# Spatial Attention and Session Day Independently Modulate Human Visual Cortical Plasticity

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## Abstract

Stimulus-specific response potentiation (SSRP) is a non-invasive form of cortical neuroplasticity that can be induced through repeated presentation of high-contrast visual stimuli. Classically, SSRP, like long-term potentiation and related plasticity mechanisms, has been hypothesized to demonstrate input specificity, such that potentiation is confined to the neural populations and synapses driven by the induction stimulus. In addition, SSRP is observed to accumulate across days in rodent models. Despite growing interest in this phenomenon, it remains unclear whether factors such as selective attention influence the magnitude or specificity of a potentiated response. This study investigated whether covert endogenous attention could modulate the strength of SSRP in high-density EEG recordings from neurotypical human participants. An established SSRP paradigm was modified to include an attention task during induction. Pre-post response amplitudes were measured with frequency-tagged (6 and 7.5 reversals-per-second) bilateral hemifield contrast-sweep checkerboard steady-state visual-evoked potential stimuli. The 10-minute high-contrast 2 Hz sign-reversing SSRP induction stimulus was presented only in the left hemifield, with the right hemifield serving as a control. On two different experimental days, each participant deployed attention either toward or away from the potentiated hemifield during induction. Response amplitudes increased

after SSRP, with increases in response amplitude manifesting in both the potentiated and control non-potentiated hemifields, weighing against the idea that SSRP is input-specific. Interestingly, significantly more response potentiation observed on the second session day compared to the first, independent of attention condition. Finally, we observed that attention enhanced SSRP in naïve but not experienced observers (i.e. on Session-Day 1 but not Session-Day 2), identifying a significant interaction between attention condition and session day. Overall our results suggest that (1) SSRP in humans may not in fact be stimulus-specific (potentiation effects are not input- or retinotopically- specific), (2) visual spatial attention has a modest enhancing effect on SSRP in first-session participants, and (3) a second session of SSRP leads to a pronounced plasticity effect compared to the first session that occludes any attentional modulation.

# Introduction

One established approach to non-invasively probe visual cortical plasticity in humans and other species is through stimulus-specific response potentiation (SSRP), characterized by enhanced visual evoked potentials (VEPs) following a period of prolonged repetitive exposure to a stimulus. It has been demonstrated that a stimulus-driven plasticity response of visual evoked potentials can be induced through a passive viewing of a high-contrast sign reversing stimulus for as little as 10 minutes [Teyler et al., 2005, Frenkel et al., 2006, Normann et al., 2007, Kirk et al., 2008, Cooke and Bear, 2010, Valstad et al., 2021]. Its induction through sensory experiences makes SSRP a valuable model for studying functional brain plasticity in vivo. SSRP mechanisms may contribute to facilitating certain forms of perceptual learning which, broadly, involves the organization, identification, and interpretation of sensory information due to visual exposure [Lu et al., 2011, Donovan et al., 2020, Hung and Carrasco, 2021]. Through these processes, the brain develops a heightened ability (lowered threshold) to detect target stimuli, potentially utilizing synaptic mechanisms similar to those observed in long term potentiation (LTP).

SSRP belongs to an umbrella of cortical plasticity that has been observed in the visual system, highlighting its rapid, yet sophisticated level of adaptation, and it shares several characteristics with Hebbian LTP, particularly dependency on NMDA receptor activation and input specificity [Kirk et al., 2008, Cooke and Bear, 2010, Kirk et al., 2021]. Treatments that extinguish LTP, such as local expression of GluR1-CT peptide or administration of zeta inhibitory peptide, also disrupt SSRP, suggesting that SSRP depends on this form of synaptic plasticity and that its modulation may provide an in vivo measure of LTP-like processes in the human cortex[Frenkel et al., 2006, Cooke and Bear, 2010]. Since SSRP and LTP mechanisms resemble each other in terms of synaptic transmission development, quantifying SSRP susceptibility holds the potential to serve as a biomarker in neuropsychiatric illness. For example, individuals with Bipolar Disorder (Type I and II), Major Depressive Disorder and Schizophrenia have been shown to exhibit impairments in SSRP [Normann et al., 2007, Jacob et al., 2021b, Valstad et al., 2021]. These findings suggest that noninvasive LTP-like plasticity probes, such as SSRP, could serve as biomarkers in clinical trials for these illnesses.

In rodents SSRP-induced response enhancement is highly selective, as slight variations in the spatial frequency, orientation, or visual contrast of the experienced stimulus do not elicit the same potentiated response, highlighting its input specificity [Frenkel et al., 2006, Kirk et al., 2008, Cooke and Bear, 2010, 2011]. Additionally, SSRP induced in one eye does not elicit the same VEP response potentiation in the untrained eye [Frenkel et al., 2006], suggesting that it arises at early stages of the visual pathway, prior to binocular integration. However, these findings are based on non-human studies, raising questions about their generalization to humans, given species-specific differences in visual system organization. To our knowledge, ocular transference of SSRP has not been investigated in humans, and while SSRP has been shown to be selective for orientation, contrast, and eye of origin, its retinotopic specificity, a logical extension of these properties, remains underexplored [Teyler et al., 2005]. Here, we examine input specificity by 1) presenting the SSRP-inducing

stimulus at a range of contrasts and collecting contrast response functions before and after induction to determine whether potentiation occurs selectively at the inducing contrast (100%), and 2) inducing SSRP in one visual hemifield and measuring visual evoked responses to stimuli in both hemifields, before and after SSRP induction.

Since SSRP reflects experience-dependent plasticity in visual cortex, it is important to consider how top-down factors such as attention may modulate its expression. Interestingly, SSRP paradigms have yet to be examined alongside established modulators of perceptual learning, and learning more broadly. Attentional state plays a fundamental role in shaping learning and memory, particularly perceptual memories [Mukai, 2011, Szpiro and Carrasco, 2015, Donovan and Carrasco, 2018]. In perceptual learning paradigms, attention has been shown to enhance neural and behavioral responses to target stimuli [Nguyen et al., 2020, Cavanaugh et al., 2022]. Moreover, paired associative stimulation (PAS)-induced plasticity in human motor cortex is enhanced when the stimulated limb is attended visually and spatially, compared to when attention is allocated elsewhere [Stefan et al., 2004]. Follow-up studies of PAS show that attention exhibits opposite effects depending on induction protocol: it enhances motor evoked potentials (MEPs) during LTP-like stimulation but reduces MEP depotentiation during LTD-like stimulation, indicating bidirectional modulation of neuroplasticity [Kamke et al., 2012, 2014]. These findings suggest that attention may act as a general modulator of experience-dependent synaptic change across cortical systems, including SSRP. To investigate this in the visual system, we examined covert endogenous spatial attention (a form of voluntary, stimulus-independent orienting toward specific retinotopic locations [Carrasco, 2011]) to determine its influence on visual cortical plasticity.

Another important feature of SSRP, observed thus far in rodents, is that potentiation accumulates across experimental days [Frenkel et al., 2006, Cooke and Bear, 2010, 2011], suggesting that consolidation processes involving sleep and time contribute to recalibrating neural circuitry strength. In humans however, previous reports describe potentiation effects emerging within minutes after a visual plastic-

ity protocol [Teyler et al., 2005, Normann et al., 2007, Klöppel et al., 2015, Valstad et al., 2020]. Our laboratory has not yet observed such immediate effects, as our initial SSRP induction study did not yield potentiation (sign-reversing gratings at 18 reversals/second presented in contrast sweeps for 900 seconds) [Ash et al., 2023], highlighting a broader issue with the paradigm: its replicability across laboratories remains inconsistent, possibly due to variability in participant population or details of visual stimulus presentation [Çavuş et al., 2012, Valstad et al., 2020, Sumner et al., 2020, Jacob et al., 2021a, Dias et al., 2022] . To assess how robust SSRP effects develop with repeated exposure, participants completed two sessions to compare the extent of potentiation after one versus two sessions of SSRP-induction.

