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Highlights

- Visual sensory tetanisation elicited robust N1b enhancement across ages
- Early-stage visual responsiveness was stable from adolescence onward
- P2a attenuation differed by age and cortical region
- Early adolescents showed sustained parieto-occipital P2a attenuation
- Higher-order visual response dynamics continue maturing through adolescence

Developmental differences in visual evoked potential responses to high-frequency sensory tetanisation across adolescence and early adulthood

Abstract

Adolescence is characterised by ongoing maturation of cortical circuits, yet little is known about how cortical responsiveness to repeated sensory stimulation changes across this developmental period. To address this question, we examined visual cortical responses to high-frequency sensory stimulation (HFS) in early adolescence, late adolescence, and adulthood using a sensory visual tetanisation paradigm. Visual evoked potentials (VEPs) were recorded from occipital and parieto-occipital regions during low-frequency visual stimulation (0.83 Hz) at baseline and 2, 4, and 20 minutes following HFS (8.87 Hz, 2 min). Post-HFS changes in the N1b and P2a VEP components, indexing early and later stages of visual processing, respectively, were used to quantify changes in cortical responsiveness. Across all age groups, N1b amplitudes showed sustained increases in both occipital and parieto-occipital regions following HFS, with no significant effects of age. In contrast, P2a amplitudes demonstrated age- and region-specific modulation. At occipital sites, all groups exhibited a comparable transient attenuation of P2a following HFS that returned to baseline levels. However, in parieto-occipital regions, P2a attenuation persisted across all post-HFS assessments in early adolescents, whereas in late adolescents and adults it recovered to baseline by 20 minutes post-HFS. These findings reveal a developmental dissociation between early and later stages of visual processing, with younger adolescents showing more sustained modulation of later neural responses. Together, the results suggest continued maturation of the mechanisms regulating cortical responsiveness to sensory stimulation across adolescence.

1. Introduction

Adolescence is a critical period for brain development, marked by substantial changes in neural connectivity and plasticity (Baker et al., 2025; Insel & Cohen, 2025). During this stage, processes such as synaptic pruning, myelination, and network reorganization reshape neural circuits, supporting cognitive, emotional, social, and perceptual maturation (Juraska & Drzewiecki, 2020; Pfeifer & Allen, 2021). Importantly, developmental changes do not occur uniformly across the brain. Sensory regions, including the visual cortex, mature earlier than association cortices; however, evidence indicates that visual cortical networks continue to undergo experience-dependent refinement throughout adolescence (Sydnor et al., 2023). This prolonged developmental trajectory suggests that neural responses to repeated sensory stimulation may also change across adolescence and early adulthood. However, age-related differences in visual cortical responsiveness to high-frequency sensory stimulation (HFS) have not yet been examined directly.

Addressing this gap is important for understanding normative developmental trajectories in visual system function. The present study therefore used electroencephalography (EEG) and a validated visual HFS paradigm (Anonymous et al, 2021) to examine age-related differences in the modulation of visual evoked potential (VEP) components from early adolescence to young adulthood. HFS paradigms are particularly relevant in this context because they provide a non-invasive method for examining experience-dependent changes in cortical responses in humans.

The use of HFS paradigms is informed by research on long-term potentiation (LTP), a key mechanism underlying synaptic strengthening that was first identified in animal models (Bliss & Lomo, 1973; Kandel & Tauc, 1965) and is widely considered to contribute to learning and memory processes (Bliss & Collingridge, 1993). Attempts to investigate analogous processes in humans led to the development of sensory tetanisation paradigms,

which use high-frequency sensory stimulation to induce sustained changes in neural responses measured non-invasively with EEG (Clapp et al., 2012; Clapp et al., 2005; Teyler et al., 2005). In the visual domain, this approach involves repeated presentation of visual stimuli at a high frequency, followed by assessment of subsequent changes in VEP amplitude. Several studies have reported alterations in VEPs following HFS, and these changes have been interpreted as reflecting experience-dependent modifications in visual cortical responding that share some characteristics with Hebbian forms of plasticity (Kirk et al., 2010; McNair et al., 2006; Ross et al., 2008; Spriggs et al., 2018). Consequently, visual HFS paradigms have been widely applied in clinical and non-clinical populations to investigate individual differences in experience-dependent cortical responsiveness (Çavuş et al., 2012; Ellis et al., 2021; Spriggs et al., 2017; Spriggs et al., 2019; Wynn et al., 2019).

However, despite its widespread use, the validity of sensory tetanisation as a direct index of LTP-like plasticity remains debated. While numerous studies have reported changes in visual evoked potentials (VEPs) following high-frequency stimulation (HFS), findings have been inconsistent, with reports of potentiation, attenuation, and null effects. In a systematic review and meta-analysis, Dias et al. (2022) concluded that the overall effect of sensory tetanisation across studies did not differ significantly from zero, highlighting substantial heterogeneity in stimulation protocols, analytical approaches, and outcome measures. Importantly, however, the authors did not recommend abandoning the paradigm. Rather, they emphasised the need for careful methodological justification and cautious interpretation of findings.

Consistent with this recommendation, the present study does not assume that HFS-induced changes in VEP amplitude provide a direct measure of synaptic LTP. Instead, HFS-related modulation of VEP components is interpreted as an index of experience-dependent changes in cortical responding. We employed a visual tetanisation paradigm that has

previously demonstrated reliable modulation of VEP components in independent samples (Anonymous et al, 2021) to examine whether cortical responses to repeated visual stimulation differ across developmental stages spanning adolescence and young adulthood. By comparing age groups, this study provides novel insight into developmental variation in experience-dependent neural adaptation while acknowledging ongoing uncertainty regarding the precise neurobiological mechanisms underlying HFS-induced VEP changes.

The importance of studying experience-dependent neural adaptation during adolescence is further supported by developmental changes in molecular and synaptic mechanisms involved in learning-related cortical modification. NMDA receptors play a central role in activity-dependent synaptic change and have been implicated in a range of experience-dependent neural processes (Lüscher & Malenka, 2012). While much research has focused on age-related declines in NMDA receptor function and synaptic adaptability during ageing (Boric et al., 2008; Radulescu et al., 2021), evidence from animal models suggests that these mechanisms also undergo substantial reorganisation across adolescence and early adulthood. For example, synaptic capacity for activity-dependent modification has been shown to vary according to developmental stage (Crair & Malenka, 1995; Izumi & Zorumski, 1995), while cortical NMDA receptor binding appears to peak during early adolescence before declining towards adulthood (Insel et al., 1990). Consistent with these findings, Schramm et al (2002) reported greater NMDA receptor-dependent synaptic modification in adolescent compared with adult mice.

