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Neural Mechanisms of Action Switching Moderate the Relationship Between Effortful Control and Aggression

Eric L. Rawls
University of New Orleans, erawls@uno.edu

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Neural Mechanisms of Action Switching Moderate the Relationship Between Effortful Control
and Aggression

A Thesis

Submitted to the Graduate Faculty of the
University of New Orleans
in partial fulfillment of the
requirements for the degree of

Master of Science
in
Psychology
Applied Biopsychology

by

Eric L. Rawls

B.S. Tulane University, 2012

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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) to decompose the chronology of cognitive functions underlying the link between effortful control and aggression. Specifically, this study investigates which ERPs moderate the effortful control - aggression association. We examined three successive ERP components (P2, N2 and P3) for stimuli that required effortful control. Results indicated that N2 activation, but not P2 or P3 activation, moderated the relationship between effortful control and aggression. These effects were present in negative and neutral contexts. This moderating effect was consistent with previous studies linking neural processing efficiency with reduced activation during cognitive control tasks. Our results suggest that efficient cognitive processing moderates the association between effortful control and aggression.

Effortful Control, Aggression, Emotion, Emotion Regulation, EEG, ERPs, LORETA

1, Introduction

Aggression and violence are complex social behaviors that exact a significant toll on human societies (Mehta & Beer, 2007). Overly aggressive behaviors have been associated with increased impulsivity and limited self-regulatory skills (Nelson & Trainor, 2007). In the context of individuals who score high on measures of interpersonal aggression, particular deficits in effortful control can be seen. Effortful control is defined in part as the process of inhibiting a dominant response in favor of a subdominant response (Rothbart & Rueda, 2005). Since much of human behavior consists of habitual or instinctive actions (Hikosaka & Isoda, 2008), effortful control is necessary specifically in those situations when a “default mode” of behavior must be overridden and different action initiated (Goldstein et al., 2007). Given the diversity and complexity of computing different action plans, it is not surprising that many areas in the brain are involved in effortful control (Rothbart et al., 2007; Bush et al., 2000). In addition, there is considerable variability in the control strategies displayed by different individuals, and many psychiatric disorders, such as substance abuse and ADHD, are thought to result partly from effortful control impairments (Wiers et al., 2013; Vaidya et al., 2014; Siegle et al., 2007). People with aggressive tendencies may have difficulty regulating their behavior during negative emotional situations, resulting in harmful interpersonal behaviors (Lewis et al., 2007). However, not all individuals who have poor effortful control are aggressive. The present study uses event-related potentials (ERPs) to decompose the chronology of cognitive functions underlying effortful control, and ascertains which of these cognitive functions contributes to the association with aggressive behavior. More specifically, the current study investigates which ERPs moderate the effortful control - aggression association. Additionally, the current study uses linear inverse models to investigate which cortical regions underlying specific ERPs moderate the association

between effortful control and aggression. Furthermore, to ascertain how effortful control-related activation patterns change depending on emotional context, this study uses an experimental paradigm intended to capture participants' control abilities during negative emotional contexts, positive emotional contexts, and relatively unemotional contexts.

A key aspect of effortful control is to switch from a dominant response to a subdominant response (Rothbart & Rueda, 2005), i.e., effectively switch action strategies. The ability to flexibly switch action strategies likely requires a number of underlying cognitive functions (e.g., Badre & Wagner, 2006; Braver et al., 2009; Eslinger & Grattan, 1993). Given that any one of these functions could show deficits that might contribute to aggressive behavior, understanding the chronology underlying effective action switching is important to set the stage for targeted treatment. Some cognitive processes that might contribute to flexible action switching are attentional orienting (Weissman et al., 2002), cognitive control (Botvinick et al., 2001), and context updating for subsequent behavioral action (Friedman et al., 2001). For example, a person needs to orient their attention towards new environmental information in order to effectively process this information. Additionally, a person needs to apply cognitive control to process or monitor conflicting information, i.e., information that would have led to the previous action strategy vs. information leading to the current (changed) action strategy. Lastly, the new information needs to be encoded to prepare for the new action strategy. These three processes can be measured using ERPs. To examine the time course of neural activation related to effective action switching, we examined three successive ERP components for a stimulus that required effortful control. P2 activation has been related to attentional orienting, and is thought to have underlying neural sources in widespread areas including occipital, temporal, and frontal regions (Britz & Pitts, 2011; Mulert et al., 2002; Vitacco et al., 2002). N2 activation has been related to

various aspects of cognitive control, such as conflict monitoring, inhibition, and emotion regulation, and is thought to have underlying neural sources in prefrontal regions, including DLPFC and ACC (Ladouceur et al., 2007; Bekker et al., 2005). P3 activation has been related to motivational processing, novelty, and compilation of higher-level decision-making processes, and is thought to have underlying neural sources in posterior regions, including occipital regions (Barry & Rushby, 2006; Volpe et al., 2007). By examining the effects of this succession of ERP components on the association between effortful control and aggression, we hope to gain a greater understanding of the impact of these temporal processes on ineffective effortful control and aggressive behavior. We hypothesize that temporally distinct patterns of neural activation will moderate the association between participant scores of effortful control and aggression. In order to elicit effortful control in the context of action switching, we used a modified AX continuous-performance task (AX-CPT; Rosvold et al., 1956). This task consists of a cue, to which participants have to provide a speeded response, then a delay period, and then a probe, to which participants have to provide a second speeded response. A preponderance of one trial type (A-X) creates a habitual response. Effortful control resources, on the other hand, are recruited when action switching is necessary in order to adjust action strategies based on new contextual information; that is, when the trial type changes (A-Y).

