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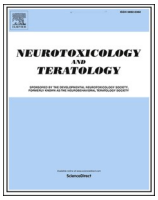

Perinatal nicotine exposure relates to stimulus-locked event-related potentials in early adolescence during an emotional go/no-go task

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ABSTRACT

Nicotine exposure is associated with negative consequences on the developing brain, both in utero and after birth. We investigated the relationship between perinatal nicotine exposure and electroencephalographic brain activity recorded during an emotional faces Go/No-Go task among adolescents. Seventy-one adolescents aged 12–15 years completed a Go/No-Go task using fearful and happy faces. Parents completed questionnaire measures of their child's temperament and self-regulation and retrospectively reported on nicotine exposure during the perinatal period. Perinatally exposed children ($n = 20$) showed increased and prolonged frontal event-related potential (ERP) differentiation in stimulus-locked analyses; that is, greater emotion and condition differentiation in comparison with their non-exposed peers ($n = 51$). However, non-exposed children showed greater late emotion differentiation recorded over posterior sites. Response-locked ERP differences were not found. ERP effects were not related to temperamental, self-regulatory, or parental education and income-related factors. This study is the first to demonstrate a relationship between perinatal nicotine exposure and ERPs in an emotional Go/No-Go task among adolescents. Findings suggest that while conflict detection remains intact for adolescents with perinatal nicotine exposure, their attentional allocation to behaviourally relevant stimuli may be magnified to beyond optimal levels, particularly when emotion is salient in information processing. Future studies can extend these findings by isolating prenatal nicotine exposure and comparing its effects to isolated postnatal exposure and clarifying the implications of the face and performance processing differences in adolescence.

Despite increased messaging around the dangers of smoking while pregnant, maternal tobacco use during pregnancy is still common (Drake et al., 2018), and its use is not without consequences for the developing brain—both in utero and after birth (England et al., 2017; Fischer and Kraemer, 2017; Knopik et al., 2012). Nicotine easily crosses the placental barrier and foetal concentrations are 15% higher than maternal concentrations (Lambers and Clark, 1996). Consequences for the foetus and/or infant include hypoxia (i.e. oxygen deprivation during labour and/or delivery), decreased cerebral oxygen saturation, decreased uterine blood flow, ischaemia (i.e. reduced blood flow), changes in myelination (i.e. neuronal insulation), reduced DNA synthesis, differential genomic methylation (i.e. addition of methyl molecules to histones reducing DNA transcription and translation), and altered neurotransmitter function (Ekblad et al., 2014; Ernst et al., 2001; Lee et al., 2015).

Prenatal exposure is associated with developmental changes to the nucleus accumbens (NAcc) and the prefrontal cortex (PFC; Brown and Kolb, 2001; Muhammad et al., 2012). Numerous studies have demonstrated changes to dendritic length, branching, density, and total number of spines in these areas. As a consequence of nicotine exposure, total spine and dendritic branching are increased in the NAcc, and there is increased dendritic complexity in both the NAcc and the PFC (Hamilton and Kolb, 2005; Muhammad et al., 2012), brain regions with strong ties to neurodevelopmental outcomes and individual differences in cognition and mental health (Kolk and Rakic, 2022). The prenatally affected dendritic complexity resembles the morphology of an adult brain resulting from chronic nicotine exposure in adulthood (Muhammad et al., 2012). Brown and Kolb (2001) also observed a 35% increase to dendritic length in the NAcc and PFC and increases in activity levels when rats were exposed to nicotine postnatally. Therefore, both pre- and

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postnatal exposure influence dendritic morphology in the NAcc and PFC. The NAcc, PFC, and occipital cortex are dopamine rich regions (Benes, 2015; Berger et al., 1988), and nicotine exposure is associated with changes to dopamine DRD4 receptors (Laucht et al., 2005) and serotonin transporter and receptor expression (Muneoka et al., 2001; Slotkin et al., 2006). Therefore, it is likely that perinatal nicotine exposure will have functional consequences (in addition to the structural consequences just described) for the activation of brain networks with high levels of dopaminergic innervation.

The neurophysiological consequences of early nicotine exposure set in motion during the perinatal period continue through adolescence. The orbitofrontal, medial frontal, and parahippocampal cortex are all thinner in adolescents prenatally exposed to cigarette smoking in comparison to their unexposed peers (Toro et al., 2008), perhaps because of disruptions to anterior cortical white matter microstructure (Jacobsen et al., 2007). This suggests that electrocortical activity originating from these regions might be disrupted. Prenatally exposed rats show reduced firing rates in the medial PFC and higher rates of impulsivity than controls (Bryden et al., 2016). Thus, exposure has lasting functional effects.

These physiological effects have later cognitive, behavioural, and emotional consequences and have been demonstrated in both cross-sectional and longitudinal research. Children born to heavy smokers show poorer processing of auditory stimuli, attention, visuospatial memory, and language comprehension than children born to non-smokers, even when adjusting or equating groups for relevant covariates (e.g. parental age at delivery, parental education; Fried et al., 1992; Jacobsen et al., 2007). Müller et al. (2013) suggest that some behavioural consequences of prenatal nicotine exposure may not be apparent until later in life, including during adolescence. Exposure is associated with a cluster of behaviours related to externalising and tied to the structure and/or function of the NAcc and PFC—higher rates of attention-deficit/hyperactivity disorder (ADHD) symptoms and deficits in response inhibition (Huijbregts et al., 2008; Schneider et al., 2011)—although, these response inhibition deficits are inconsistently found in performance on a Go/No-Go task (e.g. Dinn et al., 2004; Luijten et al., 2011). Prenatally exposed adolescents show higher levels of extravagance (one dimension of novelty seeking), higher levels of neuroticism, higher levels of impulsivity, lower levels of agreeableness (Müller et al., 2013), higher levels of externalising behaviour, and greater drug use (Lotfipour et al., 2014) than their non-exposed peers. They continue to be at higher risk than non-exposed peers for anxiety into young adulthood (Duko et al., 2022).

Behavioural differences are also observed in children exposed to smoke postnatally, even when excluding those who were prenatally exposed. Such postnatally exposed children show higher levels of hyperactivity and conduct problems while controlling for relevant confounding variables (socio-economic status [SES], parent health, parent intelligence, and obstetric complications; Gatzke-Kopp et al., 2020), poorer neurocognitive performance (Chen et al., 2013), and higher levels of autistic-like behaviours (Yang et al., 2022) relative to their non-postnatally exposed peers. This suggests that both pre and postnatal nicotine exposure are important for developmental outcomes.

Additional functional evidence for the role of prenatal nicotine exposure and substance use in PFC comes from electroencephalographic (EEG) studies, which offer the advantage of high temporal resolution. EEG is a promising, non-invasive method that can be used to assess cortical functioning (Luck, 2005). Poorer response inhibition is associated with reduced activity in the PFC (Casey et al., 1997), is more likely following nicotine exposure versus non-exposure (Huijbregts et al., 2008), and can be assessed by examining the stimulus-locked N2 and P3a event-related potential (ERP) components to stimuli that do versus do not require the inhibition of a prepotent (predominant or likely) response (e.g. a Go/No-Go task). In such a task, N2 amplitude is associated with detection of conflict and response inhibition (Gehring et al., 1993) and is maximal between 200 and 250 milliseconds (ms) post stimulus over frontal electrodes to infrequent stimuli requiring the inhibition of a prepotent response (Falkenstein, 2006). The P3a occurs between 300 and 500 ms over frontal electrodes when there is a change in the presented stimulus that requires attention and is larger for behaviourally relevant stimuli; for example, No-Go stimuli (Falkenstein et al., 1999). The N2 and P3a are associated with PFC activation (Casey et al., 1997) and risk for externalising symptoms (Hoyniak & Peterson, 2019; Venables et al., 2018). Notably, adult smokers show poorer performance on an emotionally salient Go/No-Go task and reduced No-Go N2 but not P3a amplitudes (Luitjen et al., 2011; see also, Dinn et al., 2004).

