Binocular Modulation of V1 Activity

Monocular Viewing vs. Binocular Viewing

V1 Spiking Activity

Stimulus Contrast

Time

Facilitation

Suppression

Binocular - Monocular (normalized)

50-150ms

150-250ms

Response

Contrast
Stimulating both eyes with matching stimuli 
enhances V1 responses

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Summary

Neurons in primate primary visual cortex (V1) play a key role in combining monocular inputs to form a binocular response. While much has been gleaned from studying how V1 responds to discrepant (dichoptic) images, equally important is to understand how V1 responds to concordant (dioptic) images in the two eyes. Here, we investigated the extent to which concordant, balanced, zero-disparity binocular stimulation modifies V1 responses to varying stimulus contrast using intracranial multielectrode arrays. On average, binocular stimuli evoked stronger V1 activity than their monocular counterparts. This binocular facilitation scaled most proportionately with contrast during the initial transient. As V1 responses evolved, additional contrast-mediated dynamics emerged. Specifically, responses exhibited longer maintenance of facilitation for lower contrast and binocular suppression at high contrast. These results suggest that V1 processes concordant stimulation of both eyes in at least two sequential steps: initial response enhancement followed by contrast-dependent control of excitation.
Introduction

Neurons in primary visual cortex (V1) play a key role in combining monocular information to produce a stable, binocular percept (Barton, 2004). Neurophysiological studies have provided rich insight into the flow of monocular signals and emergence of binocular responses within the primary visual pathway (Bishop and Pettigrew, 1986; Blake and Wilson, 2011; Burkhalter and Essen, 1986; DeAngelis and Newsome, 1999; Ghose and Ts’O, 1997; Henriksen et al., 2016; Horton, 2006; Lehky and Maunsell, 1996; Livingstone and Tsao, 1999; Maunsell and Essen, 1983; Pack et al., 2003; Parker et al., 2016; Parker and Cumming, 2001; Poggio, 1995; Poggio and Fischer, 1977; Schroeder et al., 1990; Smith et al., 1997; Yang et al., 2011). However, many studies of binocular combination have been geared towards understanding how the visual system responds to images that are somewhat discrepant between the two eyes, i.e., dichoptic viewing conditions. Less is understood about how V1 responds under dioptic viewing conditions, where binocular images are physically identical and fall on corresponding retinal positions.

As a matter of subjective experience, the advantages of viewing the same image with both eyes are somewhat elusive (Levelt, 1965). The simple experiment of opening and closing one eye does not elicit a dramatic change in perception. Yet, decades of research have revealed a binocular advantage in numerous psychophysical experiments (For review, see Blake et al., 1981; Blake and Fox, 1973). Psychophysical gains in performance under binocular viewing are commonly referred to as “binocular summation” (e.g., Cagenello et al., 1993). It has now been well established that binocular summation extends beyond than what would be expected from having an additional detector (i.e., probability summation) (For meta-analysis, see Baker et al., 2018; Matin, 1962; PIRENNE, 1943). Thus, it is likely that binocular summation is facilitated by a neural interaction.

The neurophysiological basis for binocular summation is thought to be an enhancement of neural responses along the primary visual pathway (Blake and Fox, 1973). Electrophysiological and neuroimaging techniques have demonstrated neural binocular summation in human V1 (Apkarian et al., 1981; Heravian et al., 1990; Hou et al., 2020; Moradi and Heeger, 2009; Pardhan et al., 1990). While enhanced compared to responses to just one eye, V1 binocular responses are typically much less than the sum of comprising
monocular responses (Ates et al., 2006; Giuseppe and Andrea, 1983; Heravian et al., 1990; Moradi and Heeger, 2009), akin to our experience of seeing with two eyes. The mechanisms of this sublinear/partial summation are not completely understood. However, modern models of binocular combination and theoretical frameworks for neural response normalization have made great inroads into the regulatory processes that facilitate and control binocular summation (Blake, 1989; Carandini and Heeger, 2012; Ding and Levi, 2021; Georgeson and Sengpiel, 2021; Hou et al., 2020; Ling and Blake, 2012; Meese et al., 2006; Said and Heeger, 2013). Foundational to such models is the parameter of stimulus contrast.

V1 neurons respond to a dynamic range of visual contrasts (Clatworthy et al., 2003). The relationship between stimulus contrast and a V1 neuron’s response is typically well-described by a sigmoidal contrast response function (CRF) (Albrecht and Hamilton, 1982; Ohzawa et al., 1985; Sengpiel and Blakemore, 1994). By measuring the ‘shifts’ in the contrast-response relationship from one sensory condition to the next, experimenters have distinguished contrast-dependent and contrast-independent mechanisms that control V1 excitation (Heuer and Britten, 2002; Sengpiel et al., 1998). For example, the addition of an orthogonal grating in the opposite eye suppressed V1 neurons in a manner that shifted the CRF down along the response-axis, rather than the contrast-axis. To the best of our knowledge, shifts in V1’s CRF under the much more common visual experience of dioptic stimulation have not been fully characterized using multi-unit electrophysiological methods.

Here, we studied the time-varying relationship between V1 binocular (dioptic) response modulation and stimulus contrast. We found that V1 population spiking activity was overall greater for binocular stimulation than for monocular stimulation, but binocular responses were considerably less than the left and right eye responses summed. The relationship between binocular modulation and stimulus contrast was dynamic. V1 binocular responses exhibited at least two sequential steps of gain modulation over monocular viewing: initial, rapid (50-100ms) summation that was more contrast-invariant followed by slower, contrast-dependent processing, all within the timeframe of a typical fixation (250 ms) (Salthouse and Ellis, 1980).
Results

We were interested in determining how V1 activity differs between stimulating one eye versus stimulating both eyes with the same image as a function of contrast. To find out, we presented sinusoidal, achromatic gratings through a mirror stereoscope (Figure 1A) either monocularly or binocularly (Figure 1B) to fixating monkeys. Gratings varied in Michelson contrast between trials [0, 0.20-0.225, 0.40-0.45, 0.80-0.90]. We used microelectrode arrays to simultaneously record extracellular voltages at multiple V1 sites. Thresholding the voltage at each site yielded a measure of neuronal activity (discretized multi-unit, see Methods for details). For each unit, we distinguished the eye that drove V1 activity the most as the dominant eye (DE; Figure 1C, dark blue) compared to the non-dominant eye (NDE; Figure 1C, light blue).

Sampling in this study varied with monkey and condition (Table 1). As a result, population measures of neuronal activity reported below include varying number of units from each monkey depending on the conditions being considered. For transparency, single-animal results are provided in tables or supplemental figures. Potential consequences of sampling-bias, which is not uncommon for studies of this kind, are considered in Discussion.

Binocular V1 responses exceed the average of monocular responses

Prior neurophysiological and imaging studies found that binocular responses constitute less than the linear sum of the converging left and right eye signals (Apkarian et al., 1981; Moradi and Heeger, 2009; Smith et al., 1997). We were curious to see if this was the case for our data as well.

Figure 1D shows each unit’s mean binocular (BIN) responses (0-250ms) plotted against the sum of its respective monocular responses (DE + NDE). The unity diagonal represents the expected binocular response if binocular summation is linear. Across our sample of multi-units (Table 1), binocular responses were approximately 56% of the summed monocular responses on average (low contrast, linear regression slope, $M = 0.55, SE = 0.011, 95\% CI [0.52,0.57], p = 1.7378e^{-130}$; med contrast, $M = 0.56, SE = 0.009, 95\% CI [0.54,0.58], p = 7.0530e^{-173}$; high contrast, $M = 0.57, SE = 0.009, 95\% CI [0.55,0.59], p = 5.8719e^{-177}$, see Table 2 for breakdown by animal; see also Supplemental Figure 2) and approximately 82% of the
quadratic sum. To control for variance in maximum firing rate between units, we calculated an additivity index at each contrast. The additivity index was derived by dividing the mean binocular response of each unit by the sum of its comprising monocular responses \([\text{BIN}_n / \Sigma(\text{LE}_n+\text{RE}_n)]\). An index value of 1 signifies that the binocular response was equivalent to the sum of comprising monocular responses. Similar to the slope coefficients, the mean additivity indexes were less than 1 but greater than 0.5 (low, \(M = 0.58, SD = 0.09, 95\% \text{ CI} [0.57,0.59]\); med, \(M = 0.59, SD = 0.08, 95\% \text{ CI} [0.58,0.60]\); high, \(M = 0.58, SD = 0.07, 95\% \text{ CI} [0.57,0.59]\)). Additivity indices did not significantly differ across contrast (Mixed model, \(F(869, 1) = 1.64, p = 0.2008\)). Thus, at the level of V1 population spiking, binocular responses show sublinear summation.

Facilitation of V1 spiking responses to balanced binocular stimulation

The above analysis replicated previous findings, showing that V1 responses tend to increase whenever a stimulus is shown to both eyes rather than one eye alone (akin to binocular summation). Given this expectation of a larger binocular response, another popular measure is to compare binocular stimulation to stimulation of whichever eye evokes the stronger response. We thus quantified this effect as well. To do so, we computed an index of “binocular modulation” as the difference in mean spiking response (0-250ms) when both eyes are stimulated, referenced to the strongest monocular (dominant eye, DE) response (see Methods and Figure 2A).