In addition, in line with the Rothbart model of emotional reactivity impacting effortful control abilities (Rothbart & Sheese, 2007), we were interested in how this neural chronology changes in the face of salient emotions. Previous studies that required aspects of effortful control have yielded prefrontal cortical activation differences depending on the emotional context of the task. For example, Monk et al. (2003) found greater ACC activation to fearful faces than neutral faces during an attention task; Ochsner et al. (2004) found ACC, VLPFC, and DLPFC activation

during emotional up-regulation and down-regulation; and lastly, Lamm and Lewis (2010) found elevated VMPFC activation for a negative condition compared to a neutral condition in a motivated go/no-go task. In order to determine if moderating effects of neural activation differ depending on emotional context, an emotional component was included in the form of neutral, positive, or negative affectively charged pictures. Notably, the negative pictures shown in this task were usually (75 percent) of a violent or threatening nature . A picture from one of these categories was presented during the delay period, i.e., independent of task requirements, so that neural activation underlying the process of action switching could be measured in the context of neutral, positive, or negative (violent or threatening) affective stimuli. We hypothesize that in the face of negatively valenced images compared to positive or neutral images, participants would show greater neural activation underlying action-switching processes and thus reveal a stronger moderating impact on aggressive behavior.

2, Method

2.1 Participants

The sample was recruited from undergraduate students taking psychology classes at the University of New Orleans. Participants were 76 undergraduate students (35 male). 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 centimeters 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.

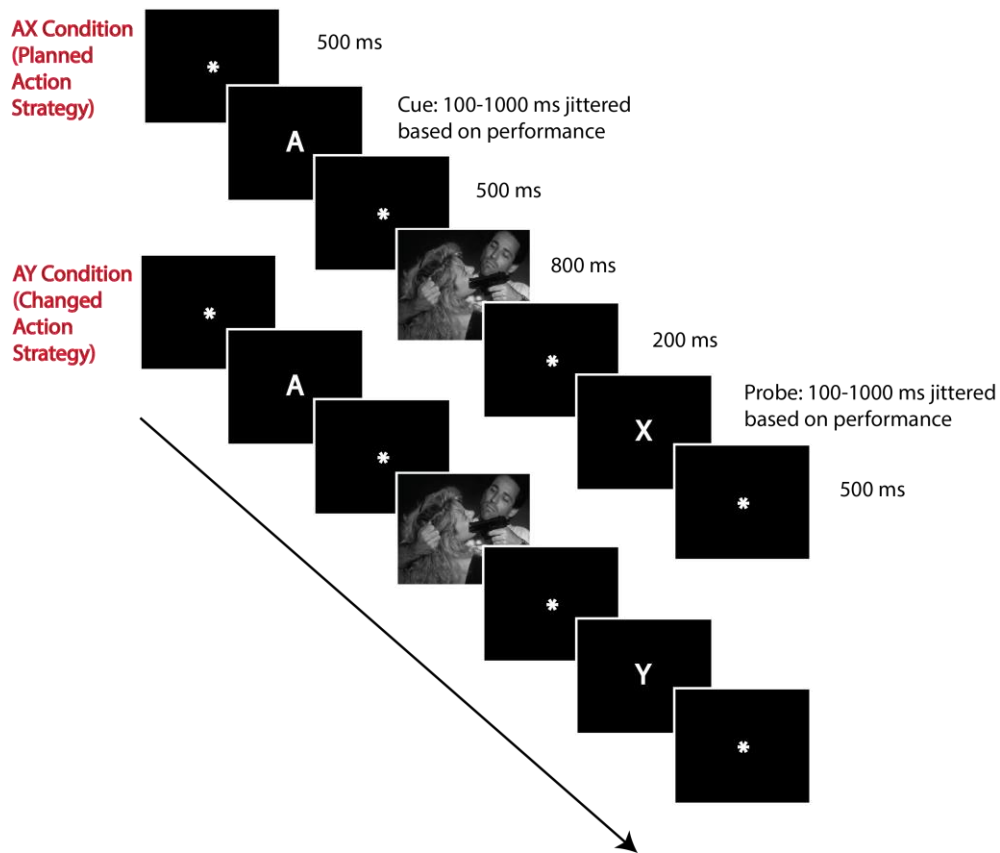
2.3 Measures & Task

2.3.1 The Adult Temperament Questionnaire Short Form (ATQ; Evans & Rothbart, 2007) is a 77-item reliable and valid self-report measure of emotional temperament and self-regulatory capacity. 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 an adult's ability to effortfully regulate their actions.

2.3.2 The Buss Perry Aggression Scale (BPAS; Buss & 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 aggressive tendencies in this sample.

2.3.3 Action Switching Task. The task was a modified AX continuous performance task (Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956). Images were presented on a 17-in monitor using E-prime Software (Psychology Software Tools, Inc., Pittsburgh, PA; Schneider, Eschman, & Zuccolotto, 2002). Stimuli were shown on a black screen and consisted of negative and neutral photos from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 2008) and single letters presented in either blue (cue) or white (probe). Negative, positive, and neutral pictures were 11 cm wide by 8 cm tall and presented in black and white (visual angle was 9.39 degrees). Letters were presented in 60-point size uppercase bold Courier New font. Trials were roughly 3.7 seconds in duration and consisted of the following events (see Figure 1): fixation (500 ms), cue (100-1000 ms), delay (1500 ms), probe (100-1000 ms), and post probe fixation (500 ms). Cue and probe trial times were adjusted dynamically based on participant performance (within each trial cue and probe trial times were always identical). The delay period was comprised of fixation (500 ms), IAPS picture (800 ms), and fixation (200 ms). Neutral, positive, and negative pictures were presented during the delay in pseudo-random order (all participants received the same random order). A diagram of the task is shown in Figure 1.

Figure 1. Modified AX-CPT Task Diagram.



The task consisted of two trial types, AX and AY, distributed randomly throughout the blocks. “A” stands for targeted cues and “X” stands for targeted probes while “Y” stands for any nontargeted probes. AX trials were the propensity setting trial type (66% of trials) and required participants to push a 2 after the cue and a 3 after the probe. AY trial types were presented less frequently (33% of trials) and required participants to push the 2 button after both the cue and the probe. Because AX trials were the propensity setting trial type, AY trials required participants to alter their usual action plans from pushing a 3 after the probe to pushing a 2 after the probe. The task was broken down into three blocks of 100 trials (300 hundred trials total) with opportunities

to rest in between each of the blocks. The task yielded two behavioral measures: performance accuracy and reaction times.

