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Neural mechanisms underlying the link between effortful control and aggression: An ERP study

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ABSTRACT

Aggression and violence are social behaviors that exact a significant toll on human societies. Individuals with aggressive tendencies display deficits in effortful control, particularly in affectively charged situations. However, not all individuals with poor effortful control are aggressive. This study uses event-related potentials (ERPs) recorded from a large sample (n = 75 undergraduates) to decompose the chronology of neural mechanisms underlying the ability to effortfully-control behavior, and then explores whether deficits in these cognitive functions might then lead to aggressive behavior. This study investigated which ERPs moderate the effortful control - aggression association. We examined three successive ERP components, the P2, N2, and P3, which have been associated with attentional orienting, response conflict, and working memory updating, for stimuli that required effortful control. N2 amplitudes were larger for trials requiring a switch from a preplanned action strategy than trials where a preplanned action strategy was followed. Furthermore, results indicated that N2 activation, but not P2 or P3 activation, moderated the relationship between effortful control and aggression. Our results suggest that small (less negative) N2s moderate the association between effortful control and aggression. These effects were present only in negative contexts, and only for high-conflict trials. Results suggest that individual differences in neural processing efficiency contributes to the execution of effortfully controlled behavior and avoidance of aggression.

1. Background

Aggression and violence are complex social behaviors that exact a significant toll on human societies (Mehta and Beer, 2010). Crime and violence on college campuses, in particular, has been the subject of much study (Coker et al., 2011; Fisher et al., 1998). This issue has been recognized on a national level with the 1990 introduction of the Jeanne Clery Disclosure of Campus Security Police and Campus Crime Statistics Act, known as the Clery Act, which requires colleges participating in Title IV financial aid programs to report statistics on campus crime rates. Aggressive and violent behaviors have been associated with increased impulsivity and limited self-regulatory skills (Nelson and Trainor, 2007). Deficits in effortful control may lead to difficulty regulating behavior, resulting in harmful interpersonal behaviors (Lewis et al., 2007). However, not all individuals who have poor effortful control are aggressive. For this reason, it is important to examine the factors that influence the link between effortful control and aggressive outcomes. In particular, it is important to investigate aggression in a sample of developing young adults on college campuses, who might be at risk for developing these behavioral problems.

Rothbart's theory of temperament defines effortful control as a temperamental characteristic that allows the selection of a subdominant response rather than a competing dominant response (Rothbart and Rueda, 2005). Since much of human behavior consists of habitual or instinctive actions (Isoda and Hikosaka, 2008; i.e., a dominant response), effortful control is necessary in those situations when a dominant mode of behavior must be overridden, and different action initiated (i.e., subdominant response; Goldstein et al., 2007). While effortful control is often emphasized as an important construct in child development, Evans and Rothbart (2007) extended this theory by developing a hierarchical temperamental model using a large sample of college undergraduates. This model showed the importance of effortful control in a sample of young adults similar to ours, therefore paving the way for an investigation of temperament and brain processes contributing to aggression on college campuses.

The ability to flexibly change action strategies from dominant to subdominant (effectively apply effortful control) likely requires multiple underlying cognitive functions (e.g., Badre and Wagner, 2006;

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Braver et al., 2009; Eslinger and Grattan, 1993). Given that deficits in any of these cognitive functions could contribute to aggressive behavior, understanding the cognitive processes underlying effective action change to a subdominant response is important to set the stage for targeted treatment. Some cognitive processes thought to contribute to our ability to effectively change action strategies are attentional orienting (e.g., Weissman et al., 2002), resolving response conflict (e.g., Nieuwenhuis et al., 2003), and working memory encoding (e.g., Friedman et al., 2001).

A person needs to orient their attention towards new environmental information (i.e., information signaling the subdominant response) in order to effectively process this information. A potential neural mechanism underlying successful attentional orienting is reflected in P2 activation (Britz and Pitts, 2011; Mulert et al., 2002; Vitacco et al., 2002), generally observed 130–290 ms post-stimulus (Kanske et al., 2011). The P2 is recruited with tasks that require attentional resources (Luck and Hillyard, 1994; Phillips and Takeda, 2009) and is modulated by emotional context (e.g., Eldar and Bar-Haim, 2010). For example, Kanske et al. (2011) demonstrated that increased attentional preference to emotional cues increased P2 amplitude.

Next, a person needs to process the response conflict between execution of the subdominant response vs. dominant response. N2 activation usually peaks 200–400 ms post-stimulus (Luck, 2014) and is a potential neural mechanism supporting successful processing of response conflict (Bekker et al., 2005; Donkers and Van Boxtel, 2004; Jonkman, 2006; Ladouceur et al., 2007). Nieuwenhuis et al. (2003) demonstrated the link between N2 amplitudes and response conflict by altering the ratio of go and nogo trials in a go/nogo task. Furthermore, the N2 is affected by emotional valence (e.g., Shackman et al., 2007; van Wouwe et al., 2011). For example, Lewis et al. (2006a, b) showed that N2 amplitudes in adolescents were consistently higher following negative emotion induction compared to a control condition. This suggests that successful processing of response conflict requires greater conflict monitoring resources when faced with negative emotion.