Additionally, posteriorally measured P3b amplitudes, which generally occur slightly after the P3a, are associated with increased attention and memory processing (Polich, 2007). The investigation of P3b amplitudes related to prenatal cocaine exposure (Crowley et al., 2009; Morie et al., 2018), substance abuse disorder (Euser et al., 2013), as well as their association to various externalising disorders (Iacono et al., 2003) have provided important insight towards understanding effects. For instance, less positive P3b amplitudes in behavioural disinhibition tasks (e.g. visual oddball and risk-taking tasks) are related to higher externalising symptoms (Iacono et al., 2003); risk for substance dependency (i.e. alcohol, nicotine, and/or illicit drugs; Euser et al., 2013); as well as disorders including ADHD, conduct disorder, and oppositional defiance disorder. Conversely, more positive P3b amplitudes are related to lower externalising symptoms and no substance dependency (Iacono et al., 2003).

Furthermore, the late-positive-potential (LPP) shares underlying mechanisms of the P300 (Hajcak and Foti, 2020), and is a slow-positive going and broadly distributed waveform observed after the presentation of emotional stimuli and has been associated with both state (e.g. subjective emotional arousal; Hajcak and Nieuwenhuis, 2006) and trait (e.g. anxiety; Kujawa et al., 2015; MacNamara and Hajcak, 2010; Moser et al., 2008; Mühlberger et al., 2009) measures including callousness and impaired recognition of fearful faces (Brislin and Patrick, 2019). Cocaine users show larger (more positive) LPPs to cocaine-related stimuli but smaller LPPs to pleasant images (e.g. smiling faces, nudes) than do controls (Dunning et al., 2011). Thus, the N2, P3a, P3b, and LPP components are all correlated with individual differences in a cluster of externalising behaviours, and several of these ERP components have been shown to be altered with substance exposure, making them important candidates for the present investigation of the correlates of perinatal nicotine exposure.

ERPs can also be measured to internally generated responses with significant associations to individual-difference factors related to the present investigation. Response-locked ERPs include the error-related negativity (ERN), which occurs when an individual makes an unintentional incorrect behavioural response (Stemmer et al., 2001; Ellis and Rothbart, 2001). The ERN reaches maximum negativity within 100 ms of the error (Blackwood and Muir, 1990; Gehring et al., 1993), is generated by mPFC structures such as the anterior cingulate (Awh and Gehring, 1999; Brázdil et al., 2005; van Noordt et al., 2015), and correlates with developmental outcomes such as behavioural control (Yeung et al., 2004) and self-regulation (Checa et al., 2014). High self-regulatory ability is associated with a more negative ERN amplitude in response to errors, while low self-regulatory ability is associated with a less negative ERN (Checa et al., 2014). Cocaine users, relative to controls, show less negative ERNs recorded during an emotionally neutral Go/No-Go task (Morie et al., 2014). A second response-locked ERP is the correct-response negativity (CRN) a less negative peak that occurs around the same latency as the ERN observed after correct responses. The CRN has comparable morphological and topographical properties to the ERN when isolated from other ongoing activity via surface Laplacian (Vidal et al., 2000).

The Pe, or error positivity, is maximal 200–400 ms over central electrodes after an incorrect response and is believed to represent the conscious recognition, or motivational salience, of making an error

(Falkenstein et al., 2000), and may also be altered as a function of nicotine exposure (see e.g. Fig. 1 of Potts et al., 2014) and cocaine addiction (Morie et al., 2014). The Pe is associated with response accuracy on an emotionally neutral continuous performance task (Thurm et al., 2020). The ERN, CRN, and Pe are all associated with individual differences in cognition and emotion, which may relate to substance exposure, and all three are modulated by emotional stimulus presentation (Simon-Thomas and Knight, 2005; Wiswede et al., 2009), underscoring the importance of examining electrophysiological correlates of substance exposure in emotionally laden tasks.

Boucher et al. (2014) were the first to capitalise on the unique strengths of EEG to assess the effect of *prenatal* nicotine exposure on response inhibition. In Boucher and colleagues' seminal study, nicotine exposure was associated with school-aged participants' reductions in stimulus-locked N2 and P3b amplitudes while completing an emotionally neutral Go/No-Go task (using letters), suggesting impairments to conflict processing. It may take more effort for a child with nicotine exposure to inhibit a prepotent response relative to someone without such exposure (Boucher et al., 2014). Moreover, it was suggested that prenatal nicotine exposure impacts PFC development, thereby leading to impairments in response inhibition that could lead to externalising behaviours (e.g. impulsivity and inattention). Greater impairments may be observed beyond school-age years, given the growth of the frontal cortex (Giedd and Rapoport, 2010).

Boucher et al. (2014) did not find that prenatal exposure was related to response-locked ERPs (ERN/CRN or Pe), only to stimulus-locked ERPs (Boucher et al., 2014). However, ERP overlays suggest a trend towards less negative ERN in exposed children (Boucher et al., 2014) and reduced punishment-motivated ERNs are observed in adult smokers relative to their non-smoking peers (Potts et al., 2014). Furthermore, it is possible that response-locked effects were not observed as Boucher et al.'s (2014) emotionally neutral task may not have adequately influenced ACC activity (Eimer and Holmes, 2007) related to differences in exposure. The ACC and PFC (among other brain regions) are associated with the conscious representation of emotional facial expressions to support the strategic control of thoughts and actions (Eimer and Holmes, 2007); thus, ACC involvement in Go/No-Go tasks may be amplified when emotional faces are used. We investigated both stimulus- and response-locked ERP associations in adolescence given the suggestive trend in Boucher and colleagues' study together with the results of Potts et al. (2014), the continued brain and ERP development beyond the school-aged sample of Boucher et al. (2014) and into young adulthood (i.e. growth and maturation of the PFC and anterior cingulate cortex; Segalowitz and Dywan, 2009), and the possibility that observed effects may vary when emotional stimuli are used (e.g. Simon-Thomas and Knight, 2005; Wiswede et al., 2009).

Boucher and colleagues assessed marijuana and alcohol use during pregnancy and retained participants with such exposures. They adjusted for children's age, sex, birth weight, education of the primary caregiver, and maternal non-verbal reasoning abilities in their analyses of covariance (Boucher et al., 2014). However, Boucher et al. (2014) did not control for temperamental qualities of the children themselves, an important consideration, especially when emotional faces are used as is the case in this study. ERP associations with perinatal exposure may differ when emotional stimuli are used given the temperamental correlates of exposure discussed earlier and literature linking temperamental differences to early posterior ERP differences in tasks involving emotional faces (Jetha et al., 2012), late LPP associations with callousness (Brislin and Patrick, 2019), and late frontal and posterior (P3a and P3b) ERP associations with all Big 5 personality traits, including extraversion (Gurrera et al., 2001). Thus, measuring ERPs within an emotional context may advance our understanding of how perinatal nicotine exposure relates to cognitive deficits that extend to the psychopathology literature.

The purpose of the present study was to assess perinatal nicotine exposure and its relationship with stimulus and response-locked ERPs recorded over frontal and posterior sites during an emotional faces Go/No-Go task while controlling for important confounding variables. As our demographic variables, personality measures, and data concerning perinatal nicotine exposure were collected in an early phase of a large population study before the publication of Boucher et al. (2014), our measures do not completely cover the ones examined by Boucher et al. (2014) nor are limited to them. The EEG phase of the study was conducted on a follow-up subsample of the original large cohort. Given the documented difficulties with emotion-regulation and/or temperamental profiles in exposed children (Müller et al., 2013; Yang et al., 2022), it may be that nicotine exposure associations with electrophysiology are especially apparent when emotional stimuli are used and may depend on temperamental qualities of the child known to affect ERPs (Brislin and Patrick, 2019; Gurerra et al., 2001; Jetha et al., 2012). One such temperamental quality, behavioural inhibition—a construct related to shyness and extraversion—has been variously linked to both increased and decreased performance on response inhibition tasks (see Henderson and Wilson, 2017 for a review). Individuals higher on extraversion tend to perform better than their low-extraversion peers when task difficulty is high (Campbell et al., 2011). A second temperamental or individual-difference factor related to the present investigation is self-regulation. Self-regulation is positively correlated with performance on response inhibition tasks (Hofmann et al., 2012) and correlates with ERPs recorded during an auditory selective attention task requiring inhibition of the to-be-ignored ear (Lackner et al., 2013). Thus, these child-level variables are important covariates and will allow for greater specificity of the nature of the perinatal nicotine exposure relationship with electrophysiology.

