Received: 14 September 2021 | Revised: 15 February 2022 | Accepted: 22 March 2022

DOI: 10.1002/jnr.25051

RESEARCH ARTICLE

JOURNAL OF Neuroscience Research

Childhood trauma exposure and reward processing in healthy adults: A functional neuroimaging study

Chanellé Juanita Hendrikse[1] | Stéfan du Plessis[1,2] | Hilmar Klaus Luckhoff[1] | Matthijs Vink[3] | Leigh Luella van den Heuvel[1,2] | Freda Scheffler[1] | Lebogang Phahladira[1] | Retha Smit[1] | Laila Asmal[1] | Soraya Seedat[1,2] | Robin Emsley[1]

[1]Department of Psychiatry, Stellenbosch University, Cape Town, South Africa

[2]Genomics of Brain Disorders Research Unit, South African Medical Research Council / Stellenbosch University, Cape Town, South Africa

[3]Departments of Experimental and Developmental Psychology, Utrecht University, Utrecht, Netherlands

Correspondence
Chanellé Juanita Hendrikse, Department of Psychiatry, Stellenbosch University, Cape Town, 8000, South Africa.
Email: cbuckle@sun.ac.za

Funding information
Research reported in this publication was supported by the South African Medical Research Council (SAMRC) for the "Shared Roots" Flagship Project (Grant no. MRC-RFA-IFSP-01-2013/SHARED ROOTS) through funding received from the South African National Treasury under its Economic Competitiveness and Support Package. The work by Leigh van den Heuvel (LVDH) and Freda Scheffler (FS) reported herein was made possible through funding by the SAMRC through its Division of Research Capacity Development under the following funding programs: (1) SAMRC CLINICIAN RESEARCHER (M.D PHD) SCHOLARSHIP PROGRAMME from funding received from the South African National Treasury (LVDH), and (2) BONGANI MAYOSI NATIONAL HEALTH SCHOLARSHIP PROGRAMME from funding received from the Public Health Enhancement Fund/South African National Department of Health (FS). The content of this publication is solely the responsibility of the authors and does not necessarily represent the official views of the SAMRC.

Abstract

The association between childhood trauma exposure and risk of developing psychopathology may in part be mediated by the effects of chronic stress on dopaminergic neurotransmission. However, little is known about the differential effects of distinct trauma types on reward processing, particularly in adults without concurrent medical or psychiatric disorders. We examined the association of childhood trauma exposure, including the differential effects of abuse and neglect, with reward processing in healthy adults ($n = 114$). Functional magnetic resonance imaging during a monetary incentive delay task was used to assess neural activity in the ventral striatum and orbitofrontal cortex in relation to reward anticipation and reward outcome, respectively. Exposure to childhood trauma, including abuse and neglect, was assessed using the Childhood Trauma Questionnaire-Short Form. We found a significant effect for abuse on ventral striatal activation during reward anticipation, adjusting for age, sex, scanner site, educational level, and household monthly income. There were no effects for abuse or neglect, independently or combined, on orbitofrontal cortex activation during reward outcome. Our findings suggest differential effects of childhood abuse on ventral striatum activation during reward anticipation in healthy adults.

KEYWORDS
functional magnetic resonance imaging, orbitofrontal cortex, RRID:SCR_003550, RRID:SCR_009537, RRID:SCR_019096, South Africa, ventral striatum

Edited by Cristina Antonella Ghiani and Adriene M. Beltz; Reviewed by Daniel Dillon
[The copyright line for this article was changed on 21 April 2022 after original online publication].

1 | INTRODUCTION

Childhood trauma is a well-known risk factor for psychiatric disorders, including posttraumatic stress disorder, depression, substance abuse, and schizophrenia (Carr et al., 2013; Curran et al., 2018; McGrath et al., 2017; McLaughlin et al., 2012). It is also associated with poor academic performance (Artz et al., 2016; Delaney-Black et al., 2002) and maladaptive risk-taking behaviors in the general population (Arens et al., 2012; Chartier et al., 2009; Danielson et al., 2010). Studies over the past few decades have provided important information on neurobiological mechanisms that may underlie these associations. One possible mechanism is stress-related hypothalamic–pituitary–adrenal (HPA) axis dysregulation (Gunnar & Quevedo, 2007; Pechtel & Pizzagalli, 2013), linked with abnormal dopaminergic neurotransmission (Bogdan et al., 2013; Duval et al., 2006; Pascucci et al., 2007; Piazza & Le Moal, 1997) and thus altered reward processing, including reward anticipation and reward outcome-related neural activity (Novick et al., 2018). At a neurodevelopmental level, reward processing is characterized by a gradual increase in reward anticipation and a decrease in reward outcome from adolescence into adulthood (Geier & Luna, 2009; Hoogendam et al., 2013). Childhood trauma exposure could affect this trajectory, leading to perturbations in reward-seeking behaviors and impulse control (Andrews et al., 2011; Cardinal et al., 2004; Vink et al., 2015) as well as increased risk of psychopathology (Birn et al., 2017; Casement et al., 2014; Elman et al., 2009; Goff et al., 2013).

It is important to note that distinct types of childhood trauma—such as abuse and neglect—could have differential effects on reward processing (Cassiers et al., 2018; Dennison et al., 2017; McLaughlin & Sheridan, 2016; McLaughlin et al., 2014). On the one hand, experiences involving early life social or cognitive deprivation, such as childhood family adversity (Boecker et al., 2014; Holz et al., 2017), institutionalization (Goff et al., 2013; Mehta et al., 2010), parental loss (Hanson, Albert, et al., 2015), or emotional neglect (Hanson, Hariri, et al., 2015), have been associated with blunted activation of striatal regions during reward anticipation. On the other hand, childhood abuse has been linked to both increased (Dennison et al., 2016) and decreased (Dillon et al., 2009) striatal activation in response to rewarding cues. However, since altered reward processing has also been described in various psychiatric disorders (Hart & Rubia, 2012; Zald & Treadway, 2017) and medical conditions (Du Plessis et al., 2015, 2018), the unique effects of childhood trauma on reward processing in healthy controls, relative to patient samples, have not been well characterized. Furthermore, prior studies have often failed to account for additional environmental risk factors, such as poverty and educational level, that may go hand in hand with childhood trauma and are known to influence neurodevelopment (Gianaros et al., 2011; Gonzalez et al., 2016; Marshall et al., 2018; Noble et al., 2013; Romens et al., 2015). This is of particular importance in developing countries where issues of social and economic inequality are worsening and school dropout rates are high (Flisher et al., 2010). Inconsistencies in the literature regarding the relationship between childhood trauma and reward processing could, therefore, be explained in part by the inclusion of participants with diverse rearing environments (Dennison et al., 2017; McLaughlin et al., 2014) and varying sociodemographic, medical, and psychiatric profiles (Hart & Rubia, 2012; Novick et al., 2018).

Significance

This is the first study to assess the differential associations of distinct childhood trauma types (including abuse and neglect) with reward processing changes in *healthy* adults living in a developing country. Contrary to prior studies which suggest an association between childhood trauma and decreased reward anticipation in the brain, we found an association between abuse exposure specifically and increased reward anticipation in the ventral striatum. This finding demonstrates a need for future studies to examine the associations of distinct trauma types with specific reward processing changes and how these changes may contribute to psychopathology risk or resilience in adulthood.

The overall aim of this study was to investigate the associations between childhood trauma exposure, including the differential effects of childhood abuse and neglect, and reward processing in adults without comorbid general medical or psychiatric disorders. We used a modified version of the well-described monetary incentive delay (MID) task (Knutson et al., 2000, 2001) to examine neural activation during reward anticipation in the ventral striatum (VS) and reward outcome in the orbitofrontal cortex (OFC) (Du Plessis et al., 2018; Hoogendam et al., 2013; Knutson et al., 2000; Van Hell et al., 2010; Vink et al., 2015). We hypothesized that childhood trauma exposure would be associated with blunted VS activation during reward anticipation (Dillon et al., 2009; Hanson, Albert, et al., 2015; Holz et al., 2017) and OFC hyperactivation during reward outcome (Boecker-Schlier et al., 2016). Furthermore, we anticipated differential effects for abuse and neglect on reward anticipation and reward outcome (Dennison et al., 2017; McLaughlin et al., 2014).