Binocular modulation index values above zero indicate binocular facilitation, whereas values below zero indicate binocular suppression. For each unit, we first pooled the data across contrast levels to achieve a grand average of V1 binocular modulation. We derived mean spiking responses over 250 ms (full duration of stimulus presentation). Figure 2B displays the grand average binocular modulation index for each unit. Across the population, binocular modulation was significantly greater than zero (\(M = 0.057, SD = 0.067, \text{ paired t-test}, t (313) = 14.95, p < 0.001, \text{ Cohen’s } d = 0.843\), see Table 3 for breakdown by animal). More than two thirds of V1 units (219 units, 69.7%) were overall facilitated when both eyes viewed the same image relative to monocular viewing.
Next, we examined the extent to which contrast modifies binocular modulation. Figure 2C shows the binocular response (mean spiking from 0-250ms) of each unit against its dominant eye response as a function of stimulus contrast. Distributions of binocular modulation values are shown in the corner histograms. We found that binocular modulation varied across stimulus contrast, Mixed model (N = 314), $F(1, 869) = 15.55, p < 0.001$ (see Table 3 for breakdown by animal). Binocular modulation was strongest for low contrast ($M = 0.034, SD = 0.081, paired t test H_0 \mu > 0, t(247) = 6.62, p < 0.001, Cohen's d = 0.42$) and medium contrast ($M = 0.034, SD = 0.072, paired t test H_0 \mu > 0, t(307) = 8.32, p < 0.001, Cohen's d = 0.47$), and weakest for high contrast ($M = 0.020, SD = 0.070, paired t test H_0 \mu > 0, t(313) = 5.10, p < 0.001, Cohen's d = 0.29$). Thus, while V1 spiking was predominantly facilitated over 250ms of binocular stimulation, this boost in activity was attenuated by stimulus contrast.

Contrast-dependency of facilitation across time

Informed by previous work (Cox et al., 2019), we suspected binocular facilitation in V1 to be transient, i.e., not lasting the entire 250 ms of stimulus viewing. However, the contrast-dependency of this dynamic remains unknown. To investigate the temporal rise and decay of facilitation across contrast, we created spike density functions (SDFs) for monocular and binocular V1 responses and compared them across time. For this analysis, we limited our sample to units for which we had balanced observations for pairwise comparisons between contrasts (N = 242 units, 234 from monkey E, see Table 1).

Figure 3A displays the population SDFs as a function of stimulus contrast (For individual monkey data, see Supplemental Figure 4A and Supplemental Figure 5B). Below each plot is the mean difference in spiking between monocular and binocular stimulation, normalized within-unit for three levels of contrast. Timepoints where binocular facilitation (above zero) was significant are indicated by a horizontal black line above the delta response (two-way paired t test, $p < 0.001$). This time varying SDF analysis revealed that the magnitude of binocular facilitation varied as a function of time at all three contrast levels. Specifically, binocular facilitation was largest at an early phase of the response and decreased over time. To further quantify this effect, we calculated the binocular modulation index over sixteen sequential
temporal windows. As expected, the binocular modulation index varied significantly with time (medium contrast, rmANOVA, $F(15, 3525) = 150, p < 0.001, n^2_G = 0.190$; see also Supplementary Figure 3).

We next estimated the magnitude of facilitation at the transient peak of V1 responses as a function of contrast. Peak magnitude varied across stimulus contrast, (rmANOVA, $F(2, 482) = 24.0, p < 0.001, n^2_G = 0.035$; Figure 3B – top). Low contrast facilitation exhibited peak magnitudes ($M = 0.28, SD = 0.14$) significantly lower than medium ($M = 0.34, SD = 0.16$) and high contrast ($M = 0.35, SD = 0.22$) peaks (low vs medium contrast, post hoc test, $t(241) = -5.74, p_{Bonferroni} < .001, Cohen’s d = -0.37$; low vs. high contrast, post hoc test, $t(247) = -6.15, p_{Bonferroni} < .001, Cohen’s d = -0.39$). Peak magnitude of facilitation at medium and high contrast were not statistically different (post hoc test, $t(241) = -1.53, p_{Bonferroni} = 0.36$). See Table 4 for within-subject pairwise comparisons.

Finally, we measured the duration of facilitation across contrasts. Duration of facilitation was defined, for each unit, as the length in samples between the peak magnitude of facilitation (see above) and the point at which the delta response crossed zero (see Methods for details). We found that duration of facilitation varied across contrast, rmANOVA, $F(2, 482) = 4.74, p = 0.009, n^2_G = 0.012$; Figure 3B – bottom). Facilitation was maintained significantly longer at low contrast (Median = 115.0 ms, $SD = 63.3$) compared to medium (Median = 87.0 ms, $SD = 59.7$) and high contrast (Median = 80.0 ms, $SD = 62.3$) (post hoc test, low vs. medium, $t(215) = 2.80, p_{Bonferroni} = .018, Cohen’s d = 0.19$; low vs. high, $t(215) = 2.45, p_{Bonferroni} = 0.045, Cohen’s d = 0.17$). Note, while both monkeys demonstrated the same trend, Monkey E’s facilitation persisted for ~50ms longer than Monkey I (Table 5). Duration of facilitation at medium and high contrast were not statistically different (post hoc test, medium vs. high, $t(215) = -0.12, p_{Bonferroni} > 0.9999$).

V1 responses at higher contrasts transition from facilitation to suppression.

Results thus far suggest that V1 binocular facilitation is a transient event that evolves over the time course of stimulation in a contrast-dependent manner. To further examine the dynamic relationship between binocular modulation and contrast, we interpolated each units’ contrast responses by fitting measured data with the Naka-Rushton function (Figure 4A, see Methods). V1 units with at least four contrast-level datapoints (234 units in Monkey E, 8 units in Monkey I, see Table 1) were used for fitting contrast response
functions (CRFs). Monocular and binocular CRFs were computed over sixteen sequential temporal windows of the V1 spiking response. Windows were 100 ms in length, each sliding by 10 ms forward in time from the preceding window.

To evaluate V1 CRFs at the population level, we generated average curves for each condition using the mean parameters (and their upper and lower bounds). Figure 4B plots the mean parameter-generated binocular and monocular CRFs as a function of time. Color and contour of the floor reflects the normalized difference between each CRF pair as it evolves over time (pink = facilitation; white = no difference, gray = suppression. Figure 4C displays three windows (50-150 ms, 100-200 ms, and 150-250 ms) for closer inspection. We designated these windows as early, intermediate, and late, respectively. In the early period of the response (50-150 ms), binocular facilitation appeared the most proportional to stimulus contrast. In the intermediate period (100-200 ms), binocular facilitation was diminished at high contrast. By the late period (150-250 ms), binocular facilitation had relinquished entirely. Instead, the late period V1 CRF exhibited binocular suppression at medium and high contrast. Given sampling, this specific population-level analysis is biased towards monkey E (234 units in Monkey E, 8 units in Monkey I, see Table 1), so we additional examined single-penetration from each subject individually (Supplemental Figure 4B and Supplemental Figure 5C). These penetrations exhibit the same overall trend as the population analysis, albeit with larger standard error given the lower number of units.

V1 binocular facilitation predominantly resembles response-gain

We next evaluated the contrast-response relationship in the context of simple forms of gain and gain-control. Two hypothetical types of gain can be gleaned from shifts in the CRF (Ling and Carrasco, 2006; Martínez-Trujillo and Treue, 2002; Ohzawa et al., 1985; Sengpiel et al., 1998; Sengpiel and Blakemore, 1994; Thiele et al., 2009). Response-gain is characterized by vertical shifts in V1’s CRF, indicating that responses increased with contrast by a constant scaling factor. Contrast-gain, on the other hand, is characterized by horizontal shifts in V1’s CRF, indicating a contrast-response relationship that depends on contrast.
To quantitatively evaluate which type of gain (response vs. contrast-gain set) is prevalent at the population level, we directly compared models that isolate the effects of response-gain and contrast-gain. In this procedure, we fixed the parameters of the Naka-Rushton equation to that of the dominant eye. We then introduced a single free parameter (G) to either multiply response (response-gain model, eq. 1 in Figure 5A) or contrast (contrast-gain model, eq. 2 in Figure 5A). Finally, we fit the mean response (over 100ms windows of the response) from the binocular condition with each model and compared their performance.

Figure 5B shows the population-level fitted binocular responses for the two models at the early, intermediate, and late phase of the response. Goodness-of-fit for each model across all sixteen windows are plotted below. Both response-gain and contrast-gain set model performance varied significantly across time (response-gain, rmANOVA, $F(2,482) = 17.8$, $p < 0.001$, $n^2_g = 0.030$; contrast-gain, rmANOVA, $F(2,482) = 40.0$, $p < 0.001$, $n^2_g = 0.065$). Response-gain performed best ($R^2 = 0.90$, 95% CI [0.88, 0.92]) and significantly better than contrast-gain ($R^2 = 0.87$, 95% CI [0.85, 0.89]) during the early phase (50-150ms, window 5) of the response (paired t test, $t(241) = 2.84$, $p = 0.00491$, Cohen’s $d = 0.18$). During the intermediate phase (100-200ms, window 10), response-gain and contrast-gain performances were comparable (response-gain, $R^2 = 0.86$, 95% CI [0.84, 0.89]; contrast-gain, $R^2 = 0.84$, 95% CI [0.82, 0.87]). In the late phase (150-250ms, window 15), performance of these simple models of gain decreased overall (response-gain, $R^2 = 0.81$, 95% CI [0.78, 0.84]; contrast-gain, $R^2 = 0.73$, 95% CI [0.69, 0.77]). Recall that the late stage of the binocular response exhibited suppression (far right Figure 4B). Binocular suppression of V1’s CRF was better captured by response-gain set (paired t test, $t(241) = 6.26$, $p < 0.001$, Cohen’s $d = 0.40$). We note that binocular suppression unfolded differently across time between the two monkeys (see Supplemental Figure 4B). However, in both cases, contrast-dependent suppression was observed following facilitation. Figure 5C shows the normalized parameter differences ($R_{\text{max}}$ and $C_{50}$) of the CRF pairs across time. As expected, differences in $R_{\text{max}}$ corresponded with response-gain performance, while differences in $C_{50}$ corresponded with contrast-gain performance.
To summarize, response-gain (increase in $R_{\text{max}}$) under binocular stimulation was most prominent at the initial peak V1 response (50-100 ms). Contrast-dependent decay of facilitation disrupted response-gain, eventually suppressing V1’s CRF. Although performance of both models was markedly reduced in the late phase (150-250ms) of the V1 response, the binocular suppression we observed was better explained by a reduction to firing rates, i.e., response-gain control.