Lastly, information about the executed subdominant response needs to be encoded to inform behavior on subsequent trials (context updating in working memory). P3 activation, generally peaking 250–600 ms post-stimulus, is a potential neural mechanism supporting the encoding of information in working memory (Saliasi et al., 2013; Vogel and Luck, 2002). The P3 is classically recruited in the oddball task and is larger when subjects must respond to infrequent task-related stimuli than for common stimuli (Squires et al., 1975). The relation of the P3 to memory consolidation was demonstrated by Vogel and Luck (2002), who showed that participants had a P2 peak but no P3 peak to blinked (missed) stimuli in an attentional blink paradigm, suggesting that the stimulus was attended but was not encoded into working memory. The P3 seems to inform future task-related behavior via working memory encoding (Donchin, 1981; Donchin and Coles, 1988; Vogel and Luck, 2002), with P3 amplitudes being greater in situations when subjects must react to an unexpected cue, such as during action switching. P3 amplitude is also sensitive to emotional context of a stimulus (e.g., Cavanagh and Geisler, 2006; Herbert et al., 2006). For example, Lewis et al. (2006a, b) showed that P3 amplitude was higher following negative emotional stimuli compared to unemotional stimuli.

To examine the time course of neural activation related to effective action change, we examined these three successive ERP components (P2, N2, & P3) in a task that requires overriding a dominant response to allow for a subdominant response (i.e., effective effortful control). Furthermore, we examined how neural activation, i.e., P2, N2, and P3 activation, underlying our ability to effectively apply effortful control differs depending on emotional context.

ERPs have been used as dependent variables in many traditional analyses and there is evidence from some paradigms and populations that ERPs have high test-retest reliability (Fabiani et al., 1987; Polich, 1986; Riesel et al., 2013; Segalowitz and Barnes, 1993) and therefore when measured in these contexts have utility as individual difference markers (Hegerl et al., 2001; Ladouceur et al., 2007; Lamm et al., 2014; Linka et al., 2005; McDermott et al., 2009; Meyer et al., 2013; Troller-Renfree et al., In press). In this study, we examined whether P2, N2, or P3 amplitudes moderated the association between an individual's effortful control capabilities and their aggressive tendencies in different emotional contexts. P2, N2, and P3 amplitudes were used as markers of individual differences in the neural mechanisms underlying attentional orienting, processing response conflict, and working memory updating respectively.

We hypothesized that 1) participants would show greater neural activation underlying action change in the face of negatively valenced images compared to positive or neutral images, suggesting that successful action change may require greater cognitive resources when faced with negative emotion. Secondly, we hypothesize that ERP activation underlying effortful control in negative emotional conditions would thus reveal a stronger moderating impact on aggressive behavior. As we were particularly interested in exploring the neural timing of these moderating effects, we do not introduce specific hypotheses about which ERP component might moderate the relationship, but instead investigate the P2-N2-P3 progression in order to examine timing of moderation effects.

2. Method

2.1. Participants

The sample was recruited from undergraduate students taking psychology classes at the University of New Orleans. Participants were 81 undergraduate students (35 male, 40 female; six participants did not provide gender and were excluded from all analyses). The mean age was 21.8 (SD = 4.9, range 18–43). Criteria for exclusion from the study were current psychiatric diagnoses, current use of psychoactive medication, and uncorrected visual impairments. All students were given extra credit to compensate for their participation. All students were English speaking. Ethical approval for the project was obtained from the University of New Orleans' Institutional Review Board.

2.2. Procedure

Participants were briefly introduced to the testing environment, after which informed consent was obtained. Participants were then seated in the testing room to complete questionnaires. After completion of the questionnaire battery, participants were seated 67 cm in front of a computer monitor. The electrode sensor net was applied. They were given a practice block of 16 trials, with the option to repeat the practice block, in order to ensure proficiency with the task.

2.3. Measures and task

2.3.1. The adult temperament questionnaire short form

The Adult Temperament Questionnaire Short Form (ATQ-SF; Evans and Rothbart, 2007) is a 77-item reliable and valid self-report measure of emotional temperament and self-regulatory capacity. Importantly, the Adult Temperament Questionnaire was initially developed for use with a sample of college undergraduates and was piloted with several samples of college undergraduates (Rothbart & Evans, 2007) – therefore, this questionnaire is theoretically suited to temperament research with our sample of undergraduates. The measure consists of 13 subscales, three of which comprise the effortful control scale: attentional control, inhibitory control, and activation control. The effortful control scale was used as a measure of a young adult's ability to effortfully regulate their actions. The internal consistency for the effortful control scale was good ($\alpha = 0.78$).

2.3.2. The Buss Perry aggression scale

The Buss Perry Aggression Scale (BPAS; Buss and Perry, 1992) is a

29-item standardized, valid, and reliable self-report measure of aggression in adults. The overall score (average of all items) was used to measure aggression in this sample. The internal consistency for this measure was good (α = 0.91). Our means and standard deviations were very similar to those normed by Buss and Perry, 1992 using a large undergraduate sample.

2.3.3. Action change task

The task was a modified AX continuous performance task (CPT; Rosvold et al., 1956). While various versions of the AX-CPT have been used in the literature, one common feature to all versions is that participants respond in a particular manner for one trial type and change response strategy for a different trial type. Thus, a behavioral propensity is established such that the more common response strategy is relatively automatic, while the less common response strategy requires effortful control due to action change requirements. Therefore, this task is meant to measure the neural activation underlying action change (switching from a dominant response to a subdominant response). For more in-depth explanation of the cognitive processing involved in the AX-CPT, the interested reader is directed to numerous articles discussing the significance of the AX-CPT (Braver, 2012; Braver and Barch, 2002; Carter et al., 1998; Hasson and Fine, 2012; Losier et al., 1996; Riccio et al., 2002, 2001).