2 | METHODS AND MATERIALS

2.1 | Participants

Participants were drawn from the healthy control group of a cross-sectional project investigating the genomic, neural, cellular, and environmental signatures common to neuropsychiatric disorders and cardiovascular disease risk ("Shared Roots" study, MRC-RFA-UFSP-01-2013). The study was approved by the Health Research Ethics Committee (HREC, N13/08/115) of the Faculty of Medicine and Health Sciences, Stellenbosch University (Cape Town, South Africa). Participants were recruited through purposive sampling in Cape Town between 2014 and 2017 and provided written informed consent. All participants were of mixed ancestry, based on self-report, as the parent study excluded other ethnic groups to avoid the effects of population stratification on genetic analyses.

Of 310 healthy adults originally recruited for the healthy control group, we included 114 in the present study. Inclusion criteria were

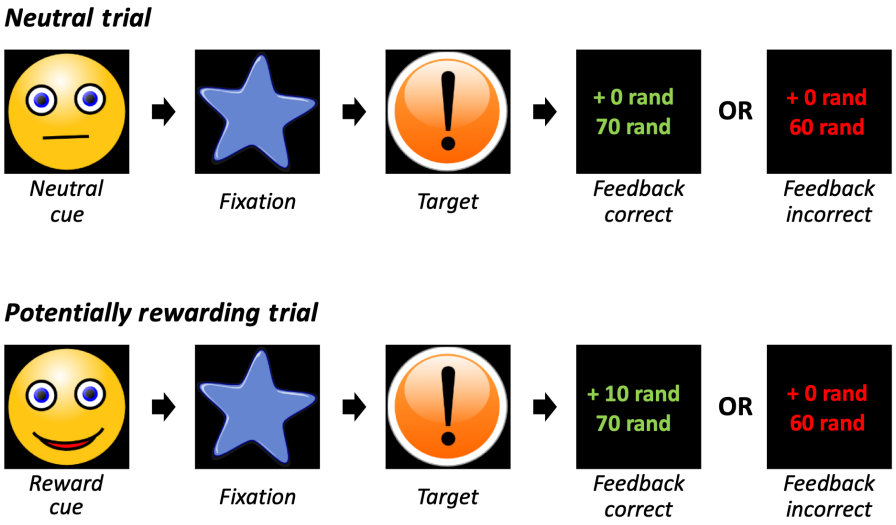

FIGURE 1 Schematic representation of the monetary incentive delay task (Knutson et al., 2000, 2001; Vink et al., 2015)

the completion of neuroimaging and clinical assessments, including screening for childhood trauma. Reasons for exclusion were as follows: (i) incomplete or unsuccessful processing of magnetic resonance imaging (MRI) scans (n = 16); (ii) age above 70 years (n = 15) (Vink et al., 2015); (iii) current diagnosis of a significant general medical condition or clinically relevant structural MRI brain abnormality (n = 33); (iv) current diagnosis of a psychiatric disorder or use of psychiatric medications (n = 61); (v) positive (n = 32) or missing (n = 3) urine toxicology result for opiates, benzodiazepines, cannabis, methamphetamine, or methaqualone on the day of scanning (Van Hell et al., 2010); (vi) incomplete primary schooling (n = 21); and (vii) missing sociodemographic data (n = 7). These exclusions left us with 160 healthy individuals, of whom we excluded a further 46 due to poor functional MRI scan quality (n = 21) or MID task performance (n = 26) (see Section 2.3.3 for further details).

2.2 | Clinical assessments

All participants completed a demographic questionnaire, the MINI International Neuropsychiatric Interview, version 6.0 (Sheehan et al., 1997), for current and lifetime psychiatric disorders, the World Health Organization STEPwise approach to surveillance (STEPS) instrument (World Health Organization, 2008), and a general medical questionnaire to assess personal medical history and record the use of concomitant medications. Handedness was measured using the Edinburgh Handedness Inventory (Oldfield, 1971). All measures were administered by study clinicians and nurses.

2.2.1 | Childhood trauma assessment

Childhood trauma exposure was assessed using the Childhood Trauma Questionnaire-Short Form (CTQ-SF) (Bernstein & Fink, 1998), which is a self-report 28-item Likert-type scale designed to assess past exposure to five types of trauma including physical, sexual, and emotional abuse, and physical and emotional neglect. The CTQ-SF has good criterion-related validity and can be used reliably across various samples (Bernstein et al., 2003).

Participants were categorized according to trauma exposure for descriptive purposes. Categorization was done as follows: The five subscales of the CTQ-SF were first scored separately, and individual participants were categorized by level of exposure to each of the trauma subtypes according to established subscale-specific thresholds (Bernstein & Fink, 1998) outlined in Table S1. The five subscales of the CTQ-SF were then collapsed into two overarching categories, namely abuse and neglect, for two reasons: First, we expected there to be a high degree of overlap between the different trauma subtypes. Second, we did not anticipate that we would have a large enough sample size to assess the unique contribution(s) of the five trauma subtypes to reward processing changes, while controlling for the potential influence of additional trauma subtypes in statistical models. Therefore, abuse exposure was determined by a score above the "moderate" threshold for any one of the three abuse-specific subscales, whereas neglect exposure was similarly inferred from the two neglect-specific subscales. Participants with abuse and/or neglect exposure were grouped under the broader category of "childhood trauma-exposed," whereas those without abuse and/or neglect exposure were categorized as "childhood trauma-unexposed."

2.3 | Neuroimaging assessments

2.3.1 | Monetary incentive delay functional MRI task

Participants completed a modified version of a well-known and reliable reward processing task (Figure 1), namely the monetary incentive delay (MID) task (Knutson et al., 2000, 2001). This task is used to study neural (i.e., blood-oxygen-level-dependent [BOLD] activation)

and behavioral responses to the anticipation and receipt of reward separately, while engaging subregions of the reward system such as the VS and OFC. At the start of each trial, a cue was presented signaling whether the participant could win money (potentially rewarding trials, $n = 30$) or not (neutral trials, $n = 30$). This cue was followed by a fixation star, target stimulus, and feedback screen. Participants were required to respond as rapidly as possible to each target by pressing a button. They were awarded 10 South African Rand (ZAR) for responding in time during reward, but not neutral, trials. The feedback screen indicated whether the participant responded in time (green lettering) or not (red lettering), whether money was won or not, as well as the cumulative reward won. Importantly, the duration of the target stimulus was variable across all trials and was adapted according to each individual participant's task performance during an initial practice session. This ensured that all participants had an equal amount of rewarded and unrewarded trials (i.e., equal task performance of about 50%) and won the same amount of money (i.e., approximately 150 ZAR) by the end of the task.

2.3.2 | Image acquisition

MRI scans were acquired at two scan sites on either a 3T Siemens Allegra (Erlangen, Germany) or a 3T Siemens Skyra (Erlangen, Germany). Scan parameters for the functional MRI scan were as follow: 360 whole-brain 2D-EPI images acquired in 9 min 35 s, repetition time (TR) = 1,600 ms, echo time (TE) = 23 ms, flip angle =72.5°, field of view (FoV) = 256 x 256 mm, 30 slices, and 4 mm isotropic voxels. High-resolution T1 ME-MPRAGE-weighted structural MRI scans were also acquired for image registration (van der Kouwe et al., 2008) and for excluding participants with clinically significant intracranial pathology (as evaluated by a radiologist and neurologist). The Siemens Allegra parameters were as follows: TR = 2,530 ms, TE_1 = 1.53 ms, TE_2 = 3.21 ms, TE_3 = 4.89 ms, TE_4 = 6.57 ms, flip angle = 7°, FoV = 256 mm, 128 slices, and 1 isotropic voxel size. The Siemens Skyra parameters were as follows: TR = 2,530 ms, TE_1 = 1.63 ms, TE_2 = 3.47 ms, TE_3 = 5.31 ms, TE_4 = 7.15 ms, flip angle = 7°, FoV = 280 mm, 128 slices, and 1 isotropic voxel size.

