

1 **Chromatic summation and receptive field properties**  
2 **of blue-on and blue-off cells in marmoset lateral**  
3 **geniculate nucleus**

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17 **Running head:** Cone summation in blue-on cells

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19

20 **Abstract**

21 The "blue-on" and "blue-off" receptive fields in retina and dorsal lateral geniculate nucleus  
22 (LGN) of diurnal primates combine signals from short-wavelength sensitive (S) cone  
23 photoreceptors with signals from medium/long wavelength sensitive (ML) photoreceptors.  
24 Three questions about this combination remain unresolved. Firstly, is the combination of S  
25 and ML signals in these cells linear or non-linear? Secondly, how does the timing of S and  
26 ML inputs to these cells influence their responses? Thirdly, is there spatial antagonism  
27 within S and ML subunits of the receptive field of these cells? We measured contrast  
28 sensitivity and spatial frequency tuning for four types of drifting sine gratings: S cone  
29 isolating, ML cone isolating, achromatic (S + ML), and counterphase chromatic (S – ML), in  
30 extracellular recordings from LGN of marmoset monkeys. We found that responses to  
31 stimuli which modulate both S and ML cones are well predicted by a linear sum of S and ML  
32 signals, followed by a saturating contrast-response relation. Differences in sensitivity and  
33 timing (i.e. vector combination) between S and ML inputs are needed to explain the  
34 amplitude and phase of responses to achromatic (S + ML) and counterphase chromatic (S –  
35 ML) stimuli. Best-fit spatial receptive fields for S and/or ML subunits in most cells (> 80%)  
36 required antagonistic surrounds, usually in the S subunit. The surrounds were however  
37 generally weak and had little influence on spatial tuning. The sensitivity and size of S and ML  
38 subunits were correlated on a cell-by-cell basis, adding to evidence that blue-on and  
39 blue-off receptive fields are specialised to signal chromatic but not spatial contrast.

40

## 41 Introduction

42 Color vision in primates originates in short- (S), medium- (M), and long-wavelength  
43 sensitive (L) cone photoreceptors in the retina. The cone signals are processed within the  
44 retina to form wavelength-selective afferent channels, including M – L opponent or  
45 "red/green" channels, in which M and L signals are differenced, and "blue/yellow" channels,  
46 driven by S cones in opposition to combinations of M and L cones. This paper addresses  
47 three outstanding questions about S-cone signalling in "blue-on/yellow off" (also known as  
48 blue-on, or more properly S-on) cells, and less commonly encountered "blue-off/yellow-on"  
49 (also known as blue-off, or more properly S-off) cells. Firstly, is the combination of S and ML  
50 signals in these cells linear or non-linear? Secondly, how does the timing of S and ML inputs  
51 to these cells influence their responses? Thirdly, is there spatial antagonism within S and ML  
52 subunits of the receptive field of these cells? Answers to these questions will improve our  
53 understanding of subcortical signals serving color vision.

54 Blue-on and blue-off cells have been the subject of longstanding study (reviewed by  
55 Dacey, 2004; Martin & Lee, 2014), and recordings from dorsal lateral geniculate nucleus  
56 (LGN) have associated blue-on and blue-off cells with the koniocellular layers of the LGN  
57 (Martin, White, Goodchild, Wilder & Sefton, 1997; Roy, Martin, Dreher, Saalman, Hu &  
58 Vidyasagar, 2009). The small bistratified (SBS) retinal ganglion cell is established as  
59 exhibiting a blue-on/yellow off receptive field (Dacey & Lee, 1994; Szmajda, Martin &  
60 Grünert, 2008; Crook, Davenport, Peterson, Packer, Detwiler & Dacey, 2009) but the  
61 anatomical substrate of blue-off cells in retina is less clear (reviewed by Dacey, 2004; Martin  
62 & Lee, 2014).

63 Firing rates of blue-on cells and blue-off cells are determined by opponent

64 (antagonistic) combination of the S and ML inputs to the receptive field (Yeh, Lee &  
65 Kremers, 1995a; Solomon, Lee, White, Rüttiger & Martin, 2005; Field et al., 2007; Tailby,  
66 Solomon & Lennie, 2008a; Crook et al., 2009). Responses to stimuli which modulate both S  
67 and ML cones should therefore be determined by the (signed) sum of the contrast delivered  
68 to the S and ML cones. This prediction was confirmed for blue-on cells in macaque retina  
69 when tested using combinations of S and ML contrast in spatially uniform fields (Crook et  
70 al., 2009). The question as to whether this finding holds across the spatial frequency tuning  
71 range of blue-on and blue-off cells has however not been specifically tested. Further, most  
72 ganglion cells, including blue-on and blue-off cells (Yeh et al., 1995a), show some degree of  
73 response saturation for high contrast stimuli (Shapley & Victor, 1978; Lee, Virsu & Elepfandt,  
74 1983; Kaplan & Shapley, 1986; Solomon et al., 2005) The specific question as to whether  
75 response saturation influences responses of blue-on and blue-off cells to combined S and  
76 ML contrast has also not been addressed.

77         Lee, Valberg and colleagues showed linear combination of saturating cone inputs can  
78 account for responses of blue-on ganglion cells to high-intensity narrow-band lights (Lee et  
79 al., 1983; Lee, Valberg, Tigwell & Tryti, 1987). In their model, illustrated schematically in  
80 Figure 1, nonlinearities in contrast response are present in the S cone and ML cone signals  
81 prior to their combination (Lee et al., 1983; Schnapf, Nunn, Meister & Baylor, 1990). If this is  
82 the case, then differences in gain and/or saturation between S and ML inputs could yield  
83 non-monotonic "supersaturating" responses to stimuli which activate both inputs. An  
84 alternative (but not mutually exclusive) possibility is that linear S cone and ML cone inputs  
85 may be first summed, then subject to nonlinear distortion. In this case, the integrated  
86 output must be monotonic with increasing contrast. These alternative models were not

87 explicitly compared in previous studies of cone inputs to macaque LGN cells (Lee et al.,  
88 1983; Lee et al., 1987). Therefore, the first question we address in this paper is: how linear is  
89 cone summation in blue-on and blue-off cells?

90 In addition to assuming linear combination of S and M inputs, most studies among  
91 those cited above had assumed that the opponent inputs arrive at the same time at the  
92 integrating site, and therefore can be modelled as a simple difference of the input functions.  
93 More recent studies, however, indicate that there is a variable degree of delay between S  
94 and ML inputs (~5 – 10 ms) to blue-on cells (Chichilnisky & Baylor, 1999; Field et al., 2007;  
95 Tailby et al., 2008a; Pietersen, Cheong, Solomon, Tailby & Martin, 2014). Such delays could  
96 help account for direction-selective achromatic responses observed in blue-on cells in  
97 macaque and marmoset LGN (Tailby et al., 2008a; Tailby, Dobbie, Hashemi-Nezhad, Forte &  
98 Martin, 2010), but responses to combined S and ML contrast have not been systematically  
99 analysed. This is the second question we address in this paper.

100 The spatial distribution of cone inputs to S cone-signaling cells in the LGN was first  
101 described as “Type II”, consisting of spatially overlapping S and ML subfields, in early work  
102 by Wiesel and Hubel (1966). Later measurements showed that the ML-off subfield is slightly  
103 larger than the S-on subfield (Solomon et al., 2005; Field et al., 2007; Crook et al., 2009).  
104 Crook et al. (2009) additionally provide evidence that inputs to the S-on subfield to blue-on  
105 SBS cells have centre-surround structure (S-on, ML-off), and blue-on cells showing  
106 band-pass response to S cone gratings have been recorded in macaque and marmoset LGN  
107 (Tailby et al., 2008a; Tailby, Szmajda, Buzás, Lee & Martin, 2008b). A population survey of  
108 the spatial transfer properties of S cone and ML cone receptive field subunits has not been  
109 made, and the degree to which blue-on and blue-off cells are specialised to transmit

110 chromatic *versus* spatial signals remains uncertain. Therefore, the third question we address  
111 in this paper is: are blue-on cells really Type II cells?

## 112 **Methods**

### 113 *Ethical approval.*

114 Procedures conformed to the Australian National Health and Medical Research Council  
115 (NHMRC) code of practice for the use and care of animals and institutional animal care and  
116 ethics committee at the University of Sydney. Procedures also conform to the code of ethics  
117 of the World Medical Association (Declaration of Helsinki).

### 118 *Animal preparation.*

119 Details of our animal preparation, recording technique, and visual stimulation environment  
120 have been published previously (Tailby et al., 2010; Pietersen et al., 2014). To summarize,  
121 extracellular recordings of single units were performed in the lateral geniculate nucleus  
122 (LGN) of common marmosets *Callithrix jacchus*. Animals were sedated with an  
123 intramuscular injection of Alfaxan (12 mg kg<sup>-1</sup>, Jurox, NSW, AUS) and Diazepam (3 mg kg<sup>-1</sup>,  
124 Roche, NSW, AUS), and anesthesia was maintained by continuous intravenous delivery of  
125 Sufentanil citrate (6 – 30 µg kg<sup>-1</sup> h<sup>-1</sup>; Sufenta Forte, Janssen Cilag, Beerse, BEL). Depth of  
126 anesthesia was monitored by continuous electroencephalography and pulse oximetry  
127 (SurgiVet, OH, USA). The animal was artificially respired with a 70%–30% mixture of  
128 NO<sub>2</sub>–Carbogen (5% CO<sub>2</sub> in O<sub>2</sub>) and head-fixed in a stereotaxic frame. A durotomy was made  
129 above the LGN and a guide tube containing the recording electrode was inserted into the  
130 brain. Action potential waveforms of single cells were discriminated by principal component  
131 analysis of amplified voltage signals from single microelectrodes (5 – 11 MΩ, FHC Inc.,

132 Bowdoin, Maine, USA). The position of each cell relative to the brain surface was recorded  
133 from a hydraulic microdrive (David Kopf Model 640). Electrolytic lesions (3–6  $\mu\text{A} \times 3\text{--}6\text{ s}$ ,  
134 electrode positive) were made to assist in track reconstruction. At the conclusion of  
135 recordings the animal was killed with an overdose of pentobarbitone sodium (80–150 mg  
136  $\text{kg}^{-1}$ , i.v.). The position of recorded cells was reconstructed histologically as described in  
137 detail in our previous publications (White, Goodchild, Wilder, Sefton & Martin, 1998;  
138 Cheong, Tailby, Solomon & Martin, 2013).