**Discussion**

We report that V1 spiking responses are predominantly facilitated (i.e., enhanced) by adding the same stimulus at a matching retinotopic position in the other eye. This binocular facilitation was centered around the transient peak of the V1 response. In line with earlier reports (Cox et al., 2019), we find that the later, sustained phase of binocular V1 responses approaches its monocular counterpart, eradicating—and at some contrast levels even reversing—any binocular facilitation. We show that initial binocular processing in V1 is better explained by a model of response-gain as opposed to contrast-gain. However, we found that simple models of contrast-response relationships break down as more complex, contrast-dependent dynamics emerge in the sustained V1 response.

**Relation to prior literature**

Research into the origins of psychophysical gains of binocular vision have pointed to binocular interactions along the primary visual pathway (Blake and Fox, 1973). Early investigations of binocular interactions found great diversity in how V1 simple and complex cells modulate under binocular stimulation (Hubel and Wiesel, 1962). Responses of binocularly activated single cells in striate cortex are shown to be either greater than the sum of monocular responses, greater than the preferred monocular response, or less than monocular responses (Barlow et al., 1967; Burns and Pritchard, 1968; Hubel and Wiesel, 1962; Pettigrew et al., 1968; Poggio and Fischer, 1977). We now understand that this diversity is functional. The type of binocular interactions in single neurons rely chiefly, among other factors, on the compatibility between retinal disparity of the stimulus and the shape of a given cell’s receptive field (Anzai et al., 1999; Barlow et al., 1967; Bishop and Pettigrew, 1986; Cumming and DeAngelis, 2001;
Binocular interactions have also been measured in humans and animals at the spatial and temporal resolutions afforded by EEG and neuroimaging. Visual evoked responses to binocular stimulation are at least 25% greater than the preferred eye or the average of the two monocular responses (Apkarian et al., 1981; Heravian et al., 1990; Sclar et al., 1986; Summa et al., 1997). Similarly, the BOLD signal in V1 is modestly greater for congruent binocular gratings compared to monocular gratings (Moradi and Heeger, 2009).

Here, we report V1 binocular interactions at the intermediate level of neural population spiking activity using multi-unit electrophysiology. We found that V1 binocular responses constituted less than the arithmetic sum of left and right eye responses (sublinear/partial binocular summation). At the same time, V1 binocular spiking was higher than the that of the population’s preferred eye (binocular facilitation). This overall increase in neural population activity for binocular stimulation is in line with the single-neuron experiments in rodents (Longordo et al., 2013; Zhao et al., 2013), cats (Grüna u, 1979; Grüna u and Singer, 1979; Hubel and Wiesel, 1962), monkeys (Poggio and Fischer, 1977), and is consistent with the idea that V1 neurons with near foveal receptive fields tend to show an excitatory bias towards zero-disparity (Poggio and Fischer, 1977). Yet, the magnitude of binocular facilitation was less than that reported in single neurons optimally stimulated with images placed at corresponding retinal positions (Burns and Pritchard, 1968). The magnitude of facilitation reported here is quantitatively closer to estimates from fMRI (Moradi and Heeger, 2009) and EEG (Harter et al., 1973). Population measurements contain neurons with different tuning preferences, which might – at least – partially explain this difference.

In addition to better spatial sampling of binocular interactions, we were also able to assess the temporal dynamics of neural binocular summation at behaviorally relevant timescales (Salthouse and Ellis, 1980). In a recent meta-analysis of psychophysical binocular summation, a negative correlation was found between psychophysical binocular summation and stimulus duration (Baker et al., 2018). That is, the
magnitude of binocular summation effects tended to decrease with exposure time. For example, psychophysical binocular summation in orientation discrimination was shown to be greatest for a brief exposure time of 50ms, which approached non-significance (indistinguishable from monocular stimulation) at durations of 100ms or longer (Bearse and Freeman, 1994). We found that V1 spiking responses were transiently facilitated (50-150ms) with peak magnitude of facilitation localized around the initial peak of the response (45-50ms). In the sustained period of the response, binocular responses were comparable to (and even less than) monocular responses, akin to the invariance of perception when opening and closing one eye. Thus, the timescales of V1 binocular enhancement reported here are consistent with the timescales reported previously for behavioral performance gains and our subjective experience of binocular vision.

Our first set of findings thus seem well in line with previous findings and the psychophysical literature on binocular summation. This convergence of studies provides a strong base that we build our findings of contrast-dependency on.

V1, contrast, and binocular gain-control

Contrast is a basic property of vision (Campbell and Robson, 1968) to which nearly all neurons in V1 increase in firing rates (Conway et al., 2002). When local contrast changes, V1 neurons adjust their dynamic range to meet the new local mean (Boynton, 2005; Ohzawa et al., 1985). This flexibility is often referred to as contrast-gain control and has been established as a fundamental operation throughout the primary visual pathway (Boynton, 2005).

A special type of contrast-gain control has been described for the transition from monocular to dichoptic viewing, i.e., when an incompatible image is ‘added’ to the other eye (Sengpiel and Blakemore, 1994). Such dichoptic stimulation has been shown to instigate inhibitory neural interactions that suppress the responses of V1 neurons, effectively shifting the V1 CRF downward along the y-axis (Sengpiel and Blakemore, 1994). Our findings for dioptic, i.e., balanced binocular, stimulation show the opposite effect. We found that dioptic stimulation led to transient facilitation of V1 responses that resemble response-gain, expressed as an upward shift of the CRF along the y-axis. Yet binocular responses were still subject to some form of control as they did not constitute a linear sum of their comprising monocular parts.
Previous empirical work has suggested that multiplicative gain control in cortical neurons can be similarly induced by either excitation or inhibition alone (Murphy and Miller, 2003). This suggests the possibility that one and the same mechanism—gain modulation of firing rates—may be responsible for the response control when the two images in the eyes either match or do not match. Future experiments that compare diotic to dichoptic V1 responses at the same temporal resolution will be needed to test this hypothesis directly.

Relation to models of binocular combination

While simple models of gain control are useful in evaluating contrast-response relationships, it is understood that the rules that govern binocular combination extend beyond a single parameter. Informed by decades of theoretical development and empirical work (Blake, 1989; Campbell and Green, 1965; Legge, 1984), the predictive power of modern models of binocular combination, such as the DSKL model (Ding and Sperling, 2006), have become progressively robust to a wide range of binocular viewing conditions (Ding et al., 2013a, 2013b; Ding and Levi, 2021, 2017; Huang et al., 2010; Yehezkel et al., 2016). Key to the success of such models has been the incorporation of multiple channels for reciprocal contrast-gain control to occur between the two eyes. Neural models of binocular combination employ similar formalisms and synergize well with the gain-control theory of binocular combination. They must additionally account for known properties of visual neurons, such as the linear spatial summation of V1 simple cells, the nonlinearities of complex cells and spike generation, and the diversity in the interocular balance of inputs to a given cell. Notably, the two-stage model (Georgeson and Sengpiel, 2021), energy models (Fleet et al., 1996; Haefner and Cumming, 2008; Ohzawa et al., 1997; READ et al., 2002; Read and Cumming, 2003; Tanabe and Cumming, 2008), and binocular/interocular normalization (Chadnova et al., 2018; Hou et al., 2020; Ling and Blake, 2012; Moradi and Heeger, 2009; Tsang and Shi, 2008) have shown promise in accounting for the neural interactions that give rise to V1 binocular responses.

Results discussed in this paper are consistent with the contrast-gain control theory of binocular combination. Specifically, binocular facilitation in V1 was attenuated by high stimulus contrast, an explicit prediction of the DSKL model (Ding and Levi, 2021). We also report on the temporal dynamics
of binocular modulation as a function of contrast. We found that the contrast-dependency of binocular modulation varied as a function of time. A potential implication of this finding is that binocular integration is comprised of sequential stages that can be operationally defined in terms of the V1 binocular contrast-response relationship. This implication could be further explored by evaluating existing neural models of binocular combination over sequential phases of the V1 response or by comparing model performance across varying levels of stimulus exposure. Based on the evolving contrast-response relationship we observed here, it is conceivable that the rate at which binocular response-gain decays in V1 could be parameterized.

Binocular response dynamics

Previously, we have shown that binocular responses in V1 exhibit at least two sequential steps that comprise initial facilitation followed by widespread differentiation between binocular concordance and discordance (Cox et al., 2019). Importantly, that work was specifically aimed at characterizing modulatory effects of non-dominant eye. Thus, unlike here, contrast in the non-dominant eye always exceeded that of the dominant eye regardless of concordance or discordance in stimulus orientation. Results here focus on the much more common visual experience of dioptic situation.

