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Citation for final published version:

Georgeson, Mark A. and Sengpiel, Frank 2021. Contrast adaptation and interocular transfer in cortical cells: a re-analysis & a two-stage gain-control model of binocular combination. *Vision Research* 185 , pp. 29-49. 10.1016/j.visres.2021.03.004

Publishers page: <http://dx.doi.org/10.1016/j.visres.2021.03.004>

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# Contrast adaptation and interocular transfer in cortical cells: a re-analysis & a two-stage gain-control model of binocular combination

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## Abstract

How do V1 cells respond to, adapt to, and combine signals from the two eyes? We tested a simple functional model that has monocular and binocular stages of divisive contrast gain control (CGC) that sit before, and after, binocular summation respectively. Interocular suppression (IOS) was another potential influence on contrast gain. Howarth, Vorobyov & Sengpiel (2009, *Cerebral Cortex*, 19, 1835–1843) studied contrast adaptation and interocular transfer in cat V1 cells. In our re-analysis we found that ocular dominance (OD) and contrast adaptation at a fixed test contrast were well described by a re-scaling of the unadapted orientation tuning curve – a simple change in response gain. We compared six variants of the basic model, and one model fitted the gain data notably better than the others did. When the dominant eye was tested, adaptation reduced cell response gain more when that eye was adapted than when the other eye was adapted. But when the non-dominant eye was tested, adapting either eye gave about the same reduction in overall gain, and there was an interaction between OD and adapting eye that was well described by the best-fitting model. Two key features of this model are that signals driving IOS arise 'early', before attenuation due to OD, while suppressive CGC signals are 'late' and so affected by OD. We show that *late* CGC confers a functional advantage: it yields partial compensation for OD, which should reduce ocular imbalance at the input to binocular summation, and improve the cell's sensitivity to variation in stereo disparity.

## Key words

Visual cortex; contrast adaptation; interocular transfer; ocular dominance; contrast gain control; binocular combination

## Acknowledgments

This re-analysis was supported by a grant to MAG from the Leverhulme Trust, EM-2017-097. The original physiological work was supported by grants to FS from the Medical Research Council (UK), G0000100, G0500186; Human Frontier Science Program, RG 0070/1999-B.

## 49 1. Introduction

50

### 51 1.1 Neural basis of binocular vision & stereopsis

52 Physiological studies over the last 60 years have established that single neurons in the  
53 primary visual cortex (V1) of cats and monkeys combine sensory inputs from the left and  
54 right eyes to provide the functional basis for binocular fusion and binocular depth  
55 perception (Anzai, Ohzawa, & Freeman, 1999; Barlow, Blakemore, & Pettigrew, 1967;  
56 Hubel & Wiesel, 1962, 1968; Ohzawa, DeAngelis, & Freeman, 1990, 1996; Pettigrew,  
57 Nikara, & Bishop, 1968; Poggio & Poggio, 1984). Many of these neurons are disparity-  
58 selective: their responses vary systematically with the degree of displacement (*binocular*  
59 *disparity*) between the spatial positions or spatial phases of the pair of input images  
60 (Anzai, Ohzawa, & Freeman, 1997, 1999a; Prince, Cumming, & Parker, 2002). A  
61 population of such cells with different disparity preferences encodes information about  
62 relative depth in 3-D scenes. For reviews of binocular cell responses in relation to  
63 stereopsis and models of stereopsis, see Marr & Poggio, 1979; Ohzawa, 1998; Poggio &  
64 Poggio, 1984; Read & Cumming, 2003; Roe, Parker, Born, & DeAngelis, 2007.

65

66 Thirty-five years ago, Ohzawa and Freeman argued that to understand the neural basis of  
67 binocular vision and stereopsis we first need good models of the signal processing that  
68 leads to binocular combination of signals from the two eyes (Freeman & Ohzawa, 1990;  
69 Ohzawa & Freeman, 1986). We adopt the same view here, and we explore the functional  
70 architecture laid out in Fig. 1, with the following themes.

71

### 72 1.2 Output nonlinearity

73 Freeman & Robson (1982) pioneered the study of binocular cortical cells in response to  
74 drifting sinewave gratings as a function of interocular phase difference (phase disparity).  
75 Using this approach, Ohzawa & Freeman (1986) found that a linear model, comprising  
76 linear spatial filtering by monocular receptive fields for each eye, followed by linear  
77 summation across the eyes, accounted well for the responses of binocular simple cells, but  
78 only if a threshold nonlinearity was applied to the signal after binocular summation  
79 (Ohzawa & Freeman, 1986; Smith, Chino, Ni, & Cheng, 1997). Later studies of  
80 orientation and direction selectivity of cortical cells led to some consensus that the form  
81 of this nonlinearity was a smooth expansive function (such as a power function) rather  
82 than half-wave rectification (e.g. Anzai, Ohzawa, & Freeman, 1999; Gardner, Anzai,  
83 Ohzawa, & Freeman, 1999). Miller & Troyer (2002) resolved the issue by showing that if  
84 the membrane potential is noisy then, in the conversion to spike rate, thresholded half-  
85 wave rectification is effectively smoothed to a power function. Such a nonlinearity  
86 confers contrast-invariant tuning, and sharpens the selectivity of the neuron for its  
87 preferred stimulus, and is a standard feature of functional models of V1 cell responses  
88 (Albrecht & Geisler, 1991; Heeger, 1992).

89

### 90 1.3 Monocular & Binocular gain controls

91 V1 cell responses to gratings are often strongly selective for interocular phase disparity,  
92 showing a peak response at a particular disparity  $\phi$ , and a minimum response at a phase  
93 disparity of  $\phi \pm 180$  deg. This is expected from a linear system that sums signals from each  
94 eye. Surprisingly, however, substantial modulation by disparity has been observed in cat  
95 & monkey cortical cells even when the contrasts are grossly mis-matched between the  
96 eyes (Ohzawa & Freeman 1986; Freeman Ohzawa 1990; Smith et al, 1997a,1997b). For  
97 the cells analysed in detail by Truchard *et al* (2000), responses were strongly modulated  
98 by disparity even when the contrast in one eye was as much as 10 or 20 times lower than  
99 in the other eye. This is indeed surprising, because the result of summing a weak  
100 sinusoidal signal with a strong one is dominated by the strong signal. The phase of the  
101 weak signal can only weakly modulate the summed response. However, monocular

102 contrast gain control changes all that. Increasing contrast is counteracted by gain  
103 reduction. Increasing contrast for one eye drives down the gain for that eye, and so the  
104 response amplitude increases rather little. Hence response amplitudes for weak and  
105 strong signals in the two eyes are more similar than the difference in their contrasts would  
106 suggest. This similarity allows the 'weak' input to modulate the binocular sum more  
107 effectively than in a linear system. Using this principle, Truchard *et al* (2000) calculated  
108 the monocular and binocular gain values ( $M$ ,  $B$  in Fig. 1) that would be needed to account  
109 for their results on a sample of cat simple cells. They found that monocular gain greatly  
110 decreased as contrast increased, while binocular gain was little affected by contrast. They  
111 concluded that contrast gain control operates mainly in the monocular pathways leading  
112 to binocular combination.

113

#### 114 *1.4 Ocular dominance*

115 Binocular V1 cells are often driven more strongly by one eye than the other (*ocular*  
116 *dominance, OD*). Surprisingly perhaps, even cells that respond exclusively to one eye  
117 when tested monocularly often exhibit strong binocular interactions and disparity  
118 sensitivity when both eyes are tested together (Ohzawa & Freeman, 1986; Smith, Chino,  
119 Ni, Ridder, & Crawford, 1997). This implies that there is a weaker but still functional  
120 input from the non-dominant eye (NE) that is 'hidden' by a response threshold or  
121 threshold-like output nonlinearity, as above. In binocular testing this input can modulate  
122 the response to the other eye, even though the NE stimulus alone may drive the spike  
123 response only weakly or not at all. With monocular testing, the output nonlinearity thus  
124 exaggerates the apparent mismatch in strength of left and right eye connections to the  
125 binocular cell (Priebe, 2008).

126

#### 127 *1.5 Contrast adaptation*

128 Exposure to the contrast of a grating reduces behavioural contrast sensitivity, and does so  
129 selectively when the adapting and test gratings are similar in orientation and spatial  
130 frequency (Blakemore & Campbell, 1969; Georgeson & Harris, 1984). In psychophysics,  
131 contrast adaptation is often taken to involve a slow build-up and slow recovery of the  
132 adaptation effect, but the dynamics are likely to be more complex than that, since even  
133 brief exposures (e.g. 1 sec) can produce brief but substantial sensitivity loss, and the time  
134 taken to recover from adaptation depends much more on the time spent adapting than it  
135 does on the contrast of the adapter (Greenlee, Georgeson, Magnussen, & Harris, 1991).

136

137 Adaptation and contrast gain control are closely linked. In psychophysics, the increase of  
138 sensitivity loss with adapting contrast, and its dependence on relative spatial frequency,  
139 were strikingly similar after (i) adapting for two minutes, or (ii) 'adapting' for 25 msec  
140 provided the test was flashed within 50 msec (Georgeson & Georgeson, 1987). This  
141 points to some shared underlying mechanism in contrast adaptation and 'instantaneous'  
142 gain control.

143

144 At the V1 cell level, exposure to contrast causes both contrast gain and response gain to  
145 decrease (Albrecht, Farrar, & Hamilton, 1984; Bonds, 1991; Ohzawa, Sclar, & Freeman,  
146 1985; Sclar, Lennie, & Depriest, 1989). More recent studies have revealed greater  
147 complexity of the suppression mechanisms involved over different spatial and temporal  
148 scales (Patterson, Wissig, & Kohn, 2013) and orientations (Westrick, Heeger, & Landy,  
149 2016), but a divisive normalization (gain control) framework still seems appropriate (for  
150 review see Solomon & Kohn, 2014), perhaps especially for the simple adaptation protocol  
151 used in our target study (Howarth, Vorobyov, & Sengpiel, 2009).

152

#### 153 *1.6 Plan for this paper*

154 We are interested in the binocular properties of V1 cells in response to luminance  
155 contrast, and in their adaptation to contrast, with the aim of formulating a relatively

156 simple, functional model that helps us to understand better the basis for binocular  
157 processing and combination of signals at the cortical cell level. To do this we:  
158 (i) re-analyzed data from Howarth, Vorobyov, & Sengpiel (2009), which appears to be the  
159 only published quantitative study to date of contrast adaptation and its interocular transfer  
160 in V1 cells;  
161 (ii) formulated a general binocular cell model (Fig. 1) along with several specific versions  
162 of it (Fig. 2) whose components are: monocular and binocular levels of gain control,  
163 ocular dominance, interocular suppression, binocular summation, and an expansive output  
164 nonlinearity;  
165 (iii) fitted these models to the data of Howarth et al (2009), constrained in part by other  
166 relevant V1 results; and  
167 (iv) compared the six model variants (Fig. 2) to infer the best-supported model, and so  
168 obtain a clearer view of how the gain control works, how it interacts with OD, and  
169 whether interocular suppression plays a role in contrast adaptation at the single-cell level.