### 139 *Visual Stimuli*

140 Stimuli comprised drifting sine gratings and sine-modulated flashing dots, displayed on a  
141 stimulus monitor (refresh rate 100 or 120 Hz) against a grey background (mean luminance  
142  $50\text{ cd/m}^2$ ) which was centred on each receptive field using a front-silvered gimbaled mirror.  
143 The driving voltage of the red, green, and blue phosphors of the stimulus monitor were  
144 adjusted to produce cone selective (“silent substitution”) gratings, using the spectral  
145 radiance distribution of the monitor phosphors, the sensitivity distribution of the marmoset  
146 cone photoreceptors, and knowledge of the spectral absorbance of the optic media and  
147 macular pigment (Brainard, 1996; Blessing, Solomon, Hashemi-Nezhad, Morris & Martin,  
148 2004; Tailby et al., 2008a). Visual stimuli were generated using custom software which also  
149 collected and sorted recorded spike waveforms and times to within 0.1 ms. (EXPO;  
150 P. Lennie, University of Rochester, Rochester, NY). The S cone selective stimulus produced  
151 60-80% contrast in S cones and less than 5% contrast in ML cones. The ML cone selective  
152 stimulus produced 60-80% contrast in ML-class cones and less than 5% contrast in S cones,  
153 relative to the nominal maximum achievable Michelson contrast of 100% for an achromatic  
154 (S + ML) stimulus, which modulated both cone classes equally. For brevity, the

155 cone-isolating stimuli are referred to as S cone and ML cone stimuli hereinafter. An  
156 “optimal” mixed (S – ML) stimulus which drove the S and ML cones in counter-phase at half  
157 the RMS cone contrast of the S cone and ML cone stimuli was also used. Drifting gratings  
158 typically had a temporal frequency of 5 Hz (range 2-15 Hz). Stimulus aperture was typically  
159 4° (range 1° – 12°), adjusted to ensure stimulation of both centre and surround.

160 Spatial frequency tuning curves were collected with drifting gratings ranging from  
161 0.1 – 12.8 cycles/degree (cyc/deg) at 50% contrast. Contrast response curves were collected  
162 with drifting gratings ranging from 2% – 70% contrast relative to the maximum achievable  
163 achromatic contrast, typically at the identified peak spatial frequency (range 0 – 2.7  
164 cyc/degree). The amplitude and phase of the first Fourier harmonic of the stimulus  
165 frequency were taken as the primary measure of the cells’ response to stimulation.

#### 166 *Analysis of contrast sensitivity*

167 For the purposes of comparing S, ML, S + ML, and S – ML contrast response functions, the  
168 amplitude of the cell’s response as a function of contrast were fit to saturating hyperbolic  
169 functions (Naka & Rushton, 1966; Sclar, Maunsell & Lennie, 1990) of the form

$$170 \quad K = \frac{M(c^n)}{c^n + c_{50}^n} + b \quad (\text{Eq. 1})$$

171 In which the spike rate  $K$  is a function of stimulus contrast  $c$ , theoretical maximum spike  
172 rate  $M$ , semisaturation contrast  $c_{50}$ , exponent  $n$ , and noise-derived discharge at zero  
173 contrast  $b$ . The semisaturation contrast  $c_{50}$  is the contrast at which the response is at half  
174 of maximum. The exponent term  $n$  controls the steepness and curvature of the curve at  
175 intermediate contrasts. Fits were performed in MATLAB (Mathworks, Natick MA) using  
176 constrained non-linear least-squares minimization in which all values were constrained to



177 be positive, the values of  $c_{50}$  were constrained to fall between 0 and 200%, and the values  
 178 of the exponent term were constrained to be less than 3. These constraints ensured that the  
 179 gain of the cell can be estimated even for low amplitude responses. When the fitted  
 180 semisaturation constant was greater than the maximum contrast, it indicated a  
 181 non-saturating response; when semisaturation constants were not constrained they could  
 182 grow without bound without substantially improving the model fit to the data. The contrast  
 183 gain is given by the derivative of Eq. 1,

$$184 \quad K' = \frac{n M c^n c_{50}^n}{c(c^n + c_{50}^n)^2} \quad (\text{Eq. 2})$$

185 and is computed at the semisaturation point  $c_{50}$  for saturating cells ( $c_{50} < 100\%$ ) and at  
 186 maximum contrast for non-saturating cells. In prior work where the value of  $n$  was fixed  
 187 to 1 (e.g. Kaplan and Shapley, 1986), the contrast gain was instead computed at low  
 188 contrast ( $c = 0$ ). It can be determined from Eq. 2 that at  $c = 0$ ,  $K'$  exists and is non-zero  
 189 if and only if  $n = 1$ .

190 For the purpose of model identification, data were also fit to a simpler version of  
 191 Eq. 1 in which the exponent was fixed at 1, and to an elaborated version of Eq. 1. (Peirce,  
 192 2007) which permits non-monotonic supersaturating responses:

$$193 \quad K = \frac{M(c^{n_1})}{c^{n_2} + c_{50}^{n_2}} + b. \quad (\text{Eq. 3})$$

194 In this model there are two exponent terms  $n_1$  and  $n_2$ , and variables are otherwise as  
 195 Eq. 1. To test the hypothesis that this equation with its increased number of free  
 196 parameters provided a significantly better fit to the data, we calculated the residual errors  
 197 for each model, following Buzás, Kóbor, Petykó, Telkes, Martin & Lénárd (2013). If the more  
 198 complex model significantly reduced the residual error over its predecessor, as assessed by

199 a 1-sided 2-sample F test for equal variances ( $p < 0.05$ ), then the more complex model was  
 200 adopted. The data were also fit to a model which implements a thresholding rather than an  
 201 expansive nonlinearity:

$$K = \max\left(\frac{M(c - c_0)}{c - c_0 + c_{50}}, 0\right) + b. \text{ (Eq.4)}$$

202 In this model the exponent term is fixed to 1, but a constant value is added to the contrast.  
 203 At contrasts below  $c_0$ , the predicted response is the noise-derived discharge at zero  
 204 contrast  $b$ .

### 205 *Analysis of chromatic summation*

206 Two extended models incorporating alternate hypothetical mechanisms for the  
 207 summation of S and ML inputs were fit to the amplitude and phase of each cell's responses.  
 208 The first model, illustrated in Figure 1A, assumes separable contrast mechanisms for S cone  
 209 and ML cone input and is given by

$$K = \frac{M_S c_S |c_S|^{n_S-1}}{|c_S|^{n_S} + c_{S,50}^{n_S}} + \frac{M_{ML} c_{ML} |c_{ML}|^{n_{ML}-1}}{|c_{ML}|^{n_{ML}} + c_{ML,50}^{n_{ML}}} + b \text{ (Eq. 5)}$$

210 In which  $K$  is the complex spike rate,  $c_S$  is S cone contrast and phase,  $M_S$  is the  
 211 theoretical saturation spike-rate (and relative phase) for S cone contrast,  $n_S$  is the  
 212 exponent of the S cone contrast function, and  $c_{S,50}$  is the semisaturation contrast for the S  
 213 cone contrast function. The notation for ML cone contrast mirrors that for S cone contrast.  
 214 The complex spike rate  $K$  is the complex sum of one Naka-Rushton curve representing S  
 215 cone input  $c_S$  and a second Naka-Rushton curve representing ML cone input  $c_{ML}$ . Prior  
 216 evidence (Yeh et al., 1995a; Solomon & Lennie, 2005) suggests that phase advance is not  
 217 prominent for blue-on cells, and so this expression ignores the phenomenon of phase

218 advance (Shapley & Victor, 1978; Benardete & Kaplan, 1999). In other words, all responses  
 219 to S cone stimuli share a single phase, as do all responses for ML cone stimuli. This model  
 220 assumes that the nonlinearities in the contrast response are present in the S and ML signals  
 221 prior to their combination, and also predicts that the overall contrast response to stimuli  
 222 having both S and ML contrasts need not be monotonic where there is a mismatch between  
 223 S and ML parameters.

224 An alternative model, illustrated in Figure 1B, assumes linear summation of cone  
 225 inputs followed by nonlinear distortion, and is given by

$$K = \frac{(M x)|x|^{(n-1)}}{(|x|^n + c_{50}^n)} + b,$$

$$x = w c_{ML} e^{i\phi_{ML}} + (1 - w)c_S e^{i\phi_S}$$

226 In which the complex spike-rate  $K$  is a function of the complex drive  $x$ , which itself is the  
 227 weighted complex sum of the S cone and ML cone contrasts. As in Eq. 1,  $M$  is theoretical  
 228 saturation spike-rate,  $c_{50}$  is semisaturation contrast,  $n$  is an exponent term, and the tonic  
 229 discharge rate is  $b$ . The ML cone fraction  $w$  varies from 1 when the cell receives  
 230 exclusively ML cone input to 0 when the cell receives exclusively S cone input, and the  
 231 response phase of the S cone and ML cone inputs are given by  $\phi_{ML}$  and  $\phi_S$ , respectively,  
 232 for S cone contrast  $c_S$  and ML cone contrast  $c_{ML}$ . This model assumes that a linear  
 233 summation of approximately linear S cone and ML cone signals occurs, and that the output  
 234 of that summation is distorted by nonlinearity either at the ganglion cell level, at the  
 235 ganglion cell to LGN synapse, or at the point of spike generation in the LGN.

236 *Analysis of spatial distribution of cone inputs*

237 We employed a straightforward extension of the standard difference-of-Gaussians (DoG)  
 238 model of Rodieck & Stone (1965) and Enroth-Cugell & Robson (1966), which accounts for  
 239 response phase by taking the complex sum of Gaussian kernels (Frishman, Freeman, Troy,  
 240 Schweitzer-Tong & Enroth-Cugell, 1987). Each kernel  $G_n$  is given by

$$G_n(\omega) = \pi r_n^2 e^{2\pi i(\phi_n + \delta_n \omega) - (\pi r_n \omega)^2} \text{ (Eq. 7)}$$

241 in which the stimulus spatial frequency is  $\omega$ , the radius of the Gaussian kernel is  $r_n$ , the  
 242 displacement of the field from the origin is  $\delta_n$ , and the response phase to a spatially  
 243 uniform stimulus is  $\phi_n$ , which is determined from the cell's dominant (S or ML) input and  
 244 response latency. The overall response spike-rate  $K$  is the complex sum of a variable  
 245 number of Gaussian kernels, each having a magnitude  $M_n$ ,

$$K = \sum_{n=1}^N M_n G_n(\omega). \text{ (Eq. 8)}$$

246 This formulation is able to support spatial-frequency-dependent changes in response phase  
 247 (Enroth-Cugell, Robson, Schweitzer-Tong & Watson, 1983). Fits were performed in MATLAB  
 248 again using a constrained non-linear least-squares algorithm. The volume  $V_n$  of a receptive  
 249 subfield, assuming circular receptive fields (but see also Chichilnisky & Baylor, 1999; Field et  
 250 al., 2007; Tailby et al., 2010), is given by

$$V_n = \pi r_n^2 M_n, \text{ (Eq. 9)}$$

251 and can be thought of as the expected response of the cell if all other excitatory or  
 252 inhibitory subfields were blocked and the entire subfield were to be stimulated by its  
 253 preferred stimulus.