2.3.3 | Image preprocessing and quality assurance

Statistical Parametric Mapping, version 12 (www.fil.ion.ucl.ac.uk/spm/, RRID:SCR_009537) was used for preprocessing and first-level statistical analysis of functional MRI scans (Hoogendam et al., 2013), which involved: (i) slice-time correction, (ii) realignment to correct for head motion, (iii) spatial normalization to the Montreal Neurological Institute T1-template brain, and (iv) spatial smoothing using an 8 mm full-width at half-maximum Gaussian kernel to accommodate interindividual differences in neuroanatomy. We utilized an in-house quality assurance tool (based on Friedman & Glover, 2006; Geissler et al., 2007; Stöcker et al., 2005), which generated region-of-interest-based signal-to-noise-ratios (SNRs), as well as whole-brain SNR maps. SNR values were compared between groups to ensure similar level of quality. Movement was evaluated using multiple parameters (Van Dijk et al., 2012) and participants were excluded based on excessive head motion (>3 mm in any direction between subsequent scans; Vink et al., 2015). Edge cases were re-evaluated and included if the SNR values were comparable and did not prove to be an outlier in our analysis. Only one such edge case was identified (head motion = 3.36 mm) and was retained as a leave-out analysis did not significantly change our findings. Finally, good region-of-interest fit was evaluated using a custom masking tool. Additionally, participants with poor MID task performance were excluded, as determined by a response count <11 for either neutral or potentially rewarding trials, or >16 correct responses for potentially rewarding trials (indicating that the participant frequently began to rapidly press the response button before, during, and after the target).

The preprocessed time-series data for all participants were analyzed using a general linear model analysis. We elected a priori to assess reward anticipation in the VS and reward outcome in the OFC, based on previous findings (Haber & Knutson, 2010; Ikemoto & Panksepp, 1999; Knutson et al., 2001, 2003; Oldham et al., 2018; Schultz et al., 1992). We chose to conduct a region-of-interest rather than a whole-brain voxel-wise analysis to limit the amount of multiple comparisons we were making. Whole-brain voxel-wise analyses with cluster-wide correction are particularly vulnerable to this issue and was recently criticized as resulting in many false positive findings (Eklund et al., 2016). Regions-of-interest were based on definitions of the AAL atlas (RRID:SCR_003550) (Tzourio-Mazoyer et al., 2002) and the Oxford-GSK-Imanova Striatal Connectivity Atlas for the VS (Tziortzi et al., 2014). *Reward anticipation* was assessed by comparing average hemodynamic change across the left and right VS over the two task conditions, that is, during and after the presentation of (i) a neutral cue across all neutral trials (i.e., neutral anticipation), and (ii) a reward cue across all potentially rewarding trials (i.e., reward anticipation). *Reward outcome* was assessed by comparing average hemodynamic change across the left and right OFC over the two task conditions, that is, during all feedback trials when (i) the button was pressed in time during a neutral trial (i.e., neutral correct outcome), and (ii) money was received for a successful reward trial (i.e., reward outcome). The onset of the factors modeling anticipation (duration range 1,529–7,479 ms) was at the presentation of the cue, while the onset of the factors modeling feedback (duration 2,000 ms) was at the presentation of the target, including the button press to the target and subsequent feedback. Motion parameters from the realignment procedure were included as factors of no interest. Low-frequency drifts were removed from the signal by applying a high-pass filter with a cut-off frequency of 128 s (Du Plessis et al., 2018).

2.4 | Statistical analysis

Statistical analyses were performed using the Statistical Package for the Social Sciences software, version 26 (www.ibm.com/products/spss-statistics, RRID:SCR_019096). Prior to performing the main analyses,

we used chi-square tests and *t* tests, as appropriate, to examine potential differences in key sample characteristics between participants with and without childhood trauma exposure. Thereafter, we evaluated the effectiveness of the MID task in the total sample using repeated measures analysis of variance (RMANOVA) models assessing the average in-scanner response time change, as well as hemodynamic changes during reward anticipation in the VS and reward outcome in OFC, over the two task conditions (i.e., neutral and potentially rewarding trials), adjusting for scanner site. No additional covariates were included in this performance analysis, since any potential between-subject variability in task performance was already accounted for by adjusting for individual performance levels. However, we used partial correlations, adjusting for scanner site, to assess the associations of relevant sociodemographic variables with the response times and accuracies (i.e., the number of correct responses) for each task condition. We anticipated faster and more accurate responses for potentially rewarding compared to neutral trials for all participants (Knutson et al., 2000, 2001).

For the main statistical analyses, we calculated total scale scores for the CTQ-SF for all participants. These total scale scores were used in RMANOVA models to examine the associations of overall childhood trauma exposure with reward anticipation and reward outcome separately. Thereafter, we ran the same RMANOVA models to assess the differential effects of childhood abuse and neglect exposure on each reward processing component. We calculated composite scores for abuse by summing the physical, sexual, and emotional abuse subscale scores, and composite scores for neglect were calculated by summing the physical and emotional neglect subscale scores (Sheridan et al., 2017). First, we entered the separate composite scores for abuse and neglect simultaneously in the RMANOVA models, replacing the CTQ-SF total scale score, as previously recommended (McLaughlin & Sheridan, 2016). Thereafter, we reran the same models entering the composite scores of each trauma type exclusively. This approach allowed us to identify reward processing changes unique to childhood abuse and not neglect, as well as demonstrate that these reward processing changes vary in relation to the severity of exposure (McLaughlin & Sheridan, 2016). We elected a priori to adjust for age, sex, educational level, monthly household income, and scanner site in all models.

3 | RESULTS

3.1 | Sample characteristics

After exclusions, the final sample (n = 114) comprised 57 healthy adults with a history of childhood trauma and 57 without a history of childhood trauma. These groups were similar in terms of age, sex, educational level, monthly household income, handedness, scanner site, and the amount of reward won during the MID task (Table 1). The childhood trauma-exposed group comprised 36 participants with abuse exposure only, six participants with neglect exposure only, and 15 participants with both abuse and neglect exposure. As expected, overlap of exposure to the five trauma subtypes was high, with 39 participants (i.e., 68% of the trauma-exposed group) reporting a history of more than one of the trauma subtypes (Table S2). For a detailed breakdown of the sociodemographic characteristics according to the five trauma subtypes, see Table S3.

3.2 | Task performance

Participants (n = 114) responded significantly faster to the target during potentially rewarding trials compared to neutral trials, as indicated by a main effect of task condition on response time, adjusting for scanner site [$F(1, 112) = 7.235$, $p = 0.008$, partial $\eta^2 = 0.061$]. Similarly, we found an expected increase in hemodynamic activity of the VS during reward anticipation [$F(1, 112) = 7.311$, $p = 0.008$, partial $\eta^2 = 0.061$] (Table S4, RMANOVA 1). In contrast, we did not initially observe a significant change in hemodynamic activity of the OFC during reward outcome [$F(1, 112) = 2.503$, $p = 0.116$] (Table S4, RMANOVA 2). However, given the potential impact of income on participants' interpretation of reward outcomes, we added household monthly income [$F(1, 111) = 4.716$, $p = 0.032$, partial $\eta^2 = 0.041$] as a covariate to this model, which rendered it significant [$F(1, 111) = 6.219$, $p = 0.014$, partial $\eta^2 = 0.053$]. In particular, participants showed increased activation of the OFC during positive outcome compared to neutral outcome trials.

Partial correlations adjusting for scanner site revealed significant positive associations between age and average response times during both neutral [$r(111) = 0.328$, $p = 0.000$) and potentially rewarding trials [$r(111) = 0.273$, $p = 0.003$]. Furthermore, educational level was positively correlated with response accuracy during potentially rewarding [$r(111) = 0.224$, $p = 0.017$), but not neutral trials [$r(111) = 0.058$, $p = 0.543$], indicating that participants with a higher educational level made more correct responses when they had the opportunity to win money. This, in turn, lead to greater total rewards among participants who completed secondary or tertiary education (M = 143 ZAR, SD = 12 ZAR) compared to those with partial secondary education (M = 136 ZAR, SD = 15 ZAR) [$t(112) = -2.664$, $p = 0.009$]; nevertheless, in reality this 7 ZAR difference is small.

