Development and Psychopathology (2022), **34**, 1054–1063
doi:10.1017/S0954579420001819

CAMBRIDGE UNIVERSITY PRESS

Regular Article

Behavioral and electrophysiological indices of inhibitory control in maltreated adolescents and nonmaltreated adolescents

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Abstract

Early adverse experiences are believed to have a profound effect on inhibitory control and the underlying neural regions. In the current study, behavioral and event-related potential (ERP) data were collected during a go/no-go task from adolescents who were involved with the child welfare system due to child maltreatment ($n = 129$) and low-income, nonmaltreated adolescents ($n = 102$). The nonmaltreated adolescents were more accurate than the maltreated adolescents on the go/no-go task, particularly on the no-go trials. Paralleling the results with typically developing populations, the nonmaltreated adolescents displayed a more pronounced amplitude of the N2 during the no-go trials than during the go trials. However, the maltreated adolescents demonstrated a more pronounced amplitude of the N2 during the go trials than during the no-go trials. Furthermore, while the groups did not differ during the go trials, the nonmaltreated adolescents displayed a more negative amplitude of the N2 than the maltreated adolescents during no-go trials. In contrast, there was not a significant group difference in amplitude of the P3. Taken together, these results provide evidence that the early adverse experiences encountered by maltreated populations impact inhibitory control and the underlying neural activity in early adolescence.

Keywords: adolescence, child maltreatment, event-related potential, go/no-go task, inhibitory control

(Received 22 April 2020; revised 26 August 2020; accepted 28 September 2020; First Published online 22 December 2020)

In 2017, approximately 674,000 children and adolescents were determined to be the victims of neglect and abuse in the United States (US Department of Health and Human Services, 2019). Furthermore, it has been estimated that 37% of the children and adolescents in the United States will experience at least one child welfare system investigation due to child maltreatment before the age of 18 (Kim, Wildeman, Jonson-Reid, & Drake, 2017). Children and adolescents involved with the child welfare system have typically been exposed to a host of early adverse experiences, including prenatal alcohol and substance exposure; physical, sexual, and emotional abuse; physical and supervisory neglect; and repeated caregiver transitions. As a consequence, these children and adolescents are at increased risk for a multitude of negative outcomes, including academic difficulties, attention and behavior problems, and alcohol and substance use (Aarons, Brown, Hough, Garland, & Wood, 2001; Clausen, Landsverk, Ganger, Chadwick, & Litrownik, 1998; Crozier & Barth, 2005; Keller, Salazar, & Courtney, 2010; Pilowsky & Wu, 2006; Zima et al., 2000). It has been theorized that the negative outcomes observed among populations who have been exposed to early adverse experiences, at least partially, result from experience-induced alterations in specific cognitive abilities and the underlying neural regions (De Bellis, 2001; Fishbein, 2000; Gunnar Fisher, & The Early Experience Stress and Prevention Network, 2006). For example, it has been speculated that experience-induced alterations in inhibitory control and the underlying neural regions contribute to some of the difficulties observed among populations exposed to early adverse experiences (Black, 1998; De Bellis, 2001; Pechtel & Pizzagalli, 2014). Therefore, the current study was designed to investigate behavioral and electrophysiological indices of inhibitory control in maltreated adolescents and low-income, nonmaltreated adolescents in early adolescence. The inclusion of a socioeconomically matched sample of nonmaltreated adolescents ensured that any observed group differences were due to child maltreatment rather than poverty, which is a significant risk factor for child maltreatment.

Inhibitory control and the underlying neural activity

Inhibitory control is a higher order cognitive ability that involves the capacity to voluntarily inhibit prepotent behavioral responses and guide appropriate behaviors through the suppression of competing, irrelevant behaviors (Casey, Tottenham, & Fossella, 2002; Durston et al., 2002). The results from neuroimaging studies indicate that specific regions of the prefrontal cortex, anterior cingulate cortex, striatum, subthalamic nucleus, and motor cortex underlie this cognitive ability (Aron, Behrens, Smith, Frank, & Poldrack, 2007; Booth et al., 2005; Bunge, Dudukovic, Thomason, Vaidya, & Gabrieli, 2002; Casey, Trainor, et al., 1997; Durston et al., 2002; Liddle, Kiehl, & Smith, 2001). There is extensive evidence to suggest that inhibitory control and the underlying neural regions have a protracted developmental course that begins in early

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Cite this article: Bruce J, Kim HK (2022). Behavioral and electrophysiological indices of inhibitory control in maltreated adolescents and nonmaltreated adolescents. *Development and Psychopathology* 34: 1054–1063, https://doi.org/10.1017/S0954579420001819

childhood and continues into emerging adulthood (Casey, Trainor, et al., 1997; Davis, Bruce, Snyder, & Nelson, 2003; Durston et al., 2006; Gogtay et al., 2004; Rubia et al., 2006; Sowell et al., 2004; Thatcher, Walker, & Giudice, 1987; Troller-Renfree et al., 2019). Superimposed on this general developmental trend, there are significant individual differences in inhibitory control that appear to be relatively stable across development (Eigsti et al., 2006; Kochanska, Murray, & Harlan, 2000). Importantly, individual differences in inhibitory control have been shown to be associated with a number of outcomes, such as academic difficulties, symptoms of social anxiety, attention and behavior problems, and alcohol and substance use (Blair & Razza, 2007; Casey, Castellanos, et al., 1997; McClelland et al., 2007; Pears, Capaldi, & Owen, 2007; Toupin, Déry, Pauzé, Mercier, & Fortin, 2000; Troller-Renfree et al., 2019; Wills & Stoolmiller, 2002).