254 Not every cell for which spatial frequency tuning was available also had chromatic contrast  
 255 response data, and so in the spatial model contrast nonlinearities are ignored; instead,

256 response amplitudes were normalized by contrast. For each cell, a sequence of  
 257 progressively more elaborate models was fit, as described in Table 1.

Model Type	N =	Constraints	Free parameters
Classic type II RF	2 (S,ML)	$r_S = r_{ML}; \delta_S = \delta_{ML};$ $\delta_n < 2 r_n$	6
Simple Opponent Cell	2 (S,ML)	$\delta_n < 2 r_n$	8
S cone Centre-Surround	3 (Sc, Ss, ML)	$r_{Sc} < r_{Ss}; \delta_{Sc} = \delta_{Ss};$ $\phi_{Sc} = \phi_{Ss} + \pi; \delta_n < 2 r_n$	10
ML cone Centre-Surround	3 (S,MLc,MLs)	$r_{MLc} < r_{MLs}; \delta_{MLc} = \delta_{MLs};$ $\phi_{MLc} = \phi_{MLs} + \pi; \delta_n$ $< 2 r_n$	10
Double-Opponent Centre-Surround	4 (Sc, Ss, MLc, MLs)	$r_{Sc} < r_{Ss}; \delta_{Sc} = \delta_{Ss};$ $r_{MLc} < r_{MLs}; \delta_{MLc} = \delta_{MLs};$ $\phi_{Sc} = \phi_{Ss} + \pi;$ $\phi_{MLc} = \phi_{MLs} + \pi;$ $\delta_n < 2 r_n$	12

258

259 For each model, residual errors were calculated and compared using 1-sided 2-sample F  
 260 tests for equal variances ( $p < 0.1$ ). The simplest model which significantly reduced the  
 261 variance of the data compared to its predecessor was adopted, as described above for  
 262 contrast responses.

### 263 *Dataset*

264 The dataset comprised recordings from 155 cells (51 blue-on cells, 6 blue-off cells, 74 P cells  
 265 and 24 M cells) from 27 animals. Responses of 68 cells to parts of the stimulus set were  
 266 previously described (Tailby et al., 2008b; Tailby et al., 2010; Pietersen et al., 2014; Cheong  
 267 & Pietersen, 2014). What is new here is our analyses of datasets comprising responses to  
 268 ML isolating stimuli and mixed chromatic (S – ML) stimuli as well as S and achromatic (S +  
 269 ML) stimuli. Most receptive fields (63.3%) were located between 2° and 12° eccentricity;  
 270 14.8% were located within 2° of the fovea and 21.9% were located at more than 12°

271 eccentricity. No systematic differences in responses to S cone stimulation were found  
272 between trichromatic female animals (identified by the presence of red-green opponent  
273 parvocellular cells), and the other dichromatic animals, all of which had a visual phenotype  
274 consistent with the presence of S cones and one cone type with peak sensitivity close to 543  
275 nm, 556 nm, or 563 nm (ML cones), and so data were pooled for analysis. Not every cell had  
276 both a contrast and a spatial frequency measurement recorded.

277         Cells were classified as blue-on, blue-off, parvocellular (P), or magnocellular (M) by  
278 the response to brief (200 or 500 ms) temporal square-wave stimuli (Pietersen et al., 2014)  
279 supplemented by measurements of spatial and temporal frequency sensitivity. Examples of  
280 a typical blue-on, P, and M response are shown in Figure 2.

281         Anatomical locations of 41%, or 64/155 cells (22/51 blue-on, 2/6 blue-off, 32/74 P,  
282 and 8/24 M) were confirmed offline with histology as described above. In cases where track  
283 location was not determined, the receptive field properties, eye dominance, encounter  
284 position, and response characteristics were used as criteria. Based on the combined  
285 anatomical and physiological criteria, one blue-on cell was located in the (ventral-most)  
286 koniocellular layer K1, 1 cell was in K2 (between the M layers), 17 cells were in K3 (between  
287 the M and P layers), 9 were in K4 (between the internal and external parvocellular layers),  
288 and 4 cells were in K6 (dorsal to the external parvocellular layer). One blue-on cell was  
289 located 'ectopically' in the ipsilateral M layer. The laminar location of 18 blue-on cells could  
290 not be determined unequivocally. Two blue-off cells were located in K3, two blue-off cells  
291 were located 'ectopically' in a P layer, and two blue-off cell locations could not be  
292 determined unequivocally. No systematic differences in blue-on or blue-off cell properties  
293 were apparent on comparing receptive fields from different layers.

294 **Results**

295 *Chromatic contrast response functions*

296 Figure 3A shows raster plots of a typical blue-on cell for 2 s presentations of 5 Hz S cone, ML  
297 cone, achromatic (S + ML) and mixed chromatic (S – ML) drifting gratings, arranged by  
298 stimulus contrast. At intermediate and high contrast the cell clearly responds in a  
299 phase-locked manner to each stimulus cycle, and the S cone response appears at close to  
300 opposite phase of the ML response. Response phase for S cone and ML cone remains  
301 constant across contrast. The lack of contrast-dependent phase advance agrees with  
302 previous data from blue-on cells in LGN of macaque and marmoset (Solomon & Lennie,  
303 2005; Tailby et al., 2008b) and retina of macaque (Yeh et al., 1995a) and capuchin monkey  
304 *Cebus apella* (Silveira et al., 1999). The cell responds only feebly to achromatic (S + ML)  
305 stimulus, but responds vigorously to the chromatic (S – ML) stimulus. Figure 3B shows the  
306 average firing rate and first 5 Fourier components of the response at maximum contrast; the  
307 zeroth and first harmonic contain the majority of the response power. The power in the  
308 higher harmonics chiefly arises from rectification of the response; when rectification is  
309 addressed by fitting a rectified sine wave to the response, the residual harmonic distortion  
310 ratio (HDR; the ratio of the sum of the squares of the 2<sup>nd</sup>-5<sup>th</sup> harmonics to the square of the  
311 fundamental) decreased by more than 20-fold. Across the population of recoded blue-on  
312 cells, correcting for rectification in the response results on average in an 8-fold decrease in  
313 HDR (data not shown). Before correcting for rectification, median values of HDR for blue-on  
314 cells are 0.57 (90% range 0.13-3.2) for S responses and 0.46 (90% range 0.12-2.0) for ML  
315 responses. After correction, these ranges decrease to 0.17 (90% range 0.02-0.88) and 0.16  
316 (90% range 0.01-1.04), respectively.

317 Figure 3C shows the amplitude of the first-harmonic responses with fitted  
318 Naka-Rushton curves, as given by Eq. 1. The response to S cone stimulation is expansive at  
319 low contrast and shows mild saturation at high contrast, yielding a sigmoid shape. Expansive  
320 nonlinearity at low contrast is less evident in the off-phase response to ML cone stimulation  
321 (Fig. 3B), but are also evident for the response to a mixed chromatic stimulus. It is  
322 interesting to note that blue-on ganglion cells in macaque retina show little sign of  
323 expansive nonlinearity (Yeh et al., 1995a; Crook et al., 2009); we return to this point in a  
324 later section.

325 Figure 3D shows the responses plotted in the complex plane, with time passing in  
326 clockwise direction. Plotting the data this way reveals that the ML cone response is in  
327 close-to-opposite phase but  $\sim 10$  deg ( $\sim 5$  ms at 5 Hz) slower than the S response, as  
328 previously reported (Tailby et al., 2010; Pietersen et al., 2014). Figure 3E-H show example  
329 responses for a blue-off, yellow-on cell. This cell has a lower baseline firing rate and overall  
330 activity, but clear phase-locking and opposite-phase responses can also be observed. The S-  
331 and ML cone response phases are opposite to those observed for the blue-on cell.

332 For blue-on cells, there were no significant differences between the distributions of  
333 the fitted Naka-Rushton semisaturation constants for curves fitted to the first-harmonic  
334 response, as compared to curves fitted to a rectification-corrected harmonic response, for  
335 either S, ML, achromatic, or mixed S – ML stimulation ( $p > 0.6$  for each case, paired  
336 Wilcoxon test). All values reported above were fitted to unmodified first-harmonic  
337 responses, as is typically reported. Figure 4 shows distributions of the fitted semisaturation  
338 constants (Fig. 4A), maximum gains (Fig. 4C), and exponent parameters (Fig. 4E) fitted using  
339 Eq. 1 to S cone and ML cone stimuli for blue-on and blue-off cells, as well as the



340 distributions of these fitted parameters for P and M cells (Fig. 4B, D, and F). Summary  
 341 statistics for the data shown in Figure 4 are given in Table 2. As expected (Kaplan & Shapley,  
 342 1986; Yeh et al., 1995b), the M cell semisaturation constants were lower, and M cell gain  
 343 was significantly higher, than those of P or blue-on cells ( $p < 0.01$  for all comparisons, 3-way  
 344 Kruskal-Wallis test [KW]). The P cell exponent terms were significantly lower than those of  
 345 blue-on or M cells ( $p < 0.01$ , KW). For blue-on cells, S cone gain was correlated with ML cone  
 346 gain (Fig. 4C,  $r = 0.77$ ,  $p < 0.01$ ) and significantly greater than ML gain ( $p = 0.01$ , 2-way KW),  
 347 suiting these cells' description as blue-on as opposed to yellow-off. Similarly, semisaturation  
 348 constants fitted to S cone responses were significantly lower than those fit to ML cone  
 349 responses ( $p < 0.01$ , 2-way KW). Of the 33 blue-on cells within  $10^\circ$  of the fovea for which a  
 350 contrast response function could be fit, 26 had a clear half-maximal response nonlinearity  
 351 ( $c_{50} < 100\%$ ) in their S response and 20 had a clear nonlinearity in their ML response.

