 2009. Emotion regulation deficits in generalized anxiety disorder, social anxiety disorder, and their co-occurrence. J. Anxiety Disord. 23 (7), 866–871.
Meyer, A., 2017. A biomarker of anxiety in children and adolescents: a review focusing on the error-related negativity (ERN) and anxiety across development. Dev. Cogn. Neurosci. 27, 58–68.
Meyer, A., Hajcak, G., 2019. A review examining the relationship between individual differences in the error-related negativity and cognitive control. Int. J. Psychophysiol. 144, 7–13.
Meyer, A., Hajcak, G., Glenn, C.R., Kujawa, A.J., Klein, D.N., 2017. Error-related brain activity is related to aversive potentiation of the startle response in children, but only the ERN is associated with anxiety disorders. Emotion 17 (3), 487–496.
Mills, K.L., Goddings, A.-L., Clasen, L.S., Giedd, J.N., Blakemore, S.-J., 2014. The developmental mismatch in structural brain maturation during adolescence. Dev. Neurosci. 36 (3–4), 147–160.
Muthén, L.K., Muthén, B.O., 2012. Mplus: Statistical Analysis With Latent Variables; User's Guide;[version 7]. Muthén et Muthén.
Neumann, A., van Lier, P.A.C., Gratz, K.L., Koot, H.M., 2010. Multidimensional assessment of emotion regulation difficulties in adolescents using the Difficulties in Emotion Regulation Scale. Assessment 17 (1), 138–149.
Nigg, J.T., 2017. Annual Research Review: on the relations among self-regulation, self-control, executive functioning, effortful control, cognitive control, impulsivity, risk-taking, and inhibition for developmental psychopathology. J. Child Psychol. Psychiatry Allied Disciplines 58 (4), 361–383.
Nylund, K.L., Asparouhov, T., Muthén, B.O., 2007. Deciding on the number of classes in latent class analysis and growth mixture modeling: a Monte Carlo simulation study. Struct. Equ. Model. A Multidiscip. J. 14 (4), 535–569.
O'Brien, S.F., Bierman, K.L., 1988. Conceptions and perceived influence of peer groups: interviews with preadolescents and adolescents. Child Dev. 59 (5), 1360–1365.
Olvet, D.M., Hajcak, G., 2009. Reliability of error-related brain activity. Brain Res. 1284, 89–99.
Oostenveld, R., Fries, P., Maris, E., Schoffelen, J.-M., 2011. FieldTrip: open source software for advanced analysis of MEG, EEG, and invasive electrophysiological data. Comput. Intell. Neurosci. 2011, 156869.
Pailing, P.E., Segalowitz, S.J., Dywan, J., Davies, P.L., 2002. Error negativity and response control. Psychophysiology 39 (2), 198–206.
Patton, J.H., Stanford, M.S., Barratt, E.S., 1995. Factor structure of the Barratt impulsiveness scale. J. Clin. Psychol. 51 (6), 768–774.
Pérez-Edgar, K., Bar-Haim, Y., McDermott, J.M., Chronis-Tuscano, A., Pine, D.S., Fox, N. A., 2010. Attention biases to threat and behavioral inhibition in early childhood shape adolescent social withdrawal. Emotion 10 (3), 349–357.
Pérez-Edgar, K., Reeb-Sutherland, B.C., McDermott, J.M., White, L.K., Henderson, H.A., Degnan, K.A., et al., 2011. Attention biases to threat link behavioral inhibition to social withdrawal over time in very young children. J. Abnorm. Child Psychol. 39 (6), 885–895.
Petersen, A.C., Crockett, L., Richards, M., Boxer, A., 1988. A self-report measure of pubertal status: reliability, validity, and initial norms. J. Youth Adolesc. 17 (2), 117–133.
Prencipe, A., Kesek, A., Cohen, J., Lamm, C., Lewis, M.D., Zelazo, P.D., 2011. Development of hot and cool executive function during the transition to adolescence. J. Exp. Child Psychol. 108 (3), 621–637.