One of the more commonly used methods for assessing inhibitory control is the go/no-go task, during which participants inhibit prepotent behavioral responses by selectively responding to target stimuli (go trials) and inhibiting responses to infrequent nontarget stimuli (no-go trials). Not surprisingly, children, adolescents, and adults are less accurate on the no-go trials that require inhibitory control than the go trials that do not require inhibitory control (Casey, Trainor, et al., 1997; Davis et al., 2003; Durston et al., 2006). In addition to a behavioral index of inhibitory control (i.e., accuracy on the no-go trials), electrophysiological indices of inhibitory control (i.e., event-related potential [ERP] data during the no-go trials) also can be assessed during the go/no-go task. In contrast to behavioral data that reflect the final output from the confluence of multiple cognitive abilities, ERP data have excellent temporal resolution (in milliseconds) and provide information about the temporal sequence of specific cognitive abilities (Luck, 2005). Much of the electrophysiological research employing the go/no-go task has focused on two stimulus-locked ERP components, the N2 and P3. The N2 is a frontocentral negative deflection that typically peaks approximately 250–400 ms after the presentation of the stimulus and is more pronounced (i.e., more negative amplitude) during the no-go trials than during the go trials. Although it is widely recognized that the N2 is associated with inhibitory control in general, the specific cognitive ability is still debated. For example, some researchers argue that it reflects inhibition of a planned response (Folstein & Van Petten, 2008) and other researchers argue that it reflects monitoring for response conflict (Nieuwenhuis, Yeung, Van Den Wildenberg, & Ridderinkhof, 2003). The results from source localization studies suggest that the N2 is most likely generated in the ventral prefrontal cortex and anterior cingulate cortex (Lamm, Zelazo, & Lewis, 2006; Nieuwenhuis et al., 2003). The P3 is a centroparietal positive deflection that typically peaks approximately 300–400 ms after the presentation of the stimulus and is more pronounced (i.e., more positive amplitude) in response to less frequent and/or more salient stimuli such as the no-go trials. It is believed to reflect response potentiation following stimulus evaluation (Nieuwenhuis, Aston-Jones, & Cohen, 2005). Evidence suggest that the P3 is most likely generated in the temporal–parietal junction and lateral prefrontal cortex (Nieuwenhuis et al., 2005).

Impact of early adverse experiences on inhibitory control and underlying neural activity

Because the neural regions underlying inhibitory control have a protracted developmental course and extensive bidirectional connections with the hypothalamic–pituitary–adrenocortical (HPA) system and other systems involved in the response to stress (Arnsten, 2009; Ghashghaei & Barbas, 2002; Herman, Ostrander, Mueller, & Figueiredo, 2005; Sullivan & Gratton, 2002), early adverse experiences are believed to have a profound influence on the development of inhibitory control and the underlying neural regions (Black, 1998; De Bellis, 2001; Pechtel & Pizzagalli, 2014). There is evidence that early adverse experiences, such as parental deprivation, reduce neuronal spine density and length in the prefrontal cortex in rodents (Helmeke et al., 2009; Holmes & Wellman, 2009). Similarly, impaired behavioral performance on inhibitory control tasks has been observed among maltreated children in foster care and children adopted from deprived institutions (Bruce, Tarullo, & Gunnar, 2009; Lewis, Dozier, Ackerman, & Sepulveda-Kozakowski, 2007; Pears, Bruce, Fisher, & Kim, 2010; Pollak et al., 2010). Findings from neuroimaging studies reveal that populations exposed to early adverse experiences also demonstrate atypical patterns of neural activation during inhibitory control tasks (Bruce et al., 2013; Mueller et al., 2010; Sheinkopf et al., 2009; Smith, Fried, Hogan, & Cameron, 2004). To date, electrophysiological indices of inhibitory control have not been examined in a maltreated population. However, children who were raised in deprived institutions and children who were prenatally exposed to alcohol have been shown to display less pronounced amplitudes of the N2 and/or P3 than their peers during the go/no-go task (Burden et al., 2009; Loman et al., 2013; McDermott, Westerlund, Zeanah, Nelson, & Fox, 2012), suggesting that early adverse experiences affect these electrophysiological indices of inhibitory control.

Objectives and hypotheses of the current study

The objective of the current study was to examine behavioral and electrophysiological indices of inhibitory control during the go/no-go task in maltreated adolescents and low-income, nonmaltreated adolescents. It is believed that the early adverse experiences encountered by maltreated populations result in acute and/or chronic activation of the HPA system and other systems involved in the response to stress, which in turn impairs the development and subsequent functioning of critical neural regions (Gunnar & Quevedo, 2007). Furthermore, it has been theorized that the neural regions underlying inhibitory control, particularly the prefrontal cortex, may be especially vulnerable to the effects of early adverse experiences because these neural regions have a protracted developmental course and a high density of glucocorticoid (hormone produced by the HPA system) receptors (Black, 1998; De Bellis, 2001; Pechtel & Pizzagalli, 2014). Consistent with this theory and with prior results with maltreated children (Lewis et al., 2007; Pears et al., 2010), it was hypothesized that the maltreated adolescents would demonstrate poorer behavioral performance during the go/no-go task than the nonmaltreated adolescents. Specifically, the maltreated adolescents were expected to be less accurate on the trials that require inhibitory control (i.e., no-go trials). Based on previous studies with other populations exposed to early adverse experiences (Burden et al., 2009; Loman et al., 2013; McDermott et al., 2012), it also was hypothesized that the maltreated adolescents would demonstrate atypical electrophysiological performance during the go/no-go task compared to the nonmaltreated adolescents. More precisely, the maltreated adolescents were expected to display less pronounced amplitudes of the N2 and P3 during the trials that require inhibitory control. Because the current study was the first study to

https://doi.org/10.1017/S0954579420001819 Published online by Cambridge University Press

examine the electrophysiological indices of inhibitory control with maltreated adolescents, the results of this study provide unique insight into the effect of the early adverse experiences typically encountered by a maltreated population on the specific cognitive abilities supporting inhibitory control.