352 Table 2

	Semisaturation (%)	Contrast gain (imp/s % <sup>-1</sup> )	Exponent
Blue-on cells S cone	63.4 ± 45.2	0.60 ± 0.42	2.4 ± 0.6
Blue-on cells ML cone	87.3 ± 46.7	0.40 ± 0.30	2.3 ± 0.7
Blue-off cells S cone	29.4 ± 17.9	0.55 ± 0.36	2.3 ± 0.8
Blue-off cells ML cone	65.6 ± 58.5	0.32 ± 0.22	2.2 ± 1.0
P cells	92.9 ± 51.0	0.35 ± 0.34	1.9 ± 0.7
M cells	22.1 ± 29.6	2.17 ± 1.34	2.4 ± 0.6

353 All reported values mean ± standard deviation

354 The scatterplot in Figure 4A raises the impression of two populations of blue-on cells with  
 355 respectively high and intermediate semisaturation constants. We found however that (apart  
 356 from the correlation of S and ML gain noted above) the joint distributions of the fitted  
 357 Naka-Rushton parameters for blue-on cells did not hold significant correlations, nor was  
 358 there evidence of clustering in the parameter space (data not shown). A low semisaturation

359 constant for S cone contrast neither predicted nor was predicted by a low semisaturation  
360 constant for ML cone contrast ( $\chi^2 = 0.04$ ,  $p = 0.833$ ). Contrast gain was mildly correlated  
361 with eccentricity for both S cone and ML cone ( $r = 0.49$  and  $r = 0.55$ , respectively); however,  
362 no other parameter was correlated with eccentricity. This result stands in contrast to the  
363 result for P cells in which semisaturation, was inversely correlated with eccentricity ( $r =$   
364  $-0.36$ ;  $p = 0.018$  vs.  $r = 0.17$ ;  $p = 0.273$ ) as previously observed (Solomon, White & Martin,  
365 1999). Blue-off cells (green cross symbols, Fig. 4A,C,E) had similar characteristics to blue-on  
366 cells but the number of cells is too small to make statistical comparisons. In comparison to a  
367 simplified model in which the exponent term is held constant (see Methods section), the  
368 expansive nonlinearity of Eq. 1 was necessary to explain the responses for a majority (29/44,  
369 65%) of blue-on cells. Interestingly, the addition of an expansive term also improved the fits  
370 for a substantial fraction (26/53, 49%) of P cells. On the other hand, the use of a  
371 supersaturating Naka-Rushton curve (Eq. 3) did not significantly improve the fit to the data  
372 for any cell, and non-monotonic responses were never observed in blue-on cells. Either an  
373 expansive or a thresholding nonlinearity (Eq. 4) could adequately fit the data overall.  
374 However, only the expansive nonlinearity model (Eq. 1) could consistently predict responses  
375 at the contrast threshold. For blue-on cells, the overall RMS error of the expansive  
376 nonlinearity model was not significantly different to the overall RMS error of the threshold  
377 model ( $3.7 \pm 1.4$  imp/s vs.  $3.9 \pm 1.5$  imp/s, respectively,  $p = 0.34$ , paired Wilcoxon test).

378         Response saturation is customarily associated with contrast gain control, which is  
379 also manifest as advance in response phase with increasing contrast (Shapley & Victor,  
380 1981; Kaplan & Benardete, 2001). On comparing response phase at half-maximum and  
381 maximum contrast for preferred chromatic direction we found substantial phase advance in

382 MC cells ( $37.4 \pm 18$  degrees,  $n = 22$ ), as would be expected (Yeh et al., 1995a; Levitt, Shumer,  
383 Sherman & Spear, 2001; Solomon, Lee & Sun, 2006). Phase advance in MC cells was  
384 significantly greater ( $p < 0.02$ , Kruskal-Wallis test) than the (negligible) phase advance in PC  
385 cells ( $6.0$  degrees  $\pm 13$ ,  $n = 46$ ) and blue-on cells ( $3.3 \pm 9$ ,  $n = 44$ ); there was no difference  
386 between PC cells and blue-on cells ( $p = 0.95$ , Kruskal-Wallis test). There was no clear sign of  
387 correlation between semisaturation constant and phase advance in blue-on cells ( $r^2 < 0.01$ ,  
388  $p = 0.82$ ). These data suggest that blue-on cells do not show contrast gain control to any  
389 great extent. A more extensive analysis of response timing may be of interest for future  
390 work.

391 *Achromatic responses arise from the summation of chromatic signals*

392 Blue-on cells integrate S cone signals with ML cone signals, and it is clear from Figure 4 that  
393 there is variation between the S cone and ML cone responses within individual cells. We  
394 showed above that this variation may be due to S and ML signals having different contrast  
395 response characteristics at the level of the outer retina. If this is the case, then the  
396 integrated responses to co-stimulation of S and ML cones is given by the sum of the  
397 responses to cone stimuli, which could lead to non-monotonic achromatic contrast  
398 responses (Fig. 1A, Eq. 5). Alternatively, the S cone and ML cone inputs to the blue-on cell  
399 may be approximately linear, in which case the integrated output cannot be expressed  
400 exactly as the sum of the component responses, but must be monotonic with increasing  
401 contrast (Fig. 1B, Eq. 6).

402 Figure 5 shows a particularly illustrative example of a blue-on cell's responses where  
403 the predictions made by these two models diverge. Figure 5A shows the cell's responses as  
404 a function of contrast with fitted Naka-Rushton curves, as given by Eq. 6, for S and ML cone

405 stimuli. Chromatic contrast is expressed relative to stimulus cone contrast (80% for S and  
406 ML cones). Figure 5B shows the measured achromatic contrast response function, along  
407 with predicted contrast response functions given by fitting the two competing models to the  
408 S and ML cone response data. The model with separable contrast mechanisms (dashed line,  
409 Fig. 5B) predicts a non-monotonic achromatic contrast response. The non-monotonic  
410 prediction arises because the expansive nonlinearity for ML-off is weaker than that for S-on,  
411 predicting response cancellation at high but not intermediate contrast. The measured data,  
412 however, falls on the curve predicted by the summation of linear cone inputs which are  
413 distorted by a nonlinearity post-integration. Figure 5C shows the amplitude and phase of  
414 the responses in the complex plane.

415 Overall, the weight of experimental evidence favors the model where summation of  
416 linear cone inputs occurs prior to non-linear distortion, as described in Eq. 6 and illustrated  
417 in Figure 1B. Across the population of blue-on cells, non-monotonic achromatic contrast  
418 responses were never observed. Most cells could be reasonably fit by either model, as  
419 would be expected if the cells are operating in the linear regime of their contrast response  
420 functions. Figure 6 shows measured vs. predicted responses to achromatic (S + ML) and  
421 mixed chromatic (S – ML) stimuli for the population of cells tested. Figure 6A shows the  
422 predictions of Eq. 6 for achromatic (S + ML) stimulation (median RMS error 8.1 imp/s),  
423 Figure 6B shows the predictions of Eq. 6 for mixed chromatic (S – ML) co-stimulation  
424 (median RMS error 5.4 imp/s), Figure 6C shows the predictions of Eq. 5 for achromatic (S +  
425 ML) stimulation (median RMS error 8.3 imp/s), and Figure 6D shows the predictions of Eq. 5  
426 for S – ML stimulation (median RMS error 11.0 imp/s). The two models perform similarly in  
427 prediction of achromatic responses ( $p = 0.66$ , 2-way KW), but the linear inputs model clearly

428 outperforms the separable contrasts model for chromatic (S – ML) stimulation ( $p < 0.01$ ,  
429 2-way KW).

#### 430 *Amplitude and timing of opponent inputs to blue-on and blue-off cells*

431 Figure 7 shows, for the data collected using 5 Hz stimulation, the amplitude and phase of  
432 responses to maximum-contrast S cone (Fig. 7A), ML cone (Fig. 7B), achromatic (Fig. 7C),  
433 and S – ML stimulation (Fig. 7D) in the complex plane. Figures 7C and 7D also show (square  
434 symbols) the distributions of achromatic and S – ML responses predicted by a vector sum of  
435 S and ML inputs. There are four main points to take from these data. Firstly, as expected,  
436 the S and ML responses cluster around close-to-opposite phase, consistent with S-on and  
437 ML-off excitation. The mean phase delay between S and ML was 187 deg, equivalent to an  
438 additional 3.6 ms at 5 Hz. Secondly, there is considerable variability in response phase  
439 across the population for all stimuli. As the data of figure 7 were collected at varying spatial  
440 frequencies, some variation in the observed response phases is due to offset of the centre  
441 of the receptive field from the centre of the stimulus patch. The true response phases to  
442 uniform stimulus are less variable than this figure implies – the circular standard deviation  
443 of the response phases to spatially uniform 5 Hz S cone stimulation is  $22.4^\circ$  ( $n = 40$ ),  
444 equivalent to  $\pm 12.4$  ms at 5 Hz. Thirdly, for most (but not all) blue-on cells the phase of the  
445 response to achromatic modulation is close to that for S cone modulation (circular  $r = 0.42$ ,  
446  $p < 0.02$ ). Finally, the fit predictions for achromatic and S – ML stimuli show heavy overlap in  
447 phase space with the measured values, most evidently for S – ML stimuli (Fig. 7D). The  
448 reader should note that these data are also consistent with the "sum-then-distort" contrast  
449 integration model described above (Eq. 6, Fig 1B, Fig. 6).

450 Figure 7E shows on a per-cell basis the amplitude and phase of responses to

451 maximum-contrast S cone, ML cone, together with measured achromatic (S + ML) response  
452 (black vector) and the vector sum prediction (magenta square symbols). Responses are  
453 normalized to the maximum response amplitude, shown at bottom of each sub-plot. Here  
454 we see that the broad distribution of response phase across the population belies some  
455 hidden regularity, by revealing that the S and ML response phases are correlated on a cell by  
456 cell basis (circular  $r = 0.61$ ,  $p < 0.01$ ). Responses to achromatic stimuli are attributable to  
457 differences in amplitude and/or timing of the S and ML inputs. Two example cells are  
458 marked in Figure 7E. The cell in the first example (open arrowhead, Fig. 7E [68, bottom left])  
459 shows identical amplitude for S and ML inputs, but the ML input lags the S input by  $209^\circ$ ,  
460 predicting a vigorous on-type achromatic response. For the second, [54, upper right] the  
461 response phase difference is  $187^\circ$  but the S input amplitude is greater than the ML input  
462 amplitude, again predicting a (weak) achromatic on-type response. These data show that  
463 the gain and timing of S and ML inputs jointly determine responses to non-cone-isolating  
464 stimuli.

#### 465 *Spatial properties of S and ML inputs to blue-on cells*

466 A variety of receptive field structures was found for blue-on cells, as illustrated in Figure 8.  
467 The simplest model (spatially coextensive S and ML subfields consistent with classical “type  
468 II” organization), best fit the responses of 15 of 47 cells (32%). An example of the receptive  
469 field structure of a type II cell is shown in Figure 8A. The associated S cone, ML cone, and  
470 achromatic tuning curves and fitted Gaussians are shown in Figure 8B, and the S and ML  
471 cone responses are plotted in the complex plane in Figure 8C. These cells rarely showed  
472 achromatic responses at any spatial frequency; however, spatially low-pass achromatic  
473 responses were observed in 5 of the 15 type II cells. In 5 cells (11%), a S-on subfield and a

474 ML-off subfield of different sizes (which were not necessarily concentric) were sufficient to  
475 explain the cell's response, consistent with the receptive field organization described in  
476 Tailby et al. (2010). An example of this receptive field structure is shown in Figure 8D, along  
477 with the associated fitted S cone, ML cone, and achromatic tuning curves (Fig. 8E) and  
478 location in the complex plane (Fig. 8F). Note the band-pass response to achromatic  
479 stimulation. In 13 cells (28%), the S cone input had spatial bandpass tuning without  
480 evidence of a corresponding bandpass characteristic for ML cone input. Responses of such  
481 cells are consistent with the presence of an S cone surround. An example of such a cell is  
482 shown in Figure 8G, along with the associated fitted S cone, ML cone, and achromatic tuning  
483 curves (Fig. 8H) and location in the complex plane (Fig. 8I). Both the achromatic and the S  
484 cone spatial frequency tuning curves show response roll-off at low spatial frequencies.  
485 Bandpass achromatic responses were present in 6 of 13 cells, with low-pass responses  
486 observed in a further 2 cells. The remainder (14 out of 47 cells, 30%) required both an S-off  
487 surround and a weak ML-on surround to explain the cell's response. An example of such a  
488 receptive field is shown in Figure 8J, along with the associated fitted S cone, ML cone, and  
489 achromatic tuning curves (fig. 8K) and location in the complex plane (fig. 8L). As we show  
490 below, overall the ML surrounds were weaker than S surrounds, and made only a small but  
491 significant improvement in fit quality. Spatial bandpass achromatic responses were more  
492 frequent in this population, being present in 9 out of 14 cells.