Quinn, P.D., Harden, K.P., 2013. Differential changes in impulsivity and sensation seeking and the escalation of substance use from adolescence to early adulthood. Dev. Psychopathol. 25 (1), 223–239.

R Core Team, 2019. R: A Language and Environment for Statistical Computing. Retrieved from. https://www.R-project.org/.
Romer, D., Betancourt, L., Giannetta, J.M., Brodsky, N.L., Farah, M., Hurt, H., 2009. Executive cognitive functions and impulsivity as correlates of risk taking and problem behavior in preadolescents. Neuropsychologia 47 (13), 2916–2926.
Ruchsow, M., Spitzer, M., Grön, G., Grothe, J., Kiefer, M., 2005. Error processing and impulsiveness in normals: evidence from event-related potentials. Brain Res. Cogn. Brain Res. 24 (2), 317–325.
Schafer, J.L., Graham, J.W., 2002. Missing data: our view of the state of the art. Psychol. Methods 7 (2), 147–177.
Schmitz, K.E., Hovell, M.F., Nichols, J.F., Irvin, V.L., Keating, K., Simon, G.M., et al., 2004. A validation study of early adolescents' pubertal self-assessments. J. Early Adolesc. 24 (4), 357–384.
Schreiber, L.R.N., Grant, J.E., Odlaug, B.L., 2012. Emotion regulation and impulsivity in young adults. J. Psychiatr. Res. 46 (5), 651–658.
Semplonius, T., Good, M., Willoughby, T., 2015. Religious and Non-religious Activity Engagement as Assets in Promoting Social Ties Throughout University: The Role of Emotion Regulation. J. Youth Adolesc. 44 (8), 1592–1606.
Shapiro, R., Siegel, A.W., Scovill, L.C., Hays, J., 1998. Risk-taking patterns of female adolescents: what they do and why. J. Adolesc. 21 (2), 143–159.
Shirtcliff, E.A., Dahl, R.E., Pollak, S.D., 2009. Pubertal development: correspondence between hormonal and physical development. Child Dev. 80 (2), 327–337.
Sisk, C.L., Zehr, J.L., 2005. Pubertal hormones organize the adolescent brain and behavior. Front. Neuroendocrinol. 26 (3–4), 163–174.
Slessareva, E., Muraven, M., 2004. Sensitivity to punishment and self-control: the mediating role of emotion. Pers. Individ. Dif. 36 (2), 307–319.
Somerville, L.H., Jones, R.M., Casey, B.J., 2010. A time of change: behavioral and neural correlates of adolescent sensitivity to appetitive and aversive environmental cues. Brain Cogn. 72 (1), 124–133.
Stahl, J., Gibbons, H., 2007. Dynamics of response-conflict monitoring and individual differences in response control and behavioral control: An electrophysiological investigation using a stop-signal task. Clin. Neurophysiol. 118 (3), 581–596.
Steinberg, L., 2008. A social neuroscience perspective on adolescent risk-taking. Dev. Rev. 28 (1), 78–106.
Steinberg, L., Albert, D., Cauffman, E., Banich, M., Graham, S., Woolard, J., 2008. Age differences in sensation seeking and impulsivity as indexed by behavior and self-report: evidence for a dual systems model. Dev. Psychol. 44 (6), 1764–1778.
Suveg, C., Zeman, J., 2004. Emotion regulation in children with anxiety disorders. J. Clin. Child Adolesc. Psychol. 33 (4), 750–759.
Tavernier, R., Willoughby, T., 2015. A longitudinal examination of the bidirectional association between sleep problems and social ties at university: the mediating role of emotion regulation. J. Youth Adolesc. 44 (2), 317–330.