170

171

## 172 2. Methods

173

### 174 2.1 Data

175 A dataset of micro-electrode responses recorded extracellularly from 54 cells in the cat  
176 primary visual cortex was available from the study of Howarth, Vorobyov, & Sengpiel  
177 (2009). Recordings were made from neurons throughout the depth of V1 (area 17), in the  
178 region representing the central visual field. To sample a wide range of ocular dominances  
179 the cortical penetrations targeted left- and right-eye columns as well as binocular zones,  
180 as determined by optical imaging of intrinsic signals. Each cell was tested with sinusoidal  
181 gratings of fixed contrast (typically 0.5 c/deg, contrast 0.5 or 1.0), filling a 20 by 20 deg  
182 display area, drifting at 2 Hz, at each of 8 orientation/directions ( $-180^0$  to  $+135^0$  in  $45^0$  deg  
183 steps). Cell response to a given test stimulus was defined as the mean firing rate  
184 (spikes/sec) during the 1.5s test periods minus the 'spontaneous rate' (mean rate during  
185 1.5s blank, zero-contrast periods). These test responses were collected after a period of  
186 adaptation to a grating of optimal spatial frequency (contrast 0.25 or 0.5) drifting in the  
187 preferred orientation and direction for the cell, and after control periods where the  
188 adapting contrast was zero. The gratings were always monocular, and all 6 possible  
189 combinations (test L or R eye, adapt L or R or neither eye) were assessed. For further  
190 details, see Howarth et al., 2009.

191

192 For the present study, cells were classified according to their ocular dominance, defined  
193 by an ocular dominance index (ODI) of the responsiveness of the cell to monocular  
194 stimuli in the less sensitive eye, relative to the more sensitive one. Specifically, the ODI  
195 was the ratio of total response to drifting gratings (summed over the 8 stimulus directions)  
196 for the non-dominant eye (NE) divided by that for the dominant eye (DE), without prior  
197 adaptation to contrast. Thus:

$$202 \quad ODI = \frac{\sum_{i=1}^8 R_{NE}(\theta_i)}{\sum_{i=1}^8 R_{DE}(\theta_i)}.$$

198 With this definition ODI ranges from 0 (no response to NE) to 1 (same response to NE  
199 and DE). For our present analyses, cells were ordered by ODI value and then divided into  
200  $N$  groups ( $N=3$  to 10, or higher), ranging from groups whose *monocular* responses were  
201 mainly confined to one eye to those driven substantially by either eye.

202

203

204

205

206

\*\*\* Fig. 1 about here \*\*\*

207

## 208 *2.2 Modelling: design and implementation*

209 An outline of the model we applied to these data is shown in Figure 1. In brief, monocular  
210 gains ( $M$ ) are reduced both by the current test contrast in a given eye and by the adapting  
211 grating (if any). Whether the current or prior contrast in one eye can reduce the  $M$  gain for  
212 the other eye depends on whether a route for interocular suppression (IOS) is present, or  
213 not. We assume that, after binocular summation, binocular gain ( $B$ ) can be reduced by  
214 adapting through either eye, and we incorporate the important finding that, unlike  $M$ ,  
215 binocular gain  $B$  is altered little or not at all by the current test stimulus (Truchard *et al*,  
216 2000).

217

218 Many cortical cells respond more vigorously to one eye than the other. Such ocular  
219 dominance (OD) has been studied intensively and much is known about the anatomy and  
220 physiology of OD in V1, its organization into cortical OD columns, and the way their  
221 development in the young animal is influenced by normal or abnormal early visual  
222 experience. But in the model scheme of Fig. 1, we do not know *a priori* where the OD  
223 asymmetry should be placed nor how it might influence the setting of gains for each eye.  
224 To address these questions we developed several more specific model structures based on  
225 Fig. 1, in which the contrast-response from one eye (NE) is attenuated relative to the  
226 dominant eye (for example,  $d_R < d_L$  in Fig. 1). In these candidate models, the monocular  
227 contrast gain control (CGC) is either driven 'early' by the unattenuated contrast (Fig. 2C),  
228 or 'late' by the contrast-response after its attenuation by OD (Fig. 2F). Three consequences  
229 of the OD attenuation, as we shall see, are (i) that the model cell responds less to  
230 stimulation through one eye (NE) than the other, (ii) that it adapts less (at stage  $B$ ) on  
231 exposure to contrast in that eye than the other, and (iii) with early CGC the adaptation of  
232 the monocular NE gain stage  $M$  is the *same* as for the dominant eye, unaffected by the  
233 degree of dominance  $d$ , while for late CGC the adaptation at level  $M$  is less when OD is  
234 more unbalanced. The ocular dominance factors  $d_L$ ,  $d_R$  and the orientation tuning  $S$  are  
235 assumed to be unaffected by prior adaptation, and we fixed test contrast at 1.0 and  
236 adapting contrast at 0.5.

237

\*\*\* Fig. 2 about here \*\*\*

238

239  
240 After the adjustments of monocular response amplitude, signals from the two eyes are  
241 summed linearly (Ohzawa & Freeman, 1986). This binocular response is subject to the  
242 second gain adjustment  $B$  and then in a final step the output values are raised to power  $p$   
243 in the conversion to firing rate  $R$ . Thus the model adopts the form of the 'contrast-  
244 gain/exponent model' (Albrecht & Geisler, 1991; Albrecht, Geisler, Frazor, & Crane,  
245 2002), which in turn is similar to the 'half-squaring normalization model' (Heeger, 1992).  
246 Our model was elaborated to incorporate binocular summation and two stages of gain  
247 control (Truchard, Ohzawa, & Freeman, 2000), as well as the ocular dominance factor.  
248 Next we describe how this model structure was implemented and evaluated quantitatively.

249

## 250 *2.3 Stage 1, nonlinear monocular responses and linear binocular combination*

251 Fig. 2 sketches six ways in which the monocular gain controls might be organized,  
252 incorporating ipsiocular contrast gain control (CGC) and (optionally) interocular  
253 suppression (IOS). Both of these gain-control mechanisms can be driven by the current  
254 (test) contrast and the prior (adapting) contrast and these driving signals might arise *early*  
255 (before attenuation due to OD), or *late* (after OD attenuation).

256

257 For simplicity of description, but with no loss of generality, we assume throughout that  
258 for each cell the left eye is the more responsive one (dominant eye, DE) while the right  
259 eye is the less responsive (non-dominant eye, NE). Initial responses  $r_L$ ,  $r_R$  in the  
260 monocular pathways leading to a given cortical cell, evoked by stimuli in the left and

261 right eyes (subscripted  $L, R$ ), were defined by a modified Naka-Rushton function that  
 262 contains extra terms representing the effect of prior adaptation to contrast in the same eye,  
 263 and possible interocular suppression. We first outline the simplest model structure (Fig.  
 264 2C, model 3) here:

$$265 \quad r_L = \frac{(1 + s) \cdot S(\theta) \cdot d_L \cdot c_L}{s + c_L + m \cdot a_L} \quad (1.3)$$

266

$$267 \quad r_R = \frac{(1 + s) \cdot S(\theta) \cdot d_R \cdot c_R}{s + c_R + m \cdot a_R} \quad (2.3)$$

268 where  $c$  is test contrast,  $a$  is adapting contrast,  $s$  is the semi-saturation constant,  $m$  is the  
 269 strength of contrast adaptation at this monocular level,  $d_L$  and  $d_R$  are attenuations of  
 270 contrast due to OD ( $d_L = 1, d_R \leq 1$ ),  $\theta$  is the test orientation/direction relative to the  
 271 preferred orientation of the cell, and  $S(\theta)$  is the tuning function that attenuates these  
 272 monocular responses by a fixed factor that depends only on test direction  $\theta$ . The scale  
 273 factor  $(1+s)$  is a convenience, ensuring that the output is 1 for an input contrast of 1 at the  
 274 preferred orientation with no adaptation. Orientation tuning is omitted from the  
 275 denominator of eqns. 1.x, 2.x (where  $x$  is model number) because there is good evidence  
 276 that the divisive gain control effect in V1 depends on stimulus contrast but shows little or  
 277 no selectivity for orientation (Bonds, 1989), and that this is so because the divisive  
 278 influence arises from a 'pool' of cells with many different preferred orientations and  
 279 spatial frequencies (Carandini & Heeger, 2012; Heeger, 1992). The outcome is a  
 280 'contrast-set gain control' (Albrecht & Geisler, 1991; Geisler & Albrecht, 1992) rather  
 281 than one in which gain depends directly on a cell's own response to a stimulus.  
 282

283 For all models,  $d_R$  attenuates the signal contrast (numerator in eqn. 2.3) but for early CGC  
 284 it plays no part in setting the monocular gain  $M$  (determined by the denominator in eqn.  
 285 1.3, 2.3). With late CGC, however, the divisive term  $c_R$  is replaced by  $c_R \cdot d_R$ , such that  
 286 lower values of  $d_R$  reduce the divisive influence, and gain  $M_R$  increases for cells with  
 287 greater ocular imbalance. This is analogous to the rise in monocular gain observed when  
 288 contrast was decreased in one eye but not the other (Truchard et al, 2000; our Fig. 3).  
 289 Note also that the empirical index for ocular dominance (ODI) is not the same as the  
 290 model's dominance parameter  $d$ , though they are closely related. ODI is based on  
 291 observed relative firing rates at the final output, while  $d$  is the hypothetical attenuation of  
 292 one monocular input signal relative to the other ( $d = d_R/d_L$ ) at an early stage (Fig. 1).  
 293

294 Evidence for interocular suppression (IOS) of responses  $r_L, r_R$  in the monocular pathways  
 295 has frequently guided the design of binocular vision models (Ding & Sperling, 2006;  
 296 Meese, Georgeson, & Baker, 2006; Moradi & Heeger, 2009; Wilson, 2017 for  
 297 psychophysics; Sengpiel, Baddeley, Freeman, Harrad, & Blakemore, 1998; Sengpiel,  
 298 Blakemore, & Harrad, 1995; Zhang et al., 2005 for physiology). IOS might seem  
 299 irrelevant for the present dataset because the two eyes were never stimulated at the same  
 300 time. But, potentially, adapting through one eye then testing the other might produce an  
 301 adaptation effect via IOS, and so we incorporated IOS into the equations for models  
 302 1,2,4,5. See Appendix for a complete list of equations for all six models.  
 303

304 The six models (Fig. 2) differ only in the monocular gain control terms (denominator of  
 305 eqns.1.x, 2.x) with no change in the numerator terms. The remaining definitions (eqns.  
 306 3,4,5 below) were common to all six models.  
 307

308 Monocular responses were a nonlinear function of contrast, but were summed linearly  
 309 across the eyes to give the output  $r_B$  of stage 1:

$$310 \quad r_B = r_L + r_R \quad (3)$$

311

312

#### 313 2.4 Stage 2, binocular adaptation and gain control

314 The final response  $R(\theta)$  of a model cell as a function of test orientation is given in two  
315 parts by (i) a response function that incorporates divisive gain reduction by responses to  
316 both the current stimulus and to the previous adapting stimulus ( $r_B$ ):

$$317 \quad R_B(\theta) = \frac{(1+z) \cdot r_B(\theta)}{1+z \cdot e_B + b \cdot r'_B} \quad (4)$$

318 followed by (ii) an output nonlinearity and amplitude scaling:

$$319 \quad R(\theta) = R_{max} \cdot (R_B)^p \quad (5)$$

320 where  $p$ ,  $z$  and  $R_{max}$  are constants,  $b$  represents the strength of adaptation at the  
321 binocular level, and  $e_B$  represents the drive (if any) from the current stimulus into this  
322 second-stage gain control. See Appendix for further rationale and justification of these  
323 model steps.