Images were presented on a 17-in monitor using E-prime Software (Psychology Software Tools, Inc., Pittsburgh, PA; Schneider et al., 2002). Stimuli were shown on a black screen in a dimly lit room. Furthermore, this task included emotionally-charged images before response action change was required allowing us to investigate the influence of emotion on action change.

Trials were between 2.7 and 4.5 s in duration and consisted of three main events (each separated by a fixation; see Fig. 1). First, a cue was presented for 100–1000 ms; this was always the letter "A" and required the same response every trial (pressing the "2" key on a response pad). After the cue, a picture was presented, consisting of negative, positive, and neutral photos from the International Affective Picture System (IAPS; Lang et al., 2008; contact corresponding author for a list of included IAPS pictures). Pictures were categorized using the Lang et al. (2008) valence and arousal ratings ("EroticCouple" images were excluded). Pictures were 11 cm wide by 8 cm tall and presented in black and white (visual angle was 9.39 degrees). Pictures were presented during the delay (between cue and probe stimuli) in pseudo-random order (all participants received the same random order). After offset of pictures, a probe was presented for 100–1000 ms, which required either execution of a prepotent response (the letter "X", which appeared on 70% of trials and required participants to press the "3" key) or withholding of a dominant response and execution of a subdominant response (the letter "Y", appearing only 30% of trials and requiring participants to press the "2" key). Participants were allowed to hold the button box in any position that was most comfortable for them. Cue and probe letters were presented in 60-point size uppercase bold Courier New font. Cues were presented in blue font and probes in white. Because participants were instructed of the color difference between cue and probe stimuli at the beginning of the task, this prevented them from losing track of which stimulus they were viewing.

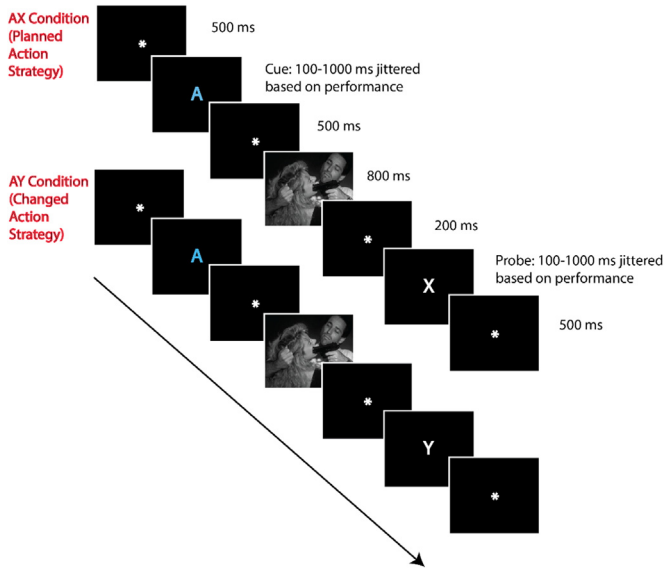

Fig. 1. Task diagram of the modified AX-CPT task.

Both cue and probe trial times were adjusted dynamically based on participant accuracy rates (within each trial, cue and probe trial times were always identical; a trial was only deemed accurate if both the cue and probe responses were correct). The procedure for this dynamic adjustment was as follows: if the participant's accuracy rate rose above 75% then the duration of the cue and probe stimuli were shortened by 20 ms every trial (to a minimum of 100 ms). If the participant's accuracy fell below 65% then the duration of the cue and probe were lengthened by 20 ms every trial (to a maximum of 1000 ms). While the task did not include jittered fixation times between stimuli, i.e., cue and probe, the dynamic adjustment had the same effect. More specifically, cue and probe stimuli presentation time length was adjusted based on their cumulative performance accuracy and therefore varied from trial-to-trial. The existence of this timing jitter ensures that the length of time between stimuli varies ensuring that the phase of oscillation upon which a subsequent stimulus falls various from trial-to-trial, a requirement for accurate measurement of the event-related potential.

The task was broken down into three blocks of 100 trials (300 hundred trials total; 100 AY trials) with opportunities to rest in between each of the blocks. The task took roughly 40 min to complete. Participants completed two practice blocks of 8 trials each in which no pictures were displayed but task performance feedback was provided to ensure task proficiency. Feedback was presented for erroneous cue/probe response patterns or late responding and consisted of a red line, presented for 200 ms. Performance feedback was only provided during the practice block and not during the actual test blocks.

2.4. EEG data collection and analyses

2.4.1. EEG data collection and processing

EEG data collection and processing procedures were consistent with Lamm et al. (2013). EEG was recorded using a 128-channel Geodesic Sensor Net and sampled at 250 Hz, using EGI software (Net Station; Electrical Geodesic, Inc., Eugene, OR [data were also processed using Net Station]). Once the impedance values for all EEG channels were reduced to below 50 kΩ, data acquisition began. During recording, all channels were referenced to Cz and after acquisition, data were re-referenced using an average reference.

Data were filtered using a FIR bandpass filter with a low-pass frequency of 50 Hz and a high-pass frequency of 0.3 Hz. To best capture eye blink artifacts, the threshold was set to 140 μV (peak-to-peak) and all trials in which this threshold was violated were excluded from analyses. Furthermore, signal activation change (peak-to-peak) exceeding 150 μV across the entire segment and fast transits exceeding a difference (peak-to-peak) of 140 μV were marked as bad and interpolated. Trials with more than 10 bad channels were excluded from analyses.