493           Figure 9 shows radius and volume of S cone and ML cone subunits for blue-on and  
494 blue-off cells, and compares these parameters with ML cone inputs to P cells. Summary  
495 statistics for the data shown in Figure 9 (A, B, C, E) are given in Table 3. For blue-on cells, the  
496 S-on subfield has a marginally smaller radius than the ML-off subfield (Fig. 9A,  $p = 0.10$ ,

497 paired Wilcoxon test), but has, on average, significantly larger volume (Fig. 9B,  $p < 0.01$ ,  
 498 paired Wilcoxon). The S-on subfield radii increased with foveal eccentricity (Kendall's tau =  
 499 0.31,  $p < 0.01$ ) as has been reported previously (Tailby et al., 2008b). Surprisingly, the  
 500 eccentricity-dependence was less pronounced for the ML-off subfields (tau = 0.15,  $p =$   
 501 0.17). The reason for this difference is not clear. The reported receptive field radii for the  
 502 S-on and ML-on subunits are within the range which would be expected based on the  
 503 dendritic arbor of the SBS RGC:  $0.50^\circ \pm 0.22^\circ$  for S cones and  $0.26^\circ \pm 0.15^\circ$  for ML cones (see  
 504 also Tailby et al., 2010).

505 **Table 3**

Blue-on cells	Radius (deg)	Volume (imp/s % <sup>-1</sup> )	Frequency
S-cone on-polarity centre	$0.38^\circ \pm 0.37^\circ$	$1.58 \pm 1.15$	100% (47/47)
ML-cone off-polarity centre	$0.40^\circ \pm 0.33^\circ$	$1.29 \pm 0.98$	100% (47/47)
S-cone off-polarity surround	$1.08^\circ \pm 0.69^\circ$	$1.09 \pm 1.13$	55% (26/47)
ML-cone on-polarity surround	$2.41^\circ \pm 5.44^\circ$	$1.33 \pm 1.56$	30% (14/47)
P cell centre (ML-cone)	$0.10^\circ \pm 0.10^\circ$	$1.19 \pm 0.48$	100% (72/72)
P cell surround (ML-cone)	$0.80^\circ \pm 0.88^\circ$	$0.91 \pm 0.40$	85% (61/72)

506 All values mean  $\pm$  SD

507 Figure 9C compares ML centre and surround radius for P cells against the S-on, S-off,  
 508 ML-off, and ML-on subunits (where detectable) of blue-on cells. Figure 9D shows the ratios  
 509 of these quantities. As expected (for review, see Martin & Lee, 2014), P cell centre radii are  
 510 significantly smaller than S-on and ML-off radii in blue-on cells ( $p < 0.01$  for both  
 511 comparisons, independent Wilcoxon). Also as expected, the S-on/ML-off ratio for blue-on  
 512 cell RF radii ( $0.993 \pm 0.408$ ) is greater than the ratio of P cell centre/surround radii ( $0.203 \pm$   
 513  $0.173$ ,  $p < 0.01$ , n-way KW), reflecting their distinct origins in retinal wiring (Field et al.,  
 514 2007; Crook et al., 2009). Where measurable, the blue-on cell S-on/S-off and ML-off/ML-on  
 515 radius ratios ( $0.347 \pm 0.218$  and  $0.365 \pm 0.186$ , respectively) were also distinguishable from



516 P cells ( $p < 0.01$  and  $p = 0.04$ , respectively); perhaps unsurprisingly, the distribution of  
517 ML-cone centre/surround ratios was more similar to the distribution of P cell  
518 centre/surround ratios.

519 Figure 9E compares ML centre and surround volumes for P cells against the S-on,  
520 S-off, ML-on and ML-off subunits (where detectable) for blue-on cells. Figure 9F shows the  
521 ratios of these quantities. In contrast to the marked differences in radii, P cell centre  
522 volumes were not significantly different from S or ML subunit volumes in blue-on cells ( $p =$   
523  $0.09$  and  $p = 0.28$  for S cone and ML cone subfields, respectively, independent Wilcoxon).  
524 Correspondingly, the S-on/ML-off volume ratio in blue-on cells ( $1.470 \pm 1.037$ ) was close to  
525 the centre/surround volume ratio in P cells ( $1.596 \pm 0.860$ ,  $p = 0.414$ , independent Wilcoxon).  
526 This result may help explain why P cells and blue-on cells both respond weakly to  
527 achromatic contrast at low spatial frequencies: the antagonistic subunit sizes are very  
528 different in P cells and blue-on cells, yet the integrated sensitivities are similar and  
529 well-matched. On the other hand, where detectable, the volume ratios S-on/S-off and  
530 ML-off/ML-on in blue-on cells were higher ( $2.613 \pm 1.952$  and  $2.286 \pm 1.070$ , respectively).  
531 In other words, the spatially antagonistic subunits in blue-on cells contribute only weakly to  
532 shaping the cells' overall response.

### 533 **Discussion**

534 We show that when stimulated with low-to-moderate S cone or ML cone contrast, blue-on  
535 cells show predominantly linear contrast response functions. Mild response saturation at  
536 high contrast and some expansive nonlinearity at low contrasts are evident.

537 The degree of response saturation in blue-on cells shows heavy overlap with that of

538 PC cells and is much weaker than that of MC cells (Fig 4A, B). The expansive nonlinearity  
539 could potentially sharpen blue-on cells' chromatic selectivity as proposed for cortical  
540 neurons (Heeger, 1992; DeValois, Cottaris, Elfar, Mahon & Wilson, 2000; Solomon, Peirce &  
541 Lennie, 2004; Solomon & Lennie, 2005), but our stimulus set was too restricted to address  
542 this possibility. The fact that blue-on ganglion cells show little sign of expansive nonlinearity  
543 (Yeh et al., 1995a; Crook et al., 2009) suggests that this distortion occurs either at the  
544 ganglion cell to LGN cell synapse or at the LGN spike output stage. Expansive nonlinearity at  
545 low contrasts is also a feature of PC cell responses in LGN of awake and anesthetized  
546 macaque monkeys (Alitto, Moore, Rathbun & Usrey, 2011), thus its presence in the PC cells  
547 and blue-on cells we recorded is not simply attributable to a species difference, or  
548 sufentanil anesthesia, or a switch of the LGN into a non-responsive "burst" mode (Sherman,  
549 1996). It is important to note however that the effects we see in LGN are much milder than  
550 the nonlinearities in cortical cell responses reported in the studies cited above and  
551 elsewhere.

552 We found that responses to simultaneous recruitment of both S cones and ML cones  
553 are consistent with integration of linear S cone and ML cone signals prior to non-linear  
554 distortion of the integrated signal. This result is consistent with measurements of linear S  
555 cone and ML cone synaptic inputs to SBS ganglion cells (Chichilnisky & Baylor, 1999; Field et  
556 al., 2007; Crook et al., 2009). In common with these previous reports, in our experimental  
557 setup we could only access a limited range of intensities (maximum  $120 \text{ cd m}^{-2}$ ), where the  
558 cone signals are likely operating in a linear range. Lee, Valberg and colleagues (1983, 1987)  
559 used narrow band spectral lights to show saturating cone inputs are needed to account for  
560 responses of blue-on cells at high intensities ( $> 500 \text{ cd m}^{-2}$ ). The presence of response

561 saturation at a pre-cortical level may also help to explain some features of cortical contrast  
562 response (Solomon & Lennie, 2005) and fits within some models of the LGN's computational  
563 contribution to vision (Dan, Atick & Reid, 1996; Mante, Frazor, Bonin, Geisler & Carandini,  
564 2005).

565         Our observation of blue-on LGN cells with varying receptive field structures may also  
566 have implications for current theories on the retinal circuitry underpinning blue-on cells.  
567 One current theory is that the SBS ganglion cell receives convergent input from a S  
568 cone-specific bipolar cell (BB) as well as one or more classes of diffuse off-type bipolar cell  
569 (DB) (Crook et al., 2009; Dacey, Crook & Packer, 2013). The S cone signal from the BB input  
570 is expected to carry an off-polarity ML cone surround (Packer, Verweij, Li, Schnapf & Dacey,  
571 2010) and the ML cone signal from the DB cells carries an on-polarity ML cone surround  
572 (Dacey, Diller, Verweij & Williams, 2000). Crook et al. (2009) proposed that mutual  
573 anihilation of these ML surround signals generate spatially co-extensive S cone and ML  
574 cone receptive fields (Crook et al., 2009, Dacey et al., 2014). Cell-specific imbalances in this  
575 mechanism could be expected to produce a distribution of ML cone surrounds, which could  
576 partially explain the variation in receptive field structure observed in blue-on cells in the  
577 LGN. The convergent BB+DB input theory, however, fails to provide an explanation for the  
578 off-polarity S cone surround that we observed in 57% of cells, nor why we did not observe  
579 blue-on cells with ML cone, but not S cone, surrounds. It is possible that the S cone surround  
580 is mediated by inhibitory amacrine cell input onto SBS ganglion cells (Ghosh & Grünert,  
581 1999); however, if this were indeed the case, why have inhibitory S cone surround  
582 responses not been reported in SBS ganglion cells? An alternative possibility is that the S  
583 cone off-polarity surround is a consequence of lateral inhibition in the LGN.

584 In summary, the image of blue-on and blue-off cells which emerges from these data is  
585 largely compatible with the blue-yellow color opponent channel originally hypothesized by  
586 Hering (Hering, 1878; Jameson & Hurvich, 1955). The (lilac-lime) axis of maximum sensitivity  
587 for blue-on cells is not exactly aligned with the perceptual blue-yellow axis proposed by  
588 Hering. But other properties of blue-on cells conform nicely to requirement of a chromatic  
589 opponent channel. Blue-on cells give responses of opposite polarity to short-wave and  
590 medium-wave regions of the spectrum. These responses show mutual antagonism, yielding  
591 weak responses to achromatic (S + ML) stimuli and strong responses to mixed chromatic  
592 (S–ML) stimuli. Further, the spatial properties of the antagonistic S-on and ML-off subunits  
593 are well-matched, yielding vigorous low-pass responses to chromatic contrast. Spatial  
594 centre-surround structure in blue-on receptive fields, where detectable, was weak and  
595 variable. One interpretation of this variability is that central mechanisms may not prioritize  
596 spatial contrast enhancement through blue-on cells. In this case the question whether  
597 blue-on cells are strictly Type II or not becomes largely academic. As a population these cells  
598 are clearly specified to favour chromatic over spatial contrast.