Furthermore, low SES (a multi-faceted construct including among its components parental education and annual income) in childhood is related to reduced attentional flexibility (Conjero & Rueda, 2018) and reduced PFC activity (Kim et al., 2013), necessitating its inclusion in the

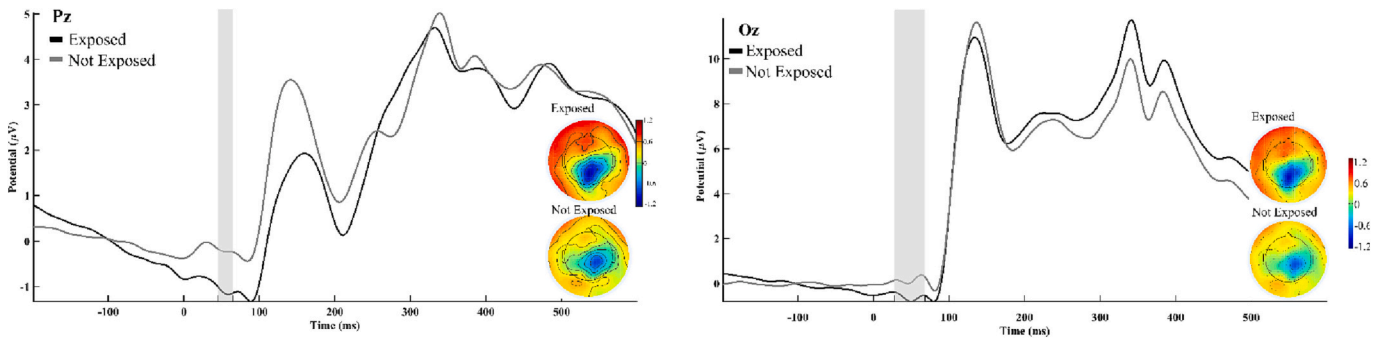

Fig. 1. Main Effect of Group on Stimulus-locked ERPs Recorded from Pz and Oz.
Note. Light grey horizontal bar represents a period of significant differences between groups that lasts for >20 ms. Topographies on the right show averaged electrical activity during time windows where group differences were observed. ERPs = event-related potentials.

present analyses. Therefore, we adjust for shyness and social affiliation (a construct related to extraversion; e.g. Tov & Koh, 2014) in addition to self-regulatory skill and socio-economic variables and look for group differences in frontal and posterior stimulus- and response-locked ERPs. We could not control for parental non-verbal reasoning abilities as in Boucher et al. (2014) as we did not collect these data.

We hypothesised that we would see differences in both stimulus- and response-locked ERPs in perinatally exposed adolescents compared to their non-exposed peers. Following Boucher et al. (2014), we expected reduced amplitudes during the timing of the stimulus-locked N2 and P3b amplitudes in addition to less negative amplitudes during the ERN and less positive amplitudes during the Pe in perinatally exposed adolescents. We expected to observe response reductions during the P3a and P3b/LPP timing in exposed participants since this pattern has been found in individuals high in externalising symptoms (e.g. Hoyniak & Peterson, 2019; Iacono et al., 2003; Venables et al., 2018).

1. Method

1.1. Participants

One-hundred four early adolescents from a predominately White community took part in a two-part study at Brock University in Ontario, Canada (Time 1, age range 10–13 years, $M_{ageT1} = 11.5$ years; Time 2 age range 12–15 years; $M_{ageT2} = 13$ years). We excluded 1 participant because of prenatal alcohol exposure, which is associated with neurodevelopmental and electrophysiological consequences (Coles and Li, 2011); 16 participants owing to performance issues or ability to engage: two for having hearing difficulties that interfered with understanding task instructions; five for withdrawing from the study owing to boredom or fatigue; two for failing to follow task instructions; seven for having poor quality data yielding fewer than 10 errors per condition after preprocessing; and 16 participants for technical difficulties saving or storing the EEG file (e.g. a server crash corrupted files). The final sample was 71 participants (37 girls and 34 boys). Birth weights ($n = 69$; not available for two participants) ranged between 1.68 and 4.46 kg. Five participants were non-singleton births, and six participants were officially underweight at birth (< 2.5 kg). Annual income ranged between $2500 ($n = 2$) and $110,000 in 2006 CAD dollars. Sample characteristics are shown in Table 1.

2. Materials and tasks

2.1. Demographics

Participants were asked to self-report their age and sex. Parents were asked to report their total household income in the previous year (2006) and to report on the highest level of schooling achieved (from 1 = 'less than grade 6' to 12 = 'graduate or professional training [graduate degree]'). These two variables served as a proxy for SES.

2.2. Smoking/alcohol index

Parents were also asked to complete a questionnaire regarding their smoking habits. We asked, 'Did the mother smoke regularly (one or more cigarettes a day) in the year before pregnancy'? If the response was 'yes', we asked, 'Did the mother stop smoking when she learned she was pregnant'? (responses were 'no'; 'yes, right away'; 'within one month'; 'within two months'; and 'after three or more months'). We asked, 'Did the mother smoke within the first year after giving birth to this child' with the same response options as above and if anyone smoked inside the home or car every day or almost every day ('yes' or 'no'). Similar to the yes–no smoking question, we asked, 'Did the mother drink alcoholic beverages regularly (one or more drinks per day) in the year before pregnancy'? If the response was 'yes', the same question and response categories as for smoking were asked to identify if and when the mother stopped drinking during pregnancy. These questions were developed for this study as an indirect way of asking about smoking and alcohol during pregnancy. If the mother smoked or drank in the year prior to pregnancy, then the child will have been exposed at least until the mother learned she was pregnant. As such, this manner of questioning was intended to reduce any social desirability response bias of simply being asked—'Did you smoke (or drink alcohol) while pregnant'?—which is more likely to elicit false negative responses. We also asked, 'Did the mother smoke within the first year after giving birth to this child'? (responses were 'no'; 'yes, right away'; 'within one month'; 'within two

Table 1
Demographic and Covariate Data as a Function of Exposure Group.

	n	% of total participants	Perinatal exposure *n* or *M (SD)*	No exposure *n* or *M (SD)*	Total *M (SD)*
Nicotine exposure					
Postnatal	8	11.30			
Exposed to second hand smoke in house and/or car					
Yes	8				
No	0				
Pre- and postnatal	12	16.90			
Stopped smoking when found out pregnant					
Never	7				
Yes, right away	2				
Within 1 month	1				
After 3 or more months	2				
Exposed to second hand smoke in house and/or car					
Yes	12				
No	0				
No exposure	51	71.80			
Type of birth					
Singleton	66	93.0	1	65	
Multiple*	5	7.0	4	1	
Sex					
Male	34	47.90	11	26	
Female	37	52.10	10	24	
Age at EEG			13.30 (0.66)	13.04 (0.80)	13.09 (0.79)
Income			74,875 (36359)	79,656 (27409)	78,309 (30002)
Highest parental education			10.5 (1.10)	10.58 (1.05)	10.56 (1.06)
Birth weight in kg*			2.98 (0.73)	3.52 (0.50)	3.36 (0.62)
BRIEF BRI			42.42 (9.75)	39.36 (9.73)	40.20 (9.77)
BRIEF MI			75.56 (20.01)	71.58 (18.95)	72.68 (19.18)
Temperament measures					
Shyness			12.42 (4.66)	11.18 (4.35)	11.53 (4.44)
Social affiliation			23.05 (3.47)	23.14 (3.48)	23.12 (3.45)

Notes. Numbers (*ns*) are provided for categorical variables, while mean (*M*) and standard deviation (*SD*) are presented for continuous variables.
EEG = electroencephalographic. BRI = Behavior Regulation Index, MI = Metacognition Index.
Postnatal and pre- and postnatal groups are collapsed into a single perinatal exposure group (in columns) to mirror EEG analyses.
* indicates significant group differences as reported in text.

months'; and 'after three or more months' to assess exposure postnatally.

2.3. Birth weight

Parents were asked for their child's birth weight (in lbs. or kg, and subsequently converted to kg), and whether the child was a singleton or multiple birth.