Recent research has further highlighted the developmental importance of NMDA receptor subunits encoded by the GRIN2 gene family (GluN2A-D). These subunits influence calcium permeability, synaptic localisation, and the balance between neural flexibility and stability. Early in development, GluN2B-containing receptors predominate and are associated with heightened sensitivity to experience, whereas increasing expression of GluN2A

contributes to the stabilisation and refinement of neural signalling across maturation (McKay et al., 2018; Sheng et al., 1994). Alterations in GRIN2 expression have also been implicated in several neurodevelopmental conditions, including autism spectrum disorder and schizophrenia (Tumdam et al., 2024), emphasising the importance of characterising normative developmental trajectories of cortical responsivity to sensory experience.

Furthermore, contrary to earlier assumptions that visual cortical development is largely complete in early childhood, recent evidence indicates that structural and functional maturation of the human visual cortex continues throughout adolescence (Murphy & Monteiro, 2024; Sydnor et al., 2023). Together, these findings suggest that adolescence may represent a period of continued change in the neurobiological mechanisms that support experience-dependent cortical adaptation, making it an important developmental window in which to investigate age-related differences in neural responses to repeated sensory stimulation.

Current Study

Despite growing interest in developmental plasticity, it remains unclear whether experience-dependent modulation of visual cortical responses differs across adolescence and early adulthood. The current study used sensory tetanisation as a non-invasive approach to examine age-related differences in changes in visual evoked responses measured using EEG following repeated sensory stimulation in early adolescents (13-14 years), late adolescents (18-19 years), and adults (25-26 years). We focused on two VEP components to assess visual cortical plasticity, the N1b and P2a. Previous research found that HFS can induce potentiation of the N1b component, a marker of early sensory processing originating from primary visual areas such as V1 (Moore et al., 2020; Teyler et al., 2005). The N1b is most prominent over occipital electrodes, making it an ideal candidate for evaluating early-stage plasticity. In addition, we examined the P2a component, which reflects later-stage visual

processing and is sensitive to attentional and integrative functions (Crowley & Colrain, 2004). The P2a is thought to originate from extrastriate and parietal regions, and its modulation may indicate broader cortical dynamics beyond primary sensory areas (Potts & Tucker, 2001). These components, reflecting different stages of visual processing, were analysed from occipital and parieto-occipital electrode clusters to capture both early sensory and later integrative cortical responses. This approach allowed us to address two key research questions.

First, does HFS-induced modulation in the human visual cortex vary across distinct stages of adolescence and early adulthood? Based on developmental evidence from animal models suggesting heightened plasticity during adolescence, we hypothesised that HFS-induced modulation of VEP responses would be greater in adolescents than adults, with the largest effects observed in the early adolescent group. Given ongoing debate regarding the extent to which HFS-induced VEP changes directly index LTP-like plasticity, these predictions are framed in terms of experience-dependent cortical response modulation rather than definitive differences in synaptic potentiation. Second, are developmental differences in cortical plasticity component- and region-specific? Given evidence for earlier maturation of primary visual cortex (V1; Huttenlocher & Dabholkar, 1997; Murphy & Monteiro, 2024), we anticipated smaller age-related differences in the N1b component, which reflects early sensory processing. In contrast, more pronounced developmental differences were expected in the P2a component, associated with higher-order visual processing, reflecting distinct developmental trajectories in more primary versus integrative visual cortical functions.

2. Methods

2.1 Participants

Fifty-eight participants completed the study; however, one early-adolescent participant was removed from analyses as there was evidence to suggest they did not adequately receive the tetanizing stimulation from the visual HFS (based on inspection of their spectral plot which indicated no peak at the tetanizing frequency or corresponding harmonics). The remaining 57 participants were split into three age groups: early-adolescents (13-14 years), late-adolescents (18-19 years), and adults (25-26 years). Early-adolescents (N = 18; males n = 9; females n = 9; mean age = 13.72 years; SD age = .46) were recruited through local schools, late-adolescents (N = 19; males n = 9; females n = 10; mean age = 18.63 years; SD age = .50) were recruited through a university participation scheme, and adults (N = 20; males n = 10; females n = 10; mean age = 25.55 years; SD age = .51) were recruited from the local community (Sheffield, UK). Exclusion criteria for all participants included history of DSM-V Axis 1 disorders, current use of antidepressants, sedatives or other medications with psychoactive properties, history of epilepsy, seizures or migraines, or other neurological illness affecting the central nervous system. All participants had normal or corrected-to-normal vision and were financially compensated for their time. Informed consent was obtained from all participants (and from parents/guardians of early-adolescent participants). The study was approved by the School of Psychology Ethics Committee of The University of Sheffield, UK.

2.2 Experimental Paradigm

This study used the same visual cortical plasticity paradigm reported in Anonymous et al (2021) which was modified from the paradigm reported in Çavuş *et al.* (2012), where VEPs were measured before and after exposure to tetanizing visual HFS. During the task (Figure 1), participants had to focus on a red central fixation point while visual stimuli were presented centrally against a white background. Stimuli were presented on a 24-inch LCD colour monitor (144 Hz refresh rate) at a viewing distance of 57cm, situated in a dimly lit room. The

paradigm consisted of two 2-minute baseline VEP assessment blocks (before the HFS), one HFS block, and three further VEP assessment blocks that occurred 2, 4 and 20 minutes after the HFS (post-HFS 1, post-HFS 2, and post-HFS 3 respectively). The purpose of the VEP assessment blocks was to assess VEPs elicited in response to the checkerboard circle before and after HFS. During VEP assessment blocks, participants were shown a pseudorandom sequence of frequently presented checkerboard circles (90 out of 100 of trials) and infrequently presented checkerboard squares (10 out of 100 of trials), with each block lasting 2 minutes. The circular checkerboard was 8cm diameter, subtending 8° of visual angle, with each check subtending 0.3°. The blue square checkerboard was 9x9cm, subtending 9° of the visual angle, with each check subtending 0.5° of the visual angle. All stimuli (circles and squares) were presented on screen for 33ms at a low frequency (0.83Hz; inter-trial period 1.17 seconds), with a central red fixation point remaining on screen throughout. To help participants maintain focus, and to monitor their attention, participants were asked to press the spacebar as quickly and accurately as possible each time the square checkerboard appeared on the screen and their response times and accuracy were recorded.