We extend our previous work by revealing a relationship between the temporal dynamics of binocular modulation and stimulus contrast. Specifically, initial facilitation of V1 to dioptic stimulation resembled response-gain, characterized by an upward shift of V1’s CRF. As V1 responses evolved, contrast-dependency emerged. Contrast-dependency was evidenced by the finding that contrast inversely correlated with facilitation duration, meaning that facilitation persisted longer for lower contrasts than higher contrasts. Furthermore, contrast positively correlated with suppression, such that V1 responses to high contrast binocular stimulation were lower than responses to monocular stimulation of the preferred eye. Finally, a model that assumes V1’s binocular response multiplicatively scales with contrast (contrast invariant) explained the initial transient but varied significantly over the time course of the stimulus presentation. These findings suggest that binocular contrast combination is a dynamic process that
involves multiple steps of processing: an initial, rapid process that is more contrast-invariant and a subsequent, slower process that is more contrast-dependent.

Limitations of the study

Data presented in this manuscript is drawn from multiple subjects but biased towards one. Therefore, populations averages of spiking activity are influenced by one subject more than the other. Subject sampling-bias is not unusual for work of this kind. For transparency, units per subject and condition are detailed in tables and statistical testes throughout the manuscript, and individual subject data is presented in supplementary figures. Nevertheless, we must consider the implications for the generalizability of findings reported here. One possibility is that our observations and corresponding conclusions truly only apply to one individual subject and thus do not represent a general processing strategy of primate visual cortex. The observations that rest most firmly on data from a single animal are those pertaining to the shape of the CRFs and the temporal dynamics of contrast-dependent decay of binocular facilitation. In the latter case, there seems to be a slight difference in the timing of transition between contrast-dependent facilitation and suppression in one animal, which can be observed by comparing single-penetration data from each subject provided in supplement. Whether this is a true individual difference, or a result of poor sampling is unclear. Future work that examines these or similar conditions in additional individuals will hopefully add weight to one interpretation or another.

Another caveat relating to the specific finding of contrast-dependent decay of binocular facilitation has to do with an inability to differentiate a contrast-dependent effects from a magnitude-dependent effects. Specifically, we report a contrast-dependent decay of initial binocular facilitation whereby higher contrasts drive a faster transition between facilitation and suppression of the binocular response compared to the monocular response. However, higher contrast stimuli also evoke more V1 spiking than lower contrasts stimuli when all other stimulus features are kept consistent. Therefore, an alternative explanation is that the magnitude of the initial transient itself determines the rate of decay of
binocular facilitation. Thoughtfully designed future experiments might be necessary to distinguish between these two mechanisms.

Author Contributions

B. A. M., A. M., and M. A. C. designed the research. K. D., J. A. W., and M. A. C. performed research. B. A. M. analyzed the data. B. A. M. prepared visualizations. B. A. M., K. D., J. A. W., B. M. C., L. D., A. M., and M. A. C. wrote the manuscript.

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Declaration of Interests

The authors declare no competing interests.

Figure Legends

**Figure 1.** Experimental design. (A) Paradigm. Monkeys fixated while viewing static sinusoidal gratings through a mirror stereoscope. Stimuli covered the previously mapped receptive field location (dashed circle) for 250ms while spiking responses were recorded from V1. (B) Stimulus conditions. Gratings appeared either in the left eye (monocular), the right eye (monocular), or both eyes simultaneously (binocular). Orientation, size, and spatial frequency of gratings were held constant throughout each experiment. Gratings varied in Michelson contrast ([0], [0.20-0.22], [0.40-0.45], [0.80-0.90]) between trials. (C) Example unit peri-stimulus time histograms (PSTH and superimposed spike density functions for each stimulus condition. Spiking activity (spikes/ s) is plotted across time for each type of stimulation (row) at each contrast (column). Shaded region encasing the plot represents +/- SEM. The eye that drove neural activity most vigorously was designated as the dominant eye (darker blue). (D) Sublinear binocular combination. Mean firing rates (N = 314 units, 234 in monkey E) to binocular stimulation are plotted against the sum of monocular firing rates (left + right eye). Solid black line represents linear summation; dashed black line represents y = 0.5x. Binocular responses were generally less than the arithmetic sum of their monocular counterparts at each contrast level and in both monkeys.

**Figure 2.** Facilitation of V1 spiking responses to balanced binocular stimulation. (A) We computed binocular modulation index to compare a unit’s strongest monocular response (its preferred eye) to its binocular response over the full stimulus duration (0-250ms). Values above 0 signify binocular facilitation while values below 0 signify binocular suppression. (B) Within-unit average (across contrast)
binocular modulation index (M = 0.057, SD = 0.067, N = 314 [234 from Monkey “E”, shown in green]). Distribution to the right shows that most V1 units were facilitated (shaded region encasing 95% CI, diamond marks the mean). (C) Binocular modulation as a function of contrast. In each panel, a unit’s binocular response is plotted against its strongest monocular response. Distribution of the binocular modulation index is shown in corner histogram; gray shaded region represents the 95% confidence interval; the black diamond marks the mean of the distribution. Facilitation was observed in most V1 units and at each contrast tested.

**Figure 3.** Contrast-dependency of V1 binocular facilitation across time. (A) Top - Population spike density functions (SDFs) for monocular (blue) and binocular stimulation (red) are shown for three contrast levels ([0.22, 0.45, 0.90]). Dotted line at zero represents stimulus onset. Shaded region represents 95% CI. Bottom - Difference between the SDFs (BIN – MON) across time, calculated within-unit and normalized to each unit’s maximum binocular response. Shaded region represents 95% CI. Note that facilitation was limited to an early phase of the response. (B) Top – Peak magnitude of facilitation increased as a function of contrast. Datapoints are shown in black (dots). Boxplot upper and lower edges indicate the 25th and 75th percentiles, respectively. Red line connects median value (red circle) of each boxplot. Whiskers extend to the most extreme data points that are not considered outliers. Bottom – duration of facilitation systematically decreased as a function of contrast. Same conventions as above.

**Figure 4.** V1 responses at higher contrasts transition from facilitation to suppression. (A) Naka-Rushton h-ratio equation used to fit individual contrast response functions (CRFs). Mean parameters were then used to estimate monocular and binocular CRFs of the V1 population. (B) Binocular and monocular CRFs were computed consecutively over 100ms windows of the V1 response, sliding by 10ms forward in time. Sixteen windows in total are shown along the y-axis (x axis is contrast, z axis is spiking response). The floor of the plot reflects the difference between the CRF pairs across time. Color bar translates these differences into strength of facilitation (pink) or suppression (gray) (C) CRFs for three temporal windows pulled from A ([50-150ms], [100-200ms], and [150-250ms]), designated as the early, intermediate, and late phase of the responses, respectively. Shaded region represents 95% CI of the parameters used to construct the curves. Facilitation appeared the most proportional to contrast in the early phase (50-150ms). At the intermediate phase (100-200ms) facilitation was diminished at high contrast. By the late phase (150-250ms), facilitation was relinquished, giving way to binocular suppression at medium and high contrast.

**Figure 5.** The effect of balanced binocular stimulation in V1 predominantly resembles a response-gain. (A) Models for how binocular stimulation interacts with contrast to modulate V1 responses. A single free parameter G multiplies either response (orange, response-gain) or contrast (green, contrast-gain) to fit binocular responses while all other parameters are fixed to the monocular (dom. eye) condition. (B) Top – model curves overlaid the 95% CI of the binocular CRF fit by the standard function. Bottom – goodness of fit (R²) for both models as a function of time. We additionally tested an alternative model of contrast-gain set (light green) that multiplies the semi-saturation constant (C₅₀). Performance of the two contrast-gain set models were comparable. (C) Difference (normalized) in the parameters Rₘₐₓ and C₅₀ of the CRFs. Binocular stimulation transiently shifts V1’s CRF upward in a manner that resembled response-gain before contrast-dependent dynamics shape the sustained response.
### Tables

**Table 1**

*Subject and Sample information*

| Subject | Sessions | Units | Low contrast | Med contrast | High contrast | CRFs |
|---------|----------|-------|--------------|--------------|--------------|------|
| “E”     | 14       | 234   | 234          | 234          | 234          | 234  |
| “I”     | 5        | 80    | 14           | 74           | 80           | 8    |
| Pooled  | 19       | 314   | 248          | 308          | 314          | 242  |

**Table 2**

*Linear regression. Slope coefficients for additivity across contrast*

| Subject | Contrast | Estimate | SE  | 95% CI Lower | 95% CI Upper | Student’s t | p    |
|---------|----------|----------|-----|--------------|--------------|-------------|------|
| “E”     | Low      | 0.5504   | 0.0015 | 0.5278      | 0.5730      | 48.0209 | <.001 |
|         | Med      | 0.5567   | 0.0112 | 0.5467      | 0.5788      | 49.7850 | <.001 |
|         | High     | 0.5667   | 0.0116 | 0.5439      | 0.5896      | 48.7941 | <.001 |
| “I”     | Low      | 0.6050   | 0.0712 | 0.4499      | 0.7601      | 0.00391 | 0.9695 |
|         | Med      | 0.5424   | 0.0241 | 0.4945      | 0.5904      | 22.5344 | <.001 |
|         | High     | 0.5400   | 0.0185 | 0.5032      | 0.5769      | 29.1959 | <.001 |
### Table 3

**Paired Samples T-Test. Binocular modulation index (0-250ms) across contrast**

| Subject | Contrast | Bayes factor<sub>10</sub> | Student’s t | df  | p     | Cohen’s d |
|---------|----------|---------------------------|-------------|-----|-------|-----------|
| “E”     | Grand    | 5.19e+29                  | 13.99       | 233 | <.001 | 0.914     |
|         | Low      | 4.43e+6                   | 6.13        | 233 | <.001 | 0.401     |
|         | Med      | 8.21e+6                   | 6.25        | 233 | <.001 | 0.408     |
|         | High     | 121                       | 3.74        | 233 | <.001 | 0.245     |
| “I”     | Grand    | 695497.5                  | 6.11        | 79  | <.001 | 0.683     |
|         | Low      | 11.7                      | 3.04        | 13  | 0.005 | 0.811     |
|         | Med      | 525811.6                  | 6.09        | 73  | <.001 | 0.708     |
|         | High     | 104.4                     | 3.68        | 79  | <.001 | 0.411     |