324

#### 325 2.5 The whole model

326 The model can be summarized by condensing eqns. 1-5 into a single equation (Fig. 1):

327

$$328 \quad R = R_{max} \cdot \{S(\theta) \cdot (c_L d_L M_L + c_R d_R M_R) \cdot B\}^p \quad (6)$$

329

330 where (taking the right eye to be NE) we have  $d_L=1$ ,  $d_R \leq 1$ . Eqns. 1.x, 2.x operate as  
331 divisive, contrast-set gain controls, because the monocular gains ( $M_L, M_R$ , eqns. 7.x, 8.x,  
332 Appendix) include a divisive term representing the test contrast ( $c_L$  or  $c_R$ ). Likewise, the  
333 binocular gain  $B$  (eqn. 9, Appendix) includes the  $r_B$  term that represents short-term gain  
334 control at the binocular stage. Gain  $B$  is adapted (reduced) by previous binocular  
335 responses ( $r'_B$ ) and (in principle) by the current response ( $r_B$ ), if  $z > 0$ . We fixed the values  
336 for  $s$  and  $z$ , guided by fitting eqns. 8.x and 9 to the gain values reported by Truchard *et al*  
337 (2000) for cat cortical cells ( $s=0.06$ ,  $z=0$ ; see legend to Fig. 3 for more detail). In  
338 particular, setting  $z=0$  prevented any control of  $B$  gain by current contrast, in line with the  
339 Truchard *et al* findings.

340

341 \*\*\* Fig. 3 about here \*\*\*

#### 342 2.6 Fitting the model

343 The fixed values adopted for  $s$  and  $z$  gave a typical, smoothly saturating contrast response  
344 (Albrecht and Geisler, 1991; Heeger, 1992; Carandini et al., 1997). And when  $z$  was a  
345 fitted parameter its value converged to zero, confirming that our *a priori* choice of  $z=0$   
346 was supported for this dataset.  $R_{max}$  was the observed peak firing rate for each cell group,  
347 but it played no part in the model-fitting of relative gain values. That left five parameters  
348 ( $m$ ,  $b$ ,  $p$ ,  $w$ ,  $d$ ) to be assigned, but  $d$  was determined directly from the *unadapted* data  
349 alone without compromising the purpose of the model-fitting. See Appendix, eqns. 12, 13  
350 for derivation of  $d$ .

351

352 We fitted model response gains to observed response gains (defined below) for the  $N$  OD  
353 cell groups simultaneously, using the method of least squares, with parameter adjustment  
354 implemented by the *Simplex* algorithm (*fminsearch* in *Matlab*). For each fitting, ten or  
355 more runs were done with jittered starting values of parameters to ensure that minimum-  
356 error solutions were not stuck in local minima. To keep the model as simple as possible  
357 (Occam's razor), and after some initial exploration, we assumed that the parameters  
358  $m, b, p, w$  did not vary across the  $N$  OD groups. Thus there were 2-4 free parameters in the  
359 fitting, but  $4N$  data points to be fitted (i.e. the  $N$  cell groups times the 4 adapting  
360 conditions whose data had not already been used in the determination of  $d$ ).

361

362 \*\*\* Fig. 4 about here \*\*\*

363

### 364 3. Results

365

#### 366 3.1 Overview of the cell response data

367 Figs. 4A,B give a summary of the physiological data, expressed as population means  
368 plotted against test orientation/direction. The descriptive tuning curve fitted to the  
369 unadapted DE data (Fig. 4A, black curve) also fitted closely to the unadapted NE data  
370 (Fig. 4B, black) and to the adapted responses of both eyes (coloured curves & symbols in  
371 Fig. 4A,B), subject only to a scaling-down of response magnitude that depended on which  
372 eye was adapted and which eye was tested, but was independent of test orientation. We  
373 refer to this scaling-down of the spike rates as a reduction in *response gain*, and we  
374 quantify these gains in Fig. 4C,D as the slope of the linear regression between responses  
375 in any given condition and those in the control condition (unadapted DE responses). If the  
376 tuning shape had changed markedly with adaptation, or between DE and NE, then the  
377 relations in Fig. 4C,D would not be linear, and our characterization in terms of response  
378 gain would be inappropriate. But as we shall see, this linearity held good not only for the  
379 population means but also quite well for sub-groups of cells with different degrees of OD,  
380 and this greatly simplified the analysis and modelling. Table 1 gives the gain factors  
381 (slopes in Fig. 4C,D) for the population mean data. Note that the gain factors describe  
382 relative responses only at the fixed high test contrast used in the experiment. Ocular  
383 dominance led to an average unadapted gain in the NE that was 43% of that in the DE.

384

385 **Table 1.** Response gains and firing rates for the population mean (54 cells)

386

<i>Adapt</i>	Response Gains, G		Firing rates, $R_{\max}$ , sp/s	
	Test DE	Test NE	Test DE	Test NE
<i>None</i>	1	0.430	36.1	14.9
<i>NE</i>	0.770	0.208	27.3	6.7
<i>DE</i>	0.359	0.227	12.3	8.5

387

388 In general, this population of cells showed interocular transfer (IOT) of contrast  
389 adaptation: adapting through one eye led to loss of response gain when testing the same  
390 eye or the other eye (Howarth et al., 2009). When the DE was tested, contrast adaptation  
391 in the same eye reduced gain much more than NE adaptation did (Fig.4C). But when the  
392 NE was tested, adaptation in either eye reduced gain by very similar amounts (Fig. 4D).  
393 This strong, asymmetric interaction cannot be described by any single figure-of-merit  
394 such as 'percentage interocular transfer'. Instead, our modelling aims to help us  
395 understand the basis for interocular transfer - in terms of gain changes at monocular and  
396 binocular levels of processing - and the influence that ocular dominance has on both  
397 contrast sensitivity and contrast adaptation.

398

399

\*\*\* Fig. 5 about here \*\*\*

400

#### 400 3.2 Three OD groups

401 Next we illustrate the tuning and gain plots obtained when the cells were divided into  
402 different OD groups (see Methods). Figs. 5A,B show that the same orientation/direction  
403 tuning curve (the template in Fig. A1, blue) fitted very well in all 18 cases (6 conditions x  
404 3 OD groups). The key point, though, is that the tuning shapes were sufficiently similar  
405 *within* each OD group that the responses mapped very well onto straight lines in Figs.  
406 5C,D, thus defining a response gain for each condition (Table 2) with fairly high  
407 precision. Differences in tuning shape between OD groups would not affect these  
408 estimates of gain, but in practice we saw no dramatic differences in tuning, even with 9 or  
409 10 groups.

410

411

412 **Table 2.** ODI and empirical response gains G for 3 OD groups (18 cells/gp)  
 413

	Group 1		Group 2		Group 3	
ODI	0.108		0.416		0.845	
	Response gains, G					
<i>Adapt</i>	Test DE	Test NE	Test DE	Test NE	Test DE	Test NE
<i>None</i>	1	0.095	1	0.378	1	0.787
<i>NE</i>	0.828	0.093	0.749	0.181	0.748	0.339
<i>DE</i>	0.374	0.002	0.336	0.194	0.376	0.449

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### 3.3 Comparing models with data

Having established the appropriateness of these response gain measures, we now compare model gains with data, to assess the contributions made by different processing stages and to determine whether the evidence favours any particular model. The cells were divided into  $N$  groups and for each  $N$  the six model variants (Fig. 2) were fitted by least-squares to the cell gains determined for each group as described above. Goodness-of-fit was quantified by  $R^2$ , and we used AIC analysis (Burnham & Anderson, 2002; Wagenmakers & Farrell, 2004) for model comparison, to assess the relative strength of evidence (the *Akaike weight*) favouring each model. AIC is especially useful where models are not nested (where one model is not a special case of another) and a nested F-test cannot be used. See legend to Fig. 6 for more on AIC. Table 3 summarizes this analysis for  $N = 3, 6$  and 9 OD groups.  $R^2$  values were high for all 6 models, implying that all the models capture much of the observed variation in gain. Presumably this is so because all 6 models share the same general binocular architecture (Fig. 1). But that is *not* an indication that the evidence favours all the models equally. The AIC calculations in Table 3 show that model 5 (late CGC, early IOS) with high Akaike weights was consistently favoured over the others, though there was some support also for model 4. Fig. 6A plots the Akaike weights against model number, showing the high weight for model 5, and a modest weighting for model 4, irrespective of the number of cell groups from 3 to 10 (indicated by different colours and ordered from left to right for each model number).

**Table 3. Steps in AIC analysis for model comparison & model selection**

Model	R <sup>2</sup>	K	SSerr	AICc	ΔAIC	Akaike weight
<i>N groups = 3, n data = 12</i>						
1. Early CGC, Late IOS	0.9756	5	0.0187	-57.56	14.040	0.001
2. Early CGC, Early IOS	0.9785	5	0.0165	-59.10	12.502	0.002
3. Early CGC, No IOS	0.9756	4	0.0187	-63.85	7.754	0.017
4. Late CGC, Late IOS	0.9896	5	0.0080	-67.80	3.797	0.122
5. Late CGC, Early IOS	<b>0.9924</b>	5	0.0058	-71.60	0.000	<b>0.811</b>
6. Late CGC, No IOS	0.9795	4	0.0157	-65.95	5.647	0.048
<i>N groups = 6, n data = 24</i>						
1. Early CGC, Late IOS	0.9552	5	0.0729	-125.77	12.369	0.002
2. Early CGC, Early IOS	0.9554	5	0.0726	-125.89	12.252	0.002
3. Early CGC, No IOS	0.9552	4	0.0729	-129.00	9.141	0.008
4. Late CGC, Late IOS	0.9694	5	0.0498	-134.94	3.206	0.162
5. Late CGC, Early IOS	<b>0.9732</b>	5	0.0436	-138.14	0.000	<b>0.807</b>
6. Late CGC, No IOS	0.9582	4	0.0681	-130.66	7.481	0.019
<i>N groups = 9, n data = 36</i>						
1. Early CGC, Late IOS	0.9362	5	0.1609	-182.77	12.554	0.001
2. Early CGC, Early IOS	0.9363	5	0.1606	-182.85	12.475	0.001
3. Early CGC, No IOS	0.9362	4	0.1609	-185.48	9.845	0.005
4. Late CGC, Late IOS	0.9520	5	0.1210	-193.03	2.300	0.235
5. Late CGC, Early IOS	<b>0.9550</b>	5	0.1135	-195.33	0.000	<b>0.742</b>
6. Late CGC, No IOS	0.9397	4	0.1521	-187.52	7.807	0.015

*Notes:*

No. of fitted parameters here was four ( $m, b, p, w$ ), reducing to three for models 3 & 6 where  $w=0$ . In all later analyses,  $p$  was fixed at  $p=2.7$ ; see legend to Fig. 6B.

The value K used in AIC is the no. of fitted parameters, plus 1.

Akaike weight can be interpreted as the probability that a given model is the best of those considered (Burnham & Anderson, 2002).