599

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605

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747

748 **Figure Legends**

749 **Figure 1**

750 Prospective models for cone opponency. A: nonlinear inputs, linearly separable output  
751 model corresponding to Eq. 5 B: linear input, nonlinear output model corresponding to Eq. 6

752 **Figure 2**

753 Cell Type classification. Example response raster plots and PSTHs showing responses to brief  
754 (0.2s) increments and decrements of ML- and S cone contrast. Representative blue-on  
755 koniocellular (A), parvocellular (B), and magnocellular (C) cells are shown. D: Response of  
756 cell (A) to S- and ML cone-isolating drifting gratings (1 cyc/deg, 5 cyc/s, 4° aperture) as a  
757 function of cone contrast. Error bars, in some cases smaller than the data point, are  $\pm$  std.  
758 error. Arrowhead shows the contrast used for the data shown in (E). E: Response of cell (A)  
759 to S- and ML cone-isolating drifting gratings (50% contrast, 5 cyc/s) as a function of grating  
760 spatial frequency. F, G: Responses of cell (B) to achromatic flashing dot and drifting gratings  
761 (7.1 cyc/s, 2° aperture (F) and 8° aperture (G)). H, I: Response of cell (C) to achromatic  
762 flashing dot and drifting gratings (5 cyc/s, 1° aperture).

763

764 **Figure 3**

765 Typical contrast responses for a blue-on and blue-off cell. A: Raster plots for 2 s  
766 presentations of 5 Hz S cone-isolating, ML cone-isolating, achromatic S+ML, and mixed S-ML  
767 2° flashing dot, organized by stimulus contrast, for a typical blue-on cell. B: Mean firing rate  
768 and first 5 Fourier components of the response at maximum contrast. The zeroth and first  
769 harmonic contain the majority of the response power. C: First-harmonic contrast response  
770 curves for the data presented in (A), showing response amplitude. Error bars, typically

771 smaller than the data point, are  $\pm$  std. error. Smooth curves show fits of Eq. 1 to the data. D:  
772 Complex plane plot for the data presented in (A), showing the amplitude and phase of the  
773 first-harmonic response. This cell has an on-phase response to S cone stimulation and an  
774 off-phase response to ML cone stimulation. E, F, G, H: Responses of a blue-off cell to a 4Hz  
775 drifting grating (0.1 cyc/deg, 8° aperture) of the above chromaticities. This cell has an  
776 off-phase response to S cone stimulation and an on-phase response to ML cone stimulation.

777

#### 778 **Figure 4**

779 Scatterplots and distributions of fitted Naka-Rushton equation parameters. A: Scatterplot of  
780 fitted semi-saturation constants for S- and ML cone-isolating stimuli for blue-on and blue-off  
781 cells. Lines of unity shown in grey. Values over 100% indicate non-saturating responses. B:  
782 Distributions of fitted semi-saturation constants for ML cone-isolating stimuli for blue-on, P,  
783 and M cells. Hatching indicates truncation of plotted distributions. C: Scatterplot of contrast  
784 gains for S- and ML cone-isolating stimuli for blue-on and blue-off cells. D: Distributions of  
785 contrast gains for ML cone-isolating stimuli for blue-on, P, and M cells. E: Scatterplot of  
786 fitted exponents for S- and ML cone-isolating stimuli for blue-on and blue-off cells. F:  
787 Distributions of fitted exponents for ML cone-isolating stimuli for blue-on, P, and M cells.

#### 788 **Figure 5**

789 Illustrative example of difference in predictions made by Eq. 5 and Eq. 6 for a selected  
790 sample cell. A: First-harmonic S- and ML cone contrast response functions. Error bars, in  
791 most cases smaller than the data point, are  $\pm$  std. error. Smooth curves show fits of Eq. 6 to  
792 the data. Open circles are S cone responses; filled circles are ML cone responses; black  
793 diamonds are achromatic responses. B: Measured and predicted first-harmonic response to  
794 achromatic stimulation. Dashed curve plots the achromatic response predicted by Eq. 5,

795 solid curve plots the achromatic response predicted by Eq. 6. C: Complex plane plot for the  
796 data presented in (A) and (B), showing the amplitude and phase of the first-harmonic  
797 response.

798 **Figure 6**

799 Predicted vs. measured response amplitudes for achromatic S+ML and mixed S – ML  
800 stimulation. A,B: Scatterplot of predicted vs. measured achromatic (A) and “optimal” S – ML  
801 (B) response amplitudes across all contrasts, excluding contrasts at which either S cone or  
802 ML cone stimulation did not evoke a response > 5 imp/s. The model assuming linear inputs,  
803 corresponding to fig. 1B, is used to generate predictions for each cell. C,D: Scatterplot of  
804 predicted vs. measured achromatic (C) and “optimal” S – ML (D) response amplitudes across  
805 contrasts. The model assuming separable nonlinear inputs, corresponding to fig. 1A, is used  
806 to generate predictions for each cell.

807 **Figure 7**

808 Predicted vs. measured complex achromatic response across all blue-on cells measured at 5  
809 Hz. A-D: Population summary of aggregate F1 response in the complex plane, for S cone-  
810 isolating (B), ML cone-isolating (C), achromatic S + ML (D), and counter-phase chromatic S –  
811 ML (E) stimuli. Open squares show predictions by Eq. 6 for (D) and (E). E: aggregate S  
812 cone-isolating, ML cone-isolating, achromatic, and predicted achromatic responses for each  
813 cell, arranged from most foveal (top left) to most peripheral (bottom right) and normalized  
814 to each cell’s response (in imp/s), given in bottom right of each plot. Clockwise motion on  
815 each dial indicates increasing phase lag. Off-phase responses fall in the shaded region of  
816 each dial. Markers are as used in (B-E). Open arrowhead (lower left) indicates a cell in which  
817 phase lag dominates the predicted achromatic response. Closed arrowhead (upper right)  
818 indicates a cell in which response gain dominates the predicted achromatic response.

819 **Figure 8**

820 Examples of observed receptive field organization for blue-on cells, corresponding to the  
821 model types listed in table 1. A: Example classical “type II” spatially coextensive receptive  
822 field for a blue-on cell at 2.8° eccentricity. B: S- and ML cone contrast response functions  
823 and measured achromatic contrast responses for the cell receptive field shown in (A). Error  
824 bars, in some cases smaller than the data point, are  $\pm$  std. error. Smooth curves show fits of  
825 Eq. 7 to the data. Open circles are S cone responses; filled circles are ML cone responses.  
826 Dashed curve shows the predicted achromatic response. C: Complex plane plot for the data  
827 presented in (A) and (B), showing the amplitude and phase of the first-harmonic response.  
828 Smooth curve shows the amplitude and phase of the fitted model. D-F: Example simple  
829 opponent blue-on cell at 0.7° eccentricity. Filled red circle shows the spatial extent of the  
830 ML cone receptive field; open blue circle shows the spatial extent of the S cone receptive  
831 field. G-I: Example cell with centre-surround organization of S cone inputs at 4.8°  
832 eccentricity. Dashed blue circle shows the spatial extent of the S cone surround. J-L:  
833 Example cell with centre-surround organization of both S cone inputs and ML cone inputs at  
834 3.9° eccentricity. Filled dashed circle shows the spatial extent of the ML cone surround.  
835

836 **Figure 9**

837 Scatterplots and distributions of fitted difference-of-Gaussians equation parameters.

838 A: Scatterplot of fitted receptive field radius for S- and ML cone inputs to blue-on and  
839 blue-off cells. Cells with classical “type II” coextensive receptive fields fall on the line of

840 unity, shown in grey. B: Scatterplot of fitted receptive field volumes for S- and ML cone

841 inputs for blue-on and blue-off cells. C: Scatterplot of centre and surround radius for S cone

842 input to blue-on cells, ML cone input to blue-on cells, P on cells, and P off cells. Marker

843 legend per (F). D: Distributions of radius ratios. From left to right: ratio of S subfield to ML

844 subfield radius for blue-on cells; ratio of ML centre radius to ML surround radius for P on

845 cells, ratio of ML centre radius to ML surround radius for P off cells; ratio of S cone centre

846 radius to S cone surround radius for blue-on cells; ratio of ML centre radius to ML surround

847 radius for blue-on cells off cells. E: Scatterplot of centre and surround volume for S cone

848 input to blue-on cells, ML cone input to blue-on cells, P on cells, and P off cells.

849 F: Distributions of volume ratios, in the same format as panel D.

850



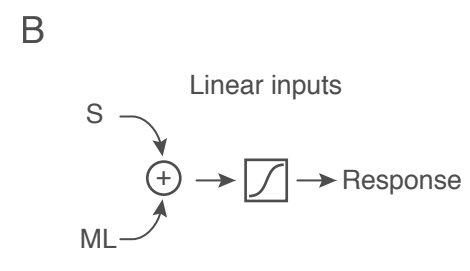
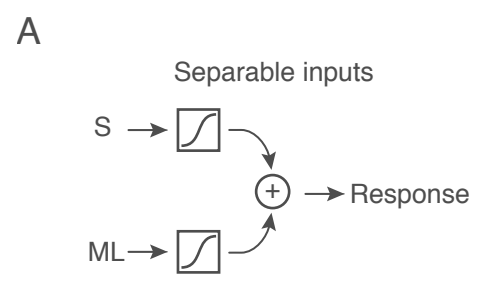