3.3 | Reward anticipation in the ventral striatum

There was no main effect of overall childhood trauma exposure [$F(1, 107) = 1.264$, $p = 0.263$] on reward anticipation in the VS (Table S4, RMANOVA 3). Moreover, no main effects of either childhood abuse [$F(1, 105) = 1.465$, $p = 0.229$] or neglect [$F(1, 105) = 0.386$, $p = 0.536$] were found when investigating the independent effects of these exposures (entered in the same model simultaneously as separate continuous variables together with an interaction term between the two) on reward anticipation in the VS. Moreover, no interaction effect of abuse and neglect on reward anticipation was found [$F(1, 105) = 0.004$, $p = 0.952$] (Table S4, RMANOVA 5).

We additionally assessed the independent effect of childhood abuse on reward anticipation in the VS without including childhood

TABLE 1 Sample characteristics: Childhood trauma-exposed versus unexposed participants and childhood abuse-exposed versus unexposed participants

	Childhood trauma exposure				Childhood abuse exposure			
Variables	**Exposed (*n* = 57)**	**Unexposed (*n* = 57)**	**Statistic**	***p***	**Exposed (*n* = 51)**	**Unexposed (*n* = 63)**	**Statistic**	***p***
Mean age in years (*SD*)	41 (13)	45 (16)	$t(108.70) = 1.338$	0.156	40 (13)	46 (15)	$t(112) = 1.896$	0.061
Sex, *n*			$X^2(1) = 0.632$	0.427			$X^2(1) = 0.639$	0.424
Male	17	21			15	23		
Female	40	36			36	40		
Educational level, *n*			$X^2(2) = 2.253$	0.324			$X^2(2) = 1.415$	0.493
Partial secondary education	33	25			29	29		
Secondary education completed	16	21			14	23		
Tertiary diploma or degree completed	8	11			8	11		
Monthly household income, *n*			$X^2(2) = 0.895$	0.639			$X^2(2) = 1.679$	0.432
<6,000 ZAR	37	33			34	36		
6,000 to 12,000 ZAR	10	14			8	16		
>12,000 ZAR	10	10			9	11		
Handedness, *n*			$X^2(2) = 3.370$	0.185			$X^2(2) = 0.745$	0.689
Left	1	2			1	2		
Right	55	55			48	60		
Ambidextrous	3	0			2	1		
Scanner site, *n*			$X^2(1) = 3.563$	0.059			$X^2(1) = 3.091$	0.079
3T Siemens Allegra	27	37			24	40		
3T Siemens Skyra	30	20			27	23		
Mean reward won in ZAR (*SD*)	140 (15)	139 (13)	$t(112) = -0.136$	0.892	140 (15)	140 (13)	$t(112) = -0.108$	0.914

Note: Participants were categorized as childhood trauma-exposed if they met the "moderate" threshold for at least one of the Childhood Trauma Questionnaire-Short Form subscales. Abuse exposure was similarly inferred from the abuse-specific subscales.

Abbreviations: *SD*, standard deviation; ZAR, South African Rand.

neglect or the interaction between abuse and neglect in the model, given that overall neglect scores were low in participants who did not report a history of abuse, resulting in only six participants with neglect exposure without concurrent abuse. We found a significant main effect for childhood abuse on reward anticipation [$F(1, 107) = 4.167$, $n = 114$, $p = 0.044$, partial $\eta^2 = 0.037$] (Table S4, RMANOVA 7), showing an increase in VS activity during reward anticipation with increased severity of abuse, adjusting for all elected confounders (Figure 2). Additionally, to confirm this unique association between childhood abuse and reward anticipation, we reran this model with childhood neglect included as a nuisance covariate. This model revealed a stronger association between childhood abuse and reward anticipation in the VS [$F(1, 106) = 6.064$, $p = 0.015$, partial $\eta^2 = 0.054$], whereas childhood neglect was not significant [$F(1, 106) = 2.004$, $p = 0.160$, partial $\eta^2 = 0.019$] (Table S4, RMANOVA 9).

3.4 | Reward outcome in the orbitofrontal cortex

There was no main effect of overall childhood trauma exposure [$F(1, 107) = 0.971$, $p = 0.327$] on reward outcome in the OFC. However, household monthly income was an important predictor of reward outcome in the OFC [$F(1, 107) = 5.803$, $n = 114$, $p = 0.018$, partial $\eta^2 = 0.051$] (Table S4, RMANOVA 4). Furthermore, no main effects of either childhood abuse [$F(1, 105) = 0.063$, $p = 0.802$] or neglect [$F(1, 105) = 0.138$, $p = 0.711$] on reward outcome were found, and there was no interaction effect of abuse and neglect on reward outcome [$F(1, 105) = 0.386$, $p = 0.536$] (Table S4, RMANOVA 6). When removing childhood neglect from the model, no main effect of childhood abuse [$F(1, 107) = 0.724$, $p = 0.397$] on reward outcome in the OFC was found, adjusting for all elected confounders (Table S4, RMANOVA 8).

4 | DISCUSSION

In the present study, we found a significant effect of childhood abuse on reward anticipation in the VS, adjusting for age, sex, educational level, monthly household income, and scanner site. We did not demonstrate the effects of overall childhood trauma and childhood neglect exposure on either reward anticipation in the VS or on reward outcome in the OFC. To the best of our knowledge, this is the first study to examine the associations between childhood trauma, including distinct trauma types, and reward processing in healthy adults recruited from a developing country.

The lack of main effects for either overall childhood trauma score and childhood neglect score on reward anticipation in the VS was unexpected considering prior evidence suggesting an association between various types of childhood stress and decreased reward anticipation in striatal regions during adolescence or adulthood (Boecker et al., 2014; Boecker-Schlier et al., 2016; Casement et al., 2014; Holz et al., 2017; Mehta et al., 2010; Morgan et al., 2014). However, an examination of overall childhood trauma might have obscured the differential effects of distinct trauma types on reward anticipation in our sample (McLaughlin & Sheridan, 2016). Indeed, childhood abuse was associated with increased VS activation during reward anticipation, suggesting increased reward sensitivity with increased levels of childhood abuse exposure. Nevertheless, the direction of this association was unexpected, since prior studies have mostly reported blunted reward anticipation in individuals with a history of childhood trauma (Boecker et al., 2014; Boecker-Schlier et al., 2016; Dillon et al., 2009; Mehta et al., 2010).

However, since our sample did not meet criteria for any psychiatric disorder, they might represent a resilient group in terms of the effects of childhood trauma on their mental health. Therefore, the increased reward sensitivity demonstrated by our participants in terms of childhood abuse exposure may not be a pathological insult (Novick

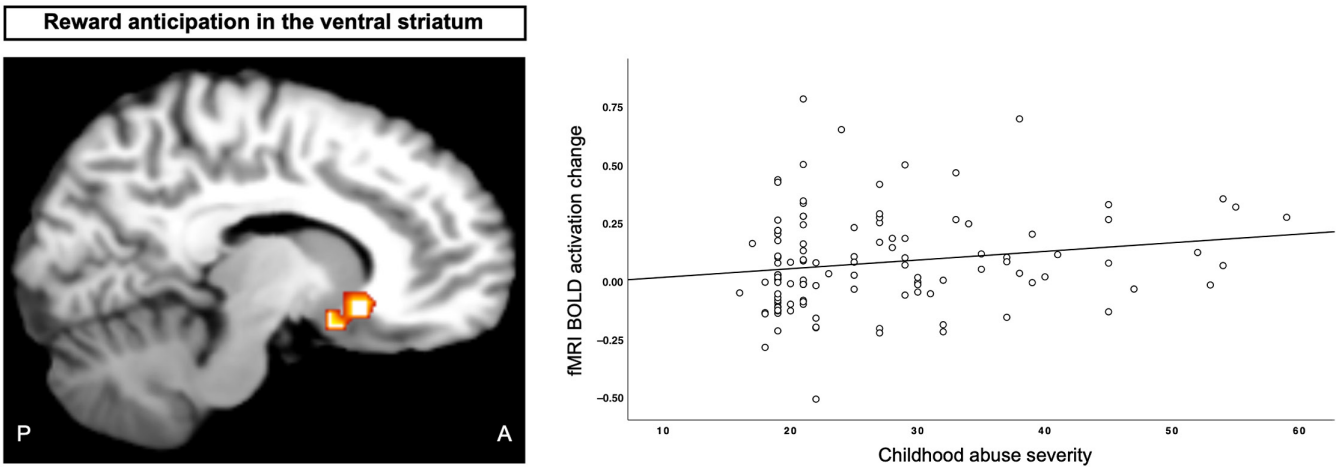

FIGURE 2 Left: Anatomical mask of the ventral striatum. Right: Change in average blood-oxygen-level-dependent (BOLD) activation in the ventral striatum from neutral to potentially rewarding trials (i.e., reward anticipation) as a function of childhood abuse severity with a linear trend line