\*\*\* Fig. 6 about here \*\*\*

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### 3.4 Choice of model parameters guided by contrast-response data

Inspection of the fitted parameters from this initial analysis revealed that for model 4 the output exponent  $p$  was surprisingly high ( $5.56 \pm 0.37$ , mean  $\pm$  s.d. over the 8 levels of grouping). This was much higher than for model 5 (mean  $2.98 \pm 0.19$ ) as well as the other models, and also higher than many estimates of mean  $p$  in the physiological literature, eg. the canonical value of 2 required by energy models and half-squaring. In addition, having  $p$  as high as 5.5 renders the present models inconsistent with data on the contrast-response of cat V1 population means. Fig. 7 replots data from two extensive studies (Albrecht & Hamilton, 1982; Crowder, Van Kleef, Dreher, & Ibbotson, 2007) and there is good agreement between the two studies (done in different continents, 25 years apart), but poor agreement with the model's contrast response when  $p=5.5$  (blue curve in Fig. 7). The best match between model and data here was given by  $p=2.7$  (Fig. 7, red curve;  $R^2 = 0.966$ ). Thus to make our models consistent with these data on unadapted contrast-responses, we fixed  $p=2.7$ , instead of making it a free parameter.

This choice of  $p$  gives the model contrast-response a semi-saturation contrast ( $c_{50}$ ) of 0.20 in accord with the cat spike-rate data (Fig. 7). But now consider the contrast-response *before* the power transformation. In the model it is in essence a simple Naka-Rushton function of the form  $r = c/(s+c)$ , whose  $c_{50}$  is  $s$ , which we set to  $s = 0.06$ . This value is fairly close to the median  $c_{50}$  of 0.076 reported by Finn et al. (2007) (their Fig. 4F) for the membrane-potential responses of 46 cat cortical cells – and is 2-3 times lower than the  $c_{50}$  for spike rates (Fig. 7). This difference could arise directly from the output nonlinearity  $p$ . When we raise the early response  $r$  to the power  $p=2.7$  it is easy to calculate that this model 'spike-rate' transformation makes the contrast-response shallower and less compressive, and increases the  $c_{50}$  from 0.06 to 0.20, in line with the evidence just described. In short, two important fixed parameters  $s = 0.06$ ,  $p = 2.7$  are supported by evidence from contrast gains (Fig. 3), and from contrast-response functions at the level of membrane potential (Finn et al., 2007) and spike rates (Fig. 7).

\*\*\* Fig. 7 about here \*\*\*

### 3.5 The 'best' model

In AIC analysis, the Akaike weight estimates the probability that a given model is the best of those considered (Burnham & Anderson, 2002). With  $p$  fixed hereafter at  $p=2.7$ , we re-fitted the models and re-plotted the Akaike weights, as shown in Fig. 6B. Model 5 (late CGC, early IOS) was now uniquely favoured (mean weight = 0.935), while support for model 4 evaporated (mean weight = 0.029). Moreover, this pattern of Akaike weights remained very similar even when there was little or no averaging across cells. With 54, 27 or 18 groups (Fig. 6C) there were just 1, 2 or 3 cells per group respectively, and even though  $R^2$  values decreased markedly in the absence of averaging, model 5 remained most likely to be the 'best' model as before (mean weight for model 5 = 0.90, model 4 = 0.06). The relative superiority of model 5 therefore cannot be attributed to any artefact of averaging across cells. See Table 4 for mean values of parameters in these model fittings. We turn now from model fitting to questions about the component processes underlying contrast adaptation.

Table 4. Best-fitting parameter values for models 1-6, with fixed  $p = 2.7$ 

Model	s	z	b	m	p	w	
M1. Early CGC, Late IOS	0.06	0	0.182	0.475	2.7	0.000	mean
	-	-	0.005	0.008	-	0.000	sd
M2. Early CGC, Early IOS	0.06	0	0.178	0.482	2.7	0.010	mean
	-	-	0.008	0.013	-	0.019	sd
M3. Early CGC, No IOS	0.06	0	0.182	0.475	2.7	0	mean
	-	-	0.005	0.008	-	-	sd
M4. Late CGC, Late IOS	0.06	0	0.171	0.522	2.7	0.085	mean
	-	-	0.007	0.009	-	0.007	sd
<b>M5. Late CGC, Early IOS</b>	<b>0.06</b>	<b>0</b>	<b>0.096</b>	<b>0.691</b>	<b>2.7</b>	<b>0.141</b>	<b>mean</b>
	-	-	0.015	0.030	-	0.012	sd
M6. Late CGC, No IOS	0.06	0	0.203	0.458	2.7	0	mean
	-	-	0.007	0.009	-	-	sd

## NOTES

1. Table shows mean and SD over the 8 cell groupings ( $N=3$  to 10). Parameters  $s, z, p$  were fixed.
2. In practice, M1 = M3 because, after fitting, parameters  $b, m, w$  emerged as the same for both. Also, M2 was very close to M1, M3 in parameters and behaviour.

490

491

492 *3.6 Is there any adaptation at the B level ?*

493 We saw earlier (Fig. 3), that there appears to be little or no control of 'instantaneous'  
 494 contrast gain at the binocular ( $B$ ) level, an idea implemented here by  $z=0$ . Hence it is  
 495 natural to ask whether the  $B$  level also has no contrast adaptation: does  $b=0$  ? To answer  
 496 this, we fitted models 1-6 as before, but with fixed  $b=0$ . We denote these modified  
 497 versions as models 7-12 respectively. It might seem obvious that a cell with no adaptation  
 498 at the binocular level ( $b=0$ ) would not exhibit interocular transfer of adaptation, but that  
 499 was true only for models 9 and 12 that had  $b=0$  and lacked interocular suppression, ie.  
 500  $w=0$ . The model fits to cat response gains for these two models were very poor (eg.  $R^2 =$   
 501  $0.57, 0.58$  for  $N=9$ ) almost entirely because (unlike the cat cells) adapting one model eye  
 502 had no effect at all on test responses through the other eye. But the question is less  
 503 obvious for the other models (7,8,10,11) that did have interocular suppression. For models  
 504 7,8,11, but not 10, we found that increases in  $m$  and  $w$  (the IOS weight) largely but not  
 505 entirely compensated for the lack of binocular-level adaptation. Interocular suppression,  
 506 persisting from the adaptation period through to the test period, thus provides a plausible  
 507 source of interocular transfer when  $B$  level adaptation is lacking. Nevertheless, from AIC  
 508 analysis over the 12 models (Fig. 6D), we should still conclude that model 5 was the one  
 509 best supported by the data while 'model 11' (ie. model 5 with  $b=0$ ) received slight  
 510 support. No others did.

511

512 Since M11 is nested within M5, we also used an F-test to determine whether M5 (with  $b$   
 513 as a free parameter) fitted significantly better than M11 ( $b=0$ ). That is, did the inclusion  
 514 of  $B$ -level adaptation in M5 justifiably improve the fit ? Table 5 suggests that it did. The  
 515 mean P-value over the 8 cell groupings was 0.02, with median  $P = 0.01$ , and only one of 8  
 516 tests gave  $P > 0.05$ . We should caution that the 8 tests were not independent, because they  
 517 were based on the same raw data, albeit combined into groups differently in each case.  
 518 Nevertheless, their consistency (7/8 tests gave  $P < 0.05$ ) reinforces the conclusion from  
 519 AIC analysis: by including adaptation at the binocular level, model 5 accounts for the  
 520 patterns of data better than models that do not.

521

F-test for nested models:

**Table 5** Does M5 fit significantly better than M11?

Nggroups	F-ratio	Ndata	d.f.	Pvalue
3	12.791	12	1,9	0.005
4	5.733	16	1,13	0.031
5	6.541	20	1,17	0.020
6	8.941	24	1,21	0.007
7	6.71	28	1,25	0.016
8	3.311	32	1,29	0.079
9	9.944	36	1,33	0.003
10	12.719	40	1,37	0.001

Mean P-value over the 8 cell groupings = 0.020, median = 0.011

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### 3.7 Is IOS a necessary feature of the model ?

We used the F-test again to ask whether IOS (with mean  $w=0.14$ , Table 4) contributed significantly to the fit of model 5. We did this by comparing M5 with M6 that did not include IOS ( $w=0$ ). The answer from Table 6 is that all the P-values were very low (mean  $P = 0.0023$ , median 0.0014), thus strongly implying that IOS contributes significantly to the contrast adaptation effect. With IOS, adapting contrast in one eye drives down the gain of the other eye's monocular ( $M$ ) mechanism, and some of that loss of gain persists to influence (reduce) the cell's later response to a test stimulus in the other eye.

F-test for nested models:

**Table 6** Does M5 fit significantly better than M6?

Nggroups	F-ratio	Ndata	d.f.	Pvalue
3	15.931	12	1,9	0.0026
4	13.301	16	1,13	0.0026
5	16.031	20	1,17	0.0008
6	13.25	24	1,21	0.0014
7	8.056	28	1,25	0.0087
8	14.46	32	1,29	0.0007
9	12.113	36	1,33	0.0014
10	15.362	40	1,37	0.0004

Mean P-value over 8 cell groupings = 0.0023, median = 0.0014

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### 3.8 Interim summary

Judged by  $R^2$  values, all six models gave a good account of the Howarth *et al* (2009) dataset in terms of variance accounted for. For example, with  $N=6$ ,  $R^2$  was 0.955 or higher, both when  $p$  was fitted (Table 3) and when fixed ( $p=2.7$ ). We think these fits are generally good because the models share the same basic structure and it is a useful one (Fig. 1). But  $R^2$  is not a good tool for model comparison and model selection; small differences in  $R^2$  can represent large differences in the extent to which one model should be preferred over another (Burnham & Anderson, 2002, pp. 95-96). For a more fine-grained comparison of the models, we used AIC analysis (Fig. 6) and found that model 5 was consistently the preferred model from this set of six models. The evidence supports the *late CGC, early IOS* scheme (Fig. 2E), such that adaptation occurs at both the  $M$  and

548 *B* levels, and interocular suppression contributes to adaptation at the *M* level. At the  
549 monocular (*M*) level, NE gain varies with OD, but DE gain does not. There is a  
550 significant contribution from adaptation at the binocular level though its strength is lower  
551 than in the other models (Table 4). Interocular transfer of adaptation is mediated by gain  
552 change at the binocular level and by the aftereffect of interocular suppression at the  
553 monocular level.

554 \*\*\* Fig. 8 about here \*\*\*

555

### 556 3.9 Trends in adapted gain as OD varied between groups

557 Fig. 8 plots cat response gains after adaptation (coloured symbols) as a function of OD,  
558 expressed as the ratio *D* of unadapted response gains (NE/DE) for each cell group. The  
559 unadapted response gains (grey squares) are important reference points, but not  
560 informative about the models because they are either 1.0 by definition (test DE) or are  
561 given from the data and choice of cell grouping, independently of any model (test NE).  
562 Each model should be assessed by how well it fits the *adapted* gains (red & cyan  
563 symbols).

564

565 Model 5 fitted the data very well for all 4 combinations of adapt eye and test eye (Fig.  
566 8A,B,C, red & cyan;  $R^2=0.949$ ,  $n=44$  data). The simplest of the alternative models (M3)  
567 predicted adapted gains (black curves) that were almost identical to M5 when the adapted  
568 eye was tested (Fig. 8A,C), but showed clear deviations from the data in the between-eye  
569 conditions (cyan symbols) in two ways. (i) After NE adaptation, M3 predicted a fall in DE  
570 gain with increasing *D* (Fig. 8A, black curve) that was steeper than observed and steeper  
571 than predicted by M5 (cyan curve). (ii) After DE adaptation, M3 predicted higher NE test  
572 gains than observed over most of the range (Fig. 8B, black line). Model 5 (late CGC,  
573 early IOS) thus emerged as the preferred model overall, evident by inspection here and by  
574 the statistical analyses presented above (Fig. 6B,C & Tables 5,6; see also Fig. S8).