We employed steady-state visually evoked potentials (ssVEPs) due to its high signal-to-noise readout of population activity and its unique ability to enable simultaneous recording activity in both hemifields with frequency-tagging [Morgan et al., 1996, Müller et al., 1998, Norcia et al., 2015, Ash et al., 2023]. These characteristics of the ssVEP enable in-vivo quantification of how attention shapes experience-dependent plasticity, observable as shifts in the contrast response function before and after induction [Ling and Carrasco, 2006] .

**Hypotheses** Based on prior studies in rodents [Frenkel et al., 2006, Cooke and Bear, 2010, 2011] and humans [Teyler et al., 2005], we predict that **(I)** SSRP will produce response potentiation in the potentiated but not control hemifield, and SSRP will be more prominent at high contrasts, reflecting LTP-like retinotopic and contrast specificity. Building on the results of [Stefan et al., 2004, Ling and Carrasco, 2006, Donovan et al., 2020, Hung and Carrasco, 2021], we further predict that **(II)** attentional deployment *toward* the induction stimulus (attention–induction congruent condition) will lead to greater plasticity—indexed by increased VEP amplitudes—than when attention is deployed *away from* (contralateral to) the induction stimulus (attention–induction incongruent condition). Finally, based on Cooke and Bear and Frenkel's SSRP investigations, we predict that **(III)** response amplitudes in the potentiated hemifield will increase across repeated days of SSRP, consistent with

cumulative effects over time.

# Methods

## Participants

26 participants (F = 15 , M = 11) were recruited from Stanford University research pool databases. Individuals 18 years of age or older, with no known neurological impairments and normal or corrected-to-normal vision, were eligible to participate. Informed consent was provided for all participants prior to any data collection. The study was approved by the Stanford IRB. Participants completed two EEG sessions and received either course credit (1 credit / 1 hour) or monetary compensation ($30 / hour).

## Data Acquisition

Prior to arrival, participants were instructed to wash their hair with shampoo (no conditioner). Vision testing was performed to confirm normal or corrected-to-normal vision. Afterwards, high-density, 128-channel electroencephalograms (EEG) were recorded using HydroCell electrode arrays and an Electrical Geodesics Net Amps 400 (Electrical Geodesics) amplifier. The EEG cap was prepared in a KCl solution for 10 minutes. Electrode caps were placed on the participant's head, and additional KCl solution was applied to individual electrodes such that all electrodes had an impedance $<$ 50 kOhm. Impedance checks were performed twice during the recording session, each session lasting 45 minutes.

## Visual Stimuli and Attentional Paradigm

Visual stimuli were displayed on a high dynamic range 60-Hz refresh rate monitor positioned 70 cm from the participant's eye level. Stimuli were generated with custom lab software XDiva. Stimuli consisted of two semicircular checkerboards each positioned laterally on each side of fixation with the stimuli width spanning 0.5° to

10° eccentricity. Each checkerboard wedge was divided into sixteen 0.628° segments along the vertical meridian relative to fixation, and 20 rings with a spatial frequency of 0.5°, creating a wedged checkerboard pattern. The semicircular design left the central area near fixation devoid of pattern elements, and both this region and the background were maintained at mean background luminance. Each hemifield stimulus was frequency-tagged to measure EEG ssVEPs. pre-/post- stimuli sign-reversal rates were : F1 = 3 Hz (6 reversals-per-second, rps) (left hemifield), F2 = 3.75 Hz (7.5 rps) (right hemifield). pre-/post- stimuli were contrast sweep stimuli presented in log steps of: 1%, 2.5%, 6.3%, 15.8%, 39.8% and 100%. Each contrast level was presented for 1.33 s (8 s of core stimulus bins). Each stimulus sweep began with a 1.33 s prelude bin at 1% contrast and ended with a 1.33 s postlude bin at 100% contrast. These stabilization periods help reduce edge artifacts in the broadband EEG for subsequent Fourier analysis. The total stimulus duration, including prelude/postlude was 10.66 s. There was a 3000 ms ± 500 ms inter-trial rest period between each stimulus. Participants were advised to limit blinking to inter-trial periods (confirmed by visual inspection of the EEG). For each session, 34-40 trials of contrast-sweep ssVEPs were acquired before SSRP, and another 34-40 trials were acquired after SSRP.

The SSRP induction stimulus was modified from the pre- post- stimulus such that the left checkerboard was presented at 100% contrast (not swept) flickering at F1 = 1 Hz (2 rps) a parameter previously reported to induce neuroplasticity. The right checkerboard, serving as the non-potentiating control stimulus was presented at 2% contrast with frequency F2 = 1.5 Hz (3 rps) . The stimuli were presented simultaneously for 30 seconds with a brief (<0.5 sec) gap, for 10 trials. The participant was then given a few seconds of rest, and the participant observed another ten 30-second trials, for a total SSRP induction duration of 10 minutes, in line with Norman et al.'s induction time [Normann et al., 2007]. During the SSRP block, participants were advised to blink when they needed to but to not close their eyes for extended periods of time. A trial example for each condition can be observed in