et al., 2018), but may rather represent an adaptive change to adversity (McLaughlin & Lambert, 2017). This notion is supported by previous studies which demonstrated weaker associations between childhood trauma exposure and anhedonia or depression in individuals with greater VS reactivity to reward (Corral-Frias et al., 2015; Dennison et al., 2016). Conversely, other studies reported associations between VS hyperactivation during reward anticipation and increased risk-taking behaviors (Kuhnen & Knutson, 2005), impulsivity (Andrews et al., 2011), depression (Casement et al., 2014), or bipolar disorder (Nusslock et al., 2012). Consequently, abnormally elevated levels of VS activation during reward anticipation could be a neural basis for the emergence of psychopathology and may have important implications for the development of preventive or therapeutic interventions (Nusslock et al., 2012). It is of course possible that those individuals with a history of childhood abuse who exhibited increased reward sensitivity in our study may have been experiencing subthreshold psychiatric symptoms or maladaptive behaviors and may be at risk of developing mental health problems in future. Furthermore, although not previously considered, educational level had an influence on task performance. The effect of childhood abuse on reward anticipation remained, however, when accounting for educational level. Interestingly, we observed a significant effect of household monthly income on reward outcome-related activity in the OFC. Although a thorough exploration of this finding is beyond the scope of this study, further study of sociodemographic factors that could affect neurodevelopment and in turn reward processing is recommended.

Our study has several limitations. First, participants reported on their childhood experiences retrospectively which may have introduced a recall bias into the data used for this study (Hardt & Rutter, 2004). Second, even though our sample size was greater than or comparable to other studies, a larger sample size would have improved our ability to detect more subtle effects. Moreover, since participants were recruited from the same catchment area, our results cannot necessarily be generalized to other populations. Third, our sample had few participants who reported experiences of neglect in the absence of abuse, undermining our ability to assess the independent effects of childhood neglect on reward processing. Fourth, since this study was cross-sectional in nature, we did not consider the associations of childhood trauma exposure with changes in reward processing over time, and causality cannot be inferred. Additionally, we did not consider the timing of childhood trauma exposure. This may be important, since differences in reward processing from childhood through adolescence into adulthood would be anticipated (Geier & Luna, 2009; Hoogendam et al., 2013). Therefore, the particular influence of childhood trauma exposure on reward processing might depend on the specific neurodevelopmental window in which the trauma is experienced (Andersen et al., 2008).

However, the strength of our study lies in the meticulous characterization of a healthy adult population with exclusion of psychiatric illnesses and medical comorbidities, not always considered in prior studies (Birn et al., 2017; Casement et al., 2015; Dillon et al., 2009; Holz et al., 2017; Morgan et al., 2014). To our knowledge, this is the first study to demonstrate the differential effects of childhood abuse and neglect exposure on reward processing in healthy adults living in a developing country, while considering important potential confounders. Future studies could examine the association of specific reward processing changes and distinct trauma types and how these associations contribute to psychopathology risk or resilience across critical stages of development. Such information may provide unique insights into the pathophysiology of trauma-related psychopathology (Nikolova et al., 2012) as well as aid in the identification of targets for preventive interventions (McLaughlin & Lambert, 2017).

DECLARATION OF TRANSPARENCY

The authors, reviewers and editors affirm that in accordance to the policies set by the *Journal of Neuroscience Research*, this manuscript presents an accurate and transparent account of the study being reported and that all critical details describing the methods and results are present.

ACKNOWLEDGMENTS

Structural MRI data analyses were performed on the Centre for High Performance Computing, Rosebank, Cape Town (http://www.chpc.ac.za/).

CONFLICT OF INTEREST

None.

AUTHOR CONTRIBUTIONS

All authors had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. *Conceptualization*, C.J.H. and S.D.; *Methodology*, C.J.H., S.D., M.V., S.S., and R.E.; *Investigation*, C.J.H., S.D., L.V.D.H., F.S., L.P., R.S., and L.A.; *Data Curation*, L.V.D.H. and R.S.; *Project Administration*, L.V.D.H. and R.S.; *Formal Analysis*, C.J.H. and S.D.P.; *Writing – Original Draft*, C.J.H., S.D., and H.K.L.; *Writing – Review & Editing*, C.J.H., S.D.P., H.K.L., M.V., L.V.D.H., F.S., L.P., R.S., L.A., S.S., and R.E.; *Supervision*, S.D.P., S.S., and R.E.; *Funding Acquisition*, S.S. and R.E.

PEER REVIEW

The peer review history for this article is available at https://publons.com/publon/10.1002/jnr.25051.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ORCID

Chanellé Juanita Hendrikse https://orcid.org/0000-0002-1494-1800
Stéfan du Plessis https://orcid.org/0000-0001-9750-5731
Freda Scheffler https://orcid.org/0000-0002-8898-8599
Retha Smit https://orcid.org/0000-0003-3966-5213

REFERENCES

Andersen, S. L., Tomada, A., Vincow, E. S., Valente, E., Polcari, A., & Teicher, M. H. (2008). Preliminary evidence for sensitive periods in the effect of childhood sexual abuse on regional brain development. *Journal of Neuropsychiatry and Clinical Neurosciences*, *20*(3), 292–301. https://doi.org/10.1176/appi.neuropsych.20.3.292

Andrews, M. M., Meda, S. A., Thomas, A. D., Potenza, M. N., Krystal, J. H., Worhunsky, P., Stevens, M. C., O'Malley, S., Book, G. A., Reynolds, B., & Pearlson, G. D. (2011). Individuals family history positive for alcoholism show functional magnetic resonance imaging differences in reward sensitivity that are related to impulsivity factors. *Biological Psychiatry*, *69*(7), 675–683. https://doi.org/10.1016/j.biopsych.2010.09.049

Arens, A. M., Gaher, R. M., & Simons, J. S. (2012). Child maltreatment and deliberate self-harm among college students: Testing mediation and moderation models for impulsivity. *American Journal of Orthopsychiatry*, *82*(3), 328–337. https://doi.org/10.1111/j.1939-0025.2012.01165.x

Artz, L., Burton, P., Ward, C. L., Leoschut, L., Phyfer, J., Lloyd, S., Kassanjee, R., & Le Mottee, C. (2016). *Sexual victimisation of children in South Africa: Final report of the Optimus Foundation study*. UBS Optimus Foundation.

Bernstein, D. P., & Fink, L. (1998). *Childhood trauma questionnaire: A retrospective self- report manual*. The Psychological Corporation.