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

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 k Ω , data acquisition began. During recording, all channels were referenced to Cz and after acquisition, data were re-referenced using an average reference.

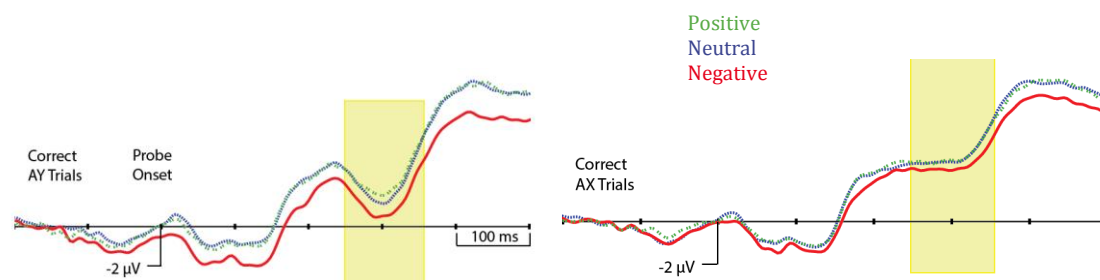
Data were filtered using a FIR bandpass filter with a low-pass frequency of 50 Hz and a high-pass frequency of .3 Hz. To best capture eye blink artifacts, the threshold was set to 140 μ V (peak-to-peak) and all trials in which this threshold was violated were excluded from analyses. Furthermore, signal activation change (peak-to-peak) exceeding 150 μ V across the entire segment and fast transits exceeding a difference (peak-to-peak) of 140 μ V 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 400 ms before to 600 ms after stimulus onset and baseline corrected for the 400 ms preceding stimulus onset. Mediofrontal P2 activation was maximal between 160 and 270 ms after stimulus onset, mediofrontal N2 activation was maximal between 270 and 390 ms after

stimulus; and parietal P3 activation was maximal between 270 and 490 ms after stimulus onset. Peak activations were therefore extracted during these time windows. The mean number of trials comprising correct AX ERPs was 42.9 (SD = 9.6; range = 40), and the mean number of trials comprising correct AY ERPs was 18.4 (SD = 4.5; range = 24).

Visualization of the correct AX and AY stimulus-locked waveforms revealed clear N1, P2, and N2 components for mediofrontal electrodes and clear P3 components for parietal electrodes (See Figure 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, 55, 62 [Pz], 72) as well as eight flanking electrodes (80, 79, 78, 77, 31, 54, 61, and 67). Because of individual differences in peak 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.

Figure 2. Grand average ERP waveforms.

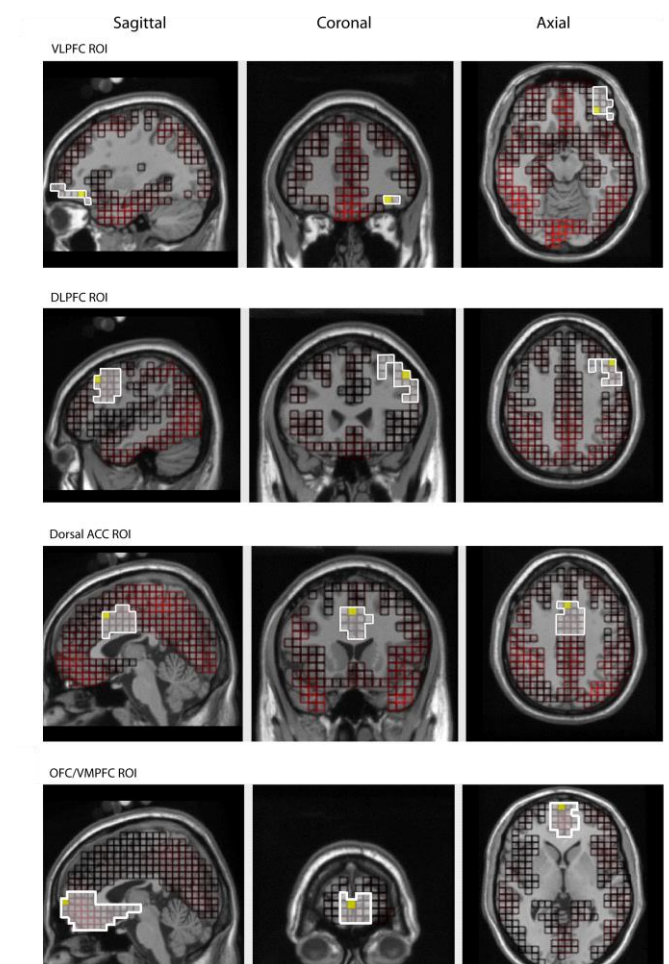


2.4.3 Source-space data analyses. A distributed inverse model that incorporates the change in activation from one electrode to another (in this case 129 electrodes) was used to

calculate the source-space activation. This type of algorithm estimates activation voxel-by-voxel and sample-by-sample and does not require any dipoles to be “fit”, thereby limiting the influence of user bias. The specific algorithm used in the current study was LORETA (Low Resolution Brain Electromagnetic Tomography), which applies a constraint to the minimum-norm solution in order to minimize the discrepancy between values of adjacent voxels (to achieve the most realistic model) within the GeoSource interface (Electrical Geodesic, Inc., Eugene, OR). A regularization constant (indicating how much noise is modeled) of 10^{-4} was applied. This amount of regularization revealed current flow patterns that matched (via visual inspection) the grand-averaged scalp topography better than other levels.