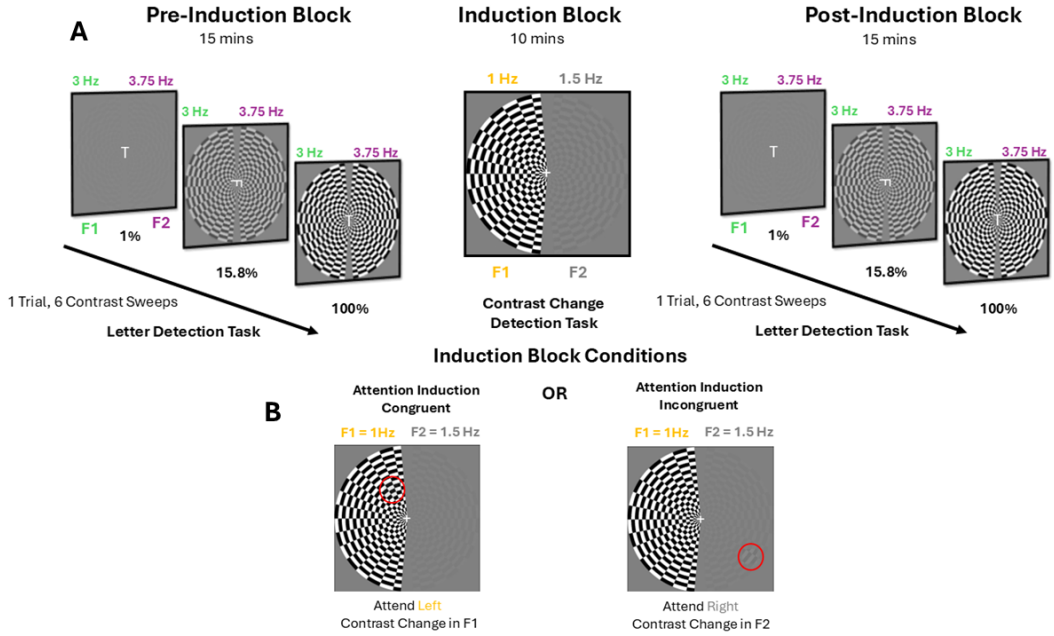


Figure 1:
Experimental Protocol. **A**. Participants first completed a pre-SSRP block, 39 contrast-sweep ssVEP trials. Next, participants completed a 10 minute SSRP block. Here the left checkerboard served as the potentiating stimuli at 100% contrast sign reversing twice a second and the right checkerboard served as the non-potentiating control (2% contrast). Finally, participants completed the post-SSRP block, which was identical to the pre-SSRP block. Contrast Response function analysis was derived from ssVEPs from the pre- and post- SSRP blocks. **B**. Illustration of the two attentional deployment conditions during induction. Left panel: Attention induction congruent in which with the participant attends *toward* the induction (left) stimulus. Right panel: Attention induction incongruent in which the participant attends away from the induction stimulus and toward the non-potentiating (right) stimulus, performing a contrast change detection task across 10 minute induction block. Attention conditions were performed in a counterbalanced order across experiment days.

*Supplementary Movies*.

In each session, participants completed a pre-SSRP Block, SSRP Block, and a post-SSRP block (**Fig. 1A**). During the pre- post-SSRP blocks, participants performed a simple letter detection task presented at the center of the screen to maintain fixation. A series of the letters L, F and T (0.25° retinotopic extent) were presented one at a time at the center of the screen approximately once per second at different orientations. Participants were instructed to make a response via a keyboard button when the letter T was presented, regardless of the orientation of the T.

Attentional deployment was manipulated during the SSRP block (**Fig. 1B**). In the **Potentiation-Attention Congruent** condition, participants were instructed to fixate on the center of the screen and deploy peripheral spatial attention to the left checkerboard (SSRP stimulus) while 2°-square 100-ms contrast decrement patches appeared randomly within the left hemifield stimulus every 5-8 seconds. Participants were instructed to make a response by pressing a button on a keypad when a contrast decrement was detected. The contrast decrement started at 18%, which decreased or increased after every 30 second trial depending on performance. Task difficulty increased on trials in which responses were made to all decrements, otherwise, task difficulty decreased, to maintain task difficulty at 70% percent correct. In the **Potentiation-Attention Incongruent** condition, participants were instructed to fixate on the center of the screen and deploy peripheral spatial attention to the right low-contrast checkerboard (non-induction stimulus) while 2°-square contrast increments appeared randomly within the right hemifield stimulus with otherwise similar features as in the attention congruent condition. The task increment started at 2% updated by staircase as above. Participants completed the two attention conditions on two separate days spaced 1-28 days apart (one participant completed the study after 38 days), the order of conditions was counterbalanced across participants.

## Signal Processing

The EEG was sampled natively at 500 Hz and then resampled at 420 Hz. Raw EEG data was processed with a 0.3-50 Hz bandpass filter with EGI system software. The data was next exported to the custom signal processing software XDiva. This software provided a digital trigger indicating the start of the trial with sub-millisecond accuracy. Artifact rejection was performed in two steps. First, the continuous filtered data were evaluated according to a sample-by-sample thresholding procedure to locate consistently noisy sensors. These channels were replaced by the average of their six nearest spatial EEG channels. Once noisy channels were interpolated in this fashion, the EEG was re-referenced from the Cz reference used during the

recording to the common average of all sensors. Second, 1 s EEG epochs that contained a large percentage of data samples exceeding the threshold (30–80 microvolts) were excluded on a sensor-by-sensor basis. Afterwards, baseline correction 1333 ms prior to trial onset was performed to further increase the signal-to-noise ratio and reduce the effect of drifting. Finally, ssVEP amplitude and phase were calculated at the 2nd, 4th, 6th and 8th harmonic by a recursive least squares adaptive filter [**?**], with weights for each component of complex values for each harmonic frequency analyzed (2F - 8F).

To reduce the 128-channel data into interpretable, physiologically plausible linear components, we applied Reliable Components Analysis (RCA) adapted for SSVEPs (**Figure 2A**). Trial-wise complex Fourier coefficients at the selected harmonics were computed for each condition. RCA seeks spatial filters that maximize trial-to-trial covariance (signal reliability) relative to within-trial covariance, formulated as a generalized eigenvalue problem. The resulting eigenvectors (spatial filters) are ordered by the amount of reliability they explain, and their forward models yield physiologically interpretable scalp topographies [Dmochowski et al., 2015]. RCA weights were trained on a group-level concatenated multi-subject sensor-by-feature matrices and then projected back to each participant's data individually through these weights to obtain component amplitudes/phases at each harmonic and contrast. This procedure emphasizes channels that contribute responses with consistent phase-locking across trials, increasing SNR and yielding components that closely match expected visual-cortical topographies. RCA was performed for each hemifield frequency tag independently, filtering at 2F1-8F1 harmonics (6, 12, 18, 24 Hz) and 2F2-8F2 harmonics (7.5, 15, 22.5, 30 Hz) as two separate runs. Depending on the hemisphere of interest, components were learned on RLS-filtered complex value data from either F1 or F2 responses across all sessions recorded (i.e. the same weighted sum of electrodes was used both pre and post SSRP measures). We note that for each hemifield frequency tag in our paradigm, the first RCA component falsely localizes to the ipsilateral occipital electrodes (**Fig. 2 A**), likely due to

dipole cancellation from the inferior and superior occipital sulcus [Ales et al., 2010]. The first reliable component (RC) for each hemifield frequency tag was analyzed.

## Contrast Response Functions

For each participant, trials obtained before and after SSRP were averaged separately. Response amplitudes were then computed as the vector average of complex Fourier coefficients for each contrast and condition at each harmonic, yielding pre- and post-SSRP contrast response functions (CRFs). CRFs were derived for the 2nd, 4th, 6th, and 8th harmonics—the 2nd harmonic containing the primary response due to frequency doubling of the sign-reversing visual stimulus—separately for each session and hemifield. Next, to capture the total steady-state response across harmonics, root-mean-square (RMS) amplitudes were calculated, yielding a phase-independent measure of overall response magnitude. This procedure integrates the contribution of multiple harmonics, providing a stable estimate of neural response strength suitable for comparison across contrasts and experimental conditions. Finally, to enable pooling across participants, post-SSRP CRFs were normalized to the maximum pre-SSRP response amplitude within the same session and hemifield. This procedure scaled all post-induction responses relative to each participant's own peak baseline response, controlling for variability in overall amplitude across sessions.

## Statistical analysis for Linear Mixed-Effects Model

Subject condition averaged RMS-normalized response amplitudes were entered into a linear mixed-effects ANOVA model to evaluate how SSRP-induced changes in VEP amplitude were influenced by attentional deployment, session day, hemifield, and contrast level. A single EEG session contributed 24 data points per participant—six contrast levels recorded before and six after SSRP induction for each hemifield. The model included fixed effects of pre/post, contrast level, hemifield, attentional deployment and session day along with their *n-way* interactions, and accounted for repeated measures within participants by modeling subject-level variability as a

random effect. This approach allowed us to examine multiple experimental factors simultaneously while controlling for inter-individual variability and within-subject dependencies inherent in repeated-measures designs. All statistical analyses were conducted in R (Version 4.4.1; R Core Team, 2023), and the model was fitted using the *lme4* package (Version 3.1.164; [Bates et al., 2015]). Fixed effects included pre/post responses, Attentional Deployment Condition, Hemifield, and Contrast Level. A random intercept for subject was included to account for inter-individual variability across 48 observations per participant.