Method

Participants

The sample in the current study included two groups of 12- to 13-year-olds: adolescents who were involved with the child welfare system due to child maltreatment ($n = 129$) and low-income, nonmaltreated adolescents ($n = 102$). To recruit the maltreated adolescents, Oregon Department of Human Services child welfare system staff provided monthly lists of all of the 12- to 13-year-olds who were victims of recent reports of neglect and/or abuse and who were living with at least one biological parent in one of seven counties. The nonmaltreated adolescents were recruited via postcards mailed to the parents of students at local middle schools. The exclusion criteria for both groups were: (a) parent or adolescent was not fluent in English and (b) parent or adolescent could not complete the assessment procedures due to a severe developmental or physical disorder. In addition, the exclusion criteria for the nonmaltreated group were: (a) family had been involved with the child welfare system (verified by parent report and child welfare system records) and (b) adolescent had not consistently lived with at least one biological parent. To ensure that group differences were not attributable to socioeconomic status, the nonmaltreated group also was required to have an annual household income equal to or less than 185% of the poverty level (i.e., cutoff to qualify for reduced price school meals via the National School Lunch Program) and a parent education equal to or less than a 4-year college degree.

Descriptive information about the adolescents is presented by group in Table 1. The maltreated adolescents and nonmaltreated adolescents did not significantly differ on age, gender, or race/ethnicity, $F(1, 229) = 2.15$, $p = .114$, Pearson $\chi^2(2, N = 231) = 1.76$, $p = .416$, and Pearson $\chi^2(1, N = 231) = 2.89$, $p = .089$, respectively. Similarly, the groups did not differ on annual household income or parent education, $F(1, 228) = 3.34$, $p = .069$, and $F(1, 229) = 0.04$, $p = .852$, respectively. The mean annual household income for both groups corresponded to $20,000–$29,000 per year, and the mean parent education for both groups corresponded to some postsecondary education but did not earn a degree or certificate. To provide an estimate of general intellectual ability, the adolescents completed the matrix reasoning subtest and vocabulary subtest of the *Wechsler Abbreviated Scale of Intelligence* – Second Edition (Wechsler, 2011). T scores from these subtests are summed to create a Full Scale Intelligence Quotient (FSIQ) with a mean of 100 and standard deviation of 15. Although both groups performed within the average range, the maltreated adolescents and nonmaltreated adolescents significantly differ on FSIQ, $F(1, 229) = 14.21$, $p = .000$. As shown in Table 1, the maltreated adolescents displayed lower FSIQs than the nonmaltreated adolescents. Thus, subsequent analyses controlled for this variable as relevant.

Table 1. Descriptive statistics for adolescent characteristics by group

Variable	Maltreated group	Nonmaltreated group
Age (years *M SD*)	13.02 0.62	12.90 0.61
Gender (% male)	51.2	46.1
Race/ethnicity (%)		
African American	0	2.9
Asian American	0.8	2.0
European American	65.9	54.9
Hispanic/Latino	11.6	28.4
Native American	2.3	1.0
Native Hawaiian/Pacific Islander	0	1.0
Multiracial/Multiethnic	19.4	9.8
Full-scale intelligence quotient (*M SD*)****	94.65 13.32	101.45 13.97

*$p < .05$. **$p < .01$. ***$p < .005$. ****$p < .001$.

Procedures

Prior to participation in the study, the adolescents provided informed assent and one of their parents provided informed permission for the adolescents' participation and informed consent for their own participation. If the State of Oregon was the legal guardian of a maltreated adolescent at the time of participation, the child welfare system caseworker (as a representative of the State) also provided informed permission for the adolescent's participation. The adolescents and parents then completed a 2½-hr laboratory-based assessment. The assessment included a standardized measure of general intellectual ability, computer-administered cognitive tasks, and questionnaires and interviews for the adolescents and questionnaires and interviews for the parents. The adolescents and parents received $35 each for completing this assessment. All study documents and procedures were approved by the Institutional Review Boards at the Oregon Social Learning Center and Oregon Public Health Division prior to beginning the study.

Measures

Go/no-go task

Behavioral data and ERP data were recorded during a computer-administered go/no-go task that was presented using the STIM Stimulus Presentation System (James Long Company, Caroga Lake, NY). For each trial, a white letter was presented on a black background for 500 ms with a fixed 1500-ms interstimulus interval. The adolescents were instructed to press the button as quickly and accurately as possible for every letter (go trials) except "X" (no-go trials). Prior to beginning the task, the adolescents completed ten practice trials that included performance feedback to ensure comprehension of the task instructions. Performance feedback was not provided during the task. The task consisted of 20 go trials, followed by a pseudorandom order of 75% go trials and 25% no-go trials. The fixed interstimulus interval and increased frequency of go trials induce the prepotent behavioral response of pressing the button that must be inhibited for the no-go trials. The adolescents completed two blocks of 180 trials (i.e., a total of 280 go trials and 80 no-go trials). Accuracy and reaction time in milliseconds were recorded for each trial using the STIM Stimulus Presentation System.

https://doi.org/10.1017/S0954579420001819 Published online by Cambridge University Press

Electroencephalogram (EEG) data acquisition and processing

Prior to collecting the EEG data, a calibration file was collected by running a 50 μV, 10-Hz calibration signal through all channels. The EEG data were recorded using a Lycra cap fitted with tin electrodes in accordance with the International 10–20 System (Jasper, 1958). Data were collected from 26 scalp electrodes and two mastoid electrodes with Cz serving as the reference electrode and AFz serving as the ground electrode. Two channels of electrooculogram (EOG) data were recorded with an electrode placed above and below the left eye for vertical EOG and an electrode placed at the outer canthus of each eye for horizontal EOG. Impedances were tested before and after EEG data collection to ensure that each electrode site had an impedance of 10 KΩ or less. The EEG data were amplified by a custom 32-channel isolated bioelectric amplifier (SA Instrumentation Company, San Diego, CA) using filter settings of 0.1 Hz and 100 Hz. The data were digitized using a sampling rate of 512 Hz with a 16-bit A/D converter (DATAQ Instruments, Inc., Akron, OH).