After the data were modeled (LORETA) for the entire cortex (2447 voxels), morphology-based regions of interest (ROIs) were generated using the Montreal Neurological Institute (MNI) average adult MRI (see Figure 3). We were interested in six ROIs: the left and right VLPFC ROI (comprised of 22 voxels each; lateral part of BA 11 and 47), the dACC ROI (comprised of 50 voxels; dorsal part of BA 24 and 32), the left and right DLPFC ROI (comprised of 63 voxels each; BA 9 and dorsal part of BA 46), and the VMPFC/OFC ROI (comprised of 147 voxels; ventromedial parts of BAs 11, 10, 14, and 13). Source waveform amplitudes (nA) for all voxels within an ROI were extracted for 400 ms before stimulus onset to 600 ms after stimulus onset and baseline corrected using the 400 ms before stimulus onset. To ensure that each participant’s maximal activation was analyzed, we chose the voxel and moment in time (within the time period during which the scalp ERP component was maximal) that showed the most activation for each ROI.

Figure 3. Morphology-based regions of interest (ROIs) generated using the Montreal Neurological Institute average adult magnetic resonance image (MRI).



2.4.4 Statistical Analyses. All EEG data with values larger or smaller than 2 SD from the mean were changed to show values of 2 SD from mean, thus preventing statistical analyses from being skewed by outliers. Regression analyses were conducted in this study in order to examine the moderating role of brain activation on the relation between effortful control and aggression. A priori t-tests revealed sex differences for some independent and dependent variables (see Table 1); therefore, sex was entered as a covariate in all EEG analyses. Additionally, for data in the

negative condition, in order to capture only brain activation that was related to both cognitive control and negative emotional state, negative AX and neutral AY brain data were entered as covariates. For data in the positive condition, in order to capture only brain activation that was related to both cognitive control and positive emotional state, positive AX and neutral AY brain data were entered as covariates. For data in the neutral condition, in order to capture only brain activation that was related to both cognitive control and a relatively unemotional context, negative and positive AY and neutral AX brain data were entered as covariates.

Table 1. Sex Differences.

Measures	<i>t</i> (64)	<i>p</i>
Negative P2	-.74	.46
Negative N2	2.82	.006
Negative P3	3.00	.004
Neutral P2	-1.87	.07
Neutral N2	1.50	.14
Neutral P3	2.77	.007
Positive P2	-2.17	.03
Positive N2	1.45	.15
Positive P3	1.96	.05
Negative AY Probe Acc	.50	.62
Neutral AY Probe Acc	.82	.42
Positive AY Probe Acc	1.18	.24
Negative AY Probe RT	-1.34	.19
Neutral AY Probe RT	-1.11	.27
Positive AY Probe RT	-.95	.35
Aggression	3.41	.001
Effortful Control	-1.50	.14

RT – Reaction Time, Acc – Accuracy

3, Results

3.1 Behavioral Results

In order to determine if there were any behavioral effects of emotional context (neutral, negative, positive), two repeated-measures ANOVAs were conducted on AY trials: 1) with performance accuracy as the dependent measure and 2) with reaction times as the dependent measure. Results were not significant for performance accuracy, $F(2,68) = .76$, $p = .47$, $\eta^2 = .02$, or reaction times, $F(2,68) = 1.52$, $p = .31$, $\eta^2 = .04$, suggesting that emotional context did not influence performance accuracy or speed during action switching.

3.2 ERP Condition Differences

In order to determine if there were any ERP differences due to emotional context (neutral, negative, positive), three repeated-measures ANOVAs were conducted: 1) P2 activation as dependent variable, 2) N2 activation as dependent variable, and 3) P3 activation as dependent variable. Since there were sex differences in the ERP data (see Table 1), sex was entered as a between-subjects factor in these analyses. Additionally, trial count was entered in all analyses as a covariate.

Results indicated that there was no main effect of emotion context on P2 amplitude, $F(2,122) = .89$, $p = .41$, $\eta^2 = .01$, nor was there a main effect of sex on P2 amplitude, $F(1,61) = 2.86$, $p = .10$, $\eta^2 = .05$.

Results for N2 amplitude showed a significant interaction of emotional condition by sex, $F(2,122) = 4.36$, $p = .02$, $\eta^2 = .07$. This result indicates that males showed less negative N2 activation in negative trials than in positive trials (Mean difference = 1.04, $p = .02$), while females showed less negative N2 activation in neutral trials than in negative trials (Mean difference = .77, $p = .04$). Additionally, males showed significantly less negative N2 activation

than females in negative trials (Mean difference = 2.42, $p = .004$). All other contrasts were not significant.

Results for P3 amplitude indicated a significant within-subjects effect of emotional condition, $F(2,122) = 5.42$, $p = .006$, $\eta^2 = .08$, as well as a significant main effect of sex, $F(1,61) = 9.14$, $p = .004$, $\eta^2 = .13$, with females showing less positive P3 amplitudes than males. Pairwise comparisons for P3 amplitude indicated that subjects had a significantly less positive activation in negative trials than in neutral trials (Mean difference = 1.43, $p < .001$) or in positive trials (Mean difference = 1.79, $p < .001$); there was no difference in P3 activation between positive and neutral conditions.

3.3 ERP Moderator Effects

Because not everyone with poor effortful control has aggressive tendencies, we also conducted a number of linear regression analyses to test whether ERP amplitudes moderate the association between effortful control and aggression. All covariates, outlined earlier, were entered in step one of the regression model. Next, ERP amplitude and effortful control (centered to decrease the possibility of multicollinearity influencing results, Aiken & West, 1991) were entered in step two. Lastly, an interaction term of ERP amplitude and effortful control was computed and entered in step 3 to test for moderation effects. Aggression was entered as the dependent variable. Correlations between individual variables are summarized in Table 2, while interaction (moderating) results are presented in Table 3. Results revealed that N2 amplitude in both negative and neutral 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 both negative conditions, $\beta = -.59$, $t(58) = -3.62$, $p = .001$, and neutral conditions, $\beta = -.53$, $t(58) = -3.45$, $p = .001$. At high levels of N2 activation (more negative), effortful control was not a significant predictor of aggression scores in negative conditions, $\beta = -.11$, $t(58) = -.55$, $p = .58$, or neutral conditions, $\beta = -.20$, $t(58) = -1.16$, $p = .25$. N2 amplitude in the positive emotional condition did not significantly moderate the association between effortful control and aggression. Additionally, there were no significant moderation effects for P2 or P3 amplitudes for any emotional conditions. Moderation plots are presented in Figure 4.