2.4. Early Adolescent Temperament Questionnaire–Revised Parent Report (EATQ-R-PR)

Parents were asked to respond to 62 prompts related to how their child behaves (Ellis and Rothbart, 2001; e.g. 'Feels shy about meeting new people', 'Wants to have close relationships with other people'), rating the typicality of the behaviour from 1 = 'almost always untrue' to 5 = 'almost always true'. No reference timeframe is given, instead, parents are asked to 'circle the answer which best describes how true each statement is for your child'. These 62 questions combine to make 10 subscales, of which the shyness (sum of five questions) and social affiliation (sum of six questions) subscales were used presently for their hypothesised associations with emotional stimulus processing. There were no missing data for this questionnaire. The EATQ-R-PR showed moderate to good internal consistency in previous (Ellis and Rothbart, 2001) and the present (shyness $\alpha = 0.87$, social affiliation $\alpha = 0.69$) research.

2.5. Behavior Rating Inventory of Executive Function (BRIEF)

The parent-report form of the BRIEF (Psychological Assessment Resources, Inc.; Gioia et al., 2000) is an 86-item measure in which parents are asked to evaluate their child's daily behaviour over the past month within eight domains of self-regulation. They are asked to report if any of the behaviours addressed in the 86 items have 'never', 'sometimes', or 'often' been a problem for their child. Fourteen questions form the parental inconsistency and parental negativity scales, which comprise the BRIEF validity checks. The inconsistency scale is a metric of contradictory or unusual responding (e.g. giving two different responses to highly similar questions). If the parent has an unusually negative response pattern, this will be reflected in the negativity scale. Negativity scores of 0–5 and inconsistency scores of 0–7 are considered acceptable (versus elevated). All participants' scores on these measures were considered acceptable (Inconsistency: $M = 2.49$, $SD = 1.95$; Negativity: $M = 0.29$, $SD = 0.90$). This study used the Behavior Rating Index (BRI) and the Metacognition Index (MI). The BRI (28 items) represents the child's ability to utilize appropriate inhibitory control in the service of shifting cognitive set, modulating emotions, and modulating behaviour. The MI (44 items) represents the child's proficiency at self-managing tasks and self-monitoring. On both scales, higher scores indicate a poorer ability to self-regulate. The scale shows acceptable to excellent internal consistency in previous (αs = 0.80–0.98; Gioia et al., 2000) and the present (both $\alpha = 0.88$) research. Mean interpolation was used for missing data.

2.6. Go/No-Go Task

Participants were seated in front of a computer monitor with a response keypad on a table in front of them. The task was completed as one practice block followed by four blocks of trials separated by short breaks. Each block lasted approximately three minutes and each break was one to three minutes in length (the participant was free to decide when they were ready to resume the task). In two blocks, they were instructed to press the button as quickly and as accurately as possible to happy faces (Go trials) and withhold response for fearful faces (No-Go trials). In two other blocks, they responded to fearful faces while withholding responses to happy faces. Block order was counterbalanced across participants (either Go happy, Go fear, Go happy, Go fear or Go fear, Go happy, Go fear, Go happy). Each block began with a slide reminding participants to respond to fearful or happy faces only as appropriate, instructions which were on the screen and read aloud by the experimenter who entered the EEG chamber during breaks.

Stimuli were comprised of 80 greyscale portrait-style images from the NimStim Set of Facial Expressions (Tottenham et al., 2009) of 20 different identities (10 men and 10 women). For each identity, two photographs were fearful, two were happy, and one of each of the happy and fearful faces were mouth-open versus mouth-closed expressions. Stimuli are available from https://macbrain.org/resources/ and have been well validated in previous studies; for example, high concordance rates between intended expression and labelled expression, high levels of test-retest reliability in adult samples (Tottenham et al., 2009), and high accuracy rates in the congruent condition of an emotional faces Stroop task in an adolescent sample (Bouhours et al., 2021). The stimuli were presented for 500 ms at the centre of the screen, followed by the appearance of fixation cross, and participants had 1200–1400 ms to respond after onset. Twenty practice trials were provided, and the experimenter validated response patterns by watching the EEG response markers appear in the recording software (BrainVision Recorder; Montreal, Quebec, Canada). Altogether, there were 480 Go trials and 160 No-Go trials, each trial type spread equally across the four blocks. Average reaction time (RT; i.e. time between onset of stimulus and button press) as well as accuracy (i.e. number correct/incorrect and percentage correct) were recorded.

3. Procedure

The data presented here are from the first phase of an ongoing longitudinal study at Brock University, Ontario, Canada. Participants and their parents were provided with the questionnaire measures detailed above to complete at home. Both participants and parents provided written consent and verbal assent as required. After completion, participants were scheduled for a three-hour EEG session at Brock University, during which they completed the Go/No-Go task along with several other measures not part of the current investigation

3.1. EEG

EEG was recorded at 121 scalp sites[1] using a Hydrocel Geodesic Sensor net (EGI, Eugene, OR) with a sampling rate of 500 Hz and 0.1–100 Hz analogue filtering. All impedances were below 50 kΩ at the outset of the recording session and were re-checked between EEG tasks. Data were re-referenced offline to the average of all sites and then pruned to exclude any time where the participant was off task (e.g. during breaks or practice periods) and excessively noisy channels (i.e. those channels whose ongoing amplitudes were aberrant from the rest for at least 80% of the recording). Data were then filtered from 1 to 30 Hz and subjected to an independent components (IC) analysis using EEGlab (AMICA; Delorme and Makeig, 2004), and ICs representing eye movement, heartbeat, or other motor activity were removed with the help of an EEGlab native plug-in (IClabel, which uses crowdsourced IC labels from EEG researchers then aggregated via latent Dirichlet allocation; Pion-Tonachini et al., 2017), although all final decisions for component removal were made by trained researchers (BT, co-author, following the guidelines laid out in labelling.ucsd.edu/tutorial/labels). After component removal, data were referenced to the average of all sites and segmented from -400 to 600 ms (response locked) or -200 to 600 ms (stimulus locked). For stimulus-locked segments, a baseline correction of -200 to 0 was applied. For response-locked segments a baseline correction of -400 to -200 ms was applied. Analyses focused on

[1] Of the 128 channels available, seven were used for physiological records (heart rate, respiration, etc.) not reported here, leaving 121 for EEG recordings.

Cz for both stimulus- and response-locked segments, as well as Pz and Oz for stimulus-locked segments.

3.2. Statistical analysis

We separated participants into two groups based on nicotine exposure: perinatal exposure (PNE, $n = 20$; 8 with only postnatal exposure and 12 with both pre- and postnatal) and control groups ($n = 51$) and conducted a series of analyses of variance (ANOVAs) to compare group's scores on questionnaire and behavioural measures (RT and accuracy).

For our EEG analysis, we checked for significant group, emotion, and condition differences across the time course including the ERN/CRN and Pe (response-locked), and N1, P2, N2 and LPP/P3 (stimulus-locked) at Cz. Similarly, at Pz and Oz we looked for significant group, emotion, and condition differences across the time course, including the timing of the P3b and LPP. Robust data-driven statistical approaches are commonplace when analysing MEG, PET, and fMRI data using programmes such as SPM (Friston, 2003), FSL (Analysis Group, FMRIB, Oxford), FieldTrip (Oostenveld et al., 2011), BrainStorm (Tadel et al., 2011), and BrainVoyager (Brain Innovations, Netherlands; Pernet et al., 2011), and are increasingly utilised in ERP research (e.g. Fields and Kuperberg, 2020; Hafer et al., 2022; van Noordt et al., 2015). Traditional hypothesis testing techniques, such as the Student's *t*-test and Pearson's r, which assess sample means and variances, are vulnerable to even small deviations from normality, which lead to higher error rates and lower statistical power (Wilcox, 2012). Traditional ERP measurements, which are by definition dependent on averaged responses to the stimuli of interest at both the subject- and group-level, are particularly limited by this constraint (Luck, 2005). Robust estimation techniques that are not constrained by assumptions regarding distribution characteristics boost both statistical power and accuracy have been developed to overcome these shortcomings of conventional hypothesis testing (e.g. narrower confidence intervals, lower Type II error rates; Wilcox, 2012).

We used the LIMO plug-in (LIMO Team et al., 2016; Pernet et al., 2011) for EEGLAB (Delorme and Makeig, 2004) to compute point-by-point differences in conditions (Go vs. No-Go and Happy vs. Fear) at the single-participant level using an ordinary least-squares design (first-level analysis). Group analyses (second level analysis) integrate parameters from the first-level analysis across participants to complete the hierarchical modelling of the data. In this second level, we utilised mixed-model ANOVAs to examine group (between subjects) and condition (within subjects) differences in ERPs, and robust regression analyses predicting beta weights by potential covariates (e.g. temperament and self-regulation variables and income). In the mixed-model ANOVAs, sphericity is accounted for using Hotelling T^2 for repeated factors (e.g. condition) and Hotelling generalised T^2 for within-by-between interactions (e.g. condition × group), which are both then transformed into *F*-values. The *F* distribution is estimated by randomly sampling participants with replacement one-thousand times from each cell of the ANOVA table, and utilises 20% trimmed means, Winsorised variances, and always assumes variance inhomogeneity (LIMO Team et al., 2016; Pernet et al., 2011).