The EEG Analysis System (James Long Company, Caroga Lake, NY) was used to calibrate, rereference, and artifact score the data. Artifact due to vertical eye movement, identified as rapid increases and decreases of 150 μV, was regressed. The EEG data were rereferenced offline using an average mastoid configuration and were digitally refiltered with a 15-Hz low-pass filter. Epochs containing signals ±200 μV and trials with reaction times of less than 200 ms or errors of commission or omission were excluded from analyses. The EEG data at Fz, FCz, Cz, and Pz were time locked to the presentation of the stimulus, corrected using a baseline window of −150 to −50 ms relative to the stimulus, and quantified separately for the go trials and no-go trials. The adolescents were required to have at least 20 artifact-free ERP trials for both trial types to be included in the analyses of the ERP data. Peak amplitude, rather than mean amplitude, was analyzed for the N2 and P3 because there was significant variability in the latency and topography of these ERP components across adolescents. The N2 was identified as the maximum negative peak between 200 and 500 ms relative to the presentation of the stimulus at Fz and FCz, and the P3 was identified as the maximum positive peak between 400 and 700 ms relative to the presentation of the stimulus at Cz and Pz. The selection of the time window and electrode sites for the ERP components was informed by previous studies that used this task with similar-aged, at-risk samples (Loman et al., 2013; McDermott et al., 2012) and refined by visual inspection of the ERP waveforms for each adolescent.

Of the 231 adolescents in the current study, one adolescent declined to complete the go/no-go task and thus was not included in the analyses of the behavioral data or ERP data. In addition, one adolescent declined to complete the EEG data acquisition, four adolescents had unusable EEG data due to an issue during data acquisition, and 13 adolescents had less than 20 artifact-free ERP trials for one or both trial types. Thus, these adolescents were not included in the analyses of the ERP data. None of the adolescents were excluded from analyses due to poor behavioral performance on the task, as all of the adolescents exceeded the established criterion for accuracy (i.e., correctly responding to at least 75% of the go trials). In sum, the analytic sample included 230 adolescents (128 maltreated adolescents and 102 nonmaltreated adolescents; 99.6% of the total sample) for the behavioral data and 212 adolescents (116 maltreated adolescents and 96 nonmaltreated adolescents; 91.8% of the total sample) for the ERP data. The percentage of the maltreated adolescents and nonmaltreated adolescents included in the analyses of the ERP data (89.9% and 94.1%, respectively) did not significantly differ, Pearson $\chi^2(2, N = 231) = 1.33$, $p = .249$. The mean number of artifact-free ERP trials for the analytic sample by group was as follows: 230.05 ($SD = 42.29$) for the maltreated group and 241.36 ($SD = 32.95$) for the nonmaltreated group for the go trials and 45.35 ($SD = 13.83$) for the maltreated group and 51.20 ($SD = 13.91$) for the nonmaltreated group for the no-go trials. The maltreated group and nonmaltreated group significantly differ in the number of artifact-free ERP trials for the go trials and no-go trials, $F(1, 210) = 4.57$, $p = .034$, and $F(1, 210) = 9.33$, $p = .003$, respectively. Thus, subsequent analyses controlled for these variables as relevant.

Table 2. Descriptive statistics for behavioral data and event-related potential (ERP) data by group

Variable	Maltreated group		Nonmaltreated group	
	M	*SD*	*M*	*SD*
Correct responses (%)				
Go trials	97.4	3.8	98.3	2.6
No-go trials	61.4	18.0	67.9	17.9
Reaction time (ms)	381.83	54.73	378.49	57.60
Peak amplitude of N2 (μV)				
Go trials at Fz	−9.74	5.91	−10.24	5.96
No-go trials at Fz	−8.72	7.19	−11.17	7.92
Go trials at FCz	−7.84	6.29	−8.57	5.98
No-go trials at FCz	−6.58	7.50	−9.53	8.26
Peak amplitude of P3 (μV)				
Go trials at Cz	7.05	5.26	6.59	4.22
No-go trials at Cz	15.07	7.44	15.48	7.13
Go trials at Pz	6.94	5.08	6.61	4.97
No-go trials at Pz	15.67	6.54	16.19	6.08

Data analysis

Prior to analyses, the behavioral data (i.e., percentage of correct responses and average reaction time) and ERP data (i.e., peak amplitude of the N2 and P3) were examined for extreme values (i.e., values more than 3 *SD* above or below the mean). This examination revealed extreme values for eight adolescents for percentage of correct responses ($n = 7$ for go trials and $n = 1$ for no-go trials), one adolescent for average reaction time, three adolescents for peak amplitude of the N2 ($n = 2$ for go trials and $n = 2$ for no-go trials), and four adolescents for peak amplitude of the P3 ($n = 4$ for go trials and $n = 0$ for no-go trials). To ensure that these extreme values did not have undue influence on the results, these adolescents were excluded from the relevant preliminary analysis. The pattern of results for these preliminary analyses was the same as the pattern of the results for the analyses that included all of the adolescents. Thus, the data from these adolescents were retained for subsequent analyses.

Descriptive data for the behavioral data and ERP data collected during the go/no-go task are presented by group in Table 2. The grand average waveforms displaying the N2 at Fz and FCz are