575

### 576 3.10 Monocular, binocular & interocular components of adaptation

577 To shed more light on underlying mechanisms, we consider model gain changes induced  
578 by adaptation at the *M* and *B* levels of processing. We begin with the simplest model M3  
579 (Fig. 9) then consider how M5 adds extra insight (Fig. 10). [Note that models M1,2,3 are  
580 effectively the same here, because they differed only in IOS; model-fitting returned  $w=0$   
581 in all three cases, meaning that IOS was silenced and all three early CGC models were  
582 rendered equivalent.]

583

584 The largest effect of adaptation on cat cell response gains came from adapting and testing  
585 the dominant eye (which we denote here as DE/DE, meaning adapt DE/test DE): observed  
586 response gain fell from 1 to about 0.36, with no evident trend across the OD groups (Fig.  
587 9D, red squares). M3 captured this effect through similar reductions of gain at the  
588 monocular level (gain 0.82, Fig. 9B, red) and at the binocular level (gain 0.85, Fig. 9C,  
589 red). Model response gain is the product of three factors ( $d_L M_L B$ ), [ $1*0.82*0.85 = 0.70$ ],  
590 raised to the power  $p$ , to give  $(0.70)^{2.7} = 0.38$ , very close to the mean observed gain. Thus  
591 two small gain changes become a larger one because they are in series, and then the  
592 expansive nonlinearity amplifies the effect - smaller gains become even smaller. In *all*  
593 models there was no trend across the OD groups for this DE/DE condition, because any  
594 influence of OD is revealed only by stimulation of NE.

595

596 \*\*\* Figs. 9 & 10 about here \*\*\*

597

598 When the dominant eye was tested after non-dominant adaptation (NE/DE), cat response  
599 gain was reduced much less overall (Fig. 9D, cyan squares) and the reduction in gain  
600 tended to be greater with increasing ocular balance *D* of the cell group. The early CGC  
601 model (cyan curve) captured the reduced magnitude of the aftereffect but over-estimated

602 this downward trend across the OD groups. There was no IOS, and so in this interocular  
603 condition monocular gains were unchanged by adaptation (Fig. 9B, cyan), but NE  
604 adaptation scaled down binocular gain  $B$ . It did so just a little when  $D$  was close to 0 (Fig.  
605 9C, cyan curve) because OD makes the adapting signal weak. When  $D$  is higher,  $B$  is  
606 scaled down more, because the adapting signal  $r_B'$  gets stronger as ocular balance  
607 approaches 1. But M3 consistently over-estimated this trend in the data, and because of  
608 their effective equivalence (noted above), M1 & M2 could do no better.

609

610 Model M5, on the other hand, was notably more accurate than M3 (Fig. 10D, or Fig. 8A,  
611 cyan curve). Early IOS reduced  $M_L$  uniformly over OD (Fig. 10B, cyan), and this gain  
612 change at the  $M$ -level meant that the predicted change of  $B$  gain was smaller (Fig. 10C),  
613 resulting in a shallower change in overall gain and a closer fit to the data (Fig. 10D or Fig.  
614 8A, cyan). In brief, interocular suppression at the monocular level accounted for some of  
615 the interocular transfer of adaptation, while binocular-level adaptation accounted for the  
616 rest. Because the IOS component of this transfer was 'early', it was also uniform across  
617 OD, and so enabled M5 to account better for the shallowness of the trend in the data – a  
618 trend created at the  $B$  level of adaptation (Fig. 10C). The possibility of there being *no*  $B$ -  
619 level component of adaptation was considered but found little support (Table 5 & Fig.  
620 6D).

621

622 Turning now to testing the non-dominant eye, we see a very different pattern of cell gains  
623 (Fig. 8B,C). Unadapted gains, by definition, increased across the OD groups (grey  
624 squares), and the observed aftereffects (reductions in gain) were of similar average  
625 magnitude after adapting the DE (Fig. 8B, cyan squares) or the NE (Fig. 8C, red squares).  
626 The simplest model M3 described the linear trend of these adapted gains fairly well (Fig.  
627 9G, 9H), but exaggerated the difference in their slopes. Nevertheless, a fairly good model  
628 should be ousted by a better one, and that is so here: M5 has a much tighter fit to the  
629 DE/NE data (Fig. 8B, cyan), and to the NE/DE data (Fig. 8A, cyan). M5 also nicely  
630 captures the upward curvature in the DE/NE data (Fig. 8B or Fig. 10H, cyan), and the  
631 lack of it in the NE/NE data (Fig. 8C or Fig. 10G, red squares).

632

### 633 3.11 The 'signature' of the best-fitting model

634 To see why M5 behaves the way it does, we need to consider the adaptive changes in  $M$ -  
635 and  $B$ -gains across different OD groups. But in the late CGC models (M4,5,6) there are  
636 large increases in absolute  $M$ -gain with decreasing  $D$  (Fig. 10F) – a direct consequence of  
637 late, OD-dependent CGC that was not a feature of early CGC (Fig. 9F). To remove this  
638 trend and make it easier to see the *change* in gain due to adaptation, Figs. 11A and 11C  
639 plot the *gain ratio*  $Q$  (adapted  $M$  gain divided by unadapted  $M$  gain) as a function of  $D$ .  
640 Fig. 11A (test DE) is unaffected by this relative measure (since unadapted DE gain = 1),  
641 but Fig. 11C (test NE) now interestingly reveals that the predicted influence of OD on  
642 monocular gain change  $M_R$  is in *opposite* directions for DE adaptation (cyan) and NE  
643 adaptation (red). This crossover interaction carries through to the final plot of predicted  
644 response gain (Fig. 11D, thin curves) and is also evident in the cat data, where at low  $D$   
645 the cyan symbols are mainly below the red but rise above at high values of  $D$ . The two  
646 predicted curves have opposite curvature, and track the corresponding data quite closely  
647 as  $D$  increases. When the number of cell groups was varied, a similar crossover in both  
648 data and model was seen in each case. Fig. S7, lower row, summarizes this for  $N = 3, 6, 9$   
649 groups.

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\*\*\* Fig. 11 about here \*\*\*

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653 We can think of this crossover (Fig. 11C,D) as the signature of models M5 (and M4),  
654 because it arises directly from the combination of *late* CGC and IOS (early or late). By  
655 our definition, *late* CGC is affected by OD (less NE/NE suppression when  $D$  is low) but

656 *early* CGC is not. For M5 and the same eye (NE/NE) condition we derive from eqn. 8.5  
657 that with test contrast  $c_R=1$  the adapted/unadapted  $M_R$  gain ratio  $Q_R$  is:

658 
$$Q_R = \frac{s + d_R}{s + d_R + m \cdot a_R \cdot d_R}$$

659 where  $m \cdot a_R$  is constant and  $d_R$  increases with  $D$  (eqn. 13). When  $d_R$  is zero  $Q_R$  is 1, but  
660 then  $Q_R$  falls as  $d_R$  increases (red curve in Fig. 11C); adaptation here is least for the most  
661 unbalanced cells. But for DE adapt, NE test, adaptation at this monocular level depends  
662 on the interocular route, and from eqn. 8.5 we get:

663 
$$Q_R = \frac{s + d_R}{s + d_R + m \cdot w \cdot a_L}$$

664 where  $m \cdot w \cdot a_L$  is a positive constant. When  $d_R$  is zero  $Q_R < 1$ , but  $Q_R$  then *rises* towards 1  
665 as  $d_R$  increases (cyan curve in Fig. 11C). In summary, this crossover can be seen as a  
666 defining characteristic of M5 – late CGC with early IOS – and its predictions are  
667 supported fairly clearly by the data (Fig. 11D).

668  
669 Similar arguments apply for M4 (late CGC, late IOS) and a similar crossover occurs in its  
670 predictions for NE test (Fig. S4). But late IOS (M4) is influenced by OD while early IOS  
671 (M5) is not. This difference results in a poorer fit to the interocular NE/DE condition for  
672 M4 than M5 (Fig. S8G). Hence we continue to focus our attention on M5 as the best  
673 explanation within this set of models.

### 674 675 3.12 Number of OD groups

676 We wondered whether the pattern of observed effects (Fig.8) might depend on the way  
677 the cells were divided into groups, or on the size of the groups. But on close examination  
678 we did not see any notable variations in the pattern of experimental results or model  
679 behaviour across the different ways of grouping. The outcomes are summarized for M5  
680 with  $N=3,6,9$  in Fig. S7 (*Supplementary material*).

681  
682

## 683 4. Discussion

684

685 We set out to increase our understanding of the functional architecture of binocular vision  
686 that allows cortical cells in V1 to respond to contrast, to adapt to contrast, and to combine  
687 inputs from the two eyes. The goal was to extend existing models by specifying how  
688 contrast adaptation may affect contrast gain both before and after binocular summation,  
689 and identifying where and how in this chain of events (Fig. 1) ocular dominance plays its  
690 part in making binocular cells less responsive to one eye than the other. To this end we re-  
691 analyzed and modelled data from Howarth et al. (2009), who studied contrast adaptation,  
692 interocular transfer, orientation/direction tuning and ocular dominance in cat cortical  
693 cells. To reduce variability and get a clearer picture of typical cell behaviour, we applied  
694 our models to responses averaged within subsets of cells that had similar degrees of  
695 ocular dominance (OD). An important simplification emerged: the orientation/direction  
696 tuning curve for these group-average responses had essentially the same shape for all OD  
697 groups and all the tested stimulus conditions, with or without adaptation of one or other  
698 eye. Adaptation, and ocular dominance, simply scaled all response rates down by a  
699 common factor which we call the *response gain*. A good model should therefore account  
700 for the pattern of changes in response gain, and maintain the tuning invariance, and strike  
701 a balance between simplicity and accuracy.

702

703 Using data and ideas from Truchard et al (2000), and from the extensive literature on  
704 suppressive gain controls, we devised simple expressions for monocular-level contrast  
705 gain controls (CGC) in which gain (for a given eye) might be driven down by the current  
706 test contrast and by prior adapting contrast in that eye or (to a lesser extent) the other eye.

707 Ocular dominance was represented by a single assumption - the contrast input to the non-  
708 dominant eye was attenuated by a fixed factor  $d$  that varied between cells, while input to  
709 the dominant eye was not influenced by OD. Indeed the model was simple and  
710 parsimonious, because the model cells were all the same except for the variation in  
711 dominance factor  $d$ . We used the model to derive  $d$  from the observed ratio  $D$  of  
712 unadapted gains in the two eyes. The cell response was shaped by the suppressive  
713 influences on the  $M$ -level CGC, arising either *early* (before OD) or *late* (after OD).  
714 Signals from the two eyes were then summed linearly and provided the excitatory drive  
715 for the cell, and the input to the binocular-level gain control. The final step, now standard  
716 in V1 models, was an accelerating power function nonlinearity, approximating the  
717 transformation from membrane potential to spike rate.