During the HFS block, designed to induce visual cortical potentiation, participants-maintained focus on the central fixation point whilst the same checkerboard circle used in VEP assessment blocks was presented rapidly for 2 minutes at a higher frequency of 8.87 Hz. Immediately following HFS, participants closed their eyes for 2 minutes before the first post-HFS VEP assessment block (post-HFS 1). Resting state EEG data was collected during the interval between post-HFS 2 and post-HFS 3 VEP assessments in eight blocks alternating between eyes open (EO) and eyes closed (EC). Each block lasted for one minute, interspersed by 20 second breaks, during which the experimenter instructed the participant to either open or close their eyes (EO – EC – EO – EC – EO – EC – EO – EC). Resting state data is not reported or analysed here.

Figure 1

2.3 EEG Methods

2.3.1 EEG Acquisition

EEG recordings were carried out using Biosemi Active Two 64-channel + CMS/DRL electrode caps and Biosemi 'Pin-Type' Ag-AgCl active electrodes, fitted according to the 10/20 electrode system, and amplified using the Biosemi Active Two AD-Box. EEG signals were recorded continuously with a sampling rate of 2048 Hz then down sampled to 512 Hz using Biosemi's decimator tool. Electrode offsets were stable and kept within ±25 μV.

2.3.2 EEG Processing

EEG data were analysed using EEGLAB (Delorme & Makeig, 2004), ERPLAB (Lopez-Calderon & Luck, 2014) and custom MATLAB scripts (R2014b, MathWorks, Natick, Massachusetts). Continuous data were re-referenced to Fz and high-pass filtered at 1 Hz. Channels that showed noise artifacts due to poor connection to the scalp were removed. Noisy segments of data (e.g., segments that contained gross artifacts such as muscle twitching or swallowing) were also removed from the data. The data were then decomposed using independent components analysis (ICA), using the *runica* algorithm, as implemented in EEGLAB. Components relating to ocular artifacts (i.e., blinking, lateral eye movements) were identified manually and removed. Any previously removed channels were interpolated back into the dataset. Data were epoched into 600ms segments (-100ms to 500ms) time-locked to the onset of the checkerboard circle, and a pre-stimulus (-100 to 0ms) baseline correction applied. Finally, epochs were averaged to create VEPs for baseline and post-HFS

assessments for each group (Figures 2 & 3). There were no significant differences between age groups in the number of channels removed during pre-processing; however, early-adolescents had significantly more trials removed due to noise/artefacts in post-HFS 2 compared to young adults (see Supplementary Information 1). Data files and processing scripts for this study are available on the Open Science Framework at the following URL: https://osf.io/q3epc/.

2.3.3 HFS-Driven Visual Steady State Response

EEG data recorded during the HFS block was processed in the same way as data recorded during VEP assessment blocks (referenced to Fz, high-pass filtered at 1 Hz, noisy channels and noisy segments of data removed, ocular artifact components identified in ICA removed, and previously removed channels interpolated). The 2-minute HFS block was epoched from the continuous dataset, and dummy triggers inserted every 2 seconds. Epochs containing excessive amounts of noise were rejected. Bespoke MATLAB scripts were used to create a power spectrum for each posterior channel for each subject. Power values in the frequency bin closest to the HFS frequency (8.87 Hz) were extracted for use in statistical analyses.

2.3.4 Selecting electrodes

In line with the approach of Teyler et al. (2005), selection of electrodes for analysis was based on which channels showed maximal N1b activity based on grand-grand averaged VEPs (collapsed across all VEP assessment blocks and across all participant groups to limit any potential bias; grand-averaged VEPs for posterior electrodes for each age group are presented in Supplemental Information 2). Maximal N1b activity was observed over channels Iz, OZ, O1, and O2. Consequently, data from these four channels were averaged together to create an occipital cluster. Based on channels selected for analysis in Spriggs et al. (2017) study of

age-related alterations in cortical plasticity in younger and older adults, this study also analysed VEP components from a parieto-occipital cluster (PO7 and PO8).

2.4 Statistical analyses

2.4.1 Selecting time windows for VEP analysis

Due to age-related differences in the latency of VEP components (Lenassi et al., 2008; Patterson Gentile et al., 2021), it was not possible to select a standard time window across all participants for each VEP component to be analysed. Consequently, the average amplitude for the N1b and P2a was calculated using the adaptive mean method (Clayson et al., 2013). This method finds the individual participants peak in a given time window, then creates a smaller window around the peak from which the average amplitude is calculated, thus allowing for a more efficient estimate of the true ERP signal when there is individual-subject variability in latency of VEP components. In this study, a grand-average VEP waveform was created for each participant that averaged across all pre- and post-HFS blocks to remove potential bias for selecting different time windows before and after HFS. Inspecting each individual participants grand-averaged waveform allowed for identification of the N1 and P2 peaks and from this select a time window for the N1b component (defined as starting at the N1 peak and ending at the midpoint between the N1 and P1 peaks) and the P2a component (defined as starting at the midpoint between the N1 and P2 peaks and ending at the P2 peak). This participant-specific time window was then used to calculate average VEP amplitude for the pre- and post-HFS blocks. This step was repeated twice; first for the occipital cluster and second for the parieto-occipital cluster. Descriptive statistics for VEP component latencies across age groups, electrode clusters, and experimental blocks, as well as comprehensive statistical analyses assessing age group differences in latency and duration of VEP components are presented in Supplementary Information 3. In summary, these results indicate that while there were no age-related differences in the latency or duration of N1b and

P2a components over the occipital cortex, significant age effects emerged over the parieto-occipital cluster. Specifically, although the duration of VEP components did not differ by age group at this site, the latency of the N1 and P2 peaks, and the midpoint between these peaks, was generally later in early adolescents compared to older groups. This suggests delayed processing in parieto-occipital regions during early adolescence, despite similar component durations.

Statistical analysis of baseline periods

Prior to examining the effects of HFS, baseline stability was assessed by comparing the two pre-HFS periods (pre-HFS 1 and pre-HFS 2). The purpose of these analyses was to determine whether VEP amplitudes remained stable prior to high frequency stimulation and to identify the most appropriate baseline measure for subsequent analyses. A series of 2 × 3 mixed-design ANOVAs were conducted separately for N1b and P2a amplitudes in the occipital and parieto-occipital clusters, with pre-HFS Block (pre-HFS 1, pre-HFS 2) as the within-subject factor and age group (early adolescent, late adolescent, adult) as the between-subject factor.