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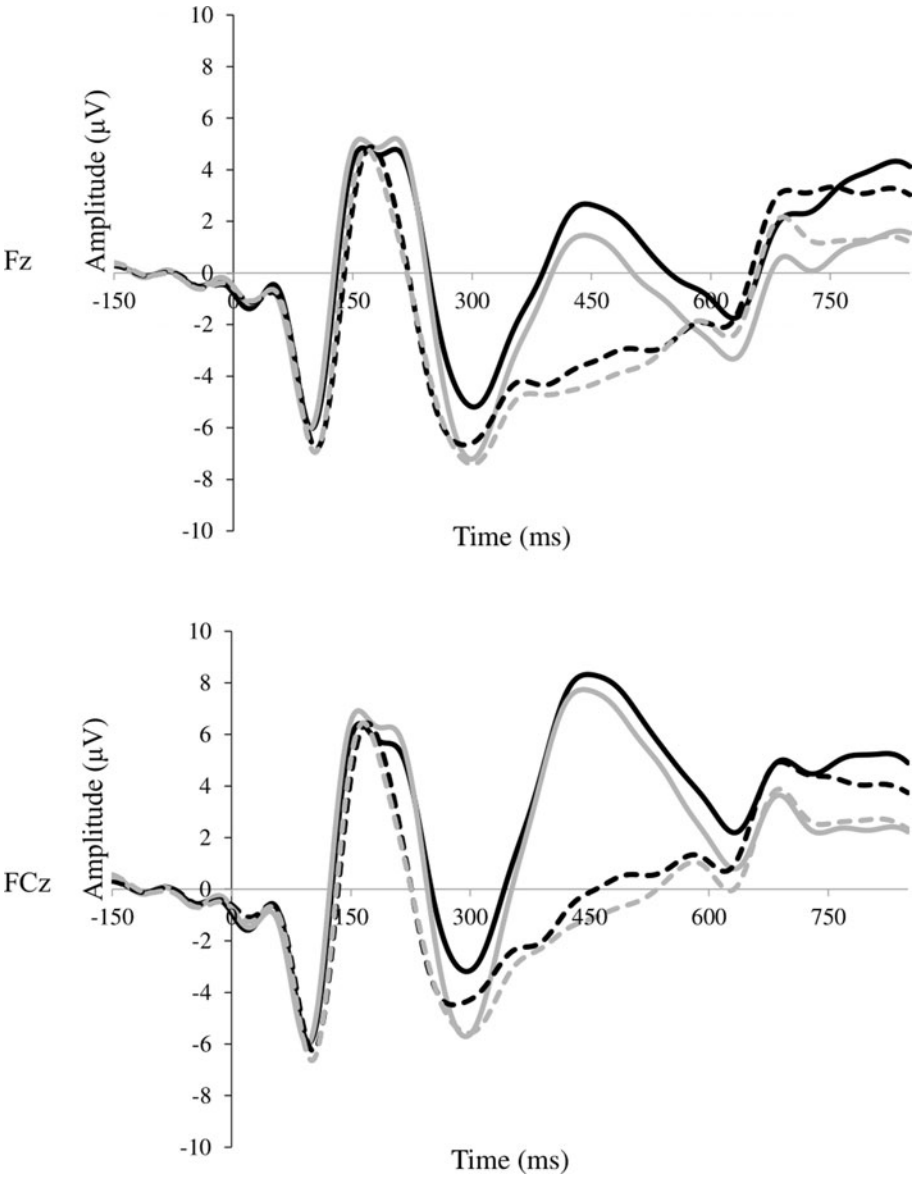

Figure 1. Grand average waveforms displaying the N2 at Fz and FCz for the go trials (dashed line) and no-go trials (solid line) for the maltreated group (black line) and nonmaltreated group (gray line).

presented by trial type and group in Figure 1, and the grand average waveforms displaying the P3 at Cz and Pz are presented by trial type and group in Figure 2. Repeated measures or one-way analyses of variance (ANOVAs) were conducted using IBM SPSS Statistics, version 25, to examine the behavioral data and ERP data. The degrees of freedom, *F* values, *p* values, and effect sizes (partial η^2) for all of the ANOVAs examining the behavioral data and ERP data are shown in Table 3. Post hoc paired comparisons using Bonferroni corrections for multiple comparisons were conducted for significant main effects and interactions. As noted above, there were significant group differences in general intellectual ability and number of artifact-free ERP trials for the go trials and no-go trials. To ensure that significant main effects of and/or interactions with group for the behavioral data and ERP data were not primarily attributable to the group difference in general intellectual ability or number of artifact-free ERP trials for the go trials and no-go trials, analyses controlled for these variables as relevant. That is, for the behavioral data, analyses with significant main effects of group and/or interactions with group were further investigated by conducting a repeated measures or one-way analysis of covariance (ANCOVA) controlling for general intellectual ability. For the ERP data, analyses with significant main effects of group and/or interactions with group were further investigated by conducting a repeated measures ANCOVA controlling for general intellectual ability and number of artifact-free ERP trials for the go trials and no-go trials. (Because the analyses of the behavioral data included all of the trials, not just the trials included in the analyses of the ERP data, the analyses of the behavioral data did not control for the number of artifact-free ERP trials for the go trials and no-go trials.)

Results

Behavioral data

Percentage of correct responses

A repeated measures ANOVA was conducted to examine percentage of correct responses on the go/no-go task with trial type (go and