Table 2. Preliminary Pearson correlation analyses (r-values) between effortful control, aggression, and brain measures.

Measures	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1 Negative P2	-															
2 Neutral P2	.65**	-														
3 Positive P2	.61**	.70**	-													
4 Negative N2	.47**	.25*	.20	-												
5 Neutral N2	.23	.37**	.16	.69**	-											
6 Positive N2	.35**	.24*	.27*	.75**	.72**	-										
7 Negative P3	.31**	.18	.13	.44**	.19	.12	-									
8 Neutral P3	.33**	.28*	.21	.40**	.20	.13	.87**	-								
9 Positive P3	.36**	.22	.24*	.38**	.18	.11	.86**	.85**	-							
10 Negative Accuracy	-.21	-.22	-.09	-.04	-.02	.16	.08	.01	.06	-						
11 Neutral Accuracy	-.20	-.30*	-.26*	-.04	-.05	.12	-.08	-.16	.00	.52**	-					
12 Positive Accuracy	-.18	-.13	-.23	-.17	-.10	-.02	-.04	-.02	.05	.45**	.58**	-				
13 Negative RT	-.24*	-.15	-.28*	-.15	-.03	-.04	-.44**	-.43**	-.41**	-.12	.08	-.03	-			
14 Neutral RT	-.24*	-.11	-.24*	-.17	-.04	-.07	-.37**	-.40**	-.37**	-.19	-.02	-.06	.92**	-		
15 Positive RT	-.20	-.04	-.22	-.13	.01	-.02	-.34**	-.36**	-.36**	-.13	-.03	-.02	.91**	.89**	-	
16 Effortful Control	.04	.09	.04	-.01	-.02	.17	-.10	-.05	-.09	-.02	.11	-.04	.01	-.03	-.06	-
17 Aggression	-.11	-.12	-.19	-.03	.01	-.06	.18	.18	.06	.14	-.05	.23	-.07	-.04	-.01	-.40**

RT – Reaction Time, Acc – Accuracy, * $p < .05$, ** $p < .01$

Table 3. Regression Showing P2 Moderation Effects Predicting Aggression.

Emotion	IV	Unstandard β	Standardized β	<i>T</i> (58)	<i>P</i>
Negative	EC	-.40	-.35	-3.06	.003**
	P2	0	.01	.05	.96
	EC*P2	.02	.07	.56	.58
Neutral	EC	-.40	-.34	-2.87	.006**
	P2	.06	.21	1.20	.23
	EC*P2	.01	.02	.18	.86
Positive	EC	-.43	-.37	-3.19	.002**
	P2	-.07	-.23	-1.45	.15
	EC*P2	-.01	-.01	-.12	.90

EC – Effortful Control, * $p < .05$, ** $p < .01$, *** $p < .001$

Table 4. Regression Showing N2 Moderation Effects Predicting Aggression.

Emotion	IV	Unstandard β	Standardized β	<i>T</i> (58)	<i>P</i>
Negative	EC	-.35	-.30	-2.71	.01**
	N2	-.06	-.22	-1.22	.23
	EC*N2	-.07	-.22	-1.99	.05*
Neutral	EC	-.29	-.25	-2.08	.04*
	N2	.04	.13	.76	.45
	EC*N2	-.08	-.26	-2.15	.04*
Positive	EC	-.39	-.34	-2.74	.01**
	N2	-.02	-.07	-.36	.72
	EC*N2	-.05	-.12	-1.05	.30

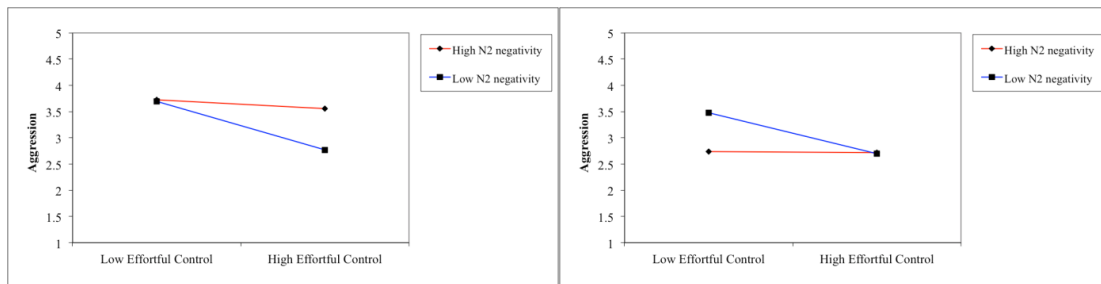
EC – Effortful Control, * $p < .05$, ** $p < .01$, *** $p < .001$

Table 5. Regression Showing P3 Moderation Effects Predicting Aggression.

Emotion	IV	Unstandard β	Standardized β	$T(58)$	P
Negative	EC	-.46	-.39	-3.31	.002**
	P3	-.14	-.61	-2.21	.03*
	EC*P3	-.03	-.12	-1.03	.31
Neutral	EC	-.41	-.36	-3.08	.003**
	P3	.09	.39	1.58	.12
	EC*P3	-.02	-.07	-.59	.56
Positive	EC	-.44	-.38	-3.42	.001**
	P3	-.10	-.43	-1.92	.06
	EC*P3	-.02	-.07	-.55	.58

EC – Effortful Control, * $p < .05$, ** $p < .01$, *** $p < .001$

Figure 4. Moderation plots: Interaction between N2 amplitudes and Effortful Control on aggression.