2.4.2. Scalp data analyses

Waveforms for correct AX and AY trials were segmented into epochs from 150 ms before to 600 ms after probe (X or Y) onset and baseline corrected for the 150 ms preceding probe onset. A data-driven approach was taken to identify the time periods for ERP measurement: a grand-average waveform was calculated for every electrode (all conditions

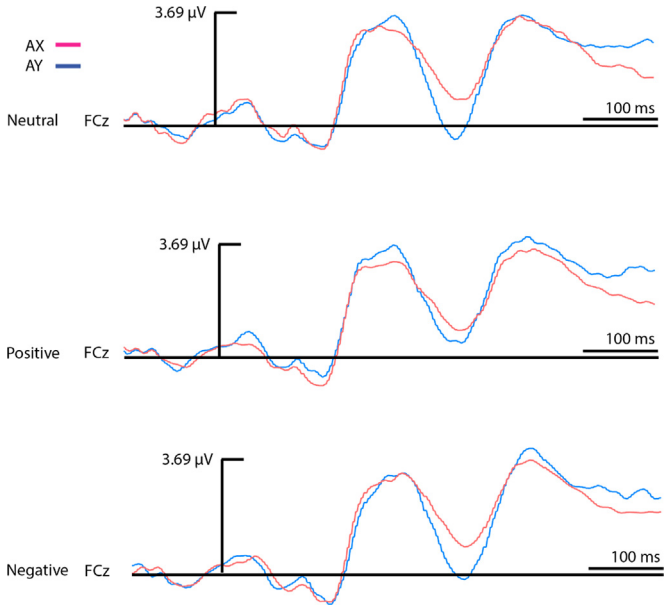

Fig. 2. Visualization of AX (preplanned action) vs. AY (action switch) ERP waveforms in each emotional condition. All waveforms are computed at FCz.

were averaged – therefore this procedure does not bias our condition-specific analyses). The time periods for the P2 and N2 were identified based on inspection of electrodes within the mediofrontal cluster (described below); the time period for the P3 was based on inspection of the grand-average waveforms at electrodes in the parietal cluster (described below). Mediofrontal P2 activation was maximal between 200 and 240 ms after stimulus onset, mediofrontal N2 activation was maximal between 300 and 330 ms after stimulus onset; and parietal P3 activation was maximal between 350 and 420 ms after stimulus onset. Mean activations were therefore extracted during these time windows. The mean number of trials comprising correct AX ERPs was 42.9 (SD = 9.6; min = 16; max = 66), and the mean number of trials comprising correct AY ERPs was 18.4 (SD = 4.5; min = 10; max = 32).

Visualization of the correct AX and AY stimulus-locked waveforms revealed clear P2 and N2 components for mediofrontal electrodes and clear P3 components for parietal electrodes (See Fig. 2 – grand averaged waves). Scalp N2 and P2 activation was exported for the following mediofrontal electrodes: four midline electrodes (VREF [Cz], 6 [FCz], 11 [Fz], and 16) as well as ten flanking electrodes (10, 18, 19, 4, 5, 12, 106, 7, 112, and 13). Scalp P3 data was exported for the following parietal electrodes: four midline electrodes (VREF [Cz], 55 [CPz], 62 [Pz], 72) as well as eight flanking electrodes (80, 79, 78, 77, 31, 54, 61, and 67). Because of individual differences in activation across electrodes, each participant's greatest (most negative or most positive, depending on ERP component) activation within these electrode clusters during AX or AY trials was analyzed.

2.4.3. Statistical analyses

Regression analyses were conducted in this study to examine the moderating role of brain activation on the relation between effortful control and aggression. Prior to moderation analysis, all independent and moderator variables were converted to z-scores to decrease the possibility of multicollinearity and scaling differences influencing results (Aiken et al., 1991; Dawson, 2014). There are frequently reported gender differences in aggressive behavior (Hyde, 1984); since gender influences are not a primary purpose of this study, we ran preliminary tests to determine whether to control for gender in the following analyses. A priori *t*-tests revealed gender differences for some independent and dependent variables (AX-Negative P2 – t(63) = 2.46, p = .02; AX-Neutral P2 – t(63) = 2.45, p = .02; AY-Negative P2 – t(63) = 2.09, p = .04; AY-Neutral P2 – t(63) = 2.19, p = .03; AX-Negative P3 – t(63) = 3.42, p = .001; AX-Neutral P3 – t(63) = 2.81, p = .007; AX-Positive P3 – t(63) = 2.66, p = .01; AY-Negative P3 – t(63) = 2.69, p = .01; AY-Neutral P3 – t(63) = 3.22, p = .002; AY-Positive P3 – t(63) = 2.57, p = .01; Aggression – t(63) = 3.41, p = .001); therefore, gender was entered as a factor in all EEG analyses (regression and ANOVA). The Greenhouse-Geisser correction was applied to all repeated-measures tests with more than one degree of freedom. All ANOVA contrasts were tested using a Bonferroni correction for multiple comparisons. Because the focus of the current study is to examine the neural correlates underlying the change in action strategy from a dominant to a subdominant response, the variable of interest for all analyses (regression and ANOVA) is AY trials. To capture only brain activation that is related to action switching, ERP amplitudes from AX trials are entered as a control in all regression analyses.