Bernstein, D. P., Stein, J. A., Newcomb, M. D., Walker, E., Pogge, D., Ahluvalia, T., Stokes, J., Handelsman, L., Medrano, M., Desmond, D., & Zule, W. (2003). Development and validation of a brief screening version of the childhood trauma questionnaire. *Child Abuse and Neglect*, *27*(2), 169–190. https://doi.org/10.1016/S0145-2134(02)00541-0

Birn, R. M., Roeber, B. J., & Pollak, S. D. (2017). Early childhood stress exposure, reward pathways, and adult decision making. *Proceedings of the National Academy of Sciences of the United States of America*, *114*(51), 201708791. https://doi.org/10.1073/pnas.1708791114

Boecker-Schlier, R., Holz, N. E., Buchmann, A. F., Blomeyer, D., Plichta, M. M., Jennen-Steinmetz, C., Wolf, I., Baumeister, S., Treutlein, J., Rietschel, M., Meyer-Lindenberg, A., Banaschewski, T., Brandeis, D., & Laucht, M. (2016). Interaction between COMT Val158Met polymorphism and childhood adversity affects reward processing in adulthood. *Neuroimage*, *132*, 556–570. https://doi.org/10.1016/j.neuroimage.2016.02.006

Boecker, R., Holz, N. E., Buchmann, A. F., Blomeyer, D., Plichta, M. M., Wolf, I., Baumeister, S., Meyer-Lindenberg, A., Banaschewski, T., Brandeis, D., & Laucht, M. (2014). Impact of early life adversity on reward processing in young adults: EEG-fMRI results from a prospective study over 25 years. *PLoS ONE*, *9*(8), 1–14. https://doi.org/10.1371/journal.pone.0104185

Bogdan, R., Nikolova, Y., & Pizzagalli, D. (2013). Neurogenetics of depression: A focus on reward processing and stress sensitivity. *Neurobiology of Disease*, *52*(1), 12–23. https://doi.org/10.1016/j.nbd.2012.05.007

Cardinal, R. N., Winstanley, C. A., Robbins, T. W., & Everitt, B. J. (2004). Limbic corticostriatal systems and delayed reinforcement. *Annals of the New York Academy of Sciences*, *1021*, 33–50. https://doi.org/10.1196/annals.1308.004

Carr, C. P., Martins, C. M. S., Stingel, A. M., Lemgruber, V. B., & Juruena, M. F. (2013). The role of early life stress in adult psychiatric disorders: A systematic review according to childhood trauma subtypes. *Journal of Nervous and Mental Disease*, *201*(12), 1007–1020. https://doi.org/10.1097/NMD.0000000000000049

Casement, M. D., Guyer, A. E., Hipwell, A. E., McAloon, R. L., Hoffmann, A. M., Keenan, K. E., & Forbes, E. E. (2014). Girls' challenging social experiences in early adolescence predict neural response to rewards and depressive symptoms. *Developmental Cognitive Neuroscience*, *8*, 18–27. https://doi.org/10.1016/j.dcn.2013.12.003

Casement, M. D., Shaw, D. S., Sitnick, S. L., Musselman, S. C., & Forbes, E. E. (2015). Life stress in adolescence predicts early adult reward-related brain function and alcohol dependence. *Social Cognitive and Affective Neuroscience*, *10*(3), 416–423. https://doi.org/10.1093/scan/nsu061

Cassiers, L. L., Bernard, B. G., Schmaal, L., Veltman, D. J., Penninx, B. W., & Van Den Eede, F. (2018). Structural and functional brain abnormalities associated with exposure to different childhood trauma subtypes: A systematic review of neuroimaging findings. *Frontiers in Psychiatry*, *9*, 329. https://doi.org/10.3389/fpyst.2018.00329

Chartier, M. J., Walker, J. R., & Naimark, B. (2009). Health risk behaviors and mental health problems as mediators of the relationship between childhood abuse and adult health. *American Journal of Public Health*, *99*(5), 847–854. https://doi.org/10.2105/AJPH.2007.122408

Corral-Frias, N. S., Nikolova, Y. S., Michalski, L. J., Baranger, D. A. A., Hariri, A. R., & Bogdan, R. (2015). Stress-related anhedonia is associated with ventral striatum reactivity to reward and transdiagnostic psychiatric symptomatology. *Psychological Medicine*, *45*(12), 2605–2617. https://doi.org/10.1017/S0033291715000525

Curran, E., Adamson, G., Rosato, M., De Cock, P., & Leavey, G. (2018). Profiles of childhood trauma and psychopathology: US National Epidemiologic Survey. *Social Psychiatry and Psychiatric Epidemiology*, *53*(11), 1207–1219. https://doi.org/10.1007/s00127-018-1525-y

Danielson, C. K., Macdonald, A., Amstadter, A. B., Hanson, R., de Arellano, M. A., Saunders, B. E., & Kilpatrick, D. G. (2010). Risky behaviors and depression in conjunction with—Or in the absence of—Lifetime history of PTSD among sexually abused adolescents. *Child Maltreatment*, *15*(1), 101–107. https://doi.org/10.1177/1077559509350075

Delaney-Black, V., Covington, C., Ondersma, S., Nordstrom-Klee, B., Templin, T., Ager, J., Janisse, J., & Sokol, R. (2002). Violence exposure, trauma and IQ and/or reading deficits among urban children. *Archives of Pediatrics and Adolescent Medicine*, *156*, 280–285. https://doi.org/10.1001/archpedi.156.3.280

Dennison, M. J., Rosen, M. L., Sambrook, K. A., Jenness, J. L., Sheridan, M. A., & McLaughlin, K. A. (2017). Differential associations of distinct forms of childhood adversity with neurobehavioral measures of reward processing: A developmental pathway to depression. *Child Development*, *90*(1), e96–e113. https://doi.org/10.1111/cdev.13011

Dennison, M. J., Sheridan, M. A., Busso, D. S., Jenness, J. L., Peverill, M., Rosen, M. L., & McLaughlin, K. A. (2016). Neurobehavioral markers of resilience to depression amongst adolescent exposed to child abuse. *Journal of Abnormal Psychology*, *125*(8), 1201–1212. https://doi.org/10.1037/abn0000215

Dillon, D. G., Holmes, A. J., Birk, J. L., Brooks, N., Lyons-Ruth, K., & Pizzagalli, D. A. (2009). Childhood adversity is associated with left basal ganglia dysfunction during reward anticipation in adulthood. *Biological Psychiatry*, *66*(3), 206–213. https://doi.org/10.1016/j.biopsych.2009.02.019

Du Plessis, S., Bossert, M., Vink, M., van den Heuvel, L., Bardien, S., Emsley, R., Buckle, C., Seedat, S., & Carr, J. (2018). Reward processing dysfunction in ventral striatum and orbitofrontal cortex in Parkinson's disease. *Parkinsonism and Related Disorders*, *48*, 82–88. https://doi.org/10.1016/j.parkreldis.2017.12.024

Du Plessis, S., Vink, M., Joska, J. A., Koutsilieri, E., Bagadia, A., Stein, D. J., & Emsley, R. (2015). HIV infection results in ventral-striatal reward system hypo-activation during cue processing. *AIDS*, *29*(11), 1335–1343. https://doi.org/10.1097/QAD.0000000000000680

Duval, F., Mokrani, M. C., Monreal-Ortiz, J. A., Fattah, S., Champeval, C., Schulz, P., & Macher, J. P. (2006). Cortisol hypersecretion in unipolar major depression with melancholic and psychotic features: Dopaminergic, noradrenergic and thyroid

correlates. *Psychoneuroendocrinology*, *31*(7), 876–888. https://doi.org/10.1016/j.psyneuen.2006.04.003

Eklund, A., Nichols, T. E., & Knutsson, H. (2016). Cluster failure: Why fMRI inferences for spatial extent have inflated false-positive rates. *Proceedings of the National Academy of Sciences of the United States of America*, *113*, 7900–7905. https://doi.org/10.1073/pnas.1602413113

Elman, I., Lowen, S., Frederick, B. B., Chi, W., Becerra, L., & Pitman, R. K. (2009). Functional neuroimaging of reward circuitry responsivity to monetary gains and losses in posttraumatic stress disorder. *Biological Psychiatry*, *66*(12), 1083–1090. https://doi.org/10.1016/j.biopsych.2009.06.006

Flisher, A. J., Townsend, L., Chikobvu, P., Lombard, C. F., & King, G. (2010). Substance use and psychosocial predictors of high school dropout in Cape Town, South Africa. *Journal of Research on Adolescence*, *20*(1), 237–255. https://doi.org/10.1111/j.1532-7795.2009.00634.x