For the occipital N1b, there was no significant effect of pre-HFS block, $F(1,54) = 1.63$, $p = .204$, $\eta^2p = .03$, and no pre-HFS Block × age group interaction, $F(2,54) = 0.28$, $p = .761$, $\eta^2p = .01$, indicating stable amplitudes across the two baseline assessments. Although a significant main effect of age group was observed, $F(2,54) = 13.39$, $p < .001$, $\eta^2p = .33$, this reflected overall developmental differences in N1b amplitude and not baseline stability.

Similarly, for the occipital P2a there was no significant effect of pre-HFS block, $F(1,54) = 0.23$, $p = .630$, $\eta^2p < .01$, and no significant interaction with age group, $F(2,54) = 1.06$, $p = .353$, $\eta^2p = .04$. Although a significant main effect of age group was observed, $F(2,54) = 3.25$, $p = .046$, $\eta^2p = .11$, there was again no evidence that baseline amplitudes changed systematically across the two pre-HFS recordings.

For the parieto-occipital N1b, neither the effect of pre-HFS block, $F(1,54) = 1.83$, $p = .181$, $\eta^2p = .03$, nor the interaction with age group, $F(2,54) = 1.66$, $p = .201$, $\eta^2p = .06$, reached significance. The main effect of age group was also non-significant, $F(2,54) = 3.11$, $p = .053$, $\eta^2p = .10$.

For the parieto-occipital P2a, there was a significant effect of pre-HFS block, $F(1,54) = 4.47$, $p = .039$, $\eta^2p = .08$, with amplitudes being larger during pre-HFS 2 ($M = 8.97$, $SE = 0.90$) than pre-HFS 1 ($M = 8.04$, $SE = 0.92$). Neither the pre-HFS Block × age group interaction, $F(2,54) = 0.90$, $p = .415$, $\eta^2p = .03$, nor the main effect of age group, $F(2,54) = 1.29$, $p = .284$, $\eta^2p = .05$, was significant.

Because baseline stability was evaluated across four separate ERP measures (occipital N1b, occipital P2a, parieto-occipital N1b, and parieto-occipital P2a), a Bonferroni-corrected significance threshold of $\alpha = .0125$ (.05/4) was applied. Following this correction, none of the baseline comparisons reached statistical significance. Although the parieto-occipital measure showed a nominally significant difference between pre-HFS 1 and pre-HFS 2 ($p = .039$), this effect did not meet the corrected significance criterion ($p < .0125$). Consequently, the baseline analyses provided no evidence of systematic differences between pre-HFS 1 and pre-HFS 2 across any of the four ERP measures examined.

Consequently, the second pre-HFS recording period was selected as the baseline reference for all subsequent analyses because it was obtained immediately prior to stimulation and therefore provides the most appropriate comparator for evaluating HFS-related changes. This approach minimises the influence of task familiarisation, adaptation to the recording environment, and other non-specific settling effects that may occur during the initial stages of testing.

Statistical Analysis of HFS Effects

All analyses examining HFS-related changes in VEP amplitude were conducted in R with analyses conducted separately for N1b and P2a amplitudes in the occipital and parieto-occipital clusters. Because ERP amplitudes were measured repeatedly from the same participants across four assessment blocks (baseline [pre-HFS 2], post-HFS 1, post-HFS2 and post-HFS 3), linear mixed-effects models were used in preference to traditional repeated-measures ANOVA. Mixed-effects modelling appropriately accounts for within-subject dependence arising from repeated observations and provides greater flexibility for handling individual variability and covariates(Baayen et al., 2008; Laird & Ware, 1982).

For each ERP component, an initial model included fixed effects of time, age group, and their interaction, together with a random intercept for participant. A second model additionally included reaction time, task accuracy, and accepted trial count as covariates to evaluate whether behavioural performance or data quality contributed to observed ERP effects. Continuous covariates included in the model (reaction time, accuracy, and number of accepted trials) were grand-mean centred prior to analysis to facilitate interpretation of model parameters.

Likelihood-ratio tests were used to compare nested models and determine whether inclusion of covariates improved model fit. Random-slope models were evaluated but were retained only when convergence criteria were met, and solutions were non-singular. Consequently, random-intercept models were used where random-slope solutions resulted in singular fits. Fixed effects were evaluated using Type III tests with Satterthwaite-adjusted degrees of freedom. Significant effects were followed by pairwise comparisons of estimated marginal means using Holm correction for multiple testing. These analyses examined (a) differences between time points within each age group and (b) age-group differences at each assessment block. A series of robustness analyses were also conducted. First, robust linear mixed-effects models were fitted to assess the influence of potential outliers. Second,

sensitivity analyses were performed after excluding observations with behavioural performance or accepted trial counts below the 5th percentile and observations with reaction times exceeding ±3 standard deviations from the sample mean. Results were considered robust when the pattern of statistical conclusions remained unchanged across these analyses.

Because the presence or absence of age group × time interactions was central to the study objectives, Bayesian model comparisons were additionally conducted. Bayes factors (BF_{10}) were calculated by comparing models including and excluding the interaction term. Following common conventions, $BF_{10} > 3$ was interpreted as evidence in favour of the interaction, $BF_{10} < 0.33$ as evidence in favour of the null model, and values between 0.33 and 3 as inconclusive.Finally, equivalence testing using the two one-sided tests (TOST) procedure was conducted for selected comparisons to distinguish between true evidence for equivalence and merely non-significant differences. This was particularly relevant when evaluating potential developmental differences in HFS-related changes.

2.4.2 Task performance

A two-way ANOVA was run to determine if there were any differences in reaction times to the target square across the VEP assessment blocks across the three age groups. Due to non-transformable ceiling effects, two non-parametric ANOVAs were conducted to assess (1) if there were age group differences in response accuracy to infrequent target squares, and (2) if there was an effect of block number on response accuracy. Descriptive statistics and additional analyses of catch-trial reaction time and accuracy are reported in Supplementary Information 4.

3. Results

3. 1. HFS-induced modulation of visual evoked potentials

3.1.1 Occipital cluster

3.1.1.1 N1b

For the occipital N1b amplitude, the covariate-adjusted mixed-effects model revealed a significant main effect of time, $F(3, 161.81) = 27.56$, $p < .001$, $\eta p^2 = .34$, indicating robust modulation of N1b amplitude following HFS. A significant main effect of age group was also observed, $F(2, 56.02) = 8.85$, $p < .001$, $\eta p^2 = .24$, whereas the time × age group interaction was not significant, $F(6, 159.96) = 0.90$, $p = .499$, $\eta p^2 = .03$. Across all age groups, N1b amplitudes were significantly enhanced at 2 min and 4 min post-HFS relative to baseline (all $ps < .01$), indicating a robust HFS effect. N1b amplitudes moved towards baseline by 20 min post-HFS, with the overall pattern of temporal modulation was comparable across groups.