3. Results

3.1. Behavioral results

To determine if there were any behavioral effects of action change requirements or emotional context, two 2 (AX or AY) X 3 (neutral, negative, positive) repeated-measures ANOVAs were conducted: 1) with performance accuracy as the dependent measure and 2) with reaction times as the dependent measure. A main effect of action change requirements was detected, $F(1,68) = 183.83$, $p < .001$, $\eta^2 = .73$, $\varepsilon = 1$, such that reaction times were faster in trials that required executing a prepotent action (AX) than trials that required changing action strategy (AY). A similar effect was present in the performance accuracy data, $F(1,68) = 126.01$, $p < .001$, $\eta^2 = .65$, $\varepsilon = 1$, such that participants were more accurate in trials that required executing a prepotent action (AX; 79% accurate) than trials that required changing action strategy (AY; 67% accurate). Reaction times and accuracies for probe stimuli are presented in Table 1. There was no main effect of emotion or emotion-by-action change requirement interaction on RTs or accuracy, suggesting that emotional context did not influence performance accuracy or speed of responding during action change. These results also suggest that any potential brain differences in emotional context are not likely to be due to task performance.

3.2. ERP condition effects

To determine if there were any ERP differences due to action change requirements or emotional context, three 2 (AX or AY) X 3 (neutral, negative, positive) repeated-measures ANOVAs were conducted on correct trials: 1) P2 activation as dependent variable, 2) N2 activation as dependent variable, and 3) P3 activation as dependent variable. Since there were gender differences in the ERP data, all analyses were run with the influence of gender controlled.

Results indicated a main effect of action change requirement (AX or AY) on P2 amplitudes, $F(1,63) = 47.78$, $p < .001$, $\eta^2 = .43$, $\varepsilon = 1$, showing that AY (action change) trials results in larger P2 amplitudes than AX (no action change) trials ($p < .001$; see Fig. 3). There was no main effect of emotion on P2 amplitudes, $F(2126) = 2.29$, $p = .11$, η^2

Table 1
Behavioral reporting, summary for unemotional, negative, and positive contexts.

Emotion	Condition	RT Mean	RT SD	ACC Mean	ACC SD
Neutral	AX	395.75	70.63	0.80	0.08
	AY	451.98	78.49	0.66	0.11
Negative	AX	397.79	70.86	0.78	0.10
	AY	456.79	83.86	0.67	0.12
Positive	AX	392.68	70.75	0.80	0.08
	AY	444.01	75.18	0.68	0.12

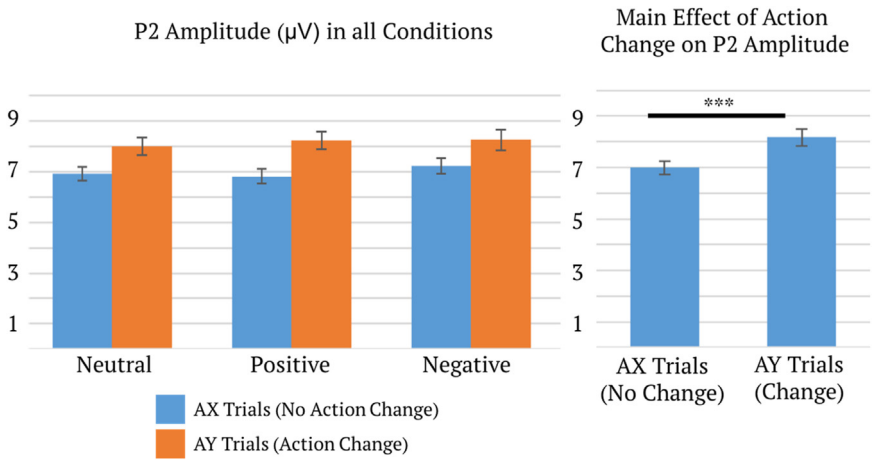

Fig. 3. Bar graph of P2 amplitude (measured at maximum of mediofrontal cluster for each participant).

= .04, ε = 0.95, nor was the interaction of action change by emotion significant, F(2126) = 0.63, p = .54, η^2 = .01, ε = 0.99.

Furthermore, there was a main effect of action change requirement on N2 amplitudes, F(1, 63) = 30.08, p < .001, η^2 = .32, ε = 1, with AY (action change) trials showing more negative N2 amplitude than AX (no action change) trials (p < .001). There was also a main effect of emotion on N2 amplitudes, F(2126) = 5.23, p = .008, η^2 = .08, ε = 0.96, with neutral trials resulting in more negative N2 amplitudes than negative trials (p = .01; see Fig. 4). The interaction of action change by emotion was not significant, F(2126) = 3.63, p = .21, η^2 = .03, ε = 0.89.

There was a main effect of action change requirement on P3 amplitudes, F(1,63) = 13.94, p < .001, η^2 = .18, ε = 1, as well as a main effect of emotion, F(2126) = 33.27, p < .001, η^2 = .35, which were both subsumed by a significant interaction of action change requirement-by- emotion, F(2126) = 4.05, p = .02, η^2 = .06. This interaction indicated that P3 amplitudes were larger for AY (action change) stimuli than AX (no action change) stimuli in neutral emotional conditions (p < .001) and positive emotional conditions (p = .001), but not in negative emotional conditions (see Fig. 5).