In the robust regression analyses, we tested for inter-subject variability of observed group-level effects by first sampling with replacement *n* electrode × time frame ERP matrices. Regression beta weights were computed for each bootstrap and then sorted from highest to lowest; from this distribution, a 95% confidence interval was derived. If the confidence interval does not include zero, the regression coefficient at that time point is considered significant. For the ANOVAs and regression analyses, times of significance were calculated at each data point at the $p < .05$ level, with windows of time considered significant if they last for 20 ms (approx. 10 data points) or more consecutively, one way that we reduced Type I error to very low levels.[2] We additionally reduced Type I error by only focusing on three electrode sites where the ERPs of interest are of greatest amplitude in previous literature—Cz, Pz, and Oz—rather than running analyses across the whole electrode montage. We did not use the Fmax or cluster-based corrections implemented in LIMO as the Fmax correction is too conservative in general, and cluster-based statistics do not perform well when groups are small as they are in this study (Pernet et al., 2015). We refer the reader to Pernet et al. (2011) for additional information on the statistical approaches presently implemented.

4. Results

Independent samples *t*-tests showed that birth weights were lower for exposed relative to unexposed participants ($t(67) = 3.49$, $p < .001$). There were significantly more twin births in the exposed relative to no-exposure groups (Wald H_0 χ^2 test for categorical variables, $Z = -2.72$, $p < .01$). Additional independent samples *t*-tests showed that when twin births were excluded, a significant difference in birth weights remained ($t(63) = 2.37$, $p = .02$) and that income did not differ between the two groups ($t(69) = 0.60$, $p = .55$), even when the two very low income participants ($2500/year) were excluded ($t(67) = 0.43$, $p = .67$; see Table 1 for descriptive statistics). There were no group differences in temperamental or self-regulatory measures (all ps > .13).

An average parental education of 10 indicates 'partial college (at least one year)' while parental education of 11 indicates 'standard college or university (undergraduate degree or diploma)'.

4.1. Task performance

We ran two separate 2 × 2 × 2 mixed-model ANOVAs to examine group (between subjects), emotion (within subjects), and response (within subjects) differences in RT and accuracy. There was no main effect of group ($p = .89$), or emotion ($p = .17$) on RT, nor was there a significant group × emotion ($p = .10$), or group × response ($p = .83$), or group × emotion × response ($p = .78$) interaction on RT. There was a significant main effect of response type, with error responses happening faster than correct responses ($F(1, 69) = 37.06$, $p < .001$). See Table 2 for ranges, means, and SDs. A significant emotion × response interaction was also observed ($F(1, 69) = 4.32$, $p = .04$), and a follow-up paired *t*-test showed that the interaction was owing to faster error than correct responses to fearful faces ($t(70) = 5.30$, $p < .001$), with no response differences to happy faces.

For accuracy, we uncovered a significant main effect of emotion ($F(1,$

Table 2
Average Reaction Times (in ms) as a Function of Condition.

Condition	Range (min–max)	Mean	Standard deviation
Errors	226.85–540.51	373.71	71.73
Fear errors	218.80–608.36	369.84	82.03
Happy errors	197.94–584.37	376.83	80.81
Corrects	213.56–597.10	401.10	80.01
Fear corrects	197.47–597.54	412.84	86.19
Happy corrects	229.64–596.65	389.37	82.47

[2] The chance rate for any one data point to reach significance is a 5% error, and to have 10 adjacent points (assuming independent tests) is $.05^{10}$ ($= 9.8 \times 10^{-14}$). In the sequence of 400 EEG sampling points tested, there are 390 10-point sequences, so this $.05^{10}$ should be multiplied by 390 ($.05^{10} \times 390 = 3.81 \times 10^{-11}$), an extremely low error rate.

69) = 8.58, $p < .01$), with happy faces ($M = 0.71$, $SE = 0.012$) having higher accuracy than fearful faces ($M = 0.67$, $SE = 0.02$); a main effect of condition, with Go trials ($M = 0.88$, $SE = 0.02$) having higher accuracy than No-Go trials ($M = 0.50$, $SE = 0.02$), $F(1, 69) = 195.79$, $p < .001$); but no significant main effect of group ($p > .99$). There were no significant two- or three-way interactions on accuracy (all $ps > .56$).

4.2. Group differences in stimulus-locked ERPs

Using the statistical approach described in the Methods section, we looked for group, emotion, and condition differences in stimulus-locked ERP amplitudes at each data point at Cz, Pz, and Oz, and present overlays for effects involving group differences.

4.2.1. Main effects

At Cz, there was a main effect of condition during the timing of the P2 (170–195 ms) and LPP (471–600 ms) with more positive amplitudes to No-Go stimuli. There was no main effect of emotion or group. Moving posteriorly, at Pz and Oz we found prolonged condition differentiation with more negative amplitudes to No-Go than Go stimuli at both electrode sites through the P3b/LPP timing (Pz: 375–526 ms; Oz: 355–497 ms). There were differences in amplitude as a function of emotion, with larger amplitudes to fearful rather than happy stimuli at Pz during the timing of the P3b (457–600 ms) and occipital sites (Oz) during somewhat earlier stimulus processing (131–400 ms). At Pz and Oz, during early stimulus processing (45–65 ms and 28–68 ms, respectively), the exposed group showed more negative amplitudes (Fig. 1); as previously described, this early effect was not observed centrally.

4.2.2. Two-way interactions

At Cz, significant emotion × condition interaction emerged during the timing of the late N1 or early P2 (152–172 ms), with only fearful faces showing more positive Go relative to No-Go amplitudes during this time window. A group × condition interaction was uncovered during the timing of the LPP (451–600 ms), with exposed participants showing larger condition differentiation during this time window relative to their unexposed peers (Fig. 2). Lastly, a significant group × emotion interaction appeared during early stimulus processing (56–76 ms). Exposed participants showed slightly larger early ERPs to happy relative to fearful faces, while unexposed participants showed no differentiation (Fig. 3). More apparent from the topographical maps, though, is that the exposed group appear to be showing a greater region of negativity for especially the faces showing fear than the non-exposed group.

An analysis of the two-way interactions at Pz revealed a significant emotion × condition interaction during the early N2 and P3b/LPP timing (Pz: 150–175, 211–263, 413–519 ms), with condition differences observed only for fearful faces. There were no group × condition differences; however, a significant group × emotion interaction was revealed at both Pz and Oz (Fig. 3), with the no-exposure group showing more positive amplitudes during the P3b/LPP timing (Pz: 341–395, 460–600 ms; Oz: 339–376, 385–435, 552–575 ms) to fearful relative to happy faces, and the exposed group showing no significant emotion differentiation at these posterior sites.

To address the possible influence of confounding variables on the group × emotion interactions at Cz, Pz and Oz, we conducted multiple regression analyses, correlating income, education, social affiliation, shyness, BRIEF MI, and BRIEF BRI separately with the Fear – Happy ERP differences (represented as beta weights from the first-level analysis) at each significant time point. No associations were found. This same analysis strategy was used for the group × condition interaction at Cz and no significant associations were found.

4.2.3. Three-way interactions

Moreover, a significant three-way group × condition × emotion interaction appeared during the timing of the P1, N1, and LPP (7–193 and 494–522 ms) at Cz, with exposed participants showing larger emotion and condition differentiation during these time windows than unexposed participants. Follow-up two-way ANOVAs were conducted separately for each group and focused solely on periods of significance in the three-way ANOVA. This analysis revealed a significant emotion × condition interaction during the P1 through P2 timing (15–191 ms) and LPP timing (494–551 ms). Decomposing this interaction further, exposed participants showed significant condition differentiation during the early stimulus processing (62–88 ms), N1 and P2 timing (105–190 ms) as well as during the LPP timing (from 494–522 ms; see the solid (not hatched) waveforms in the exposed group's overlay in Fig. 4, panel A). Note that these intervals are a few ms different than those in the three-way interaction; however, this does not practically impact the interpretation of the results, as effects are still contained within the timing of particular ERP components. For processing happy faces, exposed participants showed significant condition differentiation during the very early period (31–55 ms; see the hatched lines in the exposed group's overlay in Fig. 4, panel A). The no-exposure group did not show such condition or emotion differentiation.