Negative Emotional Condition

Unemotional Condition

3.2 N2 Source Space Analyses

Source space analyses were conducted to elucidate which cortical generators underlie the moderating effects outlined above. Source space analyses were only conducted for the N2 in negative and neutral conditions, because only this ERP in these conditions showed significant moderation at the scalp level. Linear regression analyses were conducted separately for all ROIs. Regression models were identical to the model structure outlined above, except that source space activation was used rather than scalp ERP activation. In other words, step one consisted of all covariates, step 2 consisted of main effects (source space activation underlying the N2 and effortful control values), step 3 consisted of the interaction between source space activation and effortful control, and the dependent variable was level of aggression. Source space activation and effortful control values were centered to decrease the possibility of multicollinearity influencing results (Aiken & West, 1991). Correlations between individual variables are summarized in Table 4, while interaction (moderating) results are presented in Table 5. In the negative emotion condition, source-space ROIs that significantly moderated the association between effortful control and aggression included the right DLPFC, right VLPFC, VMPFC, and ACC. When these interactions in the negative condition were probed at values of 1 SD above and below the mean, additional regression analyses revealed that this moderating effect was driven by low source space activation. At low levels of source space activation, effortful control was a significant predictor of aggression scores for DLPFC right, $\beta = -.80$, $t(58) = -4.76$, $p < .001$, VLPFC right, $\beta = -.63$, $t(58) = -3.90$, $p < .001$, VMPFC, $\beta = -.75$, $t(58) = -4.18$, $p < .001$, and dACC, $\beta = -.63$, $t(58) = -3.63$, $p < .001$. At high levels of source space activation, effortful control was not a significant predictor of aggression scores for DLPFC right, $\beta = -.08$, $t(58) = -.43$, $p = .67$, VLPFC right, $\beta = -.21$, $t(58) = -1.24$, $p = .22$, VMPFC, $\beta = .04$, $t(58) = .17$, $p = .87$,

or dACC, $\beta = -.06$, $t(58) = -.32$, $p = .75$. In the neutral emotion condition, source-space ROIs that significantly moderated the association between effortful control and aggression included the right DLPFC, VMPFC, and ACC. When these interactions in the neutral condition were probed at values of 1 SD above and below the mean, additional regression analyses revealed that this moderating effect was also driven by low source space activation. At low levels of source space activation, effortful control was a significant predictor of aggression scores for DLPFC right, $\beta = -.73$, $t(58) = -4.12$, $p < .001$, VMPFC, $\beta = -.69$, $t(58) = -4.09$, $p < .001$, and dACC, $\beta = -.68$, $t(58) = -3.66$, $p = .001$. At high levels of source space activation, effortful control was not a significant predictor of aggression scores for DLPFC right, $\beta = -.12$, $t(58) = -.56$, $p = .58$, VMPFC, $\beta = -.12$, $t(58) = -.63$, $p = .53$, or dACC, $\beta = .14$, $t(58) = .48$, $p = .63$. Moderation plots are presented in Figure 5.

Table 6. Pearson correlation analyses (r-values) between effortful control, aggression, and brain & behavioral measures.

Measures	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1 Negative DLPFC-L	-																
2 Neutral DLPFC-L	.78**	-															
3 Negative DLPFC-R	.41**	.30*	-														
4 Neutral DLPFC-R	.45**	.49**	.60**	-													
5 Negative VLPFC-L	.30*	.14	.26*	.28*	-												
6 Neutral VLPFC-L	.33**	.45**	.22	.22	.46**	-											
7 Negative VLPFC-R	0.09	.04	.19	.10	.42**	.34**	-										
8 Neutral VLPFC-R	0.07	.10	.18	.19	.37**	.58**	.49**	-									
9 Negative VMPFC	0	-.01	.12	-.03	.43**	.34**	.60**	.45**	-								
10 Neutral VMPFC	0.11	.18	.25*	.08	.37**	.60**	.46**	.63**	.58**	-							
11 Negative dACC	.45**	.35**	.73**	.57**	.14	.18	.13	.13	.22	.22	-						
12 Neutral dACC	.46**	.52**	.61**	.69**	.27*	.24*	.08	.19	.17	.25*	.64**	-					
13 Negative Acc	-.21	-.13	-.03	-.04	-.15	-.10	-.14	-.03	-.16	-.20	-.09	-.06	-				
14 Neutral Acc	-.22	-.25*	-.02	-.08	-.05	-.21	.07	-.10	-.10	-.16	-.03	-.10	.52**	-			
15 Negative RT	-.18	-.19	-.14	-.03	.10	.02	.18	-.02	.02	.00	-.19	-.16	-.12	.08	-		
16 Neutral RT	-.10	-.12	-.17	-.05	.13	.05	.23	-.04	.10	.00	-.18	-.16	-.19	-.02	.92**	-	
17 Effortful Control	.03	.03	.25*	.23	.09	-.03	.20	.09	.07	.14	.13	.23	-.02	.11	.01	-.03	-
18 Aggression	-.08	-.07	-.08	-.17	-.09	.00	.01	-.01	-.11	-.04	-.17	-.24	.14	-.05	-.07	-.04	-.40**

DLPFC-L – left dorsal lateral prefrontal cortex, DLPFC-R – right dorsal lateral prefrontal cortex, VLPFC-L – left ventral lateral prefrontal cortex, VLPFC-R – right ventral lateral prefrontal cortex, VMPFC – ventral medial prefrontal cortex, dACC – dorsal anterior cingulate cortex, RT – Reaction Time, Acc – Accuracy,

* $p < .05$, ** $p < .01$

Table 7. Moderation Effects Showing Interactions Between Source Space Activation Underlying the N2 and Effortful Control. Values are Regression Coefficients Predicting Aggression.