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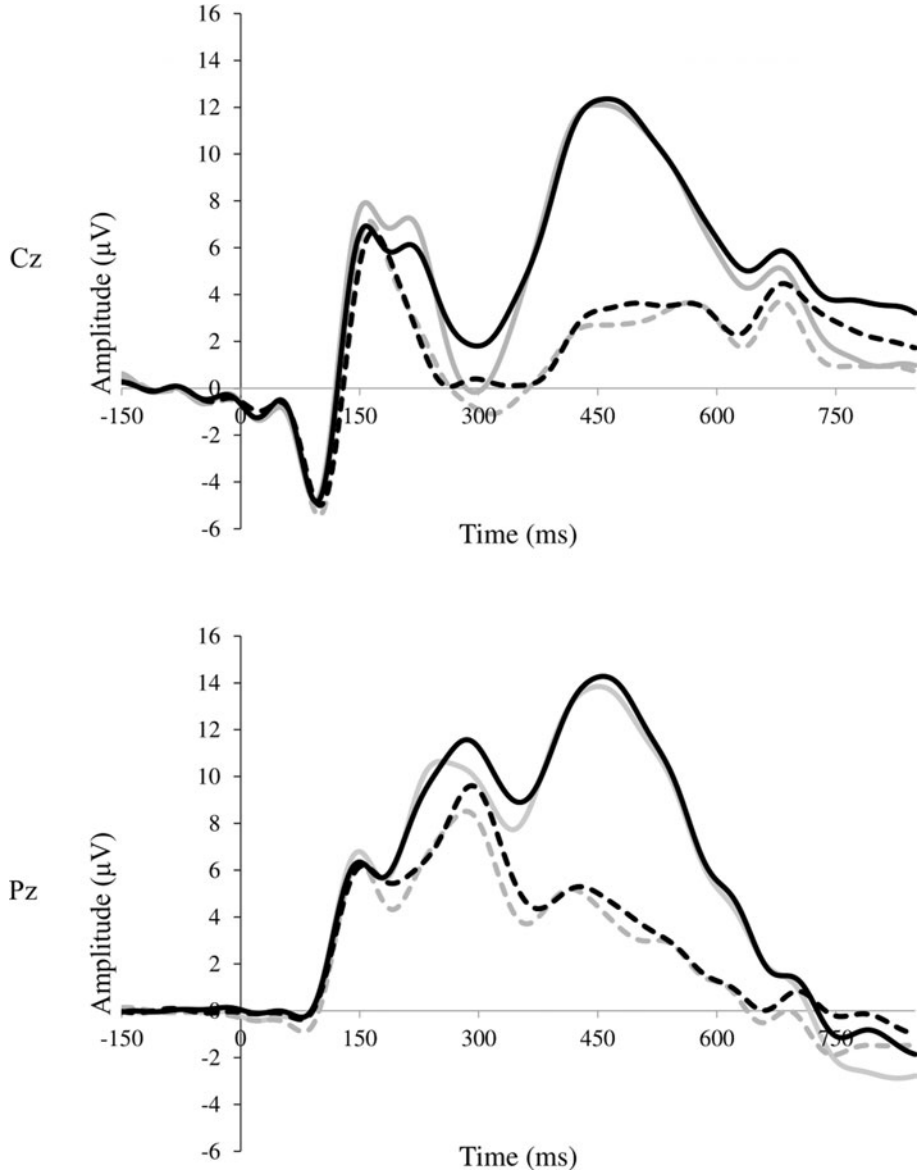

Figure 2. Grand average waveforms displaying the P3 at Cz and Pz for the go trials (dashed line) and no-go trials (solid line) for the maltreated group (black line) and nonmaltreated group (gray line).

no-go) as the within-subjects factor and group (maltreated and nonmaltreated) as the between-subjects factor. As shown in Table 3, the main effect of trial type was significant, with a higher percentage of correct responses on the go trials (M = 97.8%) than the no-go trials (M = 64.7%). The main effect of group also was significant, with the nonmaltreated adolescents (M = 83.1%) displaying a higher percentage of correct responses on the task overall than the maltreated adolescents (M = 79.4%). In addition, the interaction between trial type and group was significant. To clarify the nature of this interaction, the simple main effects of trial type and group were examined. Post hoc paired comparisons examining the simple main effect of trial type revealed that maltreated adolescents and nonmaltreated adolescents displayed a higher percentage of correct responses on the go trials than the no-go trials, $F(1, 228) = 570.94$, $p = .000$, partial $\eta^2 = .72$, and $F(1, 228) = 323.22$, $p = .000$, partial $\eta^2 = .59$, respectively. However, the difference between go trials and no-go trials appeared to be more pronounced for the maltreated adolescents than the nonmaltreated adolescents. Post hoc paired comparisons examining the simple main effect of group indicated that the nonmaltreated adolescents had a higher percentage of correct responses than the maltreated adolescents on the go trials and no-go trials, $F(1, 228) = 4.13$, $p = .043$, partial $\eta^2 = .02$, and $F(1, 228) = 7.55$, $p = .006$, partial $\eta^2 = .03$, respectively. However, the group difference appeared to be more pronounced for the no-go trials than the go trials.

To further examine the main effect of group and the interaction between trial type and group, a repeated measures ANCOVA, controlling for general intellectual ability, was conducted to examine percentage of correct responses with trial type (go and no-go) as the within-subjects factor and group (maltreated and nonmaltreated) as the between-subjects factor. Neither the main effect of general intellectual ability nor the interaction with general intellectual ability was significant. Furthermore, the main effect of group and interaction between trial type and group remained significant even after controlling for general intellectual ability.

Average reaction time

A one-way ANOVA was conducted to examine average reaction time on the go trials with group (maltreated and nonmaltreated)

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Table 3. Results of analyses of variance for behavioral and event-related potential (ERP) data

Source	*df*	*F* value	Partial η^2
Correct responses			
Trial type	1, 228	859.91****	0.79
Group	1, 228	8.35***	0.04
Trial type × Group	1, 228	6.25*	0.03
Reaction time			
Group	1, 228	0.20	0.00
Peak amplitude of N2			
Electrode site	1, 210	102.26****	0.33
Trial type	1, 210	0.08	0.00
Electrode site × Trial type	1, 210	0.33	0.00
Group	1, 210	3.70	0.02
Electrode site × Group	1, 210	1.06	0.01
Trial type × Group	1, 210	8.70***	0.04
Electrode site × Trial type × Group	1, 210	0.50	0.00
Peak amplitude of P3			
Electrode site	1, 210	1.11	0.01
Trial type	1, 210	603.99****	0.74
Electrode site × Trial type	1, 210	5.85*	0.03
Group	1, 210	0.00	0.00
Electrode site × Group	1, 210	0.04	0.00
Trial type × Group	1, 210	1.43	0.01
Electrode site × Trial type × Group	1, 210	0.00	0.00

*p < .05. **p < .01. ***p < .005. ****p < .001.

as the between-subjects factor. The main effect of group was not significant, as the groups demonstrated similar reaction times on the go trials.