## Exclusion Criteria

EEG session data was excluded if more than 30% of signal data quality was above participant signal threshold across all contrasts (n = 11). A total of 26 participant's EEG data was analyzed.

# Results

In brief, ssVEP contrast response functions (CRFs) were measured in the potentiated (left) and non-potentiated control (right) hemifields before and after a 10-minute presentation of high-contrast checkerboard stimuli restricted to the potentiated hemifield to induce stimulus-specific response potentiation (SSRP) (**Fig. 1A**). During SSRP induction, participants allocated endogenous covert attention either towards or away from the potentiating stimulus (**Fig. 1B**). Participants completed the two attention conditions on separate days, the order of which was counterbalanced across participants. All reported statistics were derived from a linear mixed-effects-models ANOVA that included pre- and post-induction responses, contrast level, hemifield, attentional deployment condition, and session day as fixed effects, with participant modeled as a random intercept.

**Hypothesis I: Retinotopic and contrast specificity Figure 2B** illustrates averaged responses across all EEG sessions (n = 26) for each hemifield. As shown,

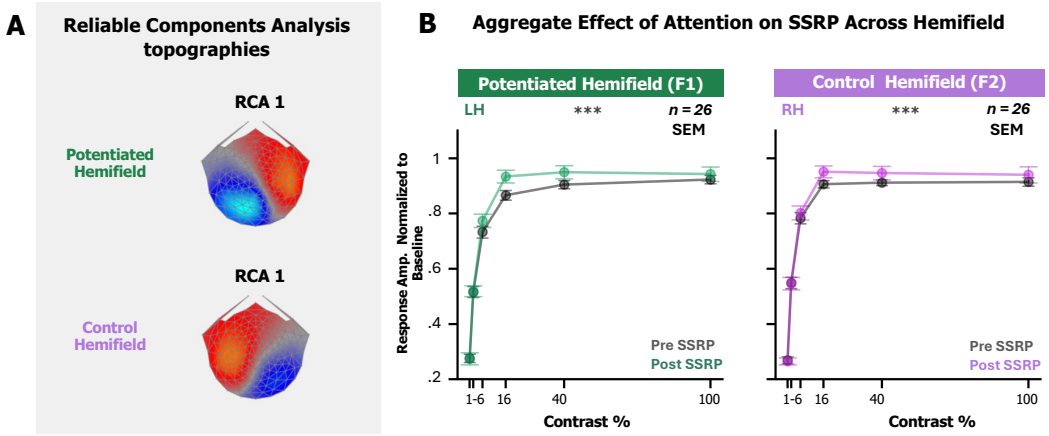


Figure 2:
**A** . Top-view topological maps (nose facing up) show the electrode weights obtained from the first RCA component for each hemifield stimulus. Notably, for each hemifield frequency tag, the first RCA component falsely localizes to the ipsilateral occipital electrodes, presumably due to dipole cancellation between the inferior and superior occipital sulci. **B** Mean contrast response functions before (black) and after (colored) the induction block in the potentiated (green) and control (purple) hemifields, data pooled across attentional conditions and session days. The y-axis indicates response amplitude normalized per-participant to pre-SSRP response-max, and the x-axis represents visual contrast. ** F = 10, $p = 0.002$, Linear mixed-effects-models ANOVA Pre vs. Post SSRP. Error bars depict s.e.m., n=26 participants.

post-SSRP responses (green for the potentiated hemifield, purple for the control hemifield) are greater than baseline (black). Consistent with this observation, our model revealed a significant overall effect of SSRP, with response amplitudes increasing after SSRP compared to baseline (F=10, p=0.002). However, contrary to previous reports, VEP potentiation was neither contrast-specific (*PrePost* × *Contrast:* $p$=0.16) nor hemifield-specific (*PrePost* × *Hemifield:* $p$=0.56), weighing against **Hypothesis I** that SSRP demonstrates LTP-like input specificity.

**Hypothesis II: Effect of attention** To visualize the effects of attentional deployment, we plotted average pre- and post-SSRP responses for each hemifield during attend-toward and attend-away conditions, irrespective of the session day on which each condition occurred (**Fig. 3A**; $n = 26$). In the potentiated hemifield (green), post-SSRP responses increased and did so similarly across attention conditions (**Fig 3A**, panels 1 and 2). In the control non-potentiated hemifield (purple), post-SSRP amplitudes also increased, with a non-significant trend toward greater increases in the Attention-Congruent condition (**Fig 3A**, panel 3) compared to the Attention-Incongruent condition (panel 4) (*PrePost* × *Attention* × *Hemifield interaction*: $p = 0.46$). There was no overall effect of attention on SSRP (*PrePost* × *Attention*: F = 0.98, $p = 0.32$). Collectively, these results suggest that, contrary to **Hypothesis II**, attention had little measurable effect on SSRP expression (but see **Interaction** section below).

**Hypothesis III: Accumulation across days** Although animal studies have shown that SSRP can accumulate across days [Frenkel et al., 2006, Cooke and Bear, 2011], repeated sessions of SSRP have not been previously performed in humans to our knowledge. We therefore plotted responses across Session Days 1 and 2, independent of the attentional condition completed during that session (**Fig. 3B**, $n = 26$). We observed a striking enhancement in post-SSRP response amplitudes in Session 2 compared to Session 1 (**Fig. 3B**, panels 2 and 4), supporting **Hypothesis III** that SSRP accumulates across repeated sessions. This session-day effect was statistically significant (*PrePost x SessionDay*: F(1, 0.1) = 4.45, $p = 0.035$) and

was observed in both hemifields, indicating no retinotopic restriction of potentiation ( *PrePost x SessionDay x Hemifield*: $p = 0.46$). Although amplitude increases appeared more pronounced at intermediate-to-high contrasts, this effect was not significant (*PrePost x SessionDay x Contrast* interaction: $p$=0.52). No significant three-way interactions were observed with Hemifield (*PrePost × Session Day × Hemifield:* $p = 0.46$) or Contrast Level (*PrePost × Session Day × Contrast Level:* $p = 0.52$), suggesting that the potentiation effect was neither retinotopically nor input specific across session days.

The increase in SSRP on Session Day 2 could be due to either a *decrease* in the pre-SSRP response or an *increase* in the post-SSRP response, as in our initial analysis responses were normalized to the pre-SSRP response maximum on each day. To distinguish these possibilities, we re-normalized each participant's CRFs across both sessions to their Session Day 1 pre-SSRP max response amplitude (**Supp. Fig. 2**). **Supp. Fig 2B** shows that increased SSRP on Session Day 2 reflected a combination of a slight *decrease* in pre-SSRP amplitude and a modest *increase* in post-SSRP amplitude relative to Session Day 1 (**Supp. Fig 2B**, panel 1).

We also examined whether the interval between sessions influenced the magnitude of SSRP accumulation. Although there was a weak trend toward greater potentiation on Session Day 2 when sessions were spaced more than four days apart (**Supp. Fig. 4**), an ANOVA comparing pre- to post-SSRP response changes across interval groups revealed no significant effect.