ROI	Emotion	Unstandard B	Standardized β	$T(58)$	P
DLPFC-L *	Negative	.36	.13	1.15	.26
EC	Neutral	-.11	-.05	-.39	.70
DLPFC-R *	Negative	1.07	.32	3.05	.003**
EC	Neutral	.83	.24	2.11	.04*
VLPFC-L *	Negative	.50	.17	1.47	.15
EC	Neutral	.006	.002	.02	.98
VLPFC-R *	Negative	.46	.23	2.10	.04 *
EC	Neutral	.16	.06	.54	.59
VMPFC * EC	Negative	.53	.28	2.50	.02*
	Neutral	.35	.26	2.32	.02*
dACC * EC	Negative	1.31	.24	2.15	.04 *
	Neutral	1.49	.27	2.07	.04*

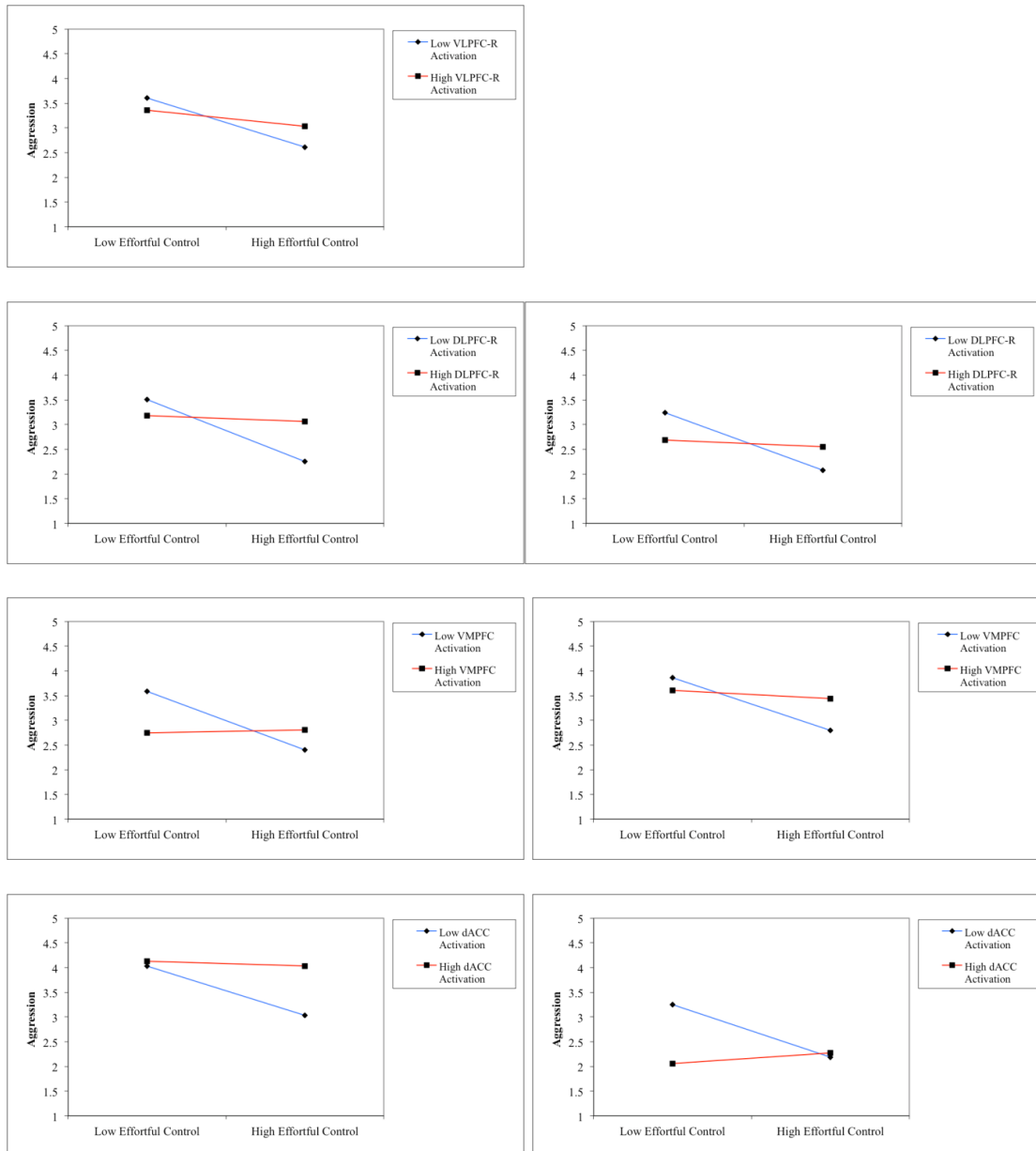
DLPFC-L – left dorsal lateral prefrontal cortex, DLPFC-R – right dorsal lateral prefrontal cortex, VLPFC-

L – left ventral lateral prefrontal cortex, VLPFC-R – right ventral lateral prefrontal cortex, VMPFC –

ventral medial prefrontal cortex, dACC – dorsal anterior cingulate cortex, EC – Effortful Control, * $p <$

.05, ** $p < .01$, *** $p < .001$

Figure 5. Moderation plots: Interaction between N2 amplitudes and Effortful Control on aggression for source-space regions.



Negative Emotional Condition

Unemotional Condition

4, Discussion

4.1 General Discussion

The present study examined the time course of neural activation underlying action switching processes, a key aspect of effortful control, and how these patterns of activation contribute to aggressive behavior. More specifically, we used ERPs and source-space activation (LORETA) 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 compared to a relatively neutral context. As predicted, brain processes underlying action switching significantly moderated the association between effortful control and aggression at both the scalp and source-space levels.