ERP data

Peak amplitude of the N2

A repeated measures ANOVA was conducted to examine peak amplitude of the N2 during the go/no-go task with electrode site (Fz and FCz) and trial type (go and no-go) as the within-subjects factors and group (maltreated and nonmaltreated) as the between-subjects factor. As shown in Table 3, the main effect of electrode was significant, with a more negative amplitude of the N2 at Fz ($M = -9.97$ μV) than at FCz ($M = -8.13$ μV). The main effect of trial type and the main effect of group were not significant. However, the interaction between trial type and group was significant. To clarify the nature of this interaction, the simple main effects of trial type and group were examined. Post hoc paired comparisons examining the simple main effect of trial type indicated that the maltreated adolescents displayed a more negative amplitude of the N2 during go trials than during no-go trials, $F(1, 210) = 5.75$, $p = .017$, partial $\eta^2 = .03$. In contrast, the nonmaltreated adolescents displayed a more negative amplitude of the N2 during no-go trials than during go trials, $F(1, 210) = 3.27$, $p = .072$, partial $\eta^2 = .02$. Furthermore, post hoc paired comparisons examining the simple main effect of group revealed that the maltreated adolescents and nonmaltreated adolescents did not differ in terms of amplitude of the N2 during go trials, $F(1, 210) = 0.57$, $p = .452$, partial $\eta^2 = .00$. Conversely, the nonmaltreated adolescents displayed a more negative amplitude of the N2 during no-go trials than the maltreated adolescents, $F(1, 210) = 6.82$, $p = .010$, partial $\eta^2 = .03$. None of the other interactions with electrode site, trial type, or group were significant.

To further examine the interaction between trial type and group, a repeated measures ANCOVA, controlling for general intellectual ability, number of ERP trials for the go trials, and number of ERP trials for the no-go trials, was conducted to examine peak amplitude of the N2 with electrode site (Fz and FCz) and trial type (go and no-go) as the within-subjects factors and group (maltreated and nonmaltreated) as the between-subjects factor. None of the main effects of or interactions with general intellectual ability, number of ERP trials for the go trials, and number of ERP trials for the no-go trials were significant. Furthermore, the interaction between trial type and group remained significant even after controlling for these variables.

Peak amplitude of the P3

A repeated measures ANOVA was conducted to examine peak amplitude of the P3 during the go/no-go task with electrode site (Cz and Pz) and trial type (go and no-go) as the within-subjects factors and group (maltreated and nonmaltreated) as the between-subjects factor. The main effects of electrode site and group were not significant. However, the main effect of trial was significant, with a more positive amplitude of the P3 during the no-go trials ($M = 15.60$ μV) than during the go trials ($M = 6.80$ μV). Furthermore, the interaction between electrode site and trial type was significant. To clarify the nature of this interaction, the simple main effects of electrode site was examined. These post hoc paired comparisons revealed that the amplitude of the P3 at Cz ($M = 6.82$ μV) and Pz ($M = 6.78$ μV) did not differ during go trials, $F(1, 210) = 0.03$, $p = .872$, partial $\eta^2 = .00$. In contrast, the amplitude of the P3 was more positive at Pz ($M = 15.93$ μV) than at Cz ($M = 15.28$ μV) during no-go trials, $F(1, 210) = 3.40$, $p = .066$, partial $\eta^2 = .02$. None of the other interactions with electrode site, trial type, or group were significant.

Discussion

There is extensive evidence demonstrating that children and adolescents who were involved with the child welfare system due to child maltreatment are at elevated risk for negative outcomes across multiple domains of functioning, including academic difficulties, attention and behavior problems, and alcohol and substance use (Aarons et al., 2001; Clausen et al., 1998; Crozier & Barth, 2005; Keller et al., 2010; Pilowsky & Wu, 2006; Zima et al., 2000). It has been speculated that experience-induced alterations in specific cognitive abilities and the underlying neural regions may contribute to the difficulties observed among maltreated children and adolescents (De Bellis, 2001; Fishbein, 2000; Gunnar & Fisher, 2006). Therefore, the current study was designed to examine behavioral and electrophysiological indices of one such cognitive ability, inhibitory control, in maltreated adolescents and low-income, nonmaltreated adolescents in early adolescence. The results of the current study contribute to the growing evidence that early adverse experiences negatively affect behavioral and electrophysiological indices of inhibitory control and provide unique information about the specific cognitive

https://doi.org/10.1017/S0954579420001819 Published online by Cambridge University Press

ability supporting inhibitory control affected in a maltreated population.

Consistent with the results of previous studies with children, adolescents, and adults (Casey, Trainor, et al., 1997; Davis et al., 2003; Durston et al., 2006), both groups of adolescents were less accurate on the no-go trials that require inhibitory control than the go trials that do not require inhibitory control. In general, the adolescents committed very few errors on the go trials. However, they performed quite poorly on the no-go trials, which suggests that successfully inhibiting a prepotent response during the go/no-go task continues to be a challenging task into early adolescence for maltreated populations and low-income populations. Furthermore, as predicted, the maltreated adolescents were less accurate on the go/no-go task than the nonmaltreated adolescents. This group difference in accuracy was more pronounced on the no-go trials than the go trials, and it remained significant even after controlling for the group difference in general intellectual ability. This pattern of results parallels the results of previous studies with other populations exposed to early adverse experiences, including maltreated children in foster care and children adopted from deprived institutions (Bruce et al., 2009; Lewis et al., 2007; Pears et al., 2010; Pollak et al., 2010). Taken together, these findings suggest that inhibitory control, as assessed by behavioral performance on inhibitory control tasks, may be particularly vulnerable to the effects of early adverse experiences.