Figure 2

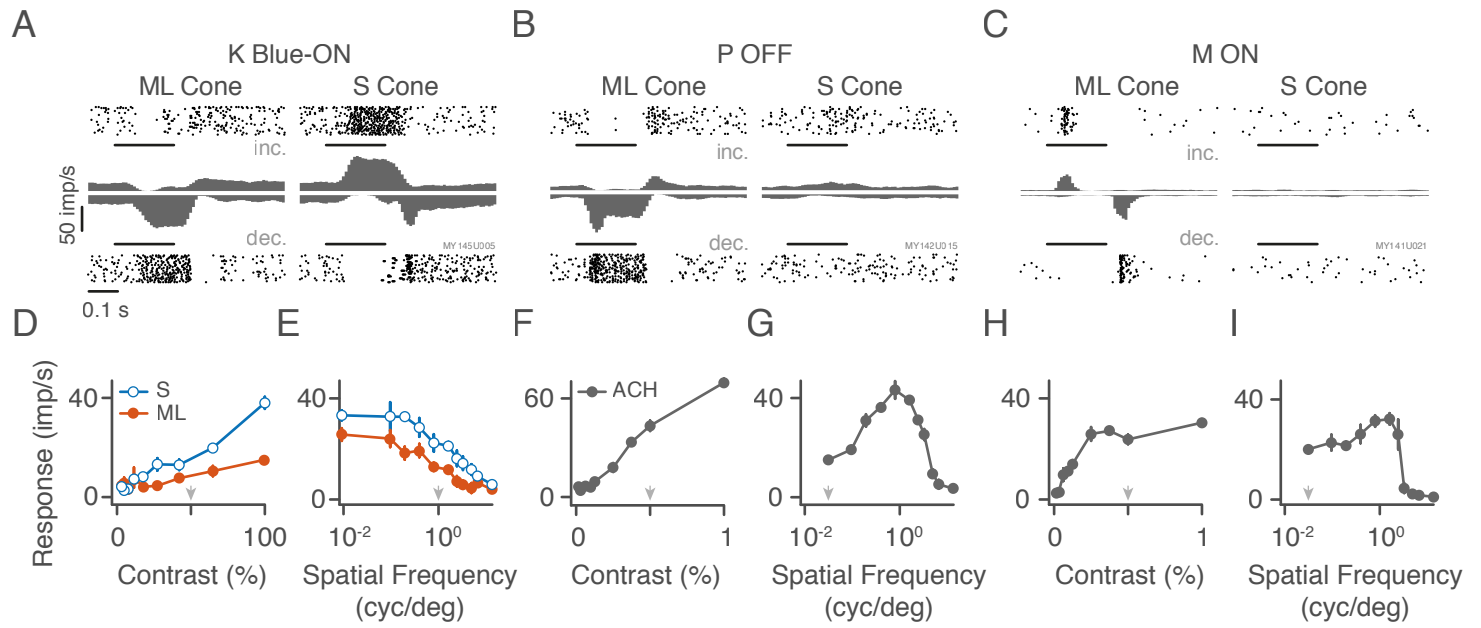


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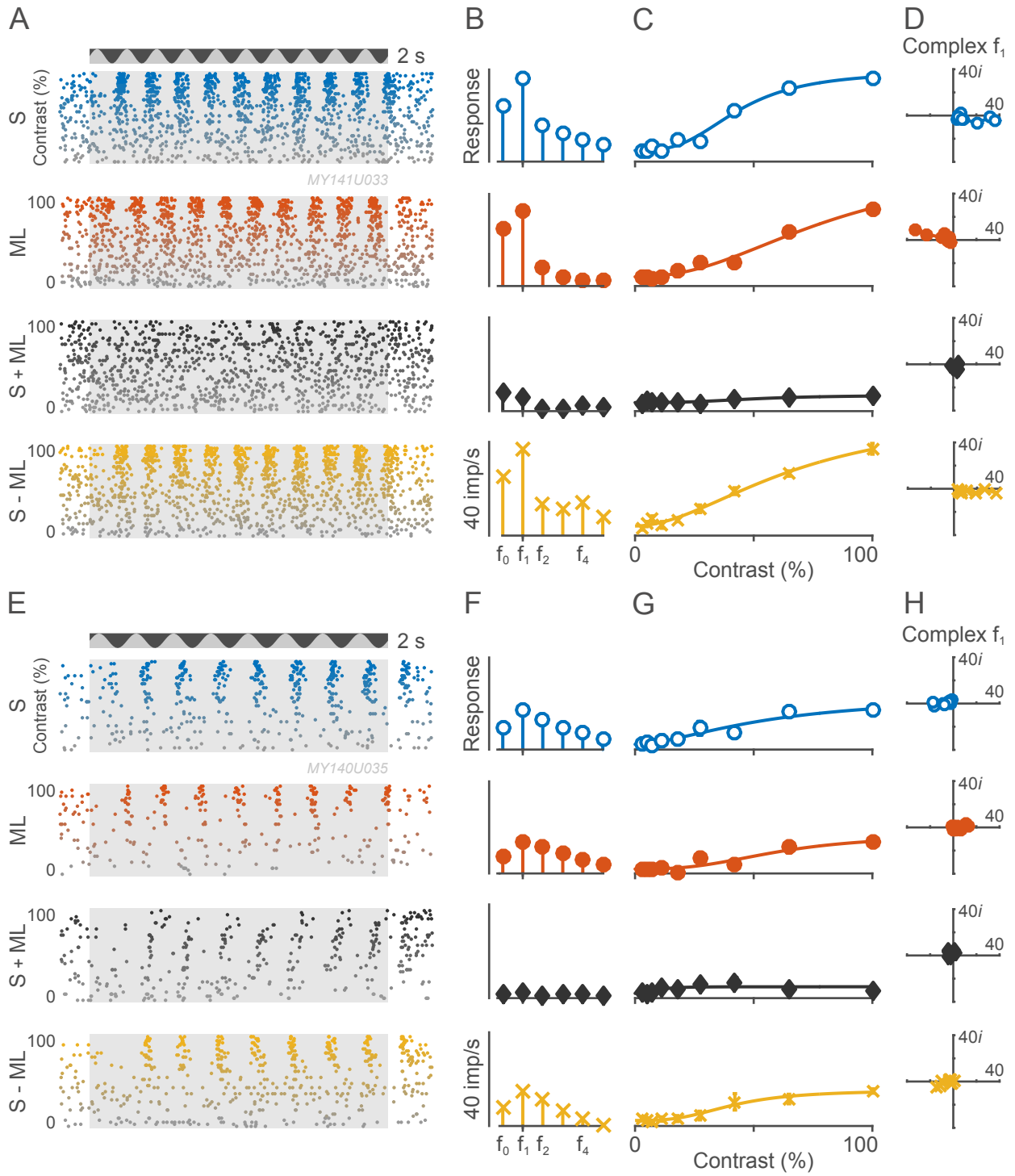


Figure 4

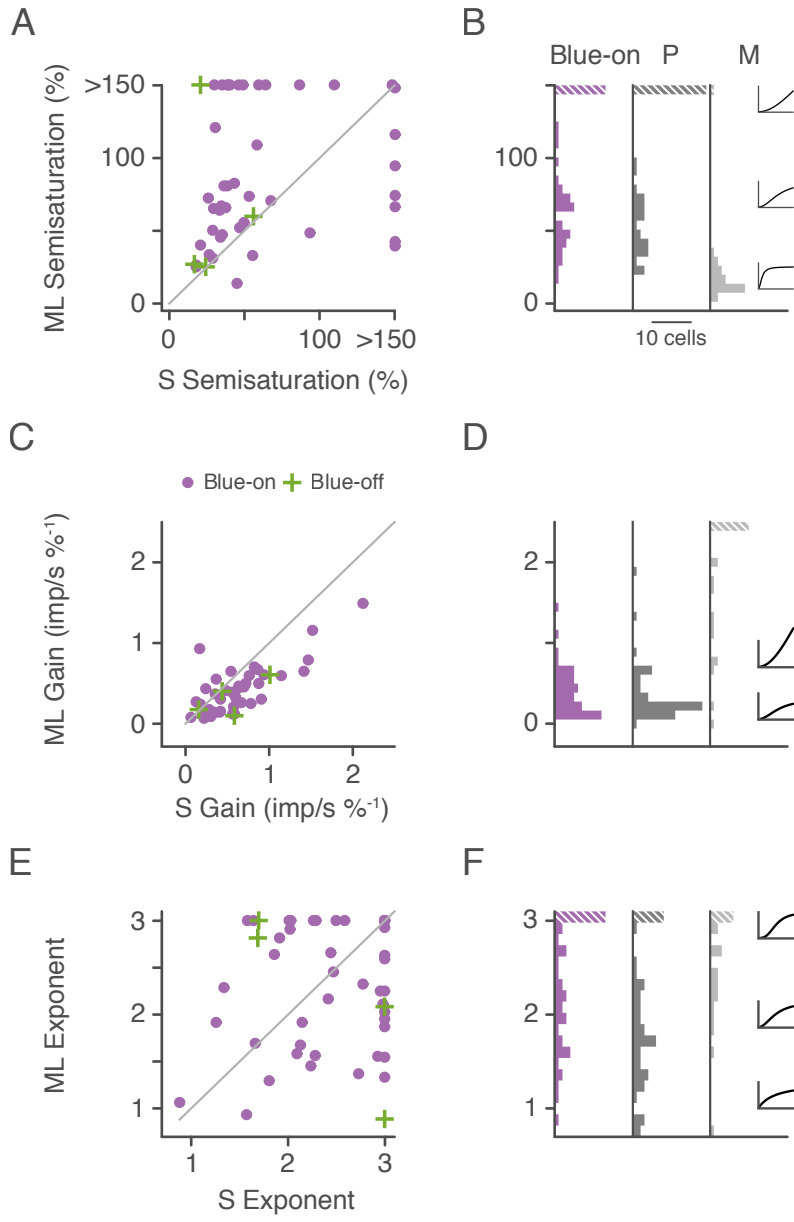


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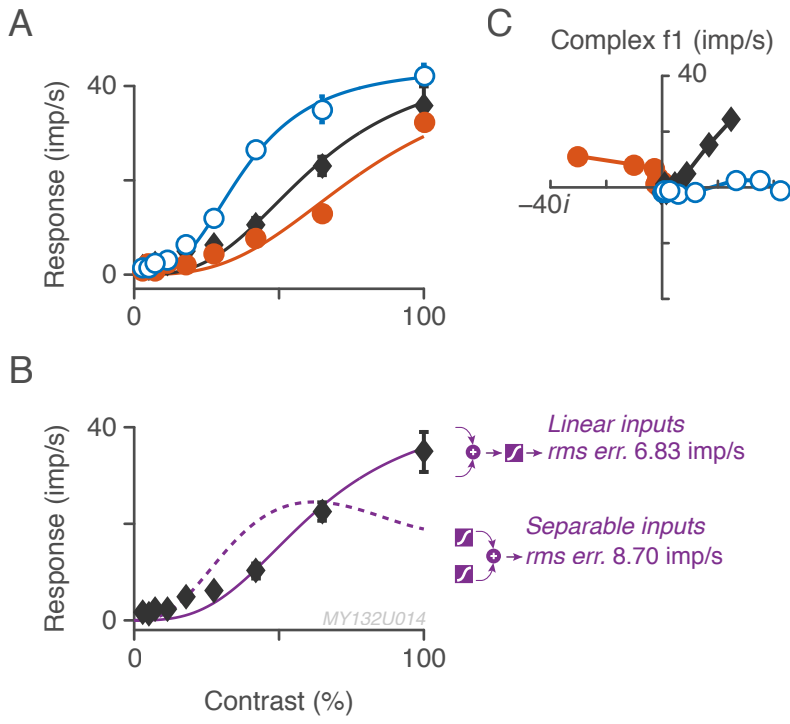