Friedman, L., & Glover, G. H. (2006). Report on a multicenter fMRI quality assurance protocol. *Journal of Magnetic Resonance Imaging*, *23*(6), 827–839. https://doi.org/10.1002/jmri.20583

Geier, C., & Luna, B. (2009). The maturation of incentive processing and cognitive control. *Pharmacologgy, Biochemistry, and Behavior*, *93*(3), 212–221. https://doi.org/10.1016/j.pbb.2009.01.021

Geissler, A., Gartus, A., Foki, T., Tahamtan, A. R., Beisteiner, R., & Barth, M. (2007). Contrast-to-noise ratio (CNR) as a quality parameter in fMRI. *Journal of Magnetic Resonance Imaging*, *25*, 1263–1270. https://doi.org/10.1002/jmri.20935

Gianaros, P. J., Manuck, S. B., Sheu, L. K., Kuan, D. C. H., Votruba-Drzal, E., Craig, A. E., & Hariri, A. R. (2011). Parental education predicts corticostriatal functionality in adulthood. *Cerebral Cortex*, *21*(4), 896–910. https://doi.org/10.1093/cercor/bhq160

Goff, B., Gee, D. G., Telzer, E. H., Humphreys, K. L., Gabard-durnam, L., Flannery, J., & Tottenham, N. (2013). Reduced nucleus accumbens reactivity and adolescent depression following early-life stress. *Neuroscience*, *249*, 129–138. https://doi.org/10.1016/j.neuroscience.2012.12.010

Gonzalez, M. Z., Allen, J. P., & Coan, J. A. (2016). Lower neighborhood quality in adolescence predicts higher mesolimbic sensitivity to reward anticipation in adulthood. *Developmental Cognitive Neuroscience*, *22*, 48–57. https://doi.org/10.1016/j.dcn.2016.10.003

Gunnar, M., & Quevedo, K. (2007). The neurobiology of stress and development. *Annual Review of Psychology*, *58*, 145–173. https://doi.org/10.1146/annurev.psych.58.110405.085605

Haber, S., & Knutson, B. (2010). The reward circuit: Linking primate anatomy and human imaging. *Neuropsychopharmacology*, *35*(1), 4–26. https://doi.org/10.1038/npp.2009.129

Hanson, J. L., Albert, D., Iselin, A. M. R., Carré, J. M., Dodge, K. A., & Hariri, A. R. (2015). Cumulative stress in childhood is associated with blunted reward-related brain activity in adulthood. *Social Cognitive and Affective Neuroscience*, *11*(3), 405–412. https://doi.org/10.1093/scan/nsv124

Hanson, J. L., Hariri, A. R., & Williamson, D. E. (2015). Blunted ventral striatum development in adolescence reflects emotional neglect and predicts depressive symptoms. *Biological Psychiatry*, *78*(9), 598–605. https://doi.org/10.1016/j.biopsych.2015.05.010

Hardt, J., & Rutter, M. (2004). Validity of adult retrospective reports of adverse childhood experiences: Review of the literature. *Journal of Child Psychology and Psychiatry*, *45*(2), 260–273. https://doi.org/10.1111/j.1469-7610.2004.00218.x

Hart, H., & Rubia, K. (2012). Neuroimaging of child abuse: A critical review. *Frontiers in Human Neuroscience*, *6*, 1–24. https://doi.org/10.3389/fnhum.2012.00052

Holz, N. E., Boecker-Schlier, R., Buchmann, A. F., Blomeyer, D., Jennen-Steinmetz, C., Baumeister, S., Plichta, M. M., Cattrell, A., Schumann, G., Esser, G., Schmidt, M., Buitelaar, J., Meyer-Lindenberg, A., Banaschewski, T., Brandeis, D., & Laucht, M. (2017). Ventral striatum and amygdala activity as convergence sites for early adversity and conduct disorder. *Social Cognitive and Affective Neuroscience*, *12*(2), 261–272. https://doi.org/10.1093/scan/nsw120

Hoogendam, J. M., Kahn, R. S., Hillegers, M. H. J., Van Buuren, M., & Vink, M. (2013). Different developmental trajectories for anticipation and receipt of reward during adolescence. *Developmental Cognitive Neuroscience*, *6*, 113–124. https://doi.org/10.1016/j.dcn.2013.08.004

Ikemoto, S., & Panksepp, J. (1999). The role of nucleus accumbens dopamine in motivated behavior: A unifying interpretation with special reference to reward-seeking. *Brain Research Reviews*, *31*(1), 6–41. https://doi.org/10.1016/s0165-0173(99)00023-5

Knutson, B., Fong, G. W., Adams, C. M., Varner, J. L., & Hommer, D. (2001). Dissociation of reward anticipation and outcome with event-related fMRI. *Neuroreport*, *12*(17), 3683–3687. https://doi.org/10.1097/00001756-200112040-00016

Knutson, B., Fong, G. W., Bennett, S. M., Adams, C. M., & Hommer, D. (2003). A region of mesial prefrontal cortex tracks monetarily rewarding outcomes: Characterization with rapid event-related fMRI. *Neuroimage*, *18*(2), 263–272. https://doi.org/10.1016/s1053-8119(02)00057-5

Knutson, B., Westdorp, A., Kaiser, E., & Hommer, D. (2000). FMRI visualization of brain activity during a monetary incentive delay task. *Neuroimage*, *12*(1), 20–27. https://doi.org/10.1006/nimg.2000.0593

Kuhnen, C. M., & Knutson, B. (2005). The neural basis of financial risk taking. *Neuron*, *47*(5), 763–770. https://doi.org/10.1016/j.neuron.2005.08.008

Marshall, N. A., Marusak, H. A., Sala-Hamrick, K. J., Crespo, L. M., Rabinak, C. A., & Thomason, M. E. (2018). Socioeconomic disadvantage and altered corticostriatal circuitry in urban youth. *Human Brain Mapping*, *39*(5), 1982–1994. https://doi.org/10.1002/hbm.23978

McGrath, J. J., McLaughlin, K. A., Saha, S., Aguilar-Gaxiola, S., al-Hamzawi, A., Alonso, J., Bruffaerts, R., de Girolamo, G., de Jonge, P., Esan, O., Florescu, S., Gureje, O., Haro, J. M., Hu, C., Karam, E. G., Kovess-Masfety, V., Lee, S., Lepine, J. P., Lim, C. C., … Kessler, R. C. (2017). The association between childhood adversities and subsequent first onset of psychotic experiences: A cross-national analysis of 23,998 respondents from 17 countries. *Psychological Medicine*, *47*(7), 1230–1245. https://doi.org/10.1017/S0033291716003263

McLaughlin, K. A., Green, J. G., Gruber, M. J., Sampson, N. A., Zaslavsky, A. M., & Kessler, R. C. (2012). Childhood adversities and first onset of psychiatric disorders in a national sample of US adolescents. *Archives of General Psychiatry*, *69*(11), 1151–1160. https://doi.org/10.1001/archgenpsychiatry.2011.2277

McLaughlin, K. A., & Lambert, H. K. (2017). Child trauma exposure and psychopathology: Mechanisms of risk and resilience. *Current Opinion in Psychology*, *14*, 29–34. https://doi.org/10.1016/j.copsyc.2016.10.004

McLaughlin, K. A., & Sheridan, M. A. (2016). Beyond cumulative risk: A dimensional approach to childhood adversity. *Current Directions in Psychological Science*, *25*(4), 239–245. https://doi.org/10.1177/0963721416655883

McLaughlin, K. A., Sheridan, M. A., & Lambert, H. K. (2014). Childhood adversity and neural development: Deprivation and threat as distinct dimensions of early experience. *Neuroscience and Biobehavioral Reviews*, *47*, 578–591. https://doi.org/10.1016/j.neubiorev.2014.10.012

Mehta, M. A., Gore-Langton, E., Golembo, N., Colvert, E., Williams, S. C. R., & Sonuga-Barke, E. (2010). Hyporesponsive reward anticipation in the basal ganglia following severe institutional deprivation early in life. *Journal of Cognitive Neuroscience*, *22*(10), 2316–2325. https://doi.org/10.1162/jocn.2009.21394