Note. H<sub>0</sub> μ Measure > 0
*Cauchy prior 0.707

### Table 4

**Paired Samples T-Test. Peak magnitude of facilitation across contrast**

| Subject | Comparison      | Mean difference | Bayes factor<sub>10</sub> | Student’s t | df  | p<sup>**</sup> | Cohen’s d |
|---------|-----------------|-----------------|---------------------------|-------------|-----|---------------|-----------|
| “E”     | Low - Med       | -0.052          | 61573.529                 | -5.41       | 233 | <.001         | -0.35     |
|         | - High          | -0.071          | 547386.053                | -5.86       | 233 | <.001         | -0.38     |
|         | Med - High      | -0.019          | 0.256                     | -1.60       | 233 | 0.34          | -0.10     |
| “I”     | Low - Med       | -0.157          | 1.319                     | -2.03       | 7   | 0.2461        | -0.72     |
|         | - High          | -0.085          | 0.734                     | -1.57       | 13  | 0.4198        | -0.42     |
|         | Med - High      | -0.018          | 0.352                     | 0.33        | 7   | 0.9999        | 0.12      |

*Cauchy prior 0.707
**Bonferroni adjusted p-value
Table 5

*Paired Samples T-Test. Duration (milliseconds) of facilitation across contrast*

| Subject | Contrast | Estimate (SD) | Comparison | Mean difference | Bayes factor* | Student’s t | df  | p** | Cohen’s d |
|---------|----------|---------------|------------|-----------------|--------------|-------------|-----|-----|-----------|
| “E”     | Low      | 106 (64.3)    | Low - Med  | 12.1            | 1.93         | 2.28        | 211 | 0.036| 0.16      |
|         | Med      | 88 (58.6)     | - High     | 11.1            | 0.91         | 1.92        | 207 | 0.084| 0.13      |
|         | High     | 81 (62.3)     | Med - High | 1.7             | 0.10         | 0.33        | 217 | 0.999| 0.03      |
| “I”     | Low      | 53.5 (40.7)   | Low - Med  | 36.0            | 3.41         | 2.30        | 7   | 0.081| 0.81      |
|         | Med      | 24 (24.4)     | - High     | 33.5            | 12.9         | 3.10        | 13  | 0.012| 0.83      |
|         | High     | 21 (19)       | Med - High | 4.9             | 0.49         | 0.46        | 7   | 0.963| 0.18      |

Note. H₀, μ Measure 1 – Measure 2 > 0
*Cauchy prior 0.707
**Bonferroni adjusted p-value

Table 6

*Goodness of fit. Simple models of binocular gain across temporal phases of V1 response*

| Subject | Gain Model | Early          | Intermediate | Late         |
|---------|------------|----------------|--------------|--------------|
|         |            | R²  | 95% CI      | R²  | 95% CI      | R²  | 95% CI      |
| “E”     | Response-    | 0.90 | [0.88, 0.92] | 0.87 | [0.85, 0.89] | 0.81 | [0.78, 0.84] |
|         | gain       |     |             |     |             |     |             |
|         | Contrast-  | 0.88 | [0.86,0.90] | 0.85 | [0.82,0.87] | 0.73 | [0.69,0.77] |
| “I”     | Response-    | 0.78 | [0.56,1.00] | 0.77 | [0.58,0.96] | 0.87 | [0.80,0.95] |
|         | gain       |     |             |     |             |     |             |
|         | Contrast-  | 0.74 | [0.50,0.96] | 0.78 | [0.56,0.99] | 0.86 | [0.75,0.97] |

Note. Naka-Rushton function fitted with three free parameters to units with at least four contrast levels.
**Transparent Methods**

**RESOURCE AVAILABILITY**

*Lead contact*
Further information and requests for resources and reagents should be directed to and will be fulfilled by the Lead Contact, Michele A. Cox (michele.a.cox@gmail.com).

*Materials availability*
This study did not generate new unique materials. All materials found in the key resource table may be obtained commercially.

*Data and code availability*
- All data reported in this paper will be shared by the lead contact upon request.
- All original code has been deposited at Zenodo and is publicly available as of the date of publication.
- Any additional information required to reanalyze the data reported in this paper is available from the lead contact upon request.

**EXPERIMENTAL MODEL AND SUBJECT DETAILS**

Two adult monkeys (Monkey E48, male, age 9; I34, female, age 12; *Macaca radiata*) were used in this study. We report on neurophysiological data recorded during 19 penetrations (14 in Monkey E48) of area V1. All procedures followed regulations by the Association for the Assessment and Accreditation of Laboratory Animal Care (AALAC), Vanderbilt University’s Institutional Animal Care and Use Committee (IACUC), and National Institutes of Health (NIH) guidelines.

**METHOD DETAILS**

*Surgical Procedures*
Prior to data collection, animals were implanted with a custom-designed plastic head holder and a plastic recording chamber (Crist Instruments) spanning over two separate surgeries. All surgeries were performed
under sterile surgical conditions using isoflurane anesthesia (1.5%–2.0%). Vital signs, including heart rate, blood pressure, SpO₂, CO₂, body temperature, and respiratory rate were monitored continuously. During surgery, the head holder and a recording chamber were attached to the skull using transcranial ceramic screws (Thomas Recording, Gießen, Germany) and self-curing dental acrylic (Lang Dental Manufacturing, Wheeling, IL). A craniotomy was performed over the parafoveal visual field representation of primary visual cortex (V1) concurrent with the position of the recording chamber. Animals received analgesics and antibiotics for postsurgical care and closely monitored by researchers, facility veterinarians, and animal care staff for at least three days following surgery.

**Visual apparatus**

Stimuli were presented on a linearized CRT monitor running at either 60 Hz (resolution 1,280 x 1,024 pixels) or 85 Hz (resolution 1,024 x 768). Animals viewed all stimuli through a custom-built mirror stereoscope, such that images on the right side of the display were viewed by the right eye and images on the left by the left eye (Figure 1A; the monitor was divided by a black, nonreflective septum). The mirrors of the stereoscope were infrared-transparent (Qian and Brascamp, 2017), enabling gaze position to be measured using infrared light-sensitive cameras (EyeLink II). To facilitate binocular fusion, an oval aperture was displayed at the edge of each half of the screen. At the beginning of each experimental session, the stereoscope was calibrated via a behavioral task (Maier et al., 2008), which required the animals to fixate on the same location in visual space while being cued in one eye only. Gaze position was measured for each fixation location and compared across eyes. When gaze position was comparable for cueing in each eye, the mirrors were considered aligned.

**Behavioral task**

A trial began once the animal fixated (self-initiated) within 1° of visual angle of a centralized fixation cue appearing in both eyes. A series of sinusoidal grating stimuli were presented to the left eye, right eye, or both eyes at a fixed location in parafoveal visual space, each lasting 250-500ms with a 500ms interval interleaved (details of stimuli are described later). If fixation was held for the duration of the trial, the
monkey received a juice reward. Alternatively, the next fixation cue appeared with a brief (1-5 s) delay. The animals were at liberty to end recording sessions at any point by halting the initiation of new trials. No other behavior was required.

**Neurophysiological procedure**

Experiments were conducted inside a radio frequency-shielded booth. During each recording session, a linear multielectrode array (U-Probe, Plexon Inc., Dallas, TX; Vector Array, NeuroNexus, Ann Arbor, MI) was inserted into V1 orthogonal to the cortical surface. Fluctuating extracellular voltages (referenced to the metallic electrode shaft) were amplified, filtered, and digitized using a 128-channel Cerebus neural signal processing system (Blackrock Microsystems, Salt Lake City, UT). Two neural signals were recorded and stored for subsequent offline analysis: a low-pass filtered signal (0.3–500 Hz) sampled at 1 kHz, corresponding to the local field potential, or LFP, and a broadband (0.3 Hz–7.5 kHz) signal sampled at 30 kHz. The neural signal processing system also recorded non-neurophysiological analog signals related to the monitor refresh (i.e., a photodiode signal; OSI Optoelectronics, Montreal, Quebec) and eye position (i.e., voltage output of eye-tracking system), which were digitized and stored at 30 and 1 kHz, respectively. These time stamps and the photodiode signal were used to align the time-varying intracranial data with the occurrence of visual events.

During recording session, the parameters of the sinusoidal grating stimuli (orientation, phase, spatial frequency, location, etc.) were customized relative to the receptive field and tuning preferences of the population of neurons recorded across the microelectrode array. Details of these procedures, including the reverse correlation-like receptive field mapping procedure and the paradigms used to identify tuning preferences have been described in detail previously (Cox et al., 2019; Westerberg et al., 2019). Note, all receptive fields were mapped binocularly, and receptive fields for a given penetration always overlapped due to the orthogonal angle of the microelectrode array to V1.
Pre-processing of spiking activity

Offline, we computed a discretized measure of multi-unit activity (MUA) by applying a time-varying threshold to the envelope of the broadband signal, with an impulse recorded at every time point where the signal envelope exceeded the threshold on each microelectrode contact. Single-unit activity was extracted using Kilosort, an unsupervised machine-learning spike-sorting algorithm (Pachitariu et al., 2016). Both techniques have been described in detail previously (Cox et al., 2019). Here, all analyses were conducted on the discretized multi-unit activity unless otherwise stated. To be included in this study, the spiking units had to fall within the bounds of V1 and exhibit a significant response to visual stimulation, determined by performing a paired-samples $t$ test ($\alpha = 0.05$) on the mean baseline activity on each trial and the mean activity during the epoch of visual stimulation (0-250 ms).