718  
719 Our account proposes three sources of gain change underlying contrast adaptation: (i)  
720 monocular gain change induced directly for one eye when that eye was adapted, (ii)  
721 monocular gain change induced between-eyes by interocular suppression (IOS) that was  
722 driven by the adapter, and (iii) binocular gain change driven by previous levels of  
723 binocular response. We modelled six different ways in which these processes might be  
724 organized (Fig. 2), and fitted these models to sets of adapted response gains derived from  
725 data of 54 cells with a wide range of ODs. We found that just one model (M5, Fig. 2E)  
726 was consistently superior to the others. It combined *late* CGC with *early* IOS, meaning  
727 that ipsiocular gain change was influenced by OD, while interocular gain change via IOS  
728 at the monocular level was not. Adaptation at the binocular level was also present, but  
729 relatively small. This combination of characteristics explained (a) the slight, but not  
730 dramatic, dependence of interocular gain change on OD when the dominant eye was  
731 tested (Fig. 8A, cyan), (b) the complete lack of OD influence on ipsiocular gain change  
732 for the dominant eye (Fig. 8A, red), (c) the overall similarity between ipsi- and inter-  
733 ocular adapted gains when the non-dominant eye was tested (Fig. 11D), coupled with (d)  
734 the modest but consistent crossover interaction between OD and adapting eye, evident for  
735 both model gains and data in Fig. 11D. We showed that this interaction in the model (Fig.  
736 11C) arose specifically from the combination of *late* CGC with IOS.

737  
738 In short, a functional model (M5), with three sources of gain control (monocular,  
739 binocular, interocular) that has 3 corresponding fitted parameters ( $m, b, w$ ) and 3 fixed  
740 parameters ( $s, z, p$ ), gives us a compact and reasonably accurate account of the way ocular  
741 dominance influences contrast adaptation and interocular transfer across a heterogeneous  
742 sample of cells in cat visual cortex.

743  
744 We showed that model M5 implies a significant role for gain change at the binocular level  
745 ( $b > 0$ ). This might suggest that contrast adaptation is somehow different from the  
746 'instantaneous' contrast gain control studied by Truchard et al (2000), who found little or  
747 no evidence for binocular-level change in contrast gain. However, our analysis (Fig. 3)  
748 suggests a simpler resolution: in the stimulus conditions studied so far, changes of  
749 monocular gain can be large, while binocular gain changes are relatively small, but not  
750 negligible. Further experimental studies comparing the characteristics of simultaneous  
751 and successive changes in contrast gain would be valuable.

#### 752 753 *4.1 Limitations and efforts to overcome them*

754 • In the experiments re-analyzed here, adapting and test contrasts were fixed at a single  
755 value, and so important features of the proposed gain controls remained untested. In part  
756 we filled the gaps by ensuring that each model's monocular contrast gains closely  
757 matched the contrast-dependence of gain measurements made by Truchard et al (2000)  
758 (Fig. 3). We also ensured good agreement between the model's unadapted contrast  
759 response function and population data from two other studies (Fig. 7).

760

761 • Adapting and test stimuli were always monocular. From the models we found that  
762 changes in gain due to adaptation were expected to be present at early (monocular) and  
763 later (binocular) levels (Figs. 9B,C, 10B,C). The exception was that when OD was  
764 extreme ( $D$  close to 0) adapting the non-dominant eye gave little change of response gain  
765 (in both model and data; Figs. 9D,10D). This was well explained by the attenuating effect  
766 of OD on the binocular-level response to the adapter. An obvious limitation, though, is  
767 that this model for responses of binocular cells has not been challenged with binocular or  
768 dichoptic stimuli. More evidence is needed, especially by combining contrast adaptation  
769 with simultaneous, dichoptic testing of both eyes.

770

771 We aim to address some of these issues in future work by comparing model performance  
772 with published data from cat cortical responses to binocular gratings that differed in  
773 contrast and/or phase between the eyes (Freeman & Robson, 1982; Kara & Boyd, 2009;  
774 Ohzawa & Freeman, 1986; Smith, Chino, Ni, & Cheng, 1997; Smith, Chino, Ni, Ridder,  
775 et al., 1997; Truchard et al., 2000), briefly discussed next.

776

#### 777 4.2 Disparity sensitivity, CGC, and compensation for OD

778 When V1 cells are tested with the same sinewave gratings drifting in the same direction in  
779 both eyes, their response often varies with the phase disparity  $\phi$  between the eyes. The  
780 response is modulated by disparity and the depth of modulation, known as the *binocular*  
781 *interaction index* (BII), is a single number that quantifies the disparity sensitivity of the  
782 cell. If left and right eye responses were proportional to contrast, and then summed, the  
783 response over disparity would, as observed, be deeply modulated when the input contrasts  
784 were equal. But if one eye's contrast were reduced its influence on the binocular sum  
785 would decrease, and the BII would fall markedly as the two contrasts became more  
786 unequal. Surprisingly, however, the BII for cells in both cat and monkey visual cortex  
787 tended to be fairly constant as the interocular contrast ratio varied (Freeman & Ohzawa,  
788 1990; Smith, Chino, Ni, & Cheng, 1997), implying some important nonlinearity in the  
789 monocular input paths (Freeman & Ohzawa, 1990).

790

791 Truchard *et al* (2000) resolved this question by showing that monocular gain controls  
792 compensate for differences in contrast between the eyes. Such cells might encode  
793 disparity without being perturbed by contrast mismatch. Freeman & Ohzawa (1990) were  
794 the first to report evidence of this kind, and they conjectured that "ocular dominance may  
795 be compensated for by such a mechanism under binocular viewing conditions". From our  
796 modelling we can take this a step further: compensation for OD occurs with *late* CGC but  
797 not early CGC. Fig. 12A shows how with early CGC the NE response is strongly  
798 attenuated (relative to DE) by factor  $d$  at all contrasts (green curve). But with late CGC  
799 the attenuation is much less than factor  $d$ , implying a substantial degree of compensation  
800 for OD (red curve). Why is the attenuation less? With late CGC the divisive signal is  $d.c$   
801 (not  $c$ ) and so there is less suppression as  $d$  gets lower. Hence monocular gain rises (cf.  
802 Fig. 10F) and this partly compensates for the signal attenuation due to OD, as Fig. 12  
803 shows (red vs green curves). This suggests an important functional role for late CGC in  
804 (partially) compensating for OD, making the inputs to binocular summation more  
805 balanced than they otherwise would be. That in turn should increase the disparity  
806 sensitivity (BII) of the cell, as it does when contrasts are physically mismatched between  
807 the eyes (Truchard et al, 2000).

808

809

\*\*\* Fig. 12 about here \*\*\*

810

#### 811 4.3 Importance of the saturation constant, $s$

812 Many or most V1 cells respond nonlinearly to contrast (e.g. Fig. 7), but some respond  
813 more linearly. For example, (i) Albrecht et al (2002) reported that in their study and others  
814 about 5% of V1 cells "demonstrated a nearly linear relationship" between response and



852

853 **Appendix**

854

855 **Equations for the six models**

856

857 Fig. 2 sketched six ways in which the monocular gain controls might be organized,  
 858 incorporating ipsiocular contrast gain control (CGC) and (optionally) interocular  
 859 suppression (IOS). We suppose that both these gain-control mechanisms can be driven by  
 860 both the current (test) contrast and the prior (adapting) contrast and that these driving  
 861 signals might arise *early* (before attenuation due to OD), or *late* (after OD attenuation).  
 862 First, we specify models 3,2,1 that have early CGC. We begin with the simplest model  
 863 (M3), and build from there.

864

865 *Model 3: Early CGC, no IOS*866 Monocular responses  $r_L, r_R$  to a stimulus with contrasts  $c_L, c_R$  are:

$$867 \quad r_L = \frac{(1+s) \cdot S(\theta) \cdot d_L \cdot c_L}{s + c_L + m \cdot a_L} \quad (1.3)$$

868

$$869 \quad r_R = \frac{(1+s) \cdot S(\theta) \cdot d_R \cdot c_R}{s + c_R + m \cdot a_R} \quad (2.3)$$

870 where  $L$  and  $R$  subscripts refer to left and right eyes,  $c$  is test contrast,  $a$  is adapting  
 871 contrast,  $d_L$  and  $d_R$  are the attenuation factors related to ocular dominance,  $s$  is a semi-  
 872 saturation constant,  $m$  is the strength parameter for contrast adaptation at this monocular  
 873 level,  $\theta$  is the test orientation/direction relative to the preferred orientation of the cell, and  
 874  $S(\theta)$  is the orientation sensitivity curve. Note that by our definition of *early* CGC the  
 875 divisor (gain control) terms are not affected by the ocular dominance factors. The  
 876 numerator - a linear response to contrast - is scaled by both  $S$  and  $d$  terms and is the same  
 877 for all models.

878

879 *Model 2: Early CGC, early IOS*

$$880 \quad r_L = \frac{(1+s) \cdot S(\theta) \cdot d_L \cdot c_L}{s + c_L + w \cdot c_R + m \cdot (a_L + w \cdot a_R)} \quad (1.2)$$

881

$$882 \quad r_R = \frac{(1+s) \cdot S(\theta) \cdot d_R \cdot c_R}{s + c_R + w \cdot c_L + m \cdot (a_R + w \cdot a_L)} \quad (2.2)$$

883

884 With IOS, left eye contrasts reduce the gain for right eye responses, and vice-versa. The  
 885 IOS terms ( $w \cdot c$ ,  $w \cdot a$ ) have been introduced 'early', without scaling by  $d_L$  or  $d_R$ . Parameter  
 886  $w$  is the weight factor for IOS.

887

888 *Model 1: Early CGC, late IOS*

$$889 \quad r_L = \frac{(1+s) \cdot S(\theta) \cdot d_L \cdot c_L}{s + c_L + w \cdot c_R \cdot d_R + m \cdot (a_L + w \cdot a_R \cdot d_R)} \quad (1.1)$$

890

$$891 \quad r_R = \frac{(1+s) \cdot S(\theta) \cdot d_R \cdot c_R}{s + c_R + w \cdot c_L \cdot d_L + m \cdot (a_R + w \cdot a_L \cdot d_L)} \quad (2.1)$$

892

893 Interocular suppression terms ( $w \cdot c_R \cdot d_R$ ,  $w \cdot a_R \cdot d_R$ , etc) have been introduced 'late', hence  
 894 scaled by the OD factors  $d_L$ ,  $d_R$ .

895

896

897

898 *Model 6: Late CGC, no IOS*

899 We now describe the corresponding equations for *late* CGC, models 6,5,4, beginning  
900 with the simplest (M6). Monocular responses  $r_L, r_R$  to a stimulus with contrasts  $c_L, c_R$  are:

$$901 \quad r_L = \frac{(1+s) \cdot S(\theta) \cdot d_L \cdot c_L}{s + c_L \cdot d_L + m \cdot a_L \cdot d_L} \quad (1.6)$$

902

$$903 \quad r_R = \frac{(1+s) \cdot S(\theta) \cdot d_R \cdot c_R}{s + c_R \cdot d_R + m \cdot a_R \cdot d_R} \quad (2.6)$$

904

905 *Model 5: Late CGC, early IOS*

906

$$907 \quad r_L = \frac{(1+s) \cdot S(\theta) \cdot d_L \cdot c_L}{s + c_L \cdot d_L + w \cdot c_R + m \cdot (a_L \cdot d_L + w \cdot a_R)} \quad (1.5)$$

908

$$909 \quad r_R = \frac{(1+s) \cdot S(\theta) \cdot d_R \cdot c_R}{s + c_R \cdot d_R + w \cdot c_L + m \cdot (a_R \cdot d_R + w \cdot a_L)} \quad (2.5)$$

910

911 In eqn. 1.5, CGC terms ( $c_L \cdot d_L$ ,  $a_L \cdot d_L$ ) are late, while IOS terms ( $w \cdot c_R$ ,  $w \cdot a_R$ ) are early and  
912 similarly for eqn 2.5 (but with L,R swapped).