Morgan, J. K., Shaw, D. S., & Forbes, E. E. (2014). Maternal depression and warmth during childhood predict age 20 neural response to reward. *Journal of American Academy of Child and Adolescent Psychiatry*, *53*(1), 108–117. https://doi.org/10.1016/j.jaac.2013.10.003

Noble, K. G., Korgaonkar, M. S., Grieve, S. M., & Brickman, A. M. (2013). Higher education is an age-independent predictor of white matter integrity and cognitive control in late adolescence. *Developmental Science*, *16*(5), 653–664. https://doi.org/10.1111/desc.12077

Nikolova, Y. S., Bogdan, R., Brigidi, B. D., & Hariri, A. R. (2012). Ventral striatum reactivity to reward and recent life stress interact to predict positive affect. *Biological Psychiatry*, *72*(2), 157–163. https://doi.org/10.1016/j.biopsych.2012.03.014

Novick, A. M., Levandowski, M. L., Laumann, L., Philip, N. S., Price, L. H., & Tyrka, A. R. (2018). The effects of early life stress on reward processing. *Journal of Psychiatric Research*, *101*, 80–103. https://doi.org/10.1016/j.jpsychires.2018.02.002

Nusslock, R., Almeida, J. R., Fobes, E. E., Versace, A., Frank, E., LaBarbara, E. J., Klein, C. R., & Phillips, M. L. (2012). Waiting to win: Elevated striatal and orbitofrontal cortical activity during reward anticipation in euthymic bipolar disorder adults. *Bipolar Disorders*, *14*(3), 249–260. https://doi.org/10.1111/j.1399-5618.2012.01012.x

Oldfield, R. C. (1971). The assessment and analysis of handedness: The Edinburgh inventory. *Neuropsychologia*, *9*(1), 97–113. https://doi.org/10.1016/0028-3932(71)90067-4

Oldham, S., Murawski, C., Fornito, A., Youssef, G., Yücel, M., & Lorenzetti, V. (2018). The anticipation and outcome phases of reward and loss processing: A neuroimaging meta-analysis of the monetary incentive delay task. *Human Brain Mapping*, *39*(8), 3398–3418. https://doi.org/10.1002/hbm.24184

Pascucci, T., Ventura, R., Latagliata, E. C., Cabib, S., & Puglisi-Allegra, S. (2007). The medial prefrontal cortex determines the accumbens dopamine response to stress through the opposing influences of norepinephrine and dopamine. *Cerebral Cortex*, *17*(12), 2796–2804. https://doi.org/10.1093/cercor/bhm008

Pechtel, P., & Pizzagalli, D. A. (2013). Disrupted reinforcement learning and maladaptive behavior in women with a history of childhood sexual abuse: A high-density event-related potential study. *JAMA Psychiatry*, *70*(5), 499–507. https://doi.org/10.1001/jamapsychiatry.2013.728

Piazza, P. V., & Le Moal, M. (1997). Glucocorticoids as a biological substrate of reward: Physiological and pathophysiological implications. *Brain Research Reviews*, *25*(3), 359–372. https://doi.org/10.1016/S0165-0173(97)00025-8

Romens, S. E., Casement, M. D., McAloon, R., Keenan, K., Hipwell, A. E., Guyer, A. E., & Forbes, E. E. (2015). Adolescent girls' neural response to reward mediates the relation between childhood financial disadvantage and depression. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *56*(11), 1177–1184. https://doi.org/10.1111/jcpp.12410

Schultz, W., Apicella, P., Scarnati, E., & Ljungberg, T. (1992). Neuronal activity in monkey ventral striatum related to the expectation of reward. *Journal of Neuroscience*, *12*(12), 4595–4610. https://doi.org/10.1523/jneurosci.12-12-04595.1992

Sheehan, D. V., Lecrubier, Y., Sheehan, K. H., Janavs, J., Weiller, E., Keskiner, A., Schinka, J., Knapp, E., Sheehan, M. F., & Dunbar, G. C. (1997). The validity of the MINI International Neuropsychiatric Interview (MINI) according to the SCID-P and its reliability. *European Psychiatry*, *12*(5), 232–241. https://doi.org/10.1016/S0924-9338(97)83297-X

Sheridan, M. A., Peverill, M., Finn, A. S., & McLaughlin, K. A. (2017). Dimensions of childhood adversity have distinct associations with neural systems underlying executive functioning. *Development and Psychopathology*, *29*(5), 1777–1794. https://doi.org/10.1017/S0954579417001390

Stöcker, T., Schneider, F., Klein, M., Habel, U., Kellerman, T., Zilles, K., & Shah, N. J. (2005). Automated quality assurance routines for fMRI data applied to a multicenter study. *Human Brain Mapping*, *25*, 237–246. https://doi.org/10.1002/hbm.20096

Tziortzi, A. C., Haber, S. N., Searle, G. E., Tsoumpas, C., Long, C. J., Shotbolt, P., Douaud, G., Jbabdi, S., Behrens, T. E., Rabiner, E., Jenkinson, M., & Gunn, R. N. (2014). Connectivity-based functional analysis of dopamine release in the striatum using diffusion-weighted MRI and positron emission tomography. *Cerebral Cortex*, *24*(5), 1165–1177. https://doi.org/10.1093/cercor/bhs397

Tzourio-Mazoyer, N., Landeau, B., Papathanassiou, D., Crivello, F., Etard, O., Delcroix, N., Mazoyer, B., & Joliot, M. (2002). Automated anatomical labeling of activations in SPM using a macroscopic anatomical parcellation of the MNI MRI single-subject brain. *Neuroimage*, *15*(1), 273–289. https://doi.org/10.1006/nimg.2001.0978

Van der Kouwe, A. J. W., Benner, T., Salat, D. H., & Fischl, B. (2008). Brain morphometry with multiecho MPRAGE. *Neuroimage*, *40*(2), 559–569. https://doi.org/10.1016/j.neuroimage.2007.12.025

Van Dijk, K. R., Sabuncu, M. R., & Buckner, R. L. (2012). The influence of head motion on intrinsic functional connectivity MRI. *Neuroimage*, *59*(1), 431–438. https://doi.org/10.1016/j.neuroimage.2011.07.44

Van Hell, H. H., Vink, M., Ossewaarde, L., Jager, G., Kahn, R. S., & Ramsey, N. F. (2010). Chronic effects of cannabis use on the human reward system: An fMRI study. *European Neuropsychopharmacology*, *20*(3), 153–163. https://doi.org/10.1016/j.euroneuro.2009.11.010

Vink, M., Kleerekooper, I., van den Wildenberg, W. P. M., & Kahn, R. S. (2015). Impact of aging on frontostriatal reward processing. *Human Brain Mapping*, *36*(6), 2305–2317. https://doi.org/10.1002/hbm.22771

World Health Organization. (2008). *The WHO STEPwise approach to noncommunicable disease risk factor for surveillance (STEPS)*. Author.

Zald, D. H., & Treadway, M. (2017). Reward processing, neuroeconomics, and psychopathology. *Annual Review of Clinical Psychology*, *13*, 471–495. https://doi.org/10.1146/annurev-clinpsy-032816-044957

SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

TABLE S1 CTQ-SF subscale-specific thresholds for the identification and severity of exposure to childhood trauma

TABLE S2 Overlap of exposure to the five trauma subtypes in the childhood trauma-exposed group ($n = 57$)

TABLE S3 Socio-demographic characteristics according to the five trauma subtypes in the total sample ($n = 114$)

TABLE S4 RMANOVA results for the total sample ($n = 114$)

Transparent Science Questionnaire for Authors

How to cite this article: Hendrikse, C. J., du Plessis, S., Luckhoff, H. K., Vink, M., van den Heuvel, L. L., Scheffler, F., Phahladira, L., Smit, R., Asmal, L., Seedat, S., Emsley, R. (2022). Childhood trauma exposure and reward processing in healthy adults: A functional neuroimaging study. *Journal of Neuroscience Research*, *100*, 1452–1462. https://doi.org/10.1002/jnr.25051