**Interaction between attention and session day** Interestingly, the mixed-effects models ANOVA detected a significant three-way interaction between PrePost, Attentional Deployment, and Session Day ($F(1, 0.15) = 8.79$, $p = .003$). This finding suggests that the sequence of attentional conditions (Day 1 Congruent, Day 2 Incongruent versus Day 1 Incongruent, Day 2 Congruent) influenced response potentiation. To visualize this interaction, data were separated by counterbalanced groups and pre- post- SSRP CRFs were plotted by attentional condition and session day for each hemifield and attention condition (**Fig. 3**). **Figure 3A** depicts data

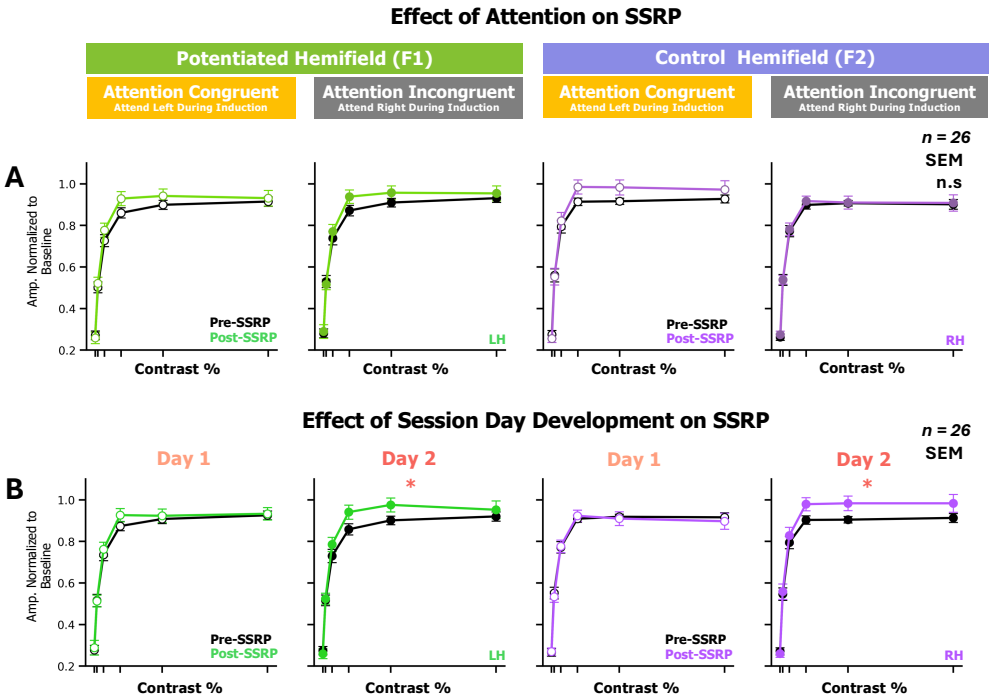


Figure 3:
**A** Average contrast-response functions (n = 26, Mean ± S.E.) measured pre-SSRP (black) and post-SSRP, (green/purple) are shown for two attentional deployment conditions: Attention-Induction Congruent (attending toward the high-contrast induction stimuli during SSRP; panels 1 and 3) and Attention-Induction Incongruent (attending away; panels 2 and 4), in the potentiated (F1, green) and control non-potentiated (F2, purple) hemifields. The y-axis represents amplitude normalized per-participant to pre-SSRP response-max, and the x-axis denotes contrast level. Attention did not robustly modulate SSRP ($PrePost \times Attention$: $p = 0.32$). **B** Contrast-response functions, arranged as in section A, but split by session day (n = 26, Mean ± S.E.). Day 1 responses are open circles (panels 1 and 3); Day 2 responses are filled circles (panels 2 and 4). Potentiation was greater on Day 2, * $p = 0.038$, $PrePost \times SessionDay$.

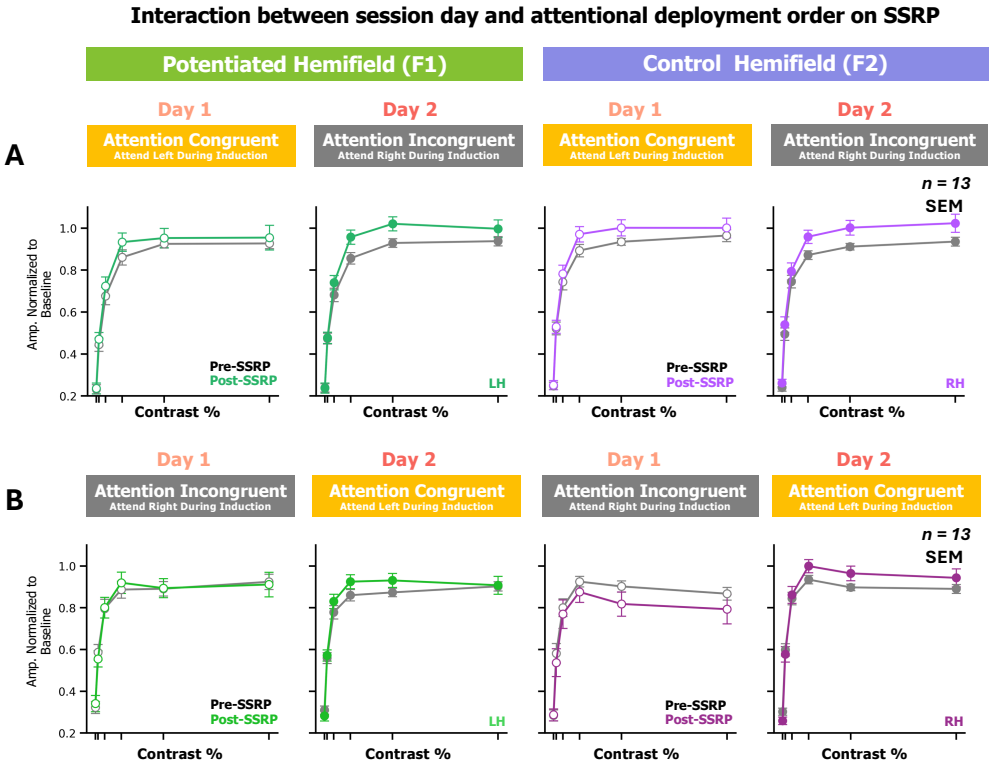


Figure 4: Participants were separated into two counterbalanced groups based on the order of attentional deployment conditions completed across session days. The y-axis represents amplitude normalized to baseline, and the x-axis denotes contrast level. **Group A** (n=13, top row) completed the Congruent condition (attending toward the induction stimulus during SSRP) for the 1st session and Incongruent (attending away from the induction stimulus) for the 2nd session. **Group B** (n=13, bottom row) completed the conditions in the reverse order, (Incongruent first, Congruent second). Session Day 1 is denoted by white filled dots along the CRF (cols. 1 and 3), while Session Day 2 is represented by filled dots (cols. 2 and 4). Hemifield is depicted in green for potentiated (F1) (cols. 1 and 2) and purple for non-potentiated (F2) (cols. 3 and 4). Attentional deployment conditions are denoted by the yellow and gray headers. On Session Day 1 (cols. 1 and 3), response amplitudes increased in the Congruent condition (top row, panels 1 and 3) but not the Incongruent condition (bottom row, panels 1 and 3), with a trend toward a paradoxical decrease in the response in the non-potentiated hemifield in this condition. On Session Day 2 (cols. 2 and 4), response amplitudes increased regardless of the attention condition in both hemifields.

from **Group A**, participants who completed the Attention-Congruent condition during Session 1 and the Attention-Incongruent condition during Session 2 (n = 13). **Figure 3B** shows **Group B**, who completed the sessions in the opposite order (n = 13). On Session Day 1, response amplitude increases post-SSRP were observed in **Group A** in both hemifields (**Fig. 3A**, panels 1 and 3), while post-SSRP responses in **Group B**, showed no potentiation on the first session day (**Fig. 3B**, panels 1 and 3). Notably, in **Group B**, CRFs in the non-potentiated hemifield (**Fig. 3B**, panel 3) showed *decreased* responses post-SSRP (in purple), suggesting a reduction in response driven by attending *away* from the SSRP-inducing stimulus. On Session Day 2, post-SSRP response potentiation emerged in both groups, in both hemifields, indicating that potentiation became more consistent across conditions on the second session (**Fig. 3**, cols. 2 and 4). This pattern conditionally confirms **Hypothesis II**, indicating that attention enhances the expression of SSRP in naive observers, but accumulated affects across days predominate and occlude any attentional effects in subsequent days of plasticity induction.