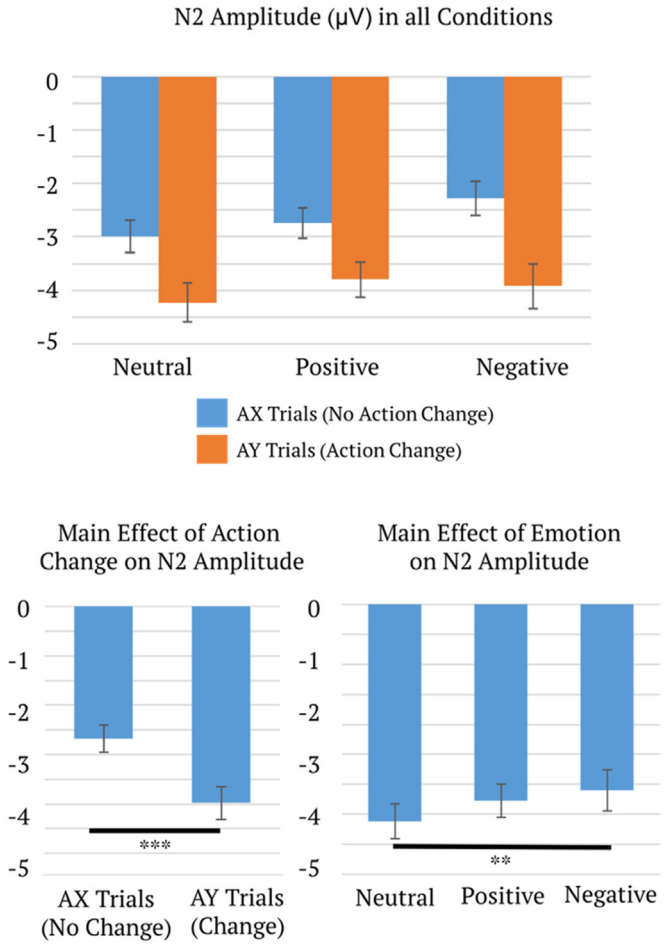

Fig. 4. Bar graph of N2 amplitude (measured at minimum of mediofrontal cluster for each participant).

3.3. ERP moderator effects

Because not everyone with poor effortful control has aggressive tendencies, we also conducted a linear regression analysis to test which ERP component (P2, N2, or P3) moderated the association between effortful control and aggression. We repeated this regression analysis for every emotional condition (neutral, negative, and positive). As action change trials (change from dominant to subdominant response) were the trial type of interest, all regression analyses used ERP activation during AY trials only. In step one of the regression analyses, we entered all variables that were controlled for (outlined earlier). In step two, we entered all three ERP components (P2 amplitude, N2 amplitude, P3 amplitude), and effortful control. Lastly, three interaction terms of ERP amplitude by effortful control were computed and entered in step 3 to test for moderation effects. Aggression was entered as the

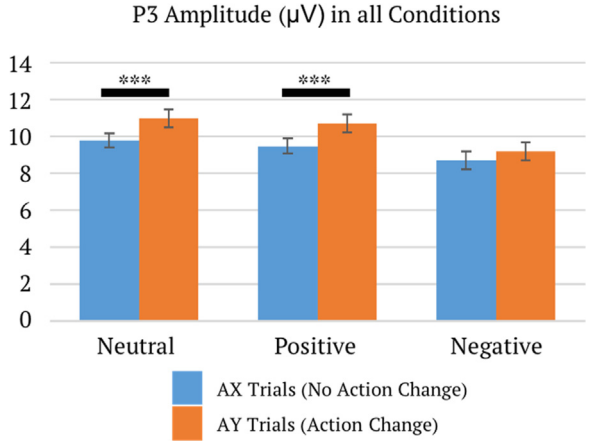

Fig. 5. Bar graph of P3 amplitude (measured at maximum of parietal cluster for each participant).

Table 2
Regression Model Summary for Unemotional, Negative, and Positive Contexts.

	Model step	F change	R^2 change	Sig. F change
Neutral	Step 1	3.41	0.19	0.01*
	Step 2	2.79	0.14	0.04*
	Step 3	1.44	0.05	0.24
Negative	Step 1	3.68	0.20	0.01*
	Step 2	3.95	0.18	0.007**
	Step 3	2.05	0.07	0.12
Positive	Step 1	3.15	0.17	0.02*
	Step 2	4.07	0.19	0.006**
	Step 3	0.48	0.02	0.70

Step 1: Control Variables. Step 2: ERP amplitudes and Effortful Control. Step 3: Interactions.
*** p < .001.
* p < .05.
** p < .01.

Table 3
Regression Model Predicting Aggression for Unemotional, Negative, and Positive Contexts.

	IV	Unstandardized β	Standardized β	t(53)	*P*
Neutral	Sex	− 0.60	− 0.33	− 2.59	0.01*
	EC	− 0.18	− 0.19	− 1.33	0.19
	P2 Amplitude	− 0.19	− 0.17	− 0.47	0.64
	N2 Amplitude	0.06	0.06	0.34	0.74
	P3 Amplitude	0.36	0.36	1.08	0.29
	P2 * EC	0.44	0.37	1.63	0.11
	N2 * EC	− 0.22	− 0.25	− 1.80	0.08
	P3 * EC	− 0.28	− 0.25	− 1.14	0.26
Negative	Sex	− 0.49	− 0.27	− 2.23	0.03^
	EC	− 0.28	− 0.30	− 2.37	0.02^
	P2 Amplitude	− 0.13	− 0.14	− 0.41	0.69
	N2 Amplitude	− 0.19	− 0.20	− 1.12	0.27
	P3 Amplitude	− 0.21	− 0.23	− 0.84	0.41
	P2 * EC	0.25	0.32	1.22	0.23
	N2 * EC	− 0.35	− 0.35	− 2.46	0.017*
	P3 * EC	− 0.13	− 0.15	− 0.61	0.54
Positive	Sex	− 0.58	− 0.31	− 2.48	0.02^
	EC	− 0.27	− 0.30	− 2.38	0.02^
	P2 Amplitude	− 0.43	− 0.46	− 1.19	0.24
	N2 Amplitude	− 0.02	− 0.02	− 0.11	0.92
	P3 Amplitude	0.10	0.10	0.30	0.77
	P2 * EC	0.30	0.30	1.12	0.27
	N2 * EC	− 0.12	− 0.14	− 0.89	0.38
	P3 * EC	− 0.20	− 0.19	− 0.79	0.43