QUANTIFICATION AND STATISTICAL ANALYSIS

V1 responses to monocular and binocular stimulation

For our purposes here, we analyzed spiking responses to stimulation of one or the other eye (monocular) or stimulation of both eyes simultaneously (binocular). All stimuli of these trials consisted of sinusoidal, monochromatic gratings. Features of the gratings, such as size, spatial frequency, and orientation, were set to values which elicited the strongest spiking response of the V1 population, informed by unit responses collected in the tuning paradigms (see Neurophysiological Procedure and Supp. Figure). For binocular presentation of gratings, all parameters (size, orientation, spatial frequency, and contrast) were identical between the two eyes and positional disparity was set to zero (resulting in an actual disparity close to zero given the flat surface of the monitor relative to the curvature of the horopter). Throughout the paper, we use the term monocular for all stimuli consisting of a grating of the units’ preferred orientation, presented to either the left or right eye in isolation. We use the term dioptic (binocular) to refer to the condition where the same grating is presented at corresponding retinal points to both eyes.

The stimulus parameter that varied experimentally was the Michelson contrast of the grating(s) between presentations. The exact contrast levels used across recording days varied slightly (e.g., we
sampled responses to more, evenly spaced, contrasts on days where the animals’ motivation was high). We thus grouped the following contrast ranges: [0, 0.20-0.225, 0.40-0.45, 0.80-0.90].

**Duration of binocular facilitation**

Duration of binocular facilitation was evaluated in efforts to assess contrast-dependency of binocular modulation across time. For this analysis, we estimated the onset and offset of facilitation from each unit’s delta spiking response (250ms). We estimated the onset of facilitation as the timepoint associated with the peak magnitude of facilitation for each unit. We estimated the offset of facilitation for each unit by identifying the timepoint after onset at which the delta response crossed zero. Duration was then computed as offset minus onset of facilitation. This procedure was repeated for each stimulus contrast level. Since we were only interested the temporal dynamics of facilitation, units that did not show facilitation (positive delta) at any timepoint were excluded from this analysis (n = 32).

**Determining ocular dominance**

Our analysis aimed to compare binocular responses of V1 neurons to their monocular counterparts. Neurons in V1 are known to differ in the magnitude they respond to stimuli presented to one eye or the other (Hubel and Wiesel, 1962). This is the *ocular dominance* of the neuron. We used the neuronal responses to monocular stimulation to compute an ocularity index that quantifies a unit’s selectivity for one versus the other eye,

\[
\text{ocularity} = \frac{\text{mean(LE)} - \text{mean(RE)}}{\text{mean(LE)} + \text{mean(RE)}}
\]

defined as differences between trial-averaged responses (*mean*, 0-250ms) of each eye divided by their sum. All nonrelevant parameters, such as orientation and contrast, were matched for this process. For each unit, we used this value to distinguish “dominant (DE) eye” and “non-dominant (NDE).
Contrast response functions (CRFs)

Contrast response functions (CRFs) portray a neuron’s mean spiking response as a function of stimulus contrast (Albrecht and Hamilton, 1982). To determine how binocular V1 spiking responses vary as a function of contrast, we measured CRFs in units for which there was four datapoints (247 in monkey E, 8 in monkey I) under monocular and binocular stimulation (Figure 1G & H). Trial-averaged spiking responses (over varying time windows) as a function of visual contrast were fit using the Naka-Rushton function:

\[ R_{R_{\text{max}}, C_{50}, n, b}(c) = \frac{R_{\text{max}} \cdot c^n}{c^n + C_{50}^n} + b \]

where \( R \) is the response of the unit, \( R_{\text{max}} \) is the projected maximum response of the unit, \( C_{50} \) is the semi-saturation constant that represents the contrast at which the output is half of the maximum response, \( n \) is the scaling exponent, and \( c \) is the contrast of the stimulus. The y-intercept \( b \) represents the maintained activity and was fixed for individual neurons to the average activity during blank trials (0 contrast = gray background). The parameters \( R_{\text{max}}, C_{50}, \) and \( n \) collectively determine the shape of the contrast response curve (Carandini and Heeger, 2012). For each unit, the trial-averaged contrast responses under binocular and monocular conditions were fit with all parameters free to vary [\( R_{\text{max}}, C_{50}, n \)].

Statistical analysis

We used custom code written in Matlab (The Mathworks Inc.) for data analysis. All statistical analysis were conducted in Jamovi version 2.0.0, an open-source statistical software. Mean piking responses (\( N = 314 \) units, 234 from Monkey E) to stimuli of varying contrast [0, 0.20-0.225, 0.40-0.45, 0.80-0.90] were compared across monocular and binocular stimulation. Mean responses were either taken as time-average spiking across full stimulus presentation (0-250ms) or 100ms windows within this timeframe, as noted. Data from monkey “I” had incomplete observations at low and medium contrast (see Table 1 for sample
information), which prevented the use of repeated measures ANOVA on pooled units from both animals.

To test effects of stimulus contrast across both animals without discarding data, we employed a mixed-model analysis of variance with contrast as a continuous predictor and the unit as a random factor (Figure 1 and Figure 2). Subsequent analyses employed repeated measures ANOVA on units with complete observations (Figure 3, Figure 4, and Figure 5). Post hoc tests were performed when appropriate to test for significant (p < 0.05) contrasts between samples (two-tailed, paired t tests) with Bonferroni correction. Performance of simple models of gain-control were evaluated by goodness of fit ($R^2$) to the observed binocular contrast responses (mean spiking over 100ms windows).
References

Albrecht, D.G., Hamilton, D.B., 1982. Striate cortex of monkey and cat: contrast response function. J Neurophysiol 48, 217–237. https://doi.org/10.1152/jn.1982.48.1.217

Anzai, A., Ohzawa, I., Freeman, R.D., 1999. Neural Mechanisms for Processing Binocular Information I. Simple Cells. J Neurophysiol 82, 891–908. https://doi.org/10.1152/jn.1999.82.2.891

Apkarian, P., Levi, D., Tyle, C.W., 1981. Binocular Facilitation in the Visual-Evoked Potential of Strabismic Amblyopes. Optometry Vision Sci 58, 820–830. https://doi.org/10.1097/00006324-198110000-00007

Ates, K., Demirtas, S., Goksoy, C., 2006. Binocular interactions in the guinea pig’s visual-evoked potentials. Brain Res 1125, 26–30. https://doi.org/10.1016/j.brainres.2006.10.016

Baker, D.H., Lygo, F.A., Meese, T.S., Georgeson, M.A., 2018. Binocular Summation Revisited: Beyond $\sqrt{2}$. Psychol Bull 144, 1186–1199. https://doi.org/10.1037/bul0000163

Barlow, H.B., Blakemore, C., Pettigrew, J.D., 1967. The neural mechanism of binocular depth discrimination. J Physiology 193, 327–342. https://doi.org/10.1113/jphysiol.1967.sp008360

Barton, R.A., 2004. Binocularity and brain evolution in primates. P Natl Acad Sci Usa 101, 10113–10115. https://doi.org/10.1073/pnas.0401955101

Bearse, M.A., Freeman, R.D., 1994. Binocular summation in orientation discrimination depends on stimulus contrast and duration. Vision Res 34, 19–29. https://doi.org/10.1016/0042-6989(94)90253-4

Bishop, P.O., Pettigrew, J.D., 1986. Neural mechanisms of binocular vision. Vision Res 26, 1587–1600. https://doi.org/10.1016/0042-6989(86)90177-x

Blake, R., 1989. A Neural Theory of Binocular Rivalry. Psychol Rev 96, 145–167. https://doi.org/10.1037/0033-295x.96.1.145

Blake, R., Fox, R., 1973. The psychophysical inquiry into binocular summation. Percept Psychophys 14, 161–185. https://doi.org/10.1037/bf03198631

Blake, R., Sloane, M., Fox, R., 1981. Further developments in binocular summation. Percept Psychophys 30, 266–276. https://doi.org/10.1037/bf03214282

Blake, R., Wilson, H., 2011. Binocular vision. Vision Res 51, 754–770. https://doi.org/10.1016/j.visres.2010.10.009

Boynton, G.M., 2005. Contrast Gain in the Brain. Neuron 47, 476–477. https://doi.org/10.1016/j.neuron.2005.08.003
Burkhalter, A., Essen, D.V., 1986. Processing of color, form and disparity information in visual areas VP and V2 of ventral extrastriate cortex in the macaque monkey. J Neurosci 6, 2327–2351. https://doi.org/10.1523/jneurosci.06-08-02327.1986

Burns, B.D., Pritchard, R., 1968. Cortical conditions for fused binocular vision. J Physiology 197, 149–171. https://doi.org/10.1113/jphysiol.1968.sp008552

Cagenello, R., Halpern, D.L., Arditi, A., 1993. Binocular enhancement of visual acuity. J Opt Soc Am 10, 1841. https://doi.org/10.1364/josaa.10.001841

Campbell, F.W., Green, D.G., 1965. Monocular versus Binocular Visual Acuity. Nature 208, 191–192. https://doi.org/10.1038/208191a0

Campbell, F.W., Robson, J.G., 1968. Application of fourier analysis to the visibility of gratings. J Physiology 197, 551–566. https://doi.org/10.1113/jphysiol.1968.sp008574