Age-group comparisons using Holm-corrected post hoc tests revealed that early adolescents exhibited larger N1b amplitudes than both late adolescents and adults at baseline (Holm-corrected $ps \leq .009$), post-HFS 1 (Holm-corrected $ps \leq .005$), post-HFS 2 (Holm-corrected $ps \leq .026$), and post-HFS 3 (Holm-corrected $ps \leq .002$), whereas no significant differences were observed between late adolescents and adults at any assessment point (all $ps \geq .307$). These findings indicate that age-group differences in overall N1b amplitude were present before HFS and remained evident throughout the post-HFS period, suggesting differences in baseline response magnitude rather than HFS-induced modulation.

Bayesian model comparison provided strong evidence against inclusion of the interaction term ($BF_{10} = 0.074$), supporting the absence of age-group differences in N1b response modulation following HFS. Reaction time was a significant covariate, $F(1, 199.06) = 4.77$, $p = .030$, $\eta p^2 = .02$, whereas accuracy, $F(1, 163.84) = 2.90$, $p = .091$, $\eta p^2 = .02$, and

accepted trial count, F(1, 169.07) = 0.53, p = .470, ηp^2 = .003, were not significant predictors. Importantly, inclusion of these covariates did not alter the pattern of findings. Sensitivity analyses excluding behavioural and data-quality outliers, together with robust mixed-effects modelling, yielded the same conclusions, indicating that the results were not driven by outlying observations or differences in data quality.

3.1.1.2 P2a

For the occipital P2a amplitude, the covariate-adjusted mixed-effects model revealed a significant main effect of time, $F(3, 161.40) = 34.47$, $p < .001$, $\eta^2_p = .39$, indicating robust modulation of P2a amplitude following HFS. Neither the main effect of age group, $F(2, 55.47) = 0.94$, $p = .397$, $\eta^2_p = .03$, nor the time × age group interaction, $F(6, 159.36) = 1.79$, $p = .105$, $\eta^2_p = .06$, reached significance.

Follow-up comparisons indicated significant reductions in P2a amplitude from baseline to both post-HFS 1 and 2 across all age groups (all Holm-corrected p's ≤ .041), demonstrating a reliable post-HFS attenuation effect. By post-HFS 3, P2a amplitudes in the late adolescent and adult groups no longer differed significantly from baseline (p's ≥ .391), whereas attenuation remained evident in the early adolescent group ($p = .021$). Although this pattern suggests more prolonged P2a modulation in early adolescents, the absence of a significant time × age group interaction indicates insufficient evidence that the temporal profile of HFS-induced P2a modulation differed reliably across age groups. Consistent with the omnibus analyses, no significant age-group differences were observed at any assessment point following correction for multiple comparisons (all Holm-corrected p's ≥ .130).

Reaction time was a significant covariate, F(1, 202.10) = 7.15, p = .008, η^2_p = .03, whereas accuracy (p = .315) and accepted trial count (p = .344) were not significant predictors. Importantly, inclusion of these covariates did not alter the overall pattern of findings. Sensitivity analyses excluding behavioural and data-quality outliers (20 of 228

observations) produced very similar results. In particular, the Time × Age Group interaction changed only modestly, from $F(6, 159.36) = 1.79$, $p = .105$, $\eta^2_p = .06$ in the primary model to $F(6, 141.06) = 1.97$, $p = .073$ in the sensitivity analysis, while the main effect of Time remained robust ($F(3, 161.40) = 34.47$, $p < .001$; sensitivity analysis: $F(3, 143.79) = 28.68$, $p < .001$). These results suggest that the overall interpretation of the findings was not driven by a small number of outlying observations or differences in data quality.

Consistent with the non-significant Time × Age Group interaction observed in the primary ($p = .105$) and sensitivity ($p = .073$) analyses; Bayesian model comparison favoured a model without the interaction ($BF_{10} = 0.277$). Together, these findings do not provide strong evidence for age-group differences in HFS-related P2a modulation.

3.1.1.3 Summary

HFS produced reliable enhancement of occipital N1b amplitudes and attenuation of occipital P2a amplitudes across all age groups. N1b enhancement and P2a attenuation were evident at 2 and 4 min post-HFS and generally returned towards baseline by 20 min post-HFS. Although early adolescents showed larger overall N1b amplitudes and descriptively more persistent P2a attenuation, neither frequentist nor Bayesian analyses supported reliable age-group differences in HFS-related modulation of either component. These conclusions were unchanged after adjustment for behavioural covariates and sensitivity analyses.

Table 1

Mean N1b and P2a Amplitudes for Occipital Cluster (standard deviation in parentheses)

VEP Component	Age Group	VEP Assessment Block			
		Pre-HFS	**Post-HFS1**	**Post-HFS2**	**Post-HFS3**
N1b	Early Adolescent	5.17	1.78	1.68	4.71

		(6.81)	(8.65)	(6.84)	(8.54)
	Late-Adolescent	-3.43	-7.01	-5.79	-5.62
		(8.35)	(9.81)	(9.73)	(9.31)
	Adult	-6.22	-10.16	-8.88	-7.91
		(6.10)	(5.94)	(6.48)	(6.83)
P2a	Early Adolescent	15.59	9.60	10.85	13.85
		(8.17)	(8.88)	(8.57)	(11.34)
	Late Adolescent	9.94	5.73	7.72	9.77
		(7.89)	(8.06)	(8.43)	(8.01)
	Adult	8.48	5.67	6.72	9.43
		(8.65)	(8.26)	(9.56)	(9.36)

Figure 2

3.1.2 Parieto-occipital cluster: N1b and P2a

3.1.2.1 N1b

For parieto-occipital N1b amplitude, the covariate-adjusted mixed-effects model revealed a significant main effect of time, $F(3, 162.43) = 21.58$, $p < .001$, $\eta^2_p = .28$, indicating robust modulation of N1b amplitude following HFS. The main effect of age group was not significant, $F(2, 56.42) = 2.00$, $p = .144$, $\eta^2_p = .07$. Critically, a significant Time × Age Group interaction was observed, $F(6, 160.20) = 2.55$, $p = .022$, $\eta^2_p = .09$, indicating that the temporal profile of N1b modulation differed across age groups. Follow-up comparisons showed significant N1b enhancement from baseline to Post-HFS 1 in all age groups (all Holm-corrected ps ≤ .009). However, the persistence of this enhancement differed across groups. In early adolescents, N1b amplitudes remained significantly enhanced relative to baseline at

both Post-HFS 2 and Post-HFS 3 (both ps < .001). Follow up comparisons showed that in adults enhancement remained significant at Post-HFS 2 (Holm-corrected p = .021) but not Post-HFS 3 (Holm-corrected p = .373). In late adolescents, enhancement was restricted to Post-HFS 1 (Holm-corrected p = .009). This pattern suggests a more sustained N1b response in early adolescents than in the older age groups.