EC = Effortful Control.
* p < = .017.
^ p < .03 (trend).

dependent variable. Regression model change summaries are presented in Table 2, while regression results are presented in Table 3. Since this analysis included three regression analyses, p-value cutoff for significance was adjusted via Bonferroni correction to control familywise error rates (0.05 / 3 = 0.017). Since a moderator which is highly correlated with the independent variable can be problematic in regression analysis, a priori Pearson correlations were computed between effortful control and ERP component (P2, N2, P3) amplitude. Values indicated no correlation with effortful control for any ERP component in any condition (all p > .17).

Results revealed that N2 amplitudes in negative conditions significantly moderated the association between effortful control and aggression. When probed at values of 1 SD above and below the mean, additional regression analyses revealed that this moderating effect was driven by low (less negative) N2 activation. At low levels of N2 activation (less negative) effortful control was a significant predictor of aggression scores in negative conditions, β = -0.68, t(53) = -4.28, p < .001. At high levels of N2 activation (more negative), effortful control was not a significant predictor of aggression scores in negative conditions, β = 0.07, t(53) = 0.33, p = .74 (see Fig. 3). N2 amplitude in neutral and positive emotional conditions did not significantly moderate the association between effortful control and aggression; P2 and P3 amplitudes did not moderate the effortful control – aggression association in any condition (Fig. 6)

4. Discussion

4.1. General discussion

In the context of a task that requires effortful control, i.e., changing from a dominant response (AX trials) to a subdominant response (AY trials), the present study examined the time course of neural activation underlying action change processes, and how these patterns of activation contribute to aggressive behavior. More specifically, we used ERPs to examine whether patterns of neural activation moderate the relationship between effortful control and aggression, and whether these moderating effects differed in emotionally salient contexts (both positive and negative) compared to relatively neutral contexts. As predicted, brain processes underlying action change significantly moderated the association between effortful control and aggression.

Given that the ability to flexibly change action strategies from a dominant response to a subdominant response likely requires a number of underlying cognitive functions (e.g., Badre and Wagner, 2006; Braver et al., 2009; Eslinger and Grattan, 1993) and that any of these functions could show deficits that might contribute to aggressive behavior, we decomposed the time course underlying action change. Our results indicate that activation during the N2 window significantly moderated

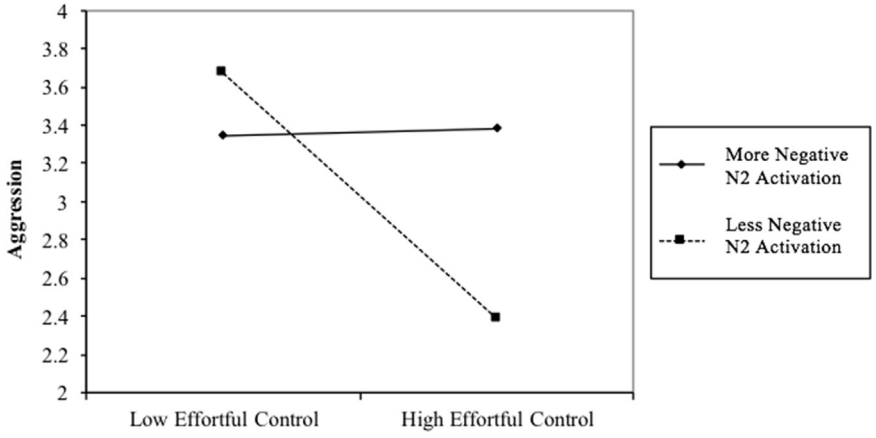

Fig. 6. N2 activation following the need for action switching moderates the association between Effortful Control and Aggression.

the effortful control – aggression relationship. Given that the N2 has been associated with aspects of cognitive control (e.g., Jonkman, 2006; Lamm et al., 2006), such as response conflict (Nieuwenhuis et al., 2003), as well as aggression (Lamm et al., 2011; Lewis et al., 2006a, b), this suggests that activation during the N2 time window might be a neural mechanism that influences resolution of response conflict over aggressive tendencies. For example, N2 activation might represent the amount of neural activation required to process the conflict between striking someone (dominant response) or walking away (subdominant response) and contribute to a person's overall effortful control, i.e., choosing a subdominant response over a dominant response. In line with this theory, our study showed the preliminary expected influence of action change requirement on N2 amplitude, where, consistent with prior research (e.g., van Wouwe et al., 2011), we showed more negative N2 amplitude for stimuli requiring a change from a preplanned action strategy.