While previous studies have examined electrophysiological indices of inhibitory control in other populations exposed to early adverse experiences (Burden et al., 2009; Loman et al., 2013; McDermott et al., 2012), the current study was the first such study with maltreated adolescents. Interestingly, the pattern of results for both groups of adolescents for the peak amplitude of the P3, which is believed to reflect response potentiation following stimulus evaluation, was consistent with previous research findings with typically developing populations (Nieuwenhuis et al., 2005). That is, the amplitude of the P3 was more pronounced during the no-go trials than during the go trials for the maltreated adolescents and nonmaltreated adolescents. In contrast, the maltreated adolescents and nonmaltreated adolescents demonstrated different patterns of results for the peak amplitude of the N2, which is believed to reflect response inhibition or conflict monitoring. Paralleling the results of previous studies with typically developing populations (Folstein & Van Petten, 2008), the nonmaltreated adolescents displayed a more pronounced amplitude of the N2 during the no-go trials than during the go trials. However, the maltreated adolescents demonstrated a more pronounced amplitude of the N2 during the go trials than during the no-go trials. Furthermore, while the maltreated adolescents and nonmaltreated adolescents did not differ in terms of amplitude of the N2 during the go trials, the nonmaltreated adolescents displayed a more negative amplitude of the N2 during no-go trials than the maltreated adolescents. Importantly, these results continued to be significant even after controlling for the group differences in general intellectual ability and the number of ERP trials included in the analyses. Although additional electrophysiological research with maltreated populations is needed, the divergent pattern of results for the amplitude of the N2 and P3 in the current study is intriguing. A possible explanation for this divergent pattern of results is that specific neural regions may be particularly sensitive to the negative effects of early adverse experiences. That is, source localization studies suggest that the N2 is generated in the ventral prefrontal cortex and anterior cingulate cortex (Lamm et al., 2006; Nieuwenhuis et al., 2003) and that the P3 is generated in the temporal–parietal junction and lateral prefrontal cortex (Nieuwenhuis et al., 2005). Perhaps the early adverse experiences encountered by maltreated adolescents have an impact on the development and subsequent functioning of the neural regions that generate the N2, but not the neural regions that generate the P3.

Taken as a whole, the results of the current study indicate that the maltreated adolescents were less accurate on the go/no-go task, particularly during the no-go trials that require inhibitory control, and displayed an atypical pattern of results on an ERP component that reflects response inhibition or conflict monitoring, but not an ERP component that reflects response potentiation, compared to the nonmaltreated adolescents. These results suggest that maltreated adolescents demonstrate an impairment in a specific cognitive ability supporting inhibitory control (rather than a more general cognitive ability such as sustained attention). There are several potential implications of the results of the current study. For example, the results may inform the development of a more precise, neurobiologically based explanatory model of the negative outcomes in maltreated populations. As noted above, difficulties with inhibitory control have been implicated in the etiology of a number of negative outcomes that have been observed among populations exposed to early adverse experiences (e.g., academic difficulties, attention and behavior problems, alcohol and substance use). Thus, inhibitory control may serve as a mechanism underlying the associations between early adverse experiences and subsequent negative outcomes among maltreated children and adolescents. Similarly, the results may illuminate a possible intervention target, contributing to the development of more effective and efficient preventive interventions for maltreated populations. That is, it may be beneficial for preventive interventions to specifically target inhibitory control (e.g., teaching skills that improve inhibitory control and/or compensate for deficits in inhibitory control) in an effort to prevent or ameliorate the negative outcomes observed among maltreated children and adolescents. To date, efforts to target inhibitory control generally have fallen into two categories: laboratory-based training (e.g., repeated practice on a computerized inhibitory control task) and ecologically-based intervention (e.g., school readiness intervention that focuses on self-regulation broadly; Bryck & Fisher, 2012). While this line of research is still its infancy, it clearly warrants additional attention given the results of the current study.

Although the current study is an important step in understanding inhibitory control and the underlying neural activity in a maltreated population, it also raises a number of critical questions for future research studies. For example, because inhibitory control continues to develop into emerging adulthood and the adolescents were assessed in early adolescence, it is not possible to determine whether the observed group differences in behavioral and electrophysiological indices of inhibitory control represent a delay (i.e., maturational lag that results in the same end state) or a deficit (i.e., persistent quantitatively or qualitatively different end state) in inhibitory control among the maltreated adolescents. Therefore, future research studies should assess inhibitory control in maltreated populations over time into (at least) emerging adulthood. Future studies also should examine the impact of specific child maltreatment experiences (e.g., type, severity, and developmental timing of child maltreatment) on behavioral and electrophysiological indices of inhibitory control. Difficulties accessing complete child welfare system records resulted in a lack of

https://doi.org/10.1017/S0954579420001819 Published online by Cambridge University Press

information about the adolescents' child maltreatment histories in the current study. However, this information is critical to understanding whether different early adverse experiences have differential effects on inhibitory control and the underlying neural regions. In addition, it is imperative to examine the associations between inhibitory control and the underlying neural activity and subsequent negative outcomes in a maltreated population. Because the adolescents in the current study were assessed into late adolescence, future analyses will investigate the relations between the behavioral and electrophysiological indices of inhibitory control assessed in early adolescence and early-onset alcohol and substance use assessed in late adolescence.

In summary, the results of the current study provide additional evidence that early adverse experiences negatively affect inhibitory control and the underlying neural activity. Specifically, compared to the nonmaltreated adolescents, the maltreated adolescents displayed poorer behavioral performance and atypical electrophysical performance on the trials that require inhibitory control. Given the purported role of inhibitory control in the development of a number of important outcomes, the impairment in inhibitory control observed among the maltreated adolescents may have a profound impact on their functioning in late adolescence and beyond. Although promising, the current study highlights the need for future research with maltreated populations. In addition to replicating the results, it will be critical to determine whether alterations in inhibitory control and the underlying neural activity increase the risk of academic difficulties, attention and behavior problems, and alcohol and substance use in maltreated children and adolescents. Furthermore, it will be important to examine the plasticity (i.e., potential for recovery) of inhibitory control and the underlying neural regions following exposure to early adverse experiences.

Acknowledgments. The authors thank Larisa Lilles for project management and the adolescents and parents for their participation.

Funding Statement. Support for this research was provided by the following grant: AA021973, NIAAA, U.S. PHS.

Conflicts of Interest. None.

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