Despite the significant interaction, direct age-group comparisons at individual assessment points did not survive correction for multiple comparisons (all Holm-corrected ps ≥ .107), indicating that the interaction reflected differences in the overall temporal pattern of change rather than significant between-group differences at specific time points.

Reaction time was a significant covariate, $F(1, 205.97) = 7.92$, $p = .005$, $\eta^2_p = .04$, whereas accuracy ($p = .255$) and accepted trial count ($p = .111$) were not significant predictors. Notably, the Time × Age Group interaction was non-significant in the unadjusted model, $F(6, 162) = 1.95$, $p = .076$, but reached significance following adjustment for reaction time, accuracy, and accepted trial count, $F(6, 160.20) = 2.55$, $p = .022$, $\eta^2_p = .09$. This pattern suggests that inclusion of the covariates reduced unexplained variance and increased sensitivity to detect age-related differences in temporal response dynamics.

Robust mixed-effects modelling and sensitivity analyses excluding behavioural and data-quality outliers yielded comparable findings, indicating that the observed interaction was not driven by outlying observations. However, Bayesian model comparison provided inconclusive evidence regarding the interaction ($BF_{10} = 1.19$), indicating that the data neither strongly supported nor strongly refuted age-group differences in HFS-related N1b modulation.

3.1.2.2 P2a

For the parieto-occipital P2a amplitude, the covariate-adjusted mixed-effects model revealed a significant main effect of time, $F(3, 162.70) = 34.98$, $p < .001$, $\eta^2_p = .39$, indicating substantial modulation of P2a amplitude following HFS. The main effect of age group was not significant, $F(2, 56.31) = 0.25$, $p = .783$, $\eta^2_p = .009$. Importantly, a significant time × age group interaction was observed, $F(6, 159.96) = 6.46$, $p < .001$, $\eta^2_p = .20$, indicating that the temporal profile of HFS-related P2a modulation differed across age groups.

Follow-up comparisons revealed significant reductions in P2a amplitude from baseline to post-HFS 1 across all age groups (all Holm-corrected p's ≤ .006); however, the persistence of this attenuation varied by age group. In early adolescents, P2a amplitudes remained significantly reduced relative to baseline at both post-HFS 2 and post-HFS 3 (both Holm-corrected p's < .001). In late adolescents, attenuation remained evident at post-HFS 2(p = .012) but not at post-HFS 3 (p = .502). Similarly, adults showed significant attenuation at post-HFS 2 (p = .004), with amplitudes returning to baseline by post-HFS 3 (p = 1.00). All reported p-values are Holm-corrected.

Although no significant age-group differences were observed at individual assessment points after correction for multiple comparisons (all Holm-corrected p's ≥ .189), the significant interaction indicates that groups differed in the duration rather than the magnitude of P2a attenuation. Specifically, early adolescents exhibited a more sustained reduction in P2a amplitude following HFS, whereas late adolescents and adults showed recovery towards baseline by the final assessment block.

Reaction time (p = .026), accuracy (p = .027), and accepted trial count (p = .033) were all significant covariates, although their effect sizes were small relative to the effects of time and the time × age group interaction. Inclusion of these covariates did not alter the pattern of findings. Sensitivity analyses and robust mixed-effects modelling yielded comparable results, indicating that the observed effects were not driven by behavioural differences, data quality,

or outlying observations. Bayesian model comparison strongly supported the inclusion of the time × age group interaction (BF_{10} = 3344.89), providing convergent evidence for age-group differences in the temporal dynamics of HFS-related P2a modulation.

3.1.2.3 Summary

Parieto-occipital N1b amplitudes showed robust modulation following HFS, with evidence that the temporal profile of this response varied across developmental stages. Although all age groups exhibited significant N1b enhancement immediately following HFS, the effect appeared most sustained in early adolescents, persisting across all post-HFS assessments, whereas modulation was shorter-lived in late adolescents and adults. However, direct age-group comparisons at individual time points were not significant after correction for multiple comparisons, and Bayesian analyses provided inconclusive evidence regarding the interaction. In contrast, P2a attenuation showed clear age-dependent temporal dynamics, with early adolescents exhibiting sustained post-HFS suppression whereas late adolescents and adults returned to baseline by the final assessment. Convergent frequentist and Bayesian evidence supported this age-group difference for the P2a. Although behavioural measures were associated with ERP amplitude, controlling for these variables did not alter the pattern of HFS-related effects, indicating that the observed modulation was not attributable to differences in task performance or data quality.

Table 2

Mean N1b and P2a Amplitudes for Parieto-Occipital Cluster (standard deviation in parentheses)

		VEP Assessment Block			
VEP Component	**Age Group**	**Pre-HFS**	**Post-HFS1**	**Post-HFS2**	**Post-HFS3**
N1b	Early Adolescent	2.72 (6.79)	-2.42 (8.85)	-.63 (6.83)	-1.42 (10.08)

	Late Adolescent	1.53 (7.14)	-.78 (8.61)	.35 (8.23)	.35 (7.55)
	Adult	-3.14 (5.45)	-5.88 (5.31)	-4.94 (5.85)	-3.98 (5.51)
P2a	Early Adolescent	11.20 (7.84)	3.67 (7.54)	5.96 (6.23)	5.43 (8.08)
	Late Adolescent	8.62 (6.26)	5.15 (6.38)	6.34 (6.51)	8.35 (6.24)
	Adult	7.07 (6.15)	4.43 (6.03)	4.73 (6.57)	7.07 (6.27)

Figure 3

3.2 High-Frequency Stimulation–Driven Visual Steady-State Response (VSSR)

To examine whether age-group differences in VEP measures could be attributed to differences in the tetanizing effect of visual stimulation, an assessment was carried out to determine the strength of the visual steady-state response (VSSR) during the HFS block. A summary VSSR measure was derived by averaging occipital channels (Oz, Iz, O1 and O2) and then parieto-occipital channels (PO7, PO8) used in the previous analyses. The power spectra for the occipital cluster are presented in Figure 4 and demonstrates that HFS-driven VSSR was observed in all age groups, as evident in the peak power value at 9Hz (the frequency bin closest to the HFS frequency of ~8.87Hz) with harmonics seen at multiples of this frequency (e.g. 18Hz, 27Hz, etc). This confirms that the visual system was effectively entrained by the tetanizing stimulus, supporting the validity of this paradigm for eliciting experience-dependent changes in cortical responsiveness, as indexed by VEPs.