# Discussion

The primary finding of this study was a significant three-way interaction between PrePost, Attentional Deployment, and Session Day, indicating that attention enhances SSRP in naive observers (Hypothesis II confirmed), but across-day accumulation of SSRP effects predominates in experienced observers (Hypothesis III confirmed) and occludes any attentional modulation of visual cortical plasticity. During the initial exposure, directing attention toward the potentiating stimulus during the initial exposure facilitated SSRP, whereas attending away from the potentiating caused a paradoxical reduction of response in the control hemifield. However, the enhancement of post-SSRP responses across both attentional conditions and hemifields conditions on the second day indicates that repeated exposure played the dominant role in facilitating potentiation across sessions. Finally, contrary to some prior reports and our **Hypothesis I**, in our paradigm SSRP was not retinotopic- or

contrast-specific.

Although animal studies of SSRP have shown exceptional input specificity as reflected by spatial frequency and orientation selectivity [Cooke and Bear, 2010, Kaneko et al., 2017], and a related form of SSRP called tetanic stimulation does show reports of retinotopic and orientation specificity in humans [Teyler et al., 2005, McNair et al., 2006], Normann et al.'s (2007) paradigm which elicits SSRP in humans, has not been systematically tested for input specificity. In our hemifield-specific design, we found no strong evidence of input specificity, as SSRP occurred in both hemifields despite induction being restricted to the left hemifield. To our knowledge, this is the first study to evaluate contrast-level–dependent SSRP effects in humans using a repeated-exposure paradigm analogous to animal models of SSRP. Although response potentiation appeared somewhat stronger at intermediate-to-high contrasts, this trend did not reach statistical significance, indicating no reliable contrast-dependent input specificity under the present protocol. The broad distribution of responses may reflect the absence of channel-specific pathways selectively engaged by simple contrast manipulations in early visual cortex. Our results tentatively suggest that stimulus-specific response potentiation may be a misnomer in the case of visual cortical plasticity induced by 10 minutes of repetitive sensory stimulation in humans. Future work employing spatial-frequency or orientation sweeps could more directly probe feature-specific channels and permit finer comparisons with rodent SSRP studies.

Plasticity processes are known to unfold gradually across sessions or days, as shown in spaced LTP paradigms [Frenkel et al., 2006, Cooke and Bear, 2010], and multi-day perceptual learning studies reporting large gains in perceptual sensitivity with repeated training [Furmanski et al., 2004, Cooke and Bear, 2010, Hua et al., 2010, Kaneko et al., 2017]. Consistent with this literature, our findings suggest that SSRP may increase across days in human participants. There is a rough similarity between our findings of enhanced SSRP on day 2, to the accumulation of response amplitude enhancements observed across multiple days of SSRP effects in rodents

[Frenkel et al., 2006]. However, the in-rodent studies did not split their measures into a pre- and post-SSRP analyses, rather pooling all responses per day to show a net increase in response amplitude across days. Our observation in contrast, shows a larger within-day change on the second day of recording, and we do not in fact see an overall increase in Session Day 2 responses when normalizing to Session Day 1 (**Supp. Fig. 2B**). The precise relationship between the across-day plasticity effects we report here and previous in-rodent studies will require clarification in future studies.

While attention modestly facilitated SSRP expression on Session Day 1, its influence diminished by Session Day 2 despite stronger overall potentiation, highlighting a dissociation between transient attentional modulation and sustained, experience-driven plasticity. This pattern suggests that attention may enhance early stages of plasticity, particularly in naïve circuits, but that repeated stimulation can elicit lasting changes independently. Rodent studies have posited a potential cholinergic contribution to attention-driven modulation of SSRP [Kaneko et al., 2017], which could be assessed in our paradigm with co-administration of acetylcholine modulators like donepezil [Rokem et al., 2010], an interesting avenue for further research.

One unexpected observation that deserves further exploration is the decrease in response amplitude within non-potentiated hemifield on Session Day 1 in the Incongruent condition (See **Fig. 3 B**, panel 3). This effect could reflect a non-local influence of the induction stimulus or, more intriguingly, a long-term depression (LTD)-like process driven by the low-contrast (2%) control stimulus that is modulated by attention. The consensus view in the field is that a high-contrast stimulus is necessary to induce SSRP, although we are not aware of a study that assessed VEP plasticity effects of repeated presentations of low contrast stimuli. A possibly analogous attentional modulation of LTD-like plasticity has been observed in the motor cortex [Kamke et al., 2012, 2014]. Future experiments can be designed to distinguish these different possibilities.

The paradigm used in the current study was primarily inspired by the protocol

originally described by Normann et al. [Normann et al., 2007] and shown to have a robust effect size in the large-scale study conducted by the Elvsåshagen group [Valstad et al., 2020]. For the sake of comparison, the distinctions between the two protocols will be delineated here. In the Normann et al. paradigm, the pre- post- SSRP stimulus consisted of a full-field 100% contrast checkerboard stimulus, 0.5° spatial frequency, sign reversing at 2 repetitions per cycle (rps). The current study's pre- post-SSRP stimulus differed in that checkerboards (approximately 0.5° SF) were semicircular extending to 10° with checks eccentricity scaled; stimuli were presented at 6 and 7.5 Hz; and contrast was swept from 2 to 100% contrast. The induction stimulus of Norman et al. [Normann et al., 2007] was the same stimulus as pre/post but presented continuously for ten minutes. The current study's induction stimulus was similar to our pre/post stimulus but with a sign reversal rate and contrast matching Norman et al (2 Hz and 100%), but differed in that it was only presented in the left hemifield. In Normann et al., there was a single baseline 20 second trial, and 20-second post- trials were presented at 2, 8, 12, 18, 22, and 28 min after the SSRP block. In the current study, in contrast, thirty-nine 10.66-second trials were presented pre- and post-, and post- stimuli were presented starting 3-4 minutes after the end of the SSRP block. We note that our paradigm may have missed delayed phases of potentiation that emerge tens of minutes later [**?**].