Taylor, J.B., Visser, T.A.W., Fueggle, S.N., Bellgrove, M.A., Fox, A.M., 2018. The error-related negativity (ERN) is an electrophysiological marker of motor impulsiveness on the Barratt Impulsiveness Scale (BIS-11) during adolescence. Dev. Cogn. Neurosci. 30, 77–86.
Tortella-Feliu, M., Balle, M., Sesé, A., 2010. Relationships between negative affectivity, emotion regulation, anxiety, and depressive symptoms in adolescents as examined through structural equation modeling. J. Anxiety Disord. 24 (7), 686–693.
Tull, M.T., Weiss, N.H., Adams, C.E., Gratz, K.L., 2012. The contribution of emotion regulation difficulties to risky sexual behavior within a sample of patients in residential substance abuse treatment. Addict. Behav. 37 (10), 1084–1092.
van den Bos, E., de Rooij, M., Miers, A.C., Bokhorst, C.L., Westenberg, P.M., 2014. Adolescents' increasing stress response to social evaluation: pubertal effects on cortisol and alpha-amylase during public speaking. Child Dev. 85 (1), 220–236.
Van der Elst, W., Ouwehand, C., van der Werf, G., Kuyper, H., Lee, N., Jolles, J., 2012. The Amsterdam Executive Function Inventory (AEFI): psychometric properties and demographically corrected normative data for adolescents aged between 15 and 18 years. J. Clin. Exp. Neuropsychol. 34 (2), 160–171.
van Duijvenvoorde, A.C.K., Huizenga, H.M., Somerville, L.H., Delgado, M.R., Powers, A., Weeda, W.D., et al., 2015. Neural correlates of expected risks and returns in risky choice across development. J. Neurosci. 35 (4), 1549–1560.
van Noordt, S.J.R., Desjardins, J.A., Segalowitz, S.J., 2015. Watch out! Medial frontal cortex is activated by cues signaling potential changes in response demands. NeuroImage 114, 356–370.
van Noordt, S.J.R., Desjardins, J.A., Gogo, C.E.T., Tekok-Kilic, A., Segalowitz, S.J., 2017. Cognitive control in the eye of the beholder: electrocortical theta and alpha modulation during response preparation in a cued saccade task. NeuroImage 145 (Pt A), 82–95.
Vervoort, L., Wolters, L.H., Hogendoorn, S.M., De Haan, E., Boer, F., Prins, P.J.M., 2010. Sensitivity of Gray's behavioral inhibition system in clinically anxious and non-anxious children and adolescents. Pers. Individ. Dif. 48 (5), 629–633.
Vijayakumar, N., Op de Macks, Z., Shirtcliff, E.A., Pfeifer, J.H., 2018. Puberty and the human brain: insights into adolescent development. Neurosci. Biobehav. Rev. 92, 417–436.
Weinberg, A., Olvet, D.M., Hajcak, G., 2010. Increased error-related brain activity in generalized anxiety disorder. Biol. Psychol. 85 (3), 472–480.
Weiss, N.H., Sullivan, T.P., Tull, M.T., 2015. Explicating the role of emotion dysregulation in risky behaviors: a review and synthesis of the literature with directions for future research and clinical practice. Curr. Opin. Psychol. 3, 22–29.
Westenberg, P.M., Drewes, M.J., Goedhart, A.W., Siebelink, B.M., Treffers, P.D.A., 2004. A developmental analysis of self-reported fears in late childhood through mid-adolescence: social-evaluative fears on the rise? J. Child Psychol. Psychiatry Allied Disciplines 45 (3), 481–495.
Wilcox, R., 2017. Introduction to Robust Estimation and Hypothesis Testing Introduction to Robust Estimation and Hypothesis Testing, 4th edition. Academic Press, San Diego, CA.
Zelazo, P.D., Qu, L., Kesek, A.C., 2010. Hot executive function: emotion and the development of cognitive control. Child Development at the Intersection of Emotion and Cognition. American Psychological Association.