913

914 *Model 4: Late CGC, late IOS*

915 Finally, equations for late CGC, late IOS contain all possible gain terms in our scheme:

916

$$917 \quad r_L = \frac{(1+s) \cdot S(\theta) \cdot d_L \cdot c_L}{s + c_L \cdot d_L + w \cdot c_R \cdot d_R + m \cdot (a_L \cdot d_L + w \cdot a_R \cdot d_R)} \quad (1.4)$$

918

$$919 \quad r_R = \frac{(1+s) \cdot S(\theta) \cdot d_R \cdot c_R}{s + c_R \cdot d_R + w \cdot c_L \cdot d_L + m \cdot (a_R \cdot d_R + w \cdot a_L \cdot d_L)} \quad (2.4)$$

920

921 The six models here are different only in the gain control (denominator terms) with no  
922 change in the numerator terms.

923

924 *Binocular summation*

925 After equations 1.x, 2.x, (where  $x$  is model number) the remaining definitions (eqns.  
926 3,4,5,6) are common to all six models. Responses to the two eyes are linearly summed:

$$927 \quad r_B = r_L + r_R \quad (3).$$

928

929 *Responses to the adapter*

930 From eqns 1-3, we can also describe Stage 1 responses  $r'$  during the adaptation period  
931 where there is no prior adaptation term, and the current contrast is  $a_L$  rather than  $c_L$ . We  
932 illustrate model 4 here; the others should be obvious.

$$933 \quad r'_L = \frac{(1+s) \cdot S(\theta) \cdot d_L \cdot a_L}{s + a_L \cdot d_L + w \cdot a_R \cdot d_R} \quad (1'.4)$$

934

$$935 \quad r'_R = \frac{(1+s) \cdot S(\theta) \cdot d_R \cdot a_R}{s + a_R \cdot d_R + w \cdot a_L \cdot d_L} \quad (2'.4)$$

936

$$937 \quad r'_B = r'_L + r'_R \quad (3')$$

938

939 where  $S(\theta) = 1$ , because the adapter was always at the cell's preferred orientation.

940

941

942 *Stage 2, binocular adaptation and gain control*

943 Much psychophysical evidence (e.g. Baker & Meese, 2012; Bjørklund & Magnussen,  
944 1981; Blake, Overton, & Lema-Stern, 1981; Vilidaitė & Baker, 2015) and a smaller  
945 number of physiological studies (Howarth et al., 2009; Maffei, Fiorentini, & Bisti, 1973)  
946 have shown that contrast adaptation transfers, at least partially, from adapting one eye to  
947 testing the other. Hence some component of adaptation is likely to lie at or after the point  
948 of binocular combination, and Stage 2 of this model represents contrast adaptation at the  
949 level of binocular integration. The final response  $R(\theta)$  of a model cell as a function of test  
950 orientation is given in two parts by (i) a response function that incorporates divisive gain  
951 reduction by responses to both the current stimulus and the previous adapting stimulus:

$$952 \quad R_B(\theta) = \frac{(1+z) \cdot r_B(\theta)}{1+z \cdot e_B + b \cdot r'_B} \quad (4)$$

953 followed by (ii) an output nonlinearity and amplitude scaling:

$$954 \quad R(\theta) = R_{max} \cdot (R_B)^p \quad (5)$$

955

956 where  $p$ ,  $z$  and  $R_{max}$  are constants,  $b$  represents the strength of adaptation at the binocular  
957 level, and  $e_B$  represents the drive from the current stimulus into this second-stage gain  
958 control. Normalization models (e.g. Heeger, 1992; reviewed by Carandini & Heeger,  
959 2012; Sawada & Petrov, 2017) typically suppose that  $e_B$  is an amalgam of responses from  
960 many nearby cells (the 'normalization pool'). An important feature is that  $e_B$  should  
961 depend on contrast but be invariant with stimulus orientation, in order that the orientation  
962 tuning is preserved over different contrast levels, as observed physiologically (Albrecht  
963 and Geisler, 1991; Heeger, 1992; Carandini et al., 1997; Scholl, Latimer, & Priebe, 2012).  
964 Pooling signals from many cells of different preferred orientations is a way to achieve that  
965 invariance, but a simple near-equivalent for our model was to assume that  $e_B =$   
966  $max(r_B(\theta))$ . The value of  $z$  controls the strength of this gain control effect, and the  $(1+z)$   
967 term on the numerator ensures that  $R_B$  is 1 when  $r_B$  is 1 in the absence of adaptation. It  
968 follows that (for monocular stimuli, as here) the peak final output  $R = R_{max}$  when  $R_B = e_B$   
969  $= r_B = c_L = 1$ , irrespective of  $p$ . Importantly, when  $b > 0$ , some trace of the previous  
970 response  $r'_B$  to the adapter acts to drive down the gain of the current response  $R_B$  to the  
971 test. Gain change at this binocular level, and/or [interocular suppression at the monocular](#)  
972 [level](#), could mediate interocular transfer of adaptation.

973

974 *Output transducer*

975 Squaring of the output values ( $p=2$ ) is a default assumption in some cortical cell models  
976 (Heeger, 1992), but physiological studies broadly agree that the value of  $p$  varies quite  
977 widely across V1 cells from about 1 to 5, with a cat population mean  $\pm$  SD of about  $2.5 \pm$   
978  $1.35$  (Albrecht & Hamilton, 1982). The weight of physiological evidence favouring a  
979 power-law output nonlinearity (Albrecht & Geisler, 1991; Contreras & Palmer, 2003;  
980 Priebe, 2008; Priebe & Ferster, 2005; Priebe, Mechler, Carandini, & Ferster, 2004; Sclar,  
981 Maunsell, & Lennie, 1990) led us [first to fit the model with  \$p\$  as a free parameter](#). In  
982 [section 3.4 we give reasons why an \*a priori\* value of  \$p\$  \( \$p = 2.7\$ , determined by other](#)  
983 [published data\) is more appropriate](#).

984

985 *Monocular & Binocular gains*

986 The whole model can be summarized by condensing eqns. 1-5 into a single equation (Fig.  
987 1):

$$988 \quad R = R_{max} \cdot \{S(\theta) \cdot (c_L d_L M_L + c_R d_R M_R) \cdot B\}^p \quad (6)$$

989

990 The monocular gain factors in eqn. 6 are obtained from eqns. 1.x, 2.x by deleting the  $S, d, c$   
991 terms in the numerator. For example, in model 4:

$$992 \quad M_L = (1+s)/(s + c_L \cdot d_L + w \cdot c_R \cdot d_R + m \cdot (a_L \cdot d_L + w \cdot a_R \cdot d_R)), \quad (7.4)$$

993

994  $M_R = (1 + s)/(s + c_R \cdot d_R + w \cdot c_L \cdot d_L + m \cdot (a_R \cdot d_R + w \cdot a_L \cdot d_L)),$  (8.4)  
 995

996 or model 5:

997  $M_L = (1 + s)/(s + c_L \cdot d_L + w \cdot c_R + m \cdot (a_L \cdot d_L + w \cdot a_R)),$  (7.5)  
 998

999  $M_R = (1 + s)/(s + c_R \cdot d_R + w \cdot c_L + m \cdot (a_R \cdot d_R + w \cdot a_L)).$  (8.5)  
 1000

1001  $B$  is obtained for all models by deleting  $r_B$  from the numerator in eqn. 4:  
 1002

1003  $B = (1 + z)/(1 + z \cdot \max(r_B) + b \cdot r'_B),$  (9)  
 1004 and  $d_L=1, d_R \leq 1.$

1005  
 1006 *Deriving dominance parameter  $d$  from unadapted data*

1007 The dominance ratio parameter  $d$  was derived from the unadapted data, as follows. In  
 1008 general, from eqn. 6 the spike-rate response to monocular contrast  $c$  in the left eye ( $c_L=c,$   
 1009  $c_R=0$ ) is:

1010  $R_L = R_{max} \cdot \{S(\theta) \cdot (cd_L M_L) \cdot B\}^p$  (10)

1011 and similarly for contrast in the right eye ( $c_R=c, c_L=0$ ):

1012  $R_R = R_{max} \cdot \{S(\theta) \cdot (cd_R M_R) \cdot B\}^p$  (11).  
 1013

1014 **With early CGC (models 1-3)** inspection of eqns. 7,8,9 shows that in the absence of  
 1015 adaptation ( $a_L = 0, a_R = 0$ )  $M_L$  (in eqn. 10) and  $M_R$  (in eqn. 11) are equal, and given that  
 1016  $z=0$  the gain  $B$  is also the same for these two monocular inputs. If we now take their  
 1017 spike-response ratio  $R_R/R_L$ , all but the  $d_L, d_R$  terms cancel out. Simple manipulation thus  
 1018 reveals that, for any test contrast and orientation, the response ratio  $R_R/R_L = \{d_R/d_L\}^p$ .  
 1019 By definition  $d = d_R/d_L$ , and so the dominance factor  $d$  is the spike-rate response ratio,  
 1020 'back-projected' through the output nonlinearity:  $d = \{R_R/R_L\}^{1/p}$ . In practice, the most  
 1021 reliable way to exploit this relation was to work with the unadapted response gains  $G_{NE},$   
 1022  $G_{DE}$  (slopes of the kind seen in Fig. 4C,D, black) because they made best use of the  
 1023 response data from all test orientations. Using these empirical unadapted gains of each  
 1024 OD cell group, **and letting  $D = G_{NE}/G_{DE}$  we computed**

1025  $d = D^{1/p}$  (12).  
 1026

1027 **For late CGC (models 4-6),** the factor  $d$  for each OD group could again be derived from  
 1028 the unadapted data via the model equations, but now depended on  $s$  and  $c$  as well as  $p$ :

1029  $d = \frac{s \cdot D^{1/p}}{s + c \cdot (1 - D^{1/p})}$  (13),

1030 where  $c$  is the fixed test contrast at which the unadapted data were collected.