The direction of the moderating effect for N2 activation was consistent with previous studies linking neural processing efficiency with reduced activation during aspects of cognitive control tasks (e.g. Casey et al., 1997; Durston et al., 2006; Lamm et al., 2013). For example, Lamm et al. (2013) found that participants who successfully deployed a reactive (to the environment) style of responding showed less prefrontal activation when required to execute last-minute environmentally-triggered action change. This decrease in prefrontal activation was interpreted to reflect increased efficiency of cognitive-control-related cortical processing due to their ongoing response style (pattern of behavior) being reactive in nature. However, individual differences in efficiency of processing should be interpreted within the specific behavior being performed. Thus, as can be seen from Fig. 4, easier trials, i.e., AX trials, show less negative N2 activation than more difficult trials, i.e., AY trials. In other words, a person who shows efficient processing on AY trials (little N2 activation) is likely to show even less negative N2 activation on AX trials. Thus, our results suggest that efficient processing of response conflict moderates the association between effortful control and aggression, i.e., high effortful control and more efficient processing was related to lower levels of aggression. More specifically, these findings, in combination with the related extant literature, suggest a nascent theory of efficiency that may inform our understanding of the neural underpinnings of cognitive control in adulthood.

Interestingly, moderation effects existed only at low levels of N2 activation (less negative), which suggests that low or efficient neural activation during action change, in conjunction with high effortful control, contributes to less aggressive outcomes. We expected to find the converse as well, that is, high neural activation amplitude and poor effortful control should result in higher aggression. Instead, the relationship between effortful control and aggression is flat at high levels of N2 activation. This suggests that our data might have a restriction of range; that is, our participants may not have shown enough variability in aggression. This argument is supported by the fact that we recruited participants from a university environment. Recruitment from a more diverse sample might reveal a greater range of aggression scores and thus potentially reveal both the *high effortful control – efficient processing – low levels of aggression effect* and the *poor effortful control – inefficient processing – high levels of aggression effect*. Future research should replicate this study on a more diverse sample.

Previous literature has found emotion-specific changes in ERP activation for negative or positive emotional contexts compared to emotionally neutral contexts (e.g., Lamm and Lewis, 2010; Lamm et al., 2013; Lewis et al., 2006a, b; van Wouwe et al., 2011). However, these studies did not compare negative and positive trials; thus, it is unclear if these effects were due to valence or arousal. The current results add to the extant literature by examining this issue within a single task, allowing direct comparison between positive, neutral, and negative contexts. For the N2, previous research has found more negative N2 activation for negative conditions than relatively neutral conditions (e.g., Lamm et al., 2013; Lewis et al., 2006a, b). Interestingly, we did not find this pattern of effects. Additionally, it is not clear why emotion differences were not found for P2 amplitudes. However, one explanation may be the structure of the task. For both the Lamm et al. (2013) and the Lewis et al. (2006a, b) studies, negative trials were grouped together into blocks while in the current study, we presented negative, neutral and positive trials randomly (not in blocks). Thus, the fact that emotional trials were presented randomly within each block might have "watered down" the impact of the negative trials. Future research should compare design differences, i.e., emotional random design vs. emotional block design, to ascertain if this is indeed the case.

Furthermore, results from the current study showed that N2 activation moderates the effortful control – aggression relationship in the negative condition but not in neutral or positive conditions. These results suggest that in the face of negatively-charged (specifically, violent or threatening) events, individuals with efficient response conflict processing are less likely to respond in an aggressive manner.

4.2. Limitations

There are limitations to the current study. First, the current study used questionnaire-based proxies to measure aggression. Questionnaire-based measures may be more subjective than biological or behavioral measures, generally for reasons relating to social desirability (Armitage and Conner, 1999; Richman et al., 1999; Sjöström and Holst, 2002). Therefore, these results should be replicated using a behavioral measure of aggression.

Second, the use of ERPs as a moderator is a relatively novel approach. Even though there is considerable evidence for the reliability of ERPs from many paradigms and the fact that a number of previous studies have used ERPs as moderators (Lamm et al., 2014; McDermott et al., 2009; and Troller-Renfree, et al., In press), because necessary psychometrics of ERPs elicited by the AX-CPT have not been examined, results from this study should be considered somewhat exploratory.

Finally, the current study had a small age range of participants. Previous neuroimaging research has shown that neural activation during cognitive control tasks differs between adolescents and adults (Eshel et al., 2007; Luna et al., 2010; Rubia et al., 2006), and therefore the young adults included in this study might not be fully representative of the adult age range. This limits generalizability of results to other age ranges. However, it is important to note that the Adult Temperament Questionnaire was initially developed and piloted with a sample of college undergraduates (Rothbart & Evans, 2007). Future work should expand on the age range of participants, to determine if moderating effects differ throughout development, including later adulthood.

4.3. Conclusions

These results suggest that neural mechanisms underlying flexible action change moderate the association between effortful control and aggression. Specifically, these results suggest that low or efficient prefrontal cortical activation contributing to effortful control can lead to less aggressive outcomes. Future studies should build upon these results by examining whether the converse is also true; that is, does high or inefficient activation during the N2 time window contribute to more aggressive outcomes? These studies should prescreen individuals to ensure that some participants are high in aggressive behavior so that there is enough variability in aggression scores for these effects to be discernable. Additionally, future research should incorporate longitudinal developmental data to ascertain whether inefficient use of regulatory resources early in life predicts future aggressive behavior problems, thereby highlighting a neural mechanism (or biomarker) that might be targeted by treatment approaches.

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