At Pz, but not Oz, we also uncovered a significant three-way group × condition × emotion interaction, lasting from 73–230 ms and 398–435 ms, which appeared to be the inverse in polarity to the interaction at Cz (Fig. 5). Follow-up tests were conducted in the same manner as for the three-way interaction at Cz. In the exposed participants, there was a two-way response × emotion interaction lasting from 101–177 ms, 205–226 ms, and 398–435 ms, whereby these participants showed significant pairwise amplitude differences between Fear Go and Fear No-Go stimuli (144–172 ms; 398–435 ms), Fear No-Go and Happy No-Go stimuli (398–435 ms), and Fear No-Go and Happy Go stimuli (398–435 ms).

To address the possible influence of confounding variables on these three-way interactions at Cz and Pz, we conducted multiple regression analyses, correlating income, education, social affiliation, shyness,

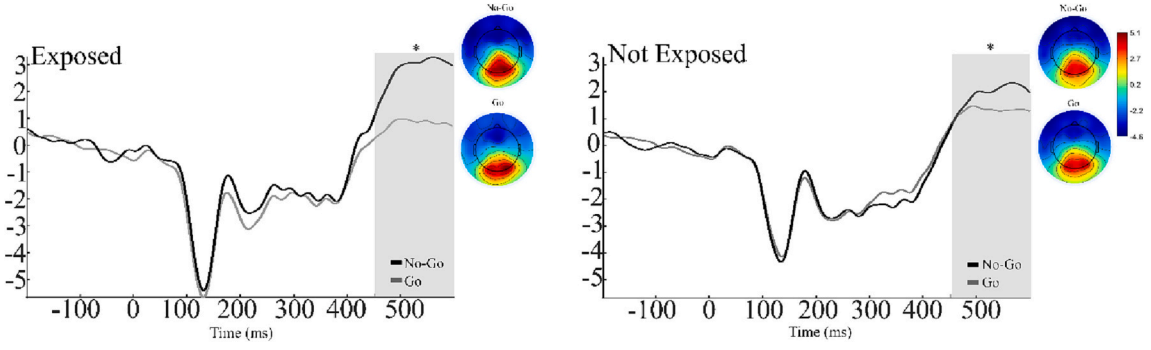

Fig. 2. Interactions of Group × Condition on Stimulus-locked ERPs Recorded from Cz.
Note. Light grey horizontal bars indicate periods of significant interactions and asterisks represent significant pairwise comparisons. Topographies on the right represent averaged electrical activity during times of significant group × condition interactions. ERPs = event-related potentials.

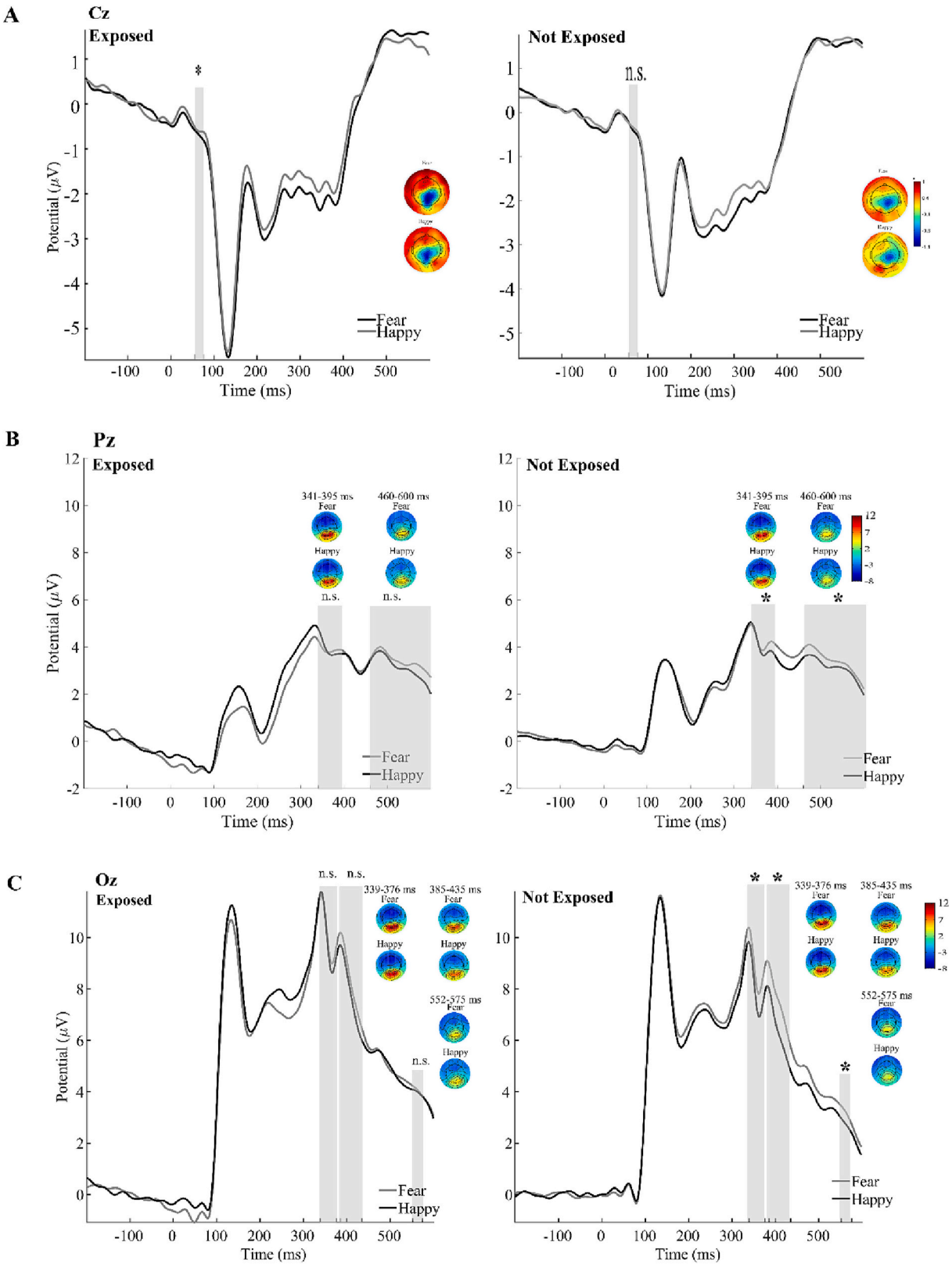

Fig. 3. Stimulus-locked Group × Emotion Interaction at Cz (row A), Pz (row B), and Oz (row C).
Note. Light grey horizontal bars represent periods of significant interactions and last for >20 ms and asterisks represent significant pairwise comparisons within those interactions. Topographies on the right of each overlay represent averaged electrical activity during times of significant group × emotion interactions.