Given that the ability to flexibly switch action strategies likely requires a number of underlying cognitive functions (e.g., Badre & Wagner, 2006; Braver, Paxton, Locke, & Barch, 2009; Eslinger & Grattan, 1993) and that any one of these functions could show deficits that might contribute to aggressive behavior, we decomposed the time course underlying action switching. Our results indicate that only activation during the N2 window significantly moderates the effortful control – aggression relationship. Given that the N2 has been associated with aspects of cognitive control (Lamm, Zelazo, & Lewis, 2006) as well as aggression (Lamm, Granic, Zelazo, & Lewis, 2011; Lewis, Granic, & Lamm, 2006), this suggests that activation during the N2 time window might be a neural mechanism that influences self-control over aggressive tendencies. However, it is not clear as to why the P2 and the P3 did not also moderate the effortful control – aggression association. Given that P2 activation has been associated with attentional orienting (Kanske, Plitschka, & Kotz, 2011; Eimer, Van Velzen, Gherri, & Press, 2006) and the fact that the current task presents all stimuli in the same location, it may be that

our task did not require enough attentional orienting. Thus, it may be that we had insufficient attentional-orienting-related variance to reveal moderational effects. It is possible that in the context of a task with active attentional orienting demands, P2 activation would play a moderating role in the effortful control - aggression relationship.

P3 activation, which has generally been associated with novelty (Friedman, Cycowicz, & Gaeta, 2001; Debener, Makeig, Delorme, & Engel, 2005), context updating (Donchin & Coles, 1988; Verleger, 1988), and motivation (Boksem et al., 2006; Potts, 2004), was not a significant moderator of the effortful control – aggression relationship. While there has been research linking P3 activation with aggression (Bartholow, Bushman, & Sestir, 2006) and externalizing behavior (Lacono et al., 2002), research linking P3 activation to effortful control is much sparser. For this reason, while P3 amplitude seems to be related to aggressive behavior, it does not seem to specifically moderate the association between effortful control and aggression.

The direction of the moderating effect for N2 activation was consistent with previous studies linking neural processing efficiency with reduced activation during cognitive control tasks (e.g. Lamm, Pine, & Fox, 2013; Casey et al., 1997; Durston et al., 2006). For example, Lamm, Pine, & Fox (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 switching. This decrease in prefrontal activation was interpreted to reflect increased efficiency of cognitive-control-related cortical processing. Thus, our results suggest that efficient cognitive control processing moderates the association between effortful control and aggression. Follow-up analyses were conducted using source space models (LORETA) to determine which neural generators drive the moderating effect. ROIs moderating the association between effortful control and aggression included DLPFC, VLPFC,

dACC, and VMPFC. The direction of these effects is also consistent with an efficiency hypothesis, in that lower (efficient) activation in these regions is associated with lower levels of aggression at high levels of effortful control. This pattern of results supports the Lamm et al. (2013) findings and suggests that improved control during the presence of last-minute environmentally triggered information, as measured by an action-switching paradigm, is related to low prefrontal cortical activation. 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.

Interestingly, moderation effects existed only at low levels of N2 activation, which suggests that low or efficient N2 activation during action switching, in conjunction with high effortful control, contributes to less aggressive outcomes. We expected to find the converse as well, that is, high N2 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 an issue with 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 less preselected (academic) 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 increases in ERP activation for negative emotional contexts compared to emotionally neutral contexts (e.g., Lamm, Pine, & Fox, 2013; Lewis et al., 2006; Lamm & Lewis, 2010). However, because these studies did not include a

positive emotional condition, it is unclear if these effects were due to valence or arousal. Similarly, Van Wouwe et al. (2010) found decreased N2 activation (less negative) for a positive compared to a relatively neutral emotional condition in a similar AX-CPT task that showed positive affective video clips. However, this study did not examine negative emotional conditions. 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. Interestingly, women showed the expected increased N2 activation in the negative condition while males did not. This pattern of results is, however, in line with research by Lithari et al. (2010), who showed that females demonstrate significantly more negative N2 amplitudes than males when passively viewing negative affective stimuli. Additionally, there was a main effect of emotion for P3 amplitude, showing that following violent imagery participants had lower P3 activation than in neutral or positive contexts. This finding might be informative for studies of emotion regulation, and suggests that following negative emotional stimuli, we are left with fewer neural resources with which to encode future actions. It is not clear why emotion differences were not found for P2 amplitude, though 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.

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

we require few cognitive-control-related resources to prevent us from lashing out aggressively. Given that our neutral stimuli were not overly emotionally arousing, e.g., a chair, it is not clear why we found significant moderation effects for this condition. It may be that our randomized presentations of emotional trials lead to an emotional carryover effect from negative to neutral trials, and that positive trials were arousing enough (positive valence) to override this emotional carryover effect. Future research should replicate this study using an emotional block design.

4.2 Limitations

There are limitations to the current study. First, the use of source-space analyses allowed us to ask region specific questions that scalp ERPs did not. However, activation patterns are estimated effects and therefore should be interpreted with caution. Furthermore, since activation is estimated, measuring activation differences for small ROIs or regions close together is difficult.

Second, the current study used questionnaire-based proxies to measure both effortful control and aggression. Questionnaire-based measures may be more subjective than biological or behavioral measures, generally for reasons relating to social desirability (Sjöström & Holst, 2002; Richmond, Kiesler, Weisband, & Drasgow, 1999; Armitage & Conner, 1999). Therefore, these results should be replicated using behavioral measures of aggression and effortful control.

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 (Rubia et al., 2006; Luna, Padmanabhan, & O’Hearn, 2010; Eshel et al., 2007), and therefore the adults included in this study might not be fully representative of the adult range. This limits generalizability of results to other age ranges. Future work should expand

on the age range of participants, in order to determine if moderating effects differ throughout development, including later adulthood.

4.3 Conclusions

These results suggest that neural mechanisms underlying flexible action switching moderate the association between effortful control and aggression. Specifically, these results suggest that low or efficient prefrontal cortical activation associated with effortful control contributes 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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Vita

The author obtained his bachelor's degree in mathematics and psychology from Tulane University in 2012. He joined the University of New Orleans psychology graduate program to pursue a PhD in Biopsychology, and became a member of Professor Connie Lamm's research group, the Developmental Cognitive Affective Psychophysiology Lab, in 2014.