Carandini, M., Heeger, D.J., 2012. Normalization as a canonical neural computation. Nat Rev Neurosci 13, 51–62. https://doi.org/10.1038/nrn3136

Chadnova, E., Reynaud, A., Clavagnier, S., Baker, D.H., Baillot, S., Hess, R.F., 2018. Interocular interaction of contrast and luminance signals in human primary visual cortex. Neuroimage 167, 23–30. https://doi.org/10.1016/j.neuroimage.2017.10.035

Clatworthy, P.L., Chirimuuta, M., Lauritzen, J.S., Tolhurst, D.J., 2003. Coding of the contrasts in natural images by populations of neurons in primary visual cortex (V1). Vision Res 43, 1983–2001. https://doi.org/10.1016/s0042-6989(03)00277-3

Conway, B.R., Hubel, D.H., Livingstone, M.S., 2002. Color Contrast in Macaque V1. Cereb Cortex 12, 915–925. https://doi.org/10.1093/cercor/12.9.915

Cox, M.A., Dougherty, K., Westerberg, J.A., Schall, M.S., Maier, A., 2019. Temporal dynamics of binocular integration in primary visual cortex. J Vision 19, 13. https://doi.org/10.1167/19.12.13

Cumming, B.G., DeAngelis, G.C., 2001. THE PHYSIOLOGY OF STEREOPSIS. Neuroscience 24, 203–238. https://doi.org/10.1146/annurev.neuro.24.1.203

DeAngelis, G.C., Newsome, W.T., 1999. Organization of Disparity-Selective Neurons in Macaque Area MT. J Neurosci 19, 1398–1415. https://doi.org/10.1523/jneurosci.19-04-01398.1999

Ding, J., Klein, S.A., Levi, D.M., 2013a. Binocular combination of phase and contrast explained by a gain-control and gain-enhancement model. J Vision 13, 13. https://doi.org/10.1167/13.2.13

Ding, J., Klein, S.A., Levi, D.M., 2013b. Binocular combination in abnormal binocular vision. J Vision 13, 14. https://doi.org/10.1167/13.2.14

Ding, J., Levi, D.M., 2021. A unified model for binocular fusion and depth perception. Vision Res 180, 11–36. https://doi.org/10.1016/j.vision.2020.11.009
Ding, J., Levi, D.M., 2017. Binocular combination of luminance profiles. J Vision 17, 4. https://doi.org/10.1167/17.13.4

Ding, J., Sperling, G., 2006. A gain-control theory of binocular combination. P Natl Acad Sci Usa 103, 1141–1146. https://doi.org/10.1073/pnas.0509629103

Fleet, D.J., Wagner, H., Heeger, D.J., 1996. Neural encoding of binocular disparity: Energy models, position shifts and phase shifts. Vision Res 36, 1839–1857. https://doi.org/10.1016/0042-6989(95)00313-4

Freeman, R.D., Ohzawa, I., 1990. On the neurophysiological organization of binocular vision. Vision Res 30, 1661–1676. https://doi.org/10.1016/0042-6989(90)90151-a

Georgeson, M.A., Sengpiel, F., 2021. Contrast adaptation and interocular transfer in cortical cells: A re-analysis & a two-stage gain-control model of binocular combination. Vision Res 185, 29–49. https://doi.org/10.1016/j.visres.2021.03.004

Ghose, G.M., Ts’O, D.Y., 1997. Form Processing Modules in Primate Area V4. J Neurophysiol 77, 2191–2196. https://doi.org/10.1152/jn.1997.77.4.2191

Giuseppe, N., Andrea, F., 1983. Binocular Interaction in Visual-Evoked Responses: Summation, Facilitation and Inhibition in a Clinical Study of Binocular Vision. Ophthalmic Res 15, 261–264. https://doi.org/10.1159/000265268

Grünau, M.V., 1979. Binocular summation and the binocularity of cat visual cortex. Vision Res 19, 813–816. https://doi.org/10.1016/0042-6989(79)90158-5

Grünau, M.W. von, Singer, W., 1979. The role of binocular neurons in the cat striate cortex in combining information from the two eyes. Exp Brain Res 34, 133–142. https://doi.org/10.1007/bf00238346

Haefner, R.M., Cumming, B.G., 2008. Adaptation to Natural Binocular Disparities in Primate V1 Explained by a Generalized Energy Model. Neuron 57, 147–158. https://doi.org/10.1016/j.neuron.2007.10.042

Harter, M.R., Seiple, W.H., Salmon, L., 1973. Binocular summation of visually evoked responses to pattern stimuli in humans. Vision Res 13, 1433–1446. https://doi.org/10.1016/0042-6989(73)90004-7

Henriksen, S., Tanabe, S., Cumming, B., 2016. Disparity processing in primary visual cortex. Philosophical Transactions Royal Soc B Biological Sci 371, 20150255. https://doi.org/10.1098/rstb.2015.0255

Heravian, J.S., Jenkins, T.C.A., Douthwaite, W.A., 1990. Binocular summation in visually evoked responses and visual acuity. Ophthal Physl Opt 10, 257–261. https://doi.org/10.1111/j.1475-1313.1990.tb00861.x

Heuer, H.W., Britten, K.H., 2002. Contrast Dependence of Response Normalization in Area MT of the Rhesus Macaque. J Neurophysiol 88, 3398–3408. https://doi.org/10.1152/jn.00255.2002
Horton, J.C., 2006. Ocular integration in the human visual cortex. Can J Ophthalmol J Can D’ophthalmologie 41, 584–593. https://doi.org/10.1016/s0008-4182(06)80027-x

Hou, C., Nicholas, S.C., Verghese, P., 2020. Contrast Normalization Accounts for Binocular Interactions in Human Striate and Extra-striate Visual Cortex. J Neurosci 40, 2753–2763. https://doi.org/10.1523/jneurosci.2043-19.2020

Huang, C.-B., Zhou, J., Zhou, Y., Lu, Z.-L., 2010. Contrast and Phase Combination in Binocular Vision. Plos One 5, e15075. https://doi.org/10.1371/journal.pone.0015075

Hubel, D.H., Wiesel, T.N., 1962. Receptive fields, binocular interaction and functional architecture in the cat’s visual cortex. J Physiology 160, 106–154. https://doi.org/10.1113/jphysiol.1962.sp006837

Legge, G.E., 1984. Binocular contrast summation—I. Detection and discrimination. Vision Res 24, 373–383. https://doi.org/10.1016/0042-6989(84)90063-4

Lehky, S.R., Maunsell, J.H.R., 1996. No binocular rivalry in the LGN of alert macaque monkeys. Vision Res 36, 1225–1234. https://doi.org/10.1016/0042-6989(95)00232-4

Levelt, 1965. On binocular rivalry. Inst. Perception Rvo-Tno, Oxford, England.

Ling, S., Blake, R., 2012. Normalization Regulates Competition for Visual Awareness. Neuron 75, 531–540. https://doi.org/10.1016/j.neuron.2012.05.032

Ling, S., Carrasco, M., 2006. Sustained and transient covert attention enhance the signal via different contrast response functions. Vision Res 46, 1210–1220. https://doi.org/10.1016/j.visres.2005.05.008

Livingstone, M.S., Tsao, D.Y., 1999. Receptive fields of disparity-selective neurons in macaque striate cortex. Nat Neurosci 2, 825–832. https://doi.org/10.1038/12199

Longordo, F., To, M.-S., Ikeda, K., Stuart, G.J., 2013. Sublinear integration underlies binocular processing in primary visual cortex. Nat Neurosci 16, 714–723. https://doi.org/10.1038/nn.3394

Maier, A., Wilke, M., Aura, C., Zhu, C., Ye, F.Q., Leopold, D.A., 2008. Divergence of fMRI and neural signals in V1 during perceptual suppression in the awake monkey. Nat Neurosci 11, 1193–1200. https://doi.org/10.1038/nn.2173

Martinez-Trujillo, J.C., Treue, S., 2002. Attentional Modulation Strength in Cortical Area MT Depends on Stimulus Contrast. Neuron 35, 365–370. https://doi.org/10.1016/s0896-6273(02)00778-x

Matin, L., 1962. Binocular Summation at the Absolute Threshold of Peripheral Vision*. J Opt Soc Am 52, 1276. https://doi.org/10.1364/josa.52.001276

Maunsell, J.H., Essen, D.C.V., 1983. Functional properties of neurons in middle temporal visual area of the macaque monkey. II. Binocular interactions and sensitivity to binocular disparity. J Neurophysiol 49, 1148–1167. https://doi.org/10.1152/jn.1983.49.5.1148

Meese, T.S., Georgeson, M.A., Baker, D.H., 2006. Binocular contrast vision at and above threshold. J Vision 6, 7–7. https://doi.org/10.1167/6.11.7
Moradi, F., Heeger, D.J., 2009. Inter-ocular contrast normalization in human visual cortex. J Vision 9, 13–13. https://doi.org/10.1167/9.3.13

Murphy, B.K., Miller, K.D., 2003. Multiplicative Gain Changes Are Induced by Excitation or Inhibition Alone. J Neurosci 23, 10040–10051. https://doi.org/10.1523/jneurosci.23-31-10040.2003

Ohzawa, I., Deangelis, G.C., Freeman, R.D., 1997. Encoding of Binocular Disparity by Complex Cells in the Cat’s Visual Cortex. J Neurophysiol 77, 2879–2909. https://doi.org/10.1152/jn.1997.77.6.2879

Ohzawa, I., Sclar, G., Freeman, R.D., 1985. Contrast gain control in the cat’s visual system. J Neurophysiol 54, 651–667. https://doi.org/10.1152/jn.1985.54.3.651

Pachitariu, M., Steinmetz, N., Kadir, S., Carandini, M., D, H.K., 2016. Kilosort: realtime spike-sorting for extracellular electrophysiology with hundreds of channels.