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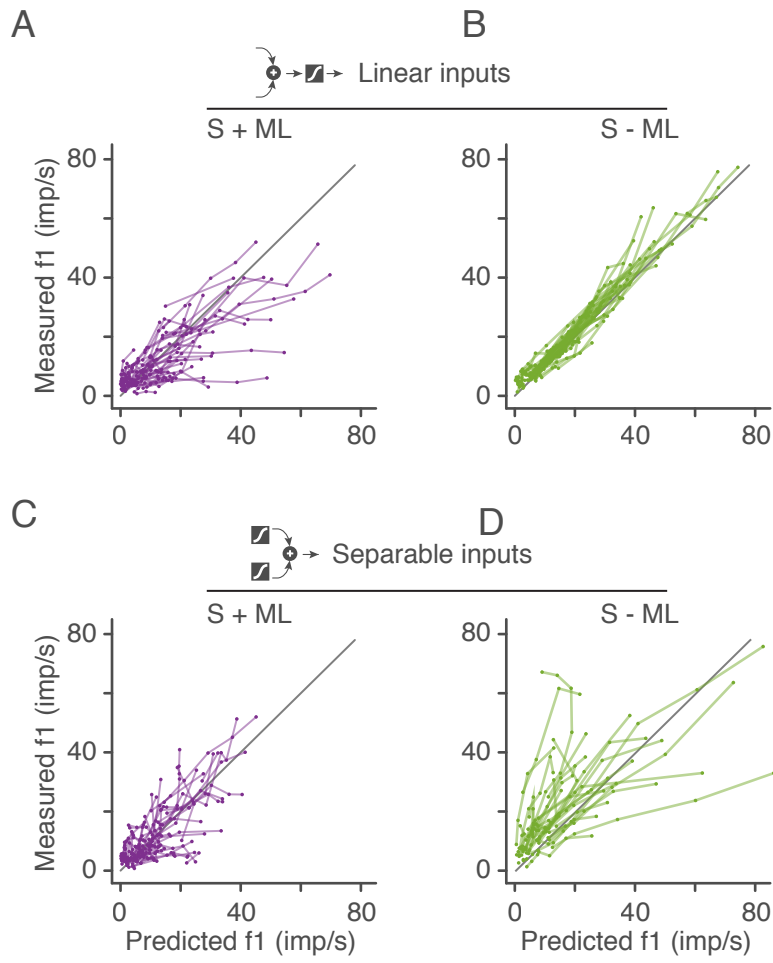
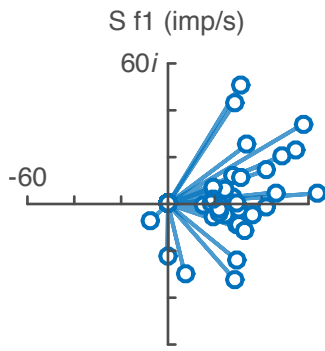
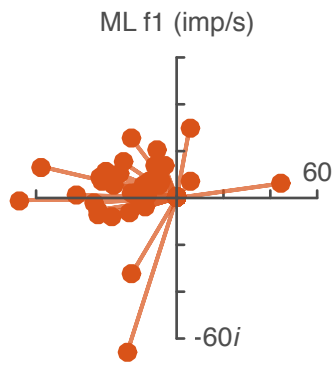


Figure 7

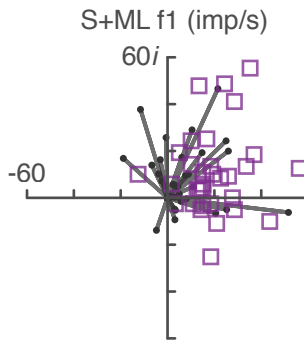
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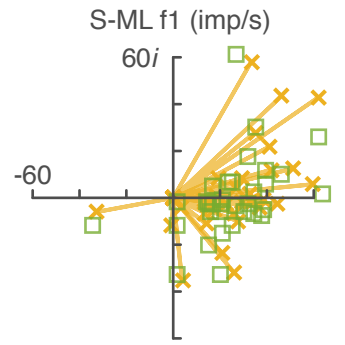
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C



D



E

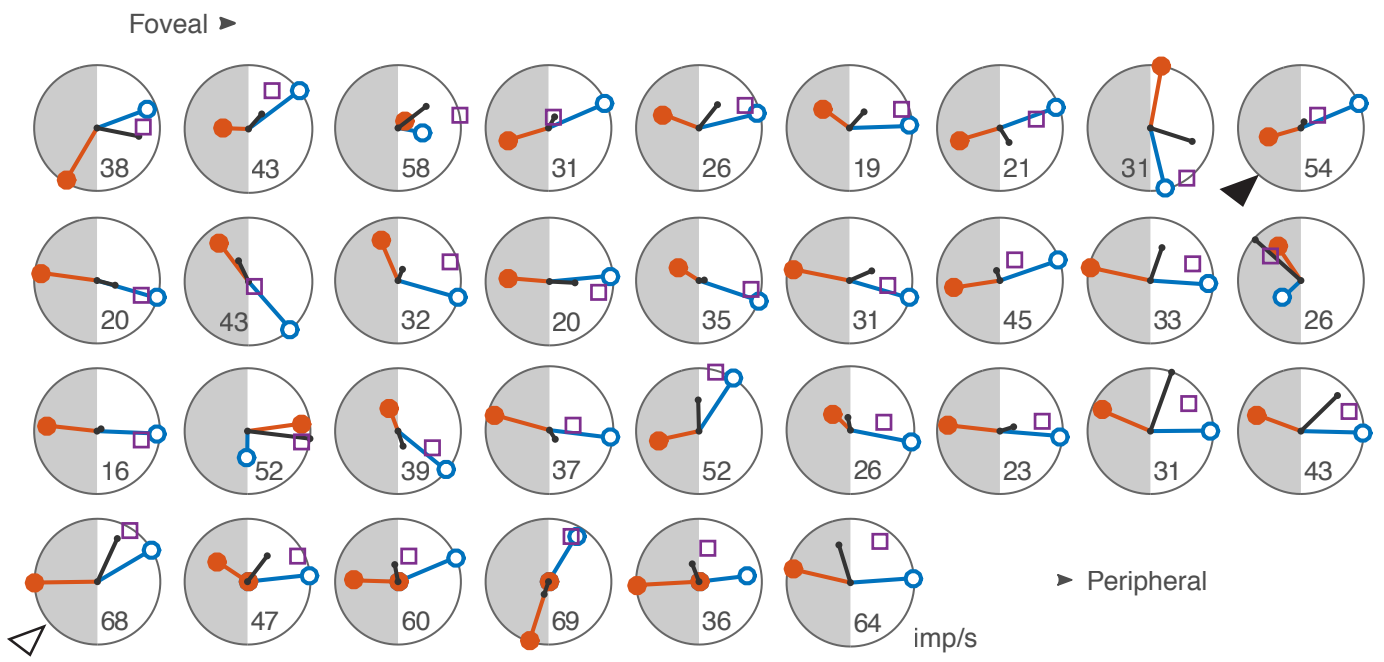


Figure 8

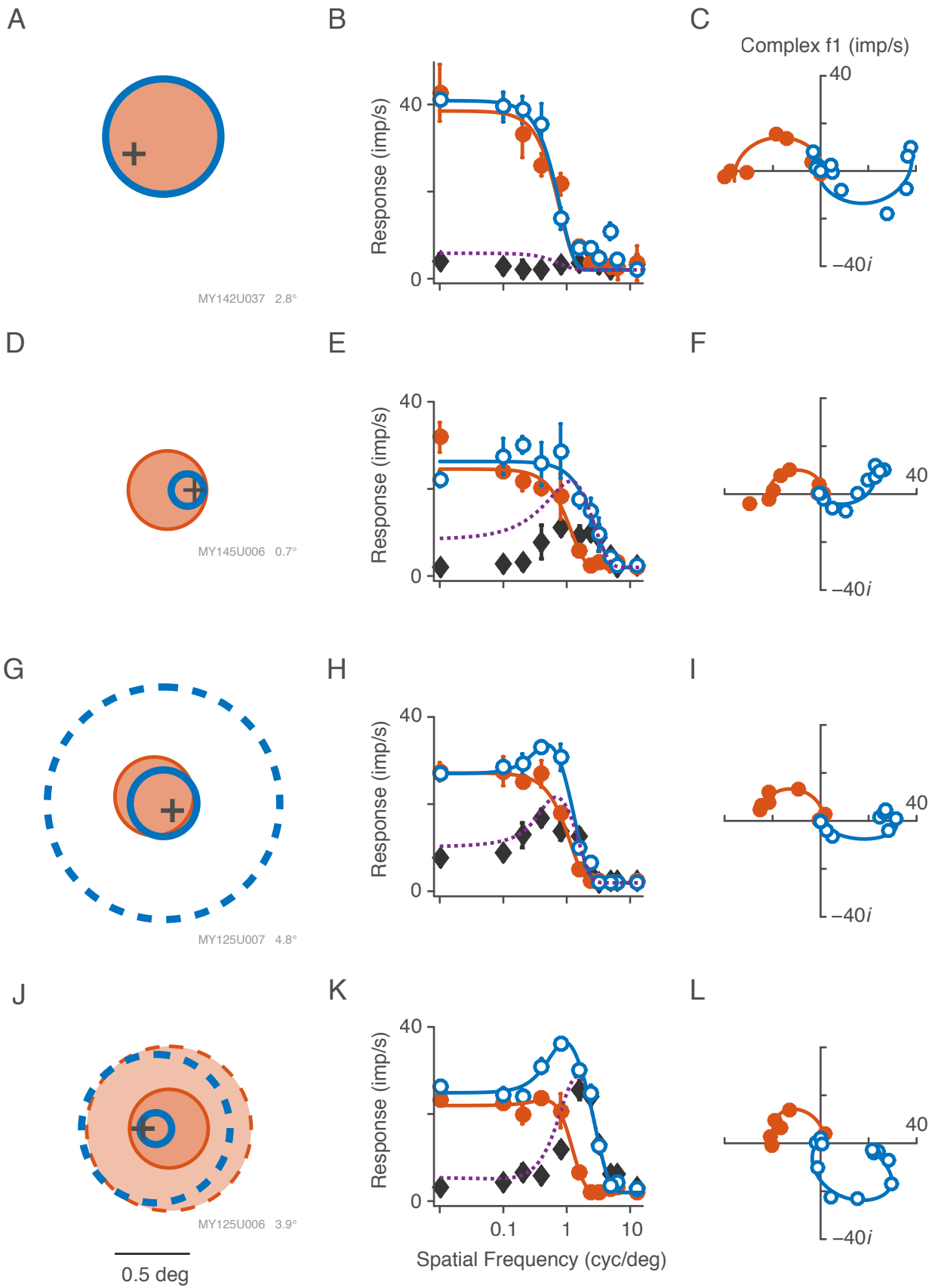




Figure 9