**Limitations** While this study advances our understanding of stimulus-specific response potentiation (SSRP), it also highlights several limitations that inform future work. **1**) Our study did not include eye-tracking, so we are not able to precisely verify fixation in the induction block. Weighing against the possibility of this impacting our results, a post-hoc analysis of the electrooculogram recordings indicated that horizontal eye movements during the induction block were minimal. Moreover, our primary observed effect of enhanced response potentiation on the Session Day 2 (**Fig. 3B**) would not be affected by issues with fixation. **2**) Participants may not have been strongly allocating attention in the task. We think this is not a major contributor to our results, because we assessed task response consistency as a rough

corollary of attention, and when we excluded participants who were not performing the task consistently, it did not alter the results. However, in future studies, a more rigorously designed attentional task with better behavioral measures and eye tracking may be more sensitive at detecting attentional effects on SSRP. **3**) The behavioral tasks implemented used during the pre-/post- blocks were primarily designed to enforce fixation rather than probe SSRP-related changes, which limits their usefulness in capturing behaviorally relevant effects of SSRP. In future studies a contrast discrimination task at threshold could be performed by participants pre-post- SSRP to compare electrophysiological plasticity effects to perceptual plasticity effects. **4**) Participants were not actually allocating attention to the potentiating stimulus. Instead they were allocating attention to the retinotopic area that encompassed the SSRP stimulus, but were trying to detect small patches of subtle contrast change within that retinotopic extent. From this perspective, the potentiating stimulus may serve as a distractor in relation to the attended targets. Therefore the attentional system may have been trying to suppress or filter out responses to the potentiating stimulus in our paradigm, which could paradoxically suppress SSRP effects. This interesting possibility will be assessed as well in future experiments. **5**) The low-contrast stimulus presented in the control right hemifield during induction may have elicitepd its own plasticity effects (as described above). If the low-contrast control stimulus in our paradigm did induce some form of plasticity, perhaps this is evident in the attention-dependent Session Day 1 response depression we observed. Future studies can assess this interesting possibility.

Finally, we can provide no comprehensive mechanistic theory to explain how variables such as stimulus contrast, repetitions per cycle, stimulus type, and eccentricity degree facilitate SSRP susceptibility. The design of our SSRP experiment remains largely empirical, with no theoretical framework to account for how temporal frequency or duration of SSRP blocks impacts the magnitude or persistence of response enhancement. This gap in mechanistic understanding highlights the critical need for further investigation into the visual system's plasticity mechanisms and

their interactions with specific stimulus properties overall.

# Supplemental Section

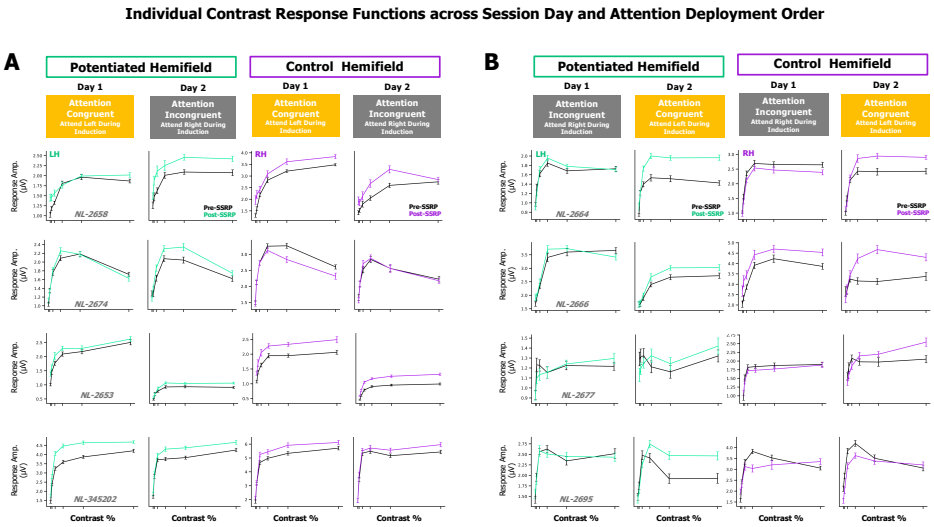


Figure 5: Individual RMS Contrast Response Functions measured Pre-SSRP (black) and Post-SSRP (colored) development for the potentiated (F1) and control (F2) hemifields across Session Day 1, Session Day 2 and the Attentional Deployment condition completed for the respective Session Day. The y-axis represents amplitude, and the x-axis denotes contrast level. Individual data are divided into two groups based on the counterbalanced order of attentional deployment conditions: **Group A** completed the **Attention SSRP Congruent condition for the 1st session** (attending toward SSRP stimuli), followed by completing the **Attention SSRP Incongruent condition** (attending away from SSRP stimuli) **for the 2nd session**. **Group B** completed the conditions in the reverse order, (**Incongruent first, Congruent second**). Each row represents a single individuals non-normalized contrast response functions (mean $\pm$ S.E) pre-SSRP (black) and post-SSRP (green, purple) across both sessions, within both hemifields. The primary headers denote what hemifield CRF's were recorded from the potentiated and control hemifield (green, purple), Session day and Attentional deployment are denoted by the secondary headers. Beneath the yellow headers represent the responses obtained during the Attention SSRP Congruent condition, beneath the gray headers represent the responses obtained during the Attention SSRP Incongruent condition. **A** The first two columns show contrast response function development Pre-SSRP (black) and Post-SSRP (green) from Session Day 1 (attending towards SSRP stimuli) to Session Day 2 (attending away from SSRP stimuli) in the potentiated hemifield. The final two columns show contrast response function development pre-SSRP (black) and post-SSRP (purple) from Session Day 1 to Session Day 2 in the control (non-potentiated hemifield). **B** In the same fashion, the first two columns show contrast response function development pre-SSRP (black) and post-SSRP (purple) from Session Day 1 (attending away from SSRP stimuli) to Session Day 2 (attending towards SSRP stimuli) in the potentiated hemifield. The final two columns show contrast response function development pre-SSRP (black) and post-SSRP (purple) from Session Day 1 to Session Day 2 in the control (non-potentiated hemifield). Overall between both groups, potentiation Post-SSRP on the second is much more pronounced compared to first session responses for both hemifields regardless of attentional deployment order, especially at higher contrasts (cols 1 and 4).