Pack, C.C., Born, R.T., Livingstone, M.S., 2003. Two-Dimensional Substructure of Stereo and Motion Interactions in Macaque Visual Cortex. Neuron 37, 525–535. https://doi.org/10.1016/s0896-6273(02)01187-x

Pardhan, S., Gilchrist, J., Douthwaite, W., Yap, M., 1990. Binocular Inhibition: Psychophysical and Electrophysiological Evidence. Optometry Vision Sci 67, 688–691. https://doi.org/10.1097/00006324-199009000-00006

Parker, A.J., Cumming, B.G., 2001. Chapter 14 Cortical mechanisms of binocular stereoscopic vision. Prog Brain Res 134, 205–216. https://doi.org/10.1016/s0079-6123(01)34015-3

Parker, A.J., Smith, J.E.T., Krug, K., 2016. Neural architectures for stereo vision. Philosophical Transactions Royal Soc B Biological Sci 371, 20150261. https://doi.org/10.1098/rstb.2015.0261

Pettigrew, J.D., Nikara, T., Bishop, P.O., 1968. Binocular interaction on single units in cat striate cortex: Simultaneous stimulation by single moving slit with receptive fields in correspondence. Exp Brain Res 6, 391–410. https://doi.org/10.1007/bf00233186

PIRENNE, M.H., 1943. Binocular and Uniocular Threshold of Vision. Nature 152, 698–699. https://doi.org/10.1038/152698a0

Poggio, G.F., 1995. Mechanisms of Stereopsis in Monkey Visual Cortex. Cereb Cortex 5, 193–204. https://doi.org/10.1093/cercor/5.3.193

Poggio, G.F., Fischer, B., 1977. Binocular interaction and depth sensitivity in striate and prestriate cortex of behaving rhesus monkey. J Neurophysiol 40, 1392–1405. https://doi.org/10.1152/jn.1977.40.6.1392

Qian, C.S., Brascamp, J.W., 2017. How to Build a Dichoptic Presentation System That Includes an Eye Tracker. J Vis Exp Jove. https://doi.org/10.3791/56033

Read, J.C.A., Cumming, B.G., 2003. Testing Quantitative Models of Binocular Disparity Selectivity in Primary Visual Cortex. J Neurophysiol 90, 2795–2817. https://doi.org/10.1152/jn.01110.2002
READ, J.C.A., PARKER, A.J., CUMMING, B.G., 2002. A simple model accounts for the response of disparity-tuned V1 neurons to anticorrelated images. Visual Neurosci 19, 735–753. https://doi.org/10.1017/s0952523802196052

Said, C.P., Heeger, D.J., 2013. A Model of Binocular Rivalry and Cross-orientation Suppression. Plos Comput Biol 9, e1002991. https://doi.org/10.1371/journal.pcbi.1002991

Salthouse, T.A., Ellis, C.L., 1980. Determinants of eye-fixation duration. Am J Psychology 93, 207–34.

Schroeder, C.E., Tenke, C.E., Arezzo, J.C., Vaughan, H.G., 1990. Binocularity in the lateral geniculate nucleus of the alert macaque. Brain Res 521, 303–310. https://doi.org/10.1016/0006-8993(90)91556-v

Sclar, G., Ohzawa, I., Freeman, R.D., 1986. Binocular summation in normal, monocularly deprived, and strabismic cats: visual evoked potentials. Exp Brain Res 62, 1–10. https://doi.org/10.1007/bf00237398

Sengpiel, F., Baddeley, R.J., Freeman, T.C.B., Harrad, R., Blakemore, C., 1998. Different mechanisms underlie three inhibitory phenomena in cat area 17. Vision Res 38, 2067–2080. https://doi.org/10.1016/s0042-6989(97)00413-6

Sengpiel, F., Blakemore, C., 1994. Interocul control of neuronal responsiveness in cat visual cortex. Nature 368, 847–850. https://doi.org/10.1038/368847a0

Smith, E.L., Chino, Y., Ni, J., Cheng, H., 1997. Binocular Combination of Contrast Signals by Striate Cortical Neurons in the Monkey. J Neurophysiol 78, 366–382. https://doi.org/10.1152/jn.1997.78.1.366

Summa, A. di, Polo, A., Tinazzi, M., Zanette, G., Bertolasi, L., Bongiovanni, L.G., Fiaschi, A., 1997. Binocular interaction in normal vision studied by pattern-reversal visual evoked potentials (PR-VEPS). Italian J Neurological Sci 18, 81–86. https://doi.org/10.1007/bf01999567

Tanabe, S., Cumming, B.G., 2008. Mechanisms Underlying the Transformation of Disparity Signals from V1 to V2 in the Macaque. J Neurosci 28, 11304–11314. https://doi.org/10.1523/jneurosci.3477-08.2008

Thiele, A., Pooremaeili, A., Delicato, L.S., Herrero, J.L., Roelfsema, P.R., 2009. Additive Effects of Attention and Stimulus Contrast in Primary Visual Cortex. Cereb Cortex New York Ny 19, 2970–2981. https://doi.org/10.1093/cercor/bhp070

Tsang, E.K.C., Shi, B.E., 2008. Normalization Enables Robust Validation of Disparity Estimates from Neural Populations. Neural Comput 20, 2464–2490. https://doi.org/10.1162/neco.2008.05-07-532

Westerberg, J.A., Cox, M.A., Dougherty, K., Maier, A., 2019. V1 microcircuit dynamics: altered signal propagation suggests intracortical origins for adaptation in response to visual repetition. J Neurophysiol 121, 1938–1952. https://doi.org/10.1152/jn.00113.2019

Yang, Y., Liu, S., Chowdhury, S.A., DeAngelis, G.C., Angelaki, D.E., 2011. Binocular Disparity Tuning and Visual-Vestibular Congruency of Multisensory Neurons in Macaque Parietal Cortex. J Neurosci 31, 17905–17916. https://doi.org/10.1523/jneurosci.4032-11.2011
Yehezkel, O., Ding, J., Sterkin, A., Polat, U., Levi, D.M., 2016. Binocular combination of stimulus orientation. Roy Soc Open Sci 3, 160534. https://doi.org/10.1098/rsos.160534

Zhao, X., Liu, M., Cang, J., 2013. Sublinear Binocular Integration Preserves Orientation Selectivity in Mouse Visual Cortex. Nat Commun 4, 2088–2088. https://doi.org/10.1038/ncomms3088

東潮二唐. 1972. 視覚領における両眼視の機能;立体視を中心ににして. 医用電子と生体工学 10, 80–87. https://doi.org/10.11239/jsmbe1963.10.80
A) **Response-gain set**

\[ R = G \times R_{\text{max}} \times c^\alpha \frac{c^\beta}{c^\gamma + C_{50}} \]  

B) **Early**, **Interm.**, **Late**

- Spiking activity (imp/sec)
- Goodness of fit

C) **Δ params (norm.)**

- Time window (100ms length)
Highlights

- Binocular facilitation in primary visual cortex (V1) is contrast dependent.
- Lower contrasts result in greater binocular facilitation overall.
- Suppression follows initial facilitation of binocular response in V1.
- Contrast modulates both facilitation duration and suppression magnitude.
### Key resources table

| REAGENT or RESOURCE | SOURCE | IDENTIFIER |
|---------------------|--------|------------|
| Deposited data      |        |            |
| Post-processing MATLAB code | This paper | DOI: https://doi.org/10.5281/zenodo.6371744 |
| Experimental models: Organisms/strains | | |
| Bonnet macaque (*Macaca radiata*) | Wake Forest University, North Carolina, USA | N/A |
| Software and algorithms | | |
| MATLAB, 2012-2014 and 2016A | MathWorks | https://www.mathworks.com |
| MonkeyLogic | David Freedman, University of Chicago; Wael Assad, Brown University | http://www.brown.edu/Research/monkeylogic/ |
| KiloSort | (Pachitariu et al., 2016)] | https://github.com/cortext-lab/KiloSort |
| Jamovi, v2.0.0 | The jamovi project, and Sebastian Jentschke | https://www.jamovi.org/ |
| Other | | |
| Vector Array | NeuroNexus | http://neuronexus.com/products/neural-probes/ |
| U-Probe, V-Probe | Plexon Inc | https://plexon.com/ |
| Recording equipment | Blackrock Microsystems | http://blackrockmicro.com/ |
| Item                                      | Brand                        | Website                                      |
|-------------------------------------------|-------------------------------|----------------------------------------------|
| Eye Link II Eye Tracker                   | SR Research                  | https://www.sr-research.com/                 |
| Cold mirrors                              | Edmund Optics                | https://www.edmundoptics.com/                |
| Data Acquisition Board PCI-6229           | National Instruments         | http://www.ni.com/en-us.html                 |
| Eye-tracking Software                     | SensoMotoric Instruments     | https://www.smvision.com/                    |
| Photodiode                                | OSI Optoelectronics          | http://www.osioptoelectronics.com/           |
| Ceramic screws                            | Thomas Recording             | https://www.thomasrecording.com/            |
| Dental Acrylic                            | Lang Dental Manufacturing    | http://www.orthodonticproductsonline.com/buyers-guide/listing/lang-dental-manufacturing-co-inc/ |
| Microdrive                                | Narishige International USA, Inc. | https://usa.narishige-group.com/              |
| Plastic Recording Chamber                 | Crist Instrument; custom-made | http://www.cristinstrument.com/ ;Vanderbilt University |