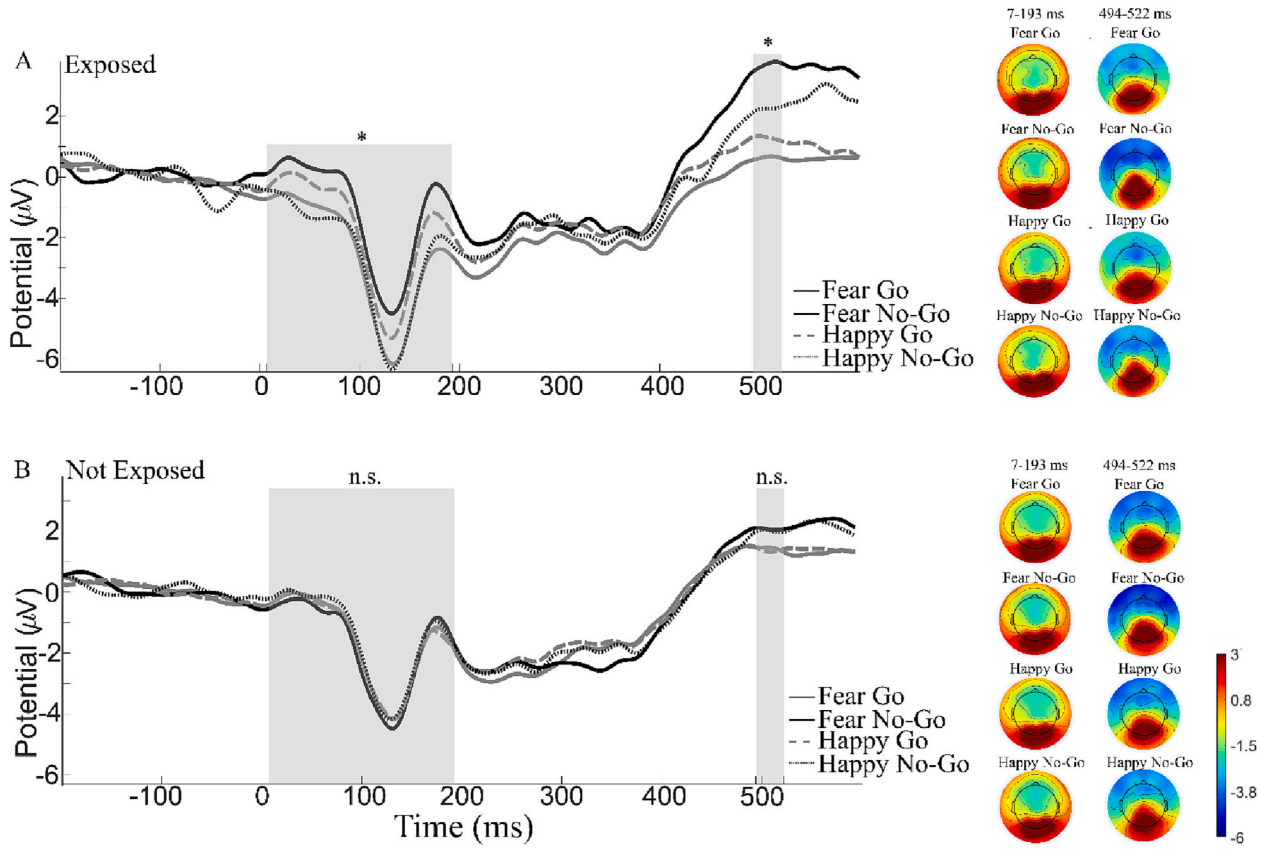

Fig. 4. Group × Condition × Emotion Interaction on Stimulus-locked ERPs Recorded from Cz.
Note. Light grey horizontal bars indicate periods of a significant three-way interaction, with asterisks representing times where follow-up tests revealed significant condition and/or emotion differences in the exposed group, and n.s. representing non-significant comparisons. Topographies on the right represent averaged electrical activity during times of significant three-way interactions. ERPs: event-related potentials.

BRIEF MI, and BRIEF BRI, separately with the emotion × condition beta weights from the first-level analysis at each significant time point for the three-way interactions. At Cz, BRIEF MI scores correlated with the emotion × condition effects from 494–515 ms; however, as noted earlier, the groups did not differ on MI, and re-running the analysis as an analysis of covariance, partialing out MI did not significantly change the results. No associations were found at Pz.

Removing six low-birthweight participants (< 2.5 kg) and removing four twins from the analyses also did not change the pattern of stimulus-locked results. Including the alcohol exposed participant also did not change the pattern of results. Please see Supplementary materials for a summary of stimulus-locked effects involving group.

4.3. Response-locked ERPs

We uncovered a main effect of response during the timing of the ERN (100–153 ms) and Pe (221–564 ms) at Cz, with more negative ERNs to error than correct responses, and more positive Pe amplitudes to errors, rather than correct responses, as expected. Amplitudes were larger to fearful rather than happy faces during two separate times during the Pe (246–314, 389–429 ms). There was no significant main effect of group and no significant two- or three-way interactions at Cz.

5. Discussion

We investigated the influence of perinatal nicotine exposure on task performance and ERPs recorded over frontal and posterior sites to emotional stimuli in adolescence. We hypothesised that we would see reductions particularly during the timing of the N2 and P3b ERPs. Additionally, we would see less negative amplitudes during the ERN in those who were exposed to nicotine and the later LPP recorded over frontal sites.

First, we ran two mixed-model ANOVAs to assess exposure effects on performance of the Go/No-Go task. In concert with the lack of behavioural differences in Boucher et al. (2014), there were no group RT or accuracy differences, suggesting that adolescents with perinatal nicotine exposure could compensate for any possible neurophysiological differences impacting behaviour in this simple task, and all groups were equally motivated to perform well. It is not uncommon for functional brain differences to be present despite no differences in behaviour (e.g. Lannoy et al., 2017; Yang et al., 2009). In fact, it would be problematic if behaviour differed across groups and would suggest that the two groups interpreted the parameters of the task differently from one another.

We also observed group differences across ERPs. We obtained stimulus-locked responses and compared amplitudes across groups and conditions at Cz, Pz, and Oz. Group differences were observed during the timing of the LPP at Cz and Pz, with the exposed group showing the largest Go/No-Go differentiation. Amplitudes during the LPP window in the exposed group differed between Go and No-Go trials for fear vs. happy faces, such that fearful faces were associated with the most positive amplitudes in No-Go trials. Our results may suggest that emotional stimuli requiring behavioural or response inhibition are more attention-capturing for exposed relative to unexposed adolescents, particularly for fearful faces. Increased reactivity to fearful faces, as demonstrated by enhanced LPP, has been observed among youth with anxiety when compared to healthy youths (Kujawa et al., 2015). Other literature has supported this finding, as social anxiety (Moser et al., 2008; Mühlberger et al., 2009) and generalised anxiety disorder (MacNamara and Hajcak, 2010) have been similarly associated with increased LPPs when processing emotionally salient stimuli (i.e. threatening faces and aversive