The 9 Hz power values ($\mu V^2/Hz$) for the occipital cluster were analysed using a one-way ANOVA to determine whether there were any age-dependent differences in average VSSR power. Results indicated that average VSSR power scores were not significantly different between early adolescents (M = 15.49, SD = 3.56), late adolescents (M = 14.64, SD = 3.02), and adults (M = 13.14, SD = 3.30), $F(2, 54) = 2.50$, $p = .092$, $\eta^2 = .085$. The 9 Hz power values ($\mu V^2/Hz$) for the parieto-occipital cluster were also analysed using a one-way ANOVA. Again, average VSSR power scores did not differ significantly between early adolescents (M = 13.20, SD = 3.32), late adolescents (M = 11.92, SD = 3.23), and adults (M = 10.78, SD = 3.86), $F(2, 54) = 2.28$, $p = .112$, $\eta^2 = .078$. This indicates no age group related differences in the magnitude of VSSR across occipital or parieto-occipital clusters, suggesting that the observed developmental differences in VEP amplitude are unlikely to be due to differences in the efficacy of the tetanizing stimulation itself.

Figure 4

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4. Discussion

In humans, sensory tetanisation has been widely used as a non-invasive approach for examining experience-dependent changes in cortical responses following repeated sensory stimulation. Although the extent to which HFS-induced VEP modulation reflects LTP-like synaptic plasticity remains debated (Dias et al., 2022), the paradigm provides a useful framework for investigating how visual cortical responses adapt to repeated stimulation across development. To the best of our knowledge, this study is the first to directly compare HFS-related changes in visual evoked potentials (VEPs) across adolescence and adulthood,

providing novel evidence of developmental differences in the modulation of visual cortical responses.

Consistent with our previous findings in adults with and without autism (Anonymous et al., 2021), we observed a reduction in P2a amplitude following HFS. Notably, P2a attenuation exhibited distinct region- and age-group-related patterns. In the occipital cluster, all age groups showed a similar pattern of attenuation following HFS, with amplitudes returning towards baseline levels over time and no evidence that this temporal profile differed across age groups. In contrast, the parieto-occipital cluster showed clear age-group differences in the persistence of P2a attenuation. Early adolescents exhibited sustained reductions in P2a amplitude across the post-HFS assessment period, whereas late adolescents and adults showed a more transient attenuation that returned to baseline by the final assessment block. These findings are consistent with the possibility that age-group differences are more apparent in later-stage visual processing networks involving extrastriate and parietal regions than in earlier sensory responses.

N1b amplitudes also exhibited robust post-HFS enhancement across both occipital and parieto-occipital regions. In the occipital cluster, N1b enhancement was strongest immediately following HFS and gradually diminished over time. At the parieto-occipital cluster, enhancement was sustained throughout the post-HFS assessment period. Despite these regional differences in temporal profile, evidence for developmental differences in N1b modulation was limited. At the occipital cluster, there was no evidence that HFS-induced N1b enhancement differed by age group. In the parieto-occipital cluster, all age groups showed N1b enhancement after HFS, with the effect appearing to last longer in early adolescents than in late adolescents and adults. However, age-group differences were not significant after correction for multiple comparisons, and Bayesian analyses provided only inconclusive evidence.

Importantly, the observed patterns of N1b and P2a modulation remained evident after accounting for reaction time, response accuracy, and accepted trial count within the mixed-effects models. Although some behavioural variables were associated with ERP amplitudes, their effects were small relative to the effects of HFS and did not alter the overall pattern of findings. Furthermore, sensitivity analyses and robust mixed-effects models yielded comparable results. Together, these findings indicate that the observed VEP modulation is unlikely to be explained solely by differences in behavioural performance, attentional engagement, or data quality. Instead, they suggest that the observed effects primarily reflect neural responses to repeated visual stimulation, with evidence for developmental differences in P2a modulation and more tentative evidence for age-related differences in the persistence of parieto-occipital N1b enhancement.

Our findings align with previous research on high-frequency visual stimulation that has found potentiation of the N1b component post-stimulation (Clapp et al., 2005; Ellis et al., 2021; McNair et al., 2006; Ross et al., 2008; but see, Spriggs et al., 2018; Teyler et al., 2005), suggesting that early visual components such as N1b are reliably modulated following HFS. The observation of comparable N1b enhancement across age groups further indicates that the neural mechanisms underlying HFS-related modulation of this component remain relatively stable from early adolescence through young adulthood. In contrast, the age-dependent modulation of the P2a component suggests that later-stage visual processing may be more influenced by developmental changes in cortical excitability or regulatory control.

We found that P2a attenuation in the parieto-occipital cluster was sustained in early adolescents but more transient in late adolescents and adults. One possibility is that these age-related differences reflect developmental variation in the neural systems supporting attentional regulation and adaptive updating of sensory representations. During adolescence, top-down control networks and inhibitory neurobiological processes continue to mature

(Caballero et al., 2016; Fuhrmann et al., 2015), potentially contributing to the prolonged modulation of later-stage visual processing following repeated stimulation. Although speculative, less mature inhibitory regulation may result in slower recovery from stimulation-induced suppression, whereas the more transient attenuation observed in older participants may reflect more efficient recalibration of cortical responses.

Interpretation of P2a modulation following HFS remains challenging, as sensory tetanisation studies have reported heterogeneous effects on later VEP components, including amplitude increases, decreases, and null findings (Dias et al., 2022). Compared with earlier sensory components, which are thought to primarily reflect initial perceptual encoding, P2a has been linked to a broader range of cognitive and perceptual processes, including attentional allocation, stimulus evaluation, perceptual matching, categorisation, and experience-dependent changes in perceptual processing (Crowley & Colrain, 2004; Freunberger et al., 2007; Potts, 2004; Potts & Tucker, 2001). As a result, P2a may be particularly sensitive to contextual influences such as developmental stage, attentional engagement, task demands, and stimulation parameters, potentially contributing to the variability observed across studies.