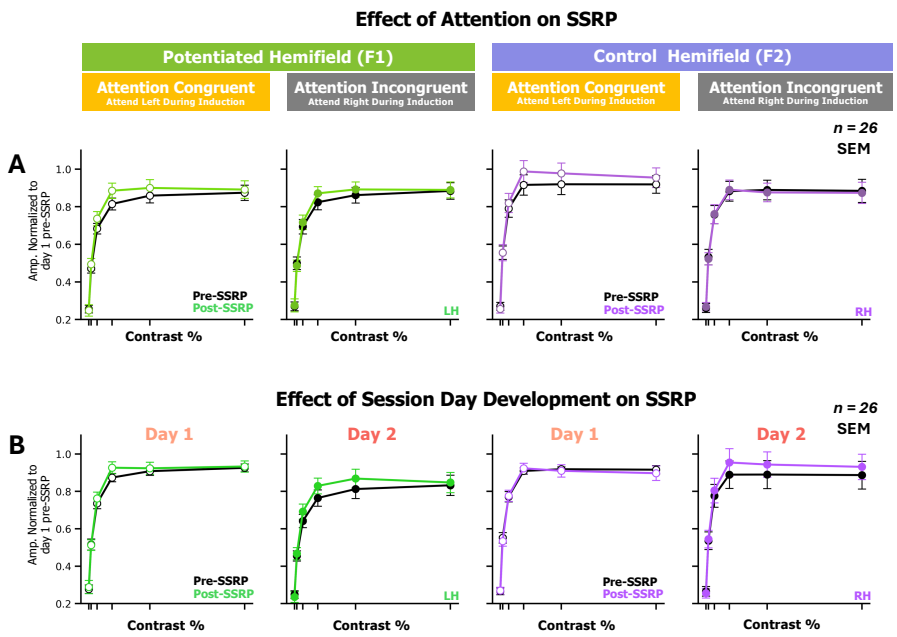


Figure 6: Session Day 1 Max-Pre Normalized CRFs by Attention Deployment Order and Session Day. The y-axis represents amplitude normalized to Session Day 1 baseline, and the x-axis denotes contrast level. **A** Average contrast-response functions (Mean $\pm$ S.E.) measured pre-SSRP (black) and post-SSRP (green/purple) are shown for two attentional deployment conditions: Attention-Induction Congruent (attending toward the high-contrast induction stimuli during SSRP; panels 1 and 3) and Attention-Induction Incongruent (attending away, panels 2 and 4), in the potentiated (F1, green) and control non-potentiated (F2, purple) hemifields. Attention did not robustly modulate SSRP. **B** CRFs, arranged as in row A, but split by session day. Day 1 responses are open circles (panels 1 and 3); Day 2 responses are filled circles (panels 2 and 4). while the trend remains the same as in Fig 3 of potentiation Post-SSRP being greater on Session Day 2 this effect was but not statistically significant with this normalization technique applied.

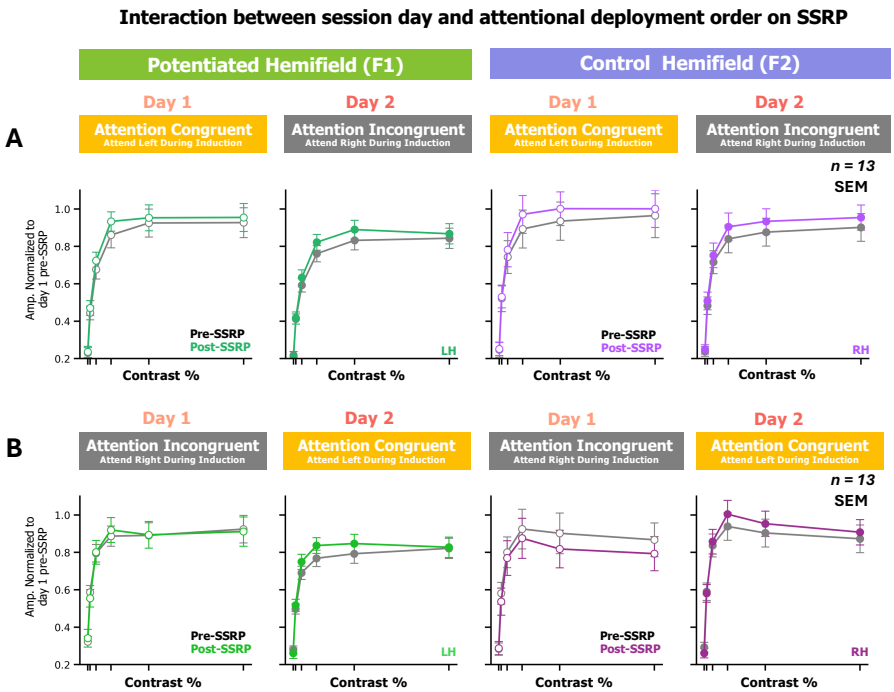


Figure 7: Session Day 1 Max-Pre Normalized CRFs Interaction between session day and attentional deployment order on SSRP. The y-axis represents amplitude normalized to Session Day 1 baseline, and the x-axis denotes contrast level. Participants were split based on the order in which they completed the two attentional deployment conditions across the two session days:**Group A** completed the Congruent condition (attending toward the induction stimulus during SSRP) for the 1st session and Incongruent (attending away from the induction stimulus) for the 2nd session. **Group B** completed the conditions in the reverse order (**Incongruent first, Congruent second**). Session Day 1 is denoted by white filled dots along the CRF, while Session Day 2 is represented by filled dots. Hemifield is depicted in green for potentiated (F1) and purple for non-potentiated (F2). Attentional deployment conditions are denoted by the yellow and gray headers. On Session Day 2 (2nd and 4th columns), response amplitudes increased in both hemifields and both attention conditions, supporting the trend as seen in **Figure 3A**, however on Session Day 2, responses Pre-SSRP are smaller in magnitude compared to Session Day 1 Pre-SSRP suggesting that some level of adaptation and plasticity affected CRF development across the first and second session day. The response change Pre-Post SSRP was not statistically significant. Additionally, we can see that Session Day 1 (cols. 1 and 3), response amplitudes increased in the Congruent condition (top row) but not the Incongruent condition (bottom row), with a trend toward a paradoxical decrease in the response Post-SSRP in the non-potentiated hemifield in this condition, supporting the trend as seen in **Figure 3B**, however the overall response change pre-post SSRP was again, not statistically significant.

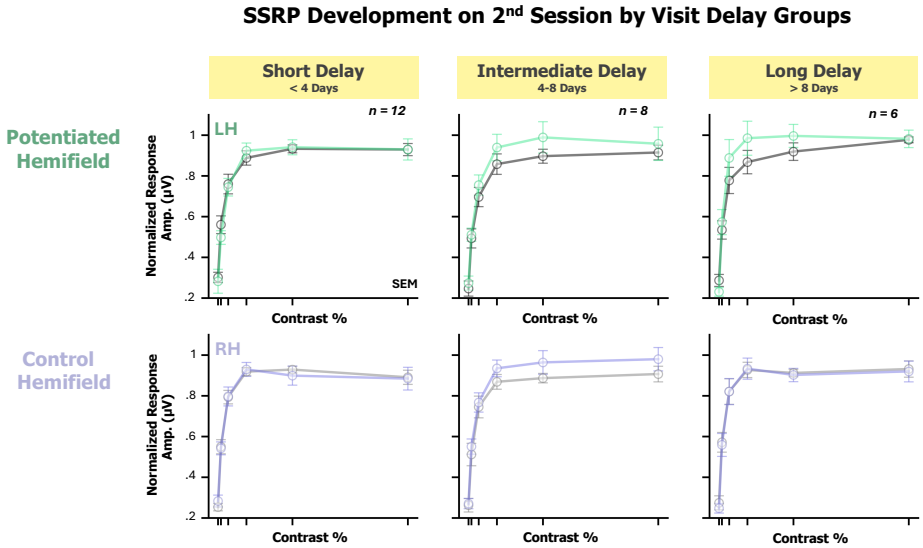


Figure 8: Session Day 2 contrast-response functions (Mean ± S.E.) measured pre-SSRP (black) and post-SSRP, (green/purple) are shown based on different session delay visit groups: Short Delay (2nd visit was less than 4 days, 1st column), Intermediate Delay (2nd visit was 4-8 days, 2nd column), Long Delay (2nd visit was more than 8 days later, 3rd column). The y-axis represents amplitude normalized baseline, and the x-axis denotes contrast level. The first row shows responses Pre-Post SSRP in the potentiated hemifield, regardless of attentional deployment condition. The second row shows responses Pre-Post SSRP in the control hemifield in the respective day delay group. In the short delay group there was no pre- post- SSRP change in either hemifield (col. 1). In the short and intermediate delay groups, on the second session day, post-SSRP response enhancement can be observed in both hemifields in the intermediate group (col. 2) and only in the potentiated hemifield in the long delay group (row 1, panel 3), indicating a robust yet spatially specific SSRP effect dependent on session day delay. However, this pattern did not reach statistical significance, and the current study protocol was not structured to manipulate inter-session delay systematically, given the limited sample size for each group.

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