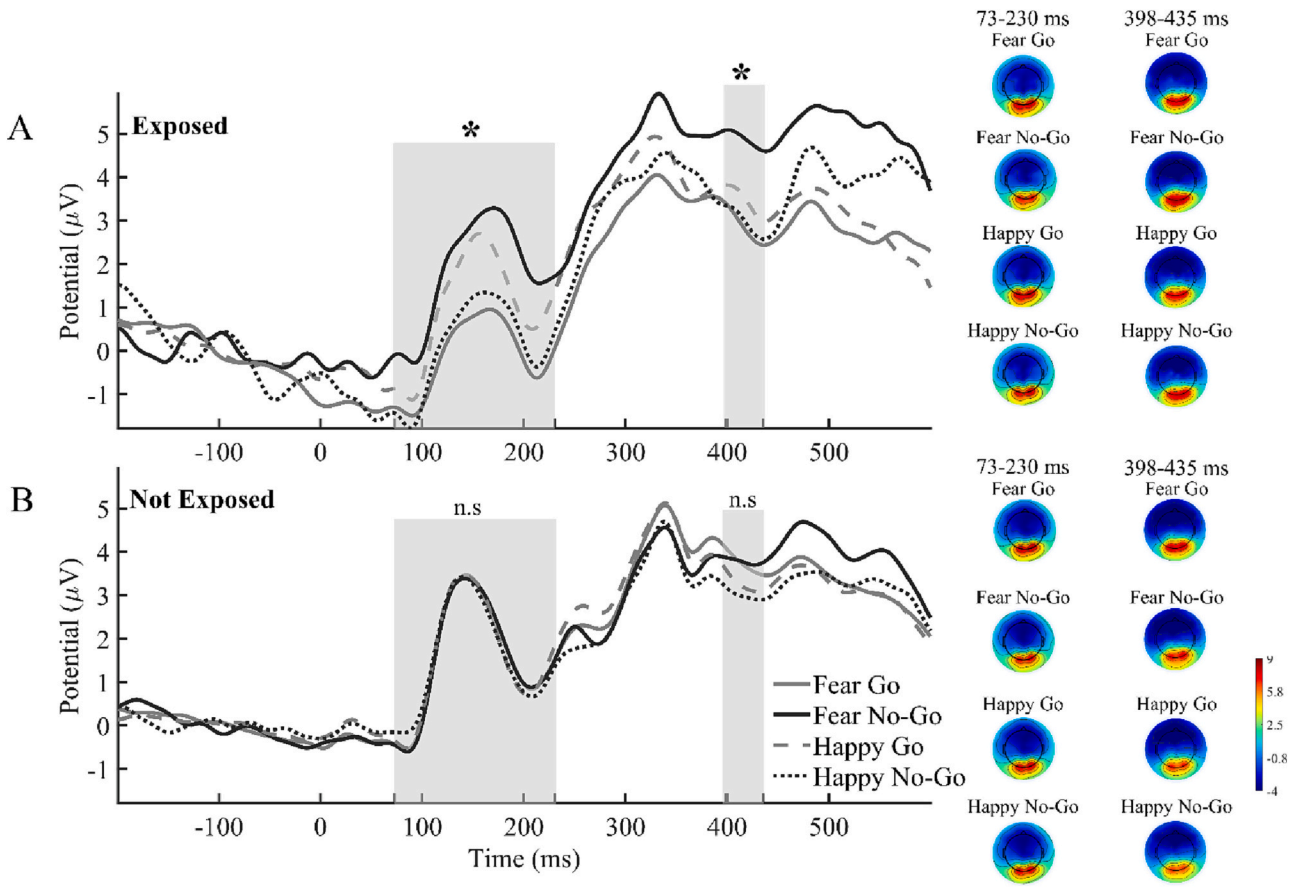

Fig. 5. Group × Condition × Emotion Interaction on Stimulus-locked ERPs Recorded from Pz.
Note. Light grey horizontal bars indicate periods of a significant three-way interaction, with asterisks representing times where follow-up tests revealed significant condition and/or emotion differences in the exposed group, and n.s. representing non-significant comparisons. Topographies on the right represent averaged electrical activity during times of significant three-way interactions. ERPs: event-related potentials.

images, respectively). Thus, prenatal nicotine exposure may impact emotional information-processing, which could increase risk for later anxiety. Indeed, a recent longitudinal study found a dose-dependent relationship between nicotine exposure and the risk for anxiety symptoms in young adulthood (Duko et al., 2022). Our findings contribute to a greater understanding of how nicotine exposure impacts neural functioning, which may be important for predicting how exposure relates to maladaptive mental health outcomes.

These stimulus-locked effects were not limited to the LPP timing but spanned across early stimulus processing (ME group at Pz and Oz, group × emotion at Cz), and LPP (group × condition at Cz, group × emotion at Pz and Oz) time windows, suggestive of increased attention to emotional or salient stimuli across the information-processing stream, even in very early stages (Kelly et al., 2008; Stolarova et al., 2006). These very early effects may be because when low-pass filters are applied, they spread amplitude changes over a wider period than they were before low-pass filtering (i.e. smoothing or flattening the waveform; Luck, 2005). This allows an effect to seemingly start very early, even a little before stimulus onset, and is a perfectly normal signal processing outcome. Consequently, we conclude that the no-exposure group has an earlier response to any stimulus happening; that is, they are more responsive at the earliest stage no matter what the condition or stimulus or that these differences are caused by differences in response preparation not observed during response-locked analyses.

Concerning the later ERPs, the LPP is sensitive to habituation (Codispoti et al., 2006; Ferrari et al., 2020), and it may be that the exposed group habituated less to the repeated presentation of emotional faces than the no-exposure group. Further, when posterior amplitudes were explored, the no-exposure group showed increased fear versus happy differentiation during the P3b timing compared to the perinatally exposed group. The posterior P3b may represent attentional resource activations promoting memory processes in temporal-parietal areas (Polich, 2007), and the no-exposure group had greater emotion (but not condition) differentiation here. This suggests that while the exposed group had initial attentional sensitisation, or a lack of habituation to emotional (especially fearful) faces over frontal sites, the no-exposure group engaged these attentional mechanisms to facilitate memory consolidation to a greater degree than the exposed group as reflected in activations recorded from posterior sites. This could mean that posterior ERPs, by virtue of lesser dopaminergic and higher norepinephrine inputs (Polich, 2007), are less sensitive to the effects of nicotine exposure. Yet, the no-exposure group showed larger fear versus happy differences in the posteriorly recorded P3b timing as previously discussed. In sum, while the exposed group showed early and sustained condition differentiation over a frontal site, the no-exposure group showed late differentiation over a posterior site. These findings may hold important implications for the impact of nicotine exposure on emotional processing.

Surprisingly, there were no group effects at the timing of the No-Go N2. The N2 is thought to reflect the initial detection of conflict to determine whether one should inhibit a response. Our findings therefore suggest that while adolescents with nicotine exposure may maintain the ability to detect conflict requiring inhibitory control, their ability to efficiently inhibit erroneous internal responses or maintain vigilance is impacted, especially when emotional processes are involved. Prenatal nicotine exposure is associated with reduced behavioural inhibition

(Huijbregts et al., 2008; Schneider et al., 2011) and externalising behaviours such as hyperactivity, conduct problems (Gatzke-Kopp et al., 2020), and substance use (Lotfipour et al., 2014). Thus, alterations to the N2, which has been implicated as an endophenotype for externalising psychopathology (Hoyniak and Petersen, 2019), were expected. Similarly, externalising behaviours and traits are associated with alterations to other ERP components. For instance, in response to stimuli depicting pain, those with high callous traits have smaller N120, P3a, and LPP amplitudes (Cheng et al., 2012). In addition, reduced ERP amplitudes in response to fearful faces are associated with high callousness (Brislin and Patrick, 2019). These findings were not observed in our study, and the pattern of ERP alterations observed may be more indicative of an increased risk for internalising disorders, such as anxiety. Additional research in higher-risk and clinical populations is required to explore this distinction.

While our frontal stimulus-locked results are not consistent with Boucher et al. (2014), they are consistent with the results of Bennett et al. (2009), who found children with prenatal exposure increased activation in many frontal brain regions during a non-emotional Go/No-Go task and may be consistent with animal research showing increased dendritic branching and complexity in the NAcc and PFC as a result of exposure (Hamilton and Kolb, 2005: Muhammad et al., 2012). Unfortunately, having used fMRI with its relatively lower temporal resolution, they could not separate stimulus- versus response-locked activity. Thus, further research is required to elucidate the conditions with which children and adolescents with varying levels of nicotine exposure show higher or lower electrophysiological activation in prefrontal regions.

Regarding response-locked analyses, we compared amplitudes during the ERN/CRN but did not find reduced differentiation in the exposed group, and thus did not have support for this part of our hypothesis. Boucher et al. (2014) similarly did not find significant ERN group differences; however, overlays suggested less negative ERNs in exposed children. Like Boucher et al. (2014), we coded exposure dichotomously. As such, less is known regarding the individual differences associated with exposure, which may provide more insight into ERN/CRN amplitude differentiation. This suggests small effects worth following up on and/or individual-difference factors (e.g. severity of exposure) in impacting response-locked effects.

Our findings coincide with other literature suggesting perinatal nicotine exposure alters electrocortical activity differently for stimulus and response-locked ERPs. To our knowledge, this study is the first to investigate perinatal exposure and its effects on adolescent ERPs to emotional stimuli. This study adds to previous research suggesting that perinatal nicotine exposure affects frontal and posterior recorded stimulus-locked ERPs. Moreover, perinatal nicotine exposure may impact attentional processing in tasks requiring response inhibition, particularly when emotional stimuli are concerned. It has been recently suggested that prenatal nicotine exposure increases the risk for anxiety in young adulthood (Duko et al., 2022). In line with the literature suggesting an association between ERPs and anxiety (i.e. Kujawa et al., 2015; MacNamara and Hajcak, 2010; Moser et al., 2008; Mühlberger et al., 2009; Schindler and Bublatzky, 2020), our results may contribute to an increased understanding of how perinatal nicotine exposure influences processes involved in later mental health.

5.1. Limitations and future directions

We could not separate the effects of prenatal versus postnatal versus combined nicotine exposures nor the length of exposure owing to sample size limitations. Future studies may consider quantification of nicotine exposure in a dosage-dependent manner, investigate interactions with genetic polymorphisms (Chaarani et al., 2019), and continue to tease apart interactions with emotional versus neutral stimuli on group ERP differences. Future studies would benefit from a longitudinal design examining in more detail the implications of the face and performance processing differences in adolescence.

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Declaration of Competing Interest

The authors declare that there are no conflicts of interest.

Data availability

The data that has been used are confidential.

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Appendix A. Supplementary data

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