Within this framework, the reduction in P2a amplitude may reflect habituation or reduced attentional engagement following repeated visual stimulation, rather than plasticity alone. The longer-lasting attenuation observed in early adolescents may suggest slower maturation of attentional and inhibitory mechanisms, while the recovery of P2a amplitudes towards baseline in older groups may indicate more efficient regulation of these processes. Similar P2a attenuation has previously been observed in adults with and without autism (Anonymous et al., 2021). Nevertheless, given that P2a is sensitive to a range of developmental, cognitive, and task-related factors, further research is needed to clarify the mechanisms underlying these effects.s

It is important to note that in this study we observed age-related differences in VEP component latency over the parieto-occipital cortex, with early adolescents showing significantly later N1b and P2a peaks compared to older groups (these differences were not present over the occipital cortex). However, latency differences did not extend to component duration, and the timing shifts were relatively small and consistent across blocks. Latency variability across development is well-documented and may reflect ongoing maturation of visual pathways and neural transmission speed (Lenassi et al., 2008; Patterson Gentile et al., 2021). However, given the regional specificity and consistency of the post-HFS effects, these latency shifts are unlikely to explain the observed amplitude changes in the VEP components, including both potentiation and attenuation patterns. Additionally, our use of the adaptive mean method, shown to provide more individualized and accurate estimates of ERP components, particularly in populations with greater latency variability (Clayson et al., 2013), further supports the reliability of our findings. Taken together, these results suggest that the observed differences in VEP modulation are unlikely to be artefacts of developmental differences in component latency or measurement precision.

Before interpreting these findings in terms of developmental differences in cortical plasticity, several considerations are important. Although sensory tetanisation was originally developed as a human analogue of LTP induction and numerous studies have reported post-HFS changes in VEP amplitudes, there remains substantial debate regarding the mechanisms underpinning these effects. Dias et al. (2022) demonstrated considerable variability across studies and found that average tetanisation effects were not reliably different from zero, raising questions about whether HFS-induced VEP modulation can be interpreted as a direct marker of synaptic potentiation. Consequently, the present findings should not be viewed as evidence for developmental differences in LTP itself. Rather, they demonstrate developmental differences in how visual cortical responses are modulated following repeated

high-frequency stimulation within a well-characterized experimental paradigm. Although these response changes may reflect plasticity-related mechanisms, alternative explanations, including changes in adaptation, excitation-inhibition balance, attentional allocation, or broader network-level dynamics, cannot be excluded.

Limitations and future directions

The interpretational challenges associated with sensory tetanisation discussed above highlight the importance of further validating HFS-induced VEP modulation as a marker of experience-dependent cortical change. Whilst the present study employed a well-characterised visual tetanisation paradigm previously shown to produce reliable modulation of VEP responses (anonymous et al, 2021), differences in stimulation parameters, stimulus characteristics, and analytical approaches across studies may contribute to variability in the magnitude and duration of observed effects (Dias et al., 2022; Galuba et al., 2025; Sumner et al., 2020). Future research should therefore prioritise methodological standardisation and replication across independent samples. In addition, combining EEG with complementary techniques could help clarify the mechanisms underlying HFS-related changes in VEP responses. For example, neurochemical and neuroimaging approaches, such as magnetic resonance spectroscopy, EEG-fMRI coupling, or other multimodal methods, may provide converging evidence regarding the contribution of excitatory and inhibitory processes to the observed response modulation (Cooke & Bear, 2010; Malenka & Bear, 2004).

A key limitation in our design is the absence of a sham or no-HFS control condition. Consequently, we cannot determine with certainty whether the observed post-HFS changes were specifically attributable to high-frequency stimulation or whether they may have been influenced by repeated stimulus exposure, habituation, sensitisation, or other time-related factors. To address this concern, we examined baseline stability by comparing the two low-frequency VEP assessment blocks that preceded HFS. Across the four ERP measures

examined, there was no evidence of systematic change between pre-HFS 1 and pre-HFS 2 after correction for multiple comparisons. Although a nominally significant increase in parieto-occipital P2a amplitude was observed between baseline assessments, this effect did not survive correction and was in the opposite direction to the post-HFS attenuation observed following stimulation. Taken together, these findings suggest that the post-HFS effects are unlikely to be explained solely by repeated low-frequency stimulus presentation or simple habituation effects. However, in the absence of an appropriate control condition, alternative explanations cannot be excluded. Future studies should incorporate sham or slow-presentation control conditions to establish the specificity of HFS-induced changes in VEP responses.

Additionally, the absence of behavioural correlates of VEP changes observed after our sensory tetanisation protocol limits interpretation of how these neural changes relate to perceptual or cognitive outcomes. Including task-based measures in future work or longitudinal follow-up could help determine whether VEP changes predict learning, adaptation, or developmental trajectories. Future research should also explore whether individual differences in attentional control, arousal, or inhibitory tone contribute to the observed age-related differences in P2a modulation. Investigating these dynamics in clinical populations, such as individuals with neurodevelopmental disorders or sensory processing differences, may further elucidate the functional relevance of these components .

Furthermore, several factors that may influence visual cortical plasticity were not assessed in the present study and should be considered when interpreting the observed age-group differences. We did not collect measures of pubertal status, sleep quantity or quality, socioeconomic factors, or other developmental variables that may contribute to individual differences in cortical responsivity. These factors may partially account for variability within and between age groups and therefore represent important considerations for future research.

Additionally, participants in the adolescent and adult groups were recruited through different channels, which may have introduced unmeasured differences between samples. Consequently, the current findings should be interpreted as preliminary evidence from a cross-sectional comparison, and replication in longitudinal studies with more comprehensive developmental characterisation is needed to clarify developmental influences on HFS-induced changes in VEP responses that are consistent with, but not definitive evidence of, underlying synaptic plasticity.

Conclusions

The present findings provide preliminary evidence that HFS-induced modulation of VEP components differs across age groups. Specifically, early adolescents showed more sustained changes in visual cortical responses following HFS than late adolescents and young adults, although the pattern of effects varied across ERP components. These findings suggest that age may influence neurophysiological responses to repeated visual stimulation. However, given the cross-sectional design, the absence of measures such as pubertal status, sleep, and other developmental factors, and ongoing debate regarding the interpretation of sensory tetanisation paradigms, the results should not be interpreted as evidence of developmental trajectories or maturational mechanisms. Rather, they highlight age-group differences that warrant further investigation. Replication in larger, longitudinal samples with more comprehensive developmental characterisation will be necessary to determine the extent to which these differences reflect developmental changes in visual cortical functioning.

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