1031  
 1032 \*\*\* Fig. A1 about here \*\*\*  
 1033

1034 *Orientation/direction tuning*

1035 The tuning curve  $S(\theta)$  was derived by first fitting a function  $r(\theta)$  to the population-mean  
 1036 (unadapted) DE response data, and then normalizing it. The fitted function  $r(\theta)$  (Fig. A1,  
 1037 blue curve) had a Gaussian central lobe in the preferred direction ( $\theta=0$ ), and a similar lobe  
 1038 of lower amplitude in the non-preferred direction (Carandini & Ferster, 2000; Gardner,  
 1039 Anzai, Ohzawa, & Freeman, 1999). It was defined over the range  $-180 \leq \theta \leq 180$  as:

1040  $r(\theta) = r_0 \cdot \left\{ \alpha + \exp\left(\frac{-\theta^2}{2\sigma^2}\right) + \beta \cdot \exp\left(\frac{-(\theta - 180)^2}{2\sigma^2}\right) \right.$   
 1041  $\left. + \beta \cdot \exp\left(\frac{-(\theta + 180)^2}{2\sigma^2}\right) \right\}$  (14)

1042 where  $\theta$  is the orientation/direction of the moving test stimulus in degrees,  $\alpha$  was a small  
1043 upward offset of the whole tuning curve,  $\sigma$  represents orientation bandwidth,  $\beta$  describes  
1044 direction selectivity as the relative amplitude of response in the non-preferred direction  
1045 ( $\theta=\pm 180^\circ$ ) and  $r_0$  is a scale factor. There was a very close fit ( $R^2 = 0.999$ ) of this  
1046 descriptive function to the unadapted DE response (Fig. A1, circles, average of all 54  
1047 cells). Best-fitting tuning parameters were:  $\alpha = 0.145$ ,  $\beta = 0.501$ ,  $\sigma = 23.0^\circ$  and  $r_0 = 31.5$   
1048 spikes/s, corresponding to a peak rate  $r_{max}$  of 36.1 spikes/s. These values were used to  
1049 create the tuning curve  $S(\theta)$  for the model, normalized to a peak of 1:

$$S(\theta) = \{r(\theta)/r_{max}\}^{1/p}$$

1051 where the power ( $1/p$ ) compensates for the fact that the spike-rate data to which  $r(\theta)$  is  
1052 fitted are observed *after* the output nonlinearity, while the tuning curve  $S$  in the model lies  
1053 before it. It is now well-established that the selectivity of cortical neurons is sharpened by  
1054 an expansive output nonlinearity (Albrecht & Geisler, 1991; Anderson, 2000; Carandini  
1055 & Ferster, 2000; Heeger, 1992; Miller & Troyer, 2002). Because the exponent  $p$  is greater  
1056 than 1,  $S$  is broader and with a higher baseline than the observed response tuning  $r$ , as Fig.  
1057 A1 shows (cf. Finn, Priebe, & Ferster, 2007, their Fig. 1).

1058  
1059 Tuning parameters for population-mean unadapted NE responses ( $\alpha = 0.181$ ,  $\beta = 0.512$ ,  $\sigma$   
1060  $= 24.6^\circ$ ) were very similar to those for DE given above, and the tuning parameters  $\alpha$ ,  $\beta$ ,  $\sigma$   
1061 varied very little with adapting condition, most likely because the adapter was always at  
1062 the preferred orientation of the cell. Adapting to non-preferred orientations is known to  
1063 have complex effects on both orientation tuning and bandwidth (Crowder et al., 2006;  
1064 Dragoi, Sharma, & Sur, 2000; Patterson et al., 2013) outside the scope of the present  
1065 work.

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1070 **References**

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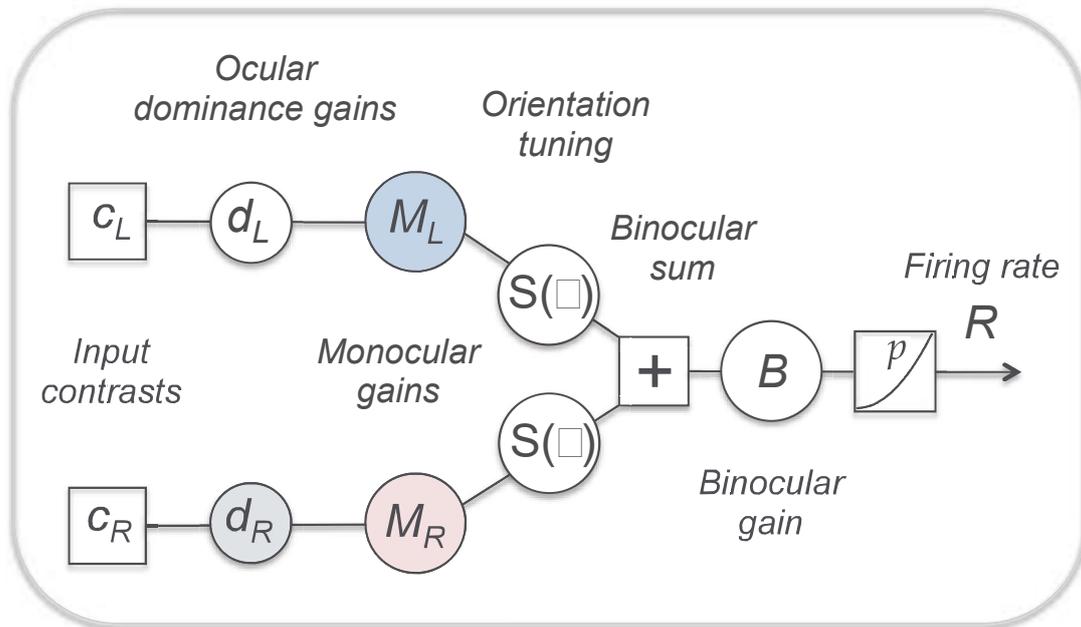
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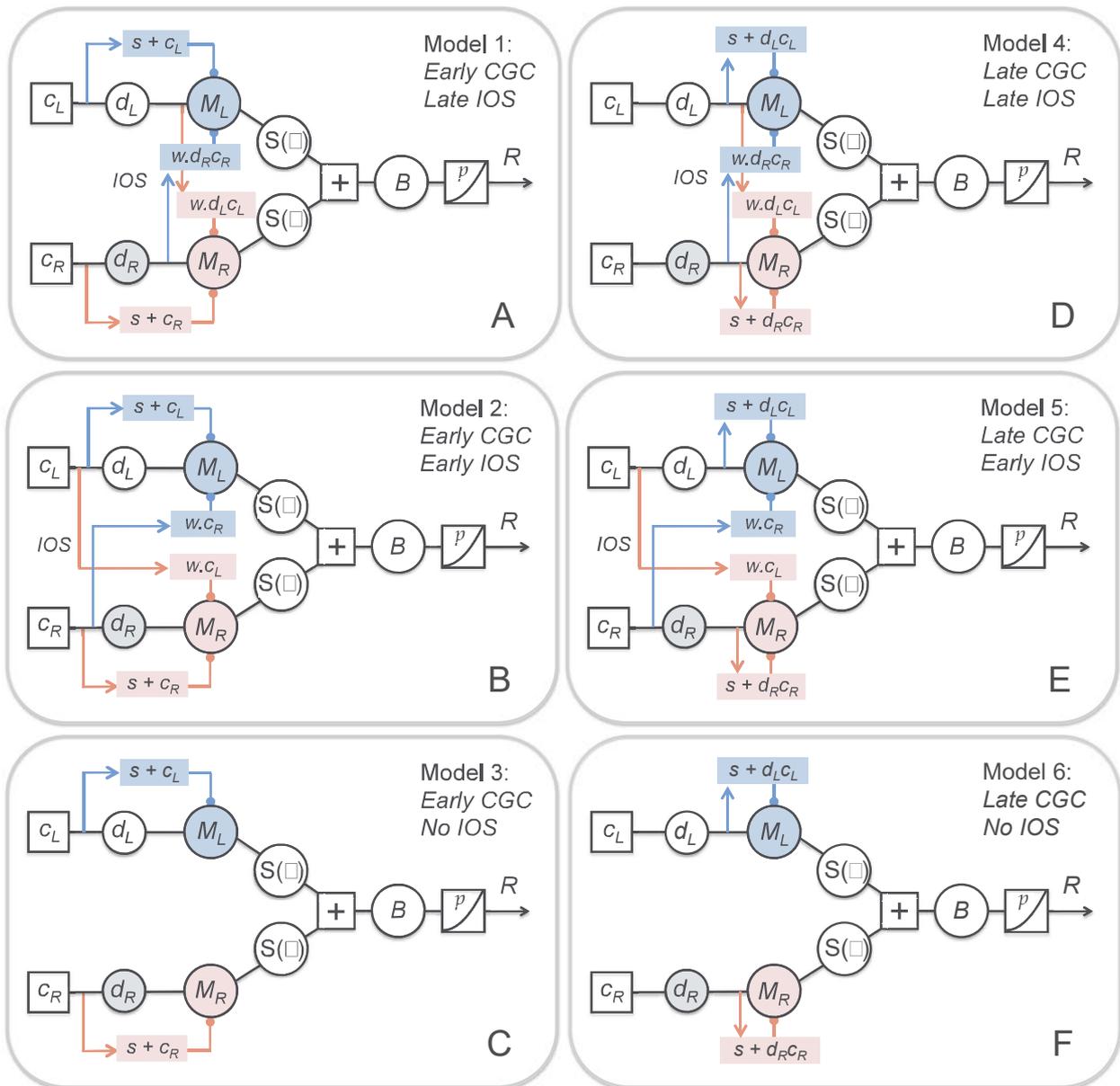


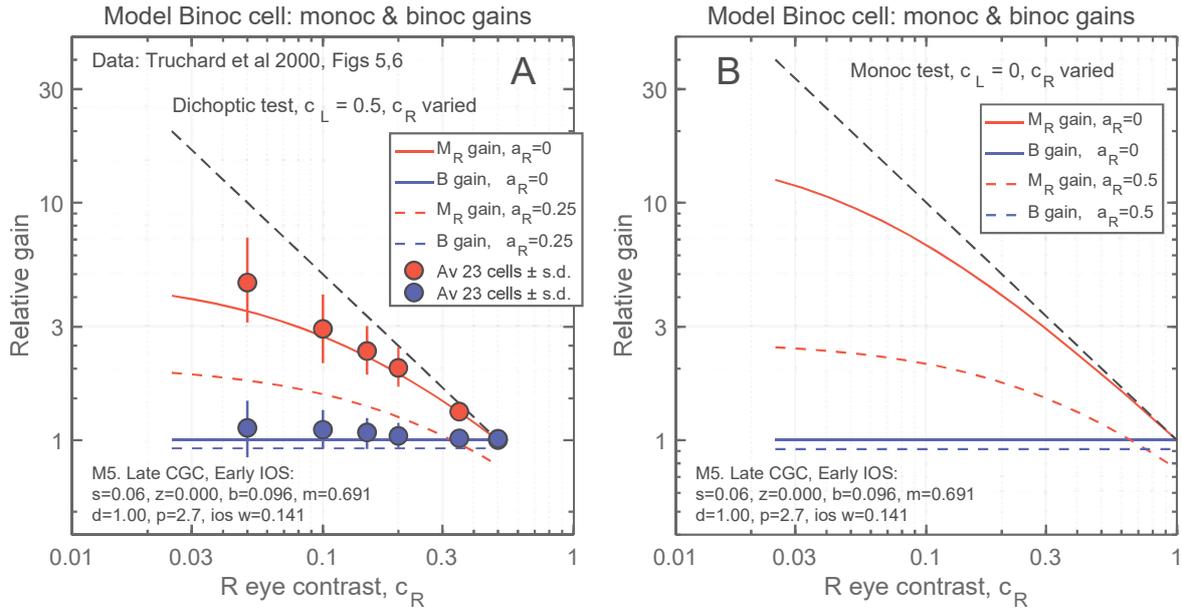
## Schematic model for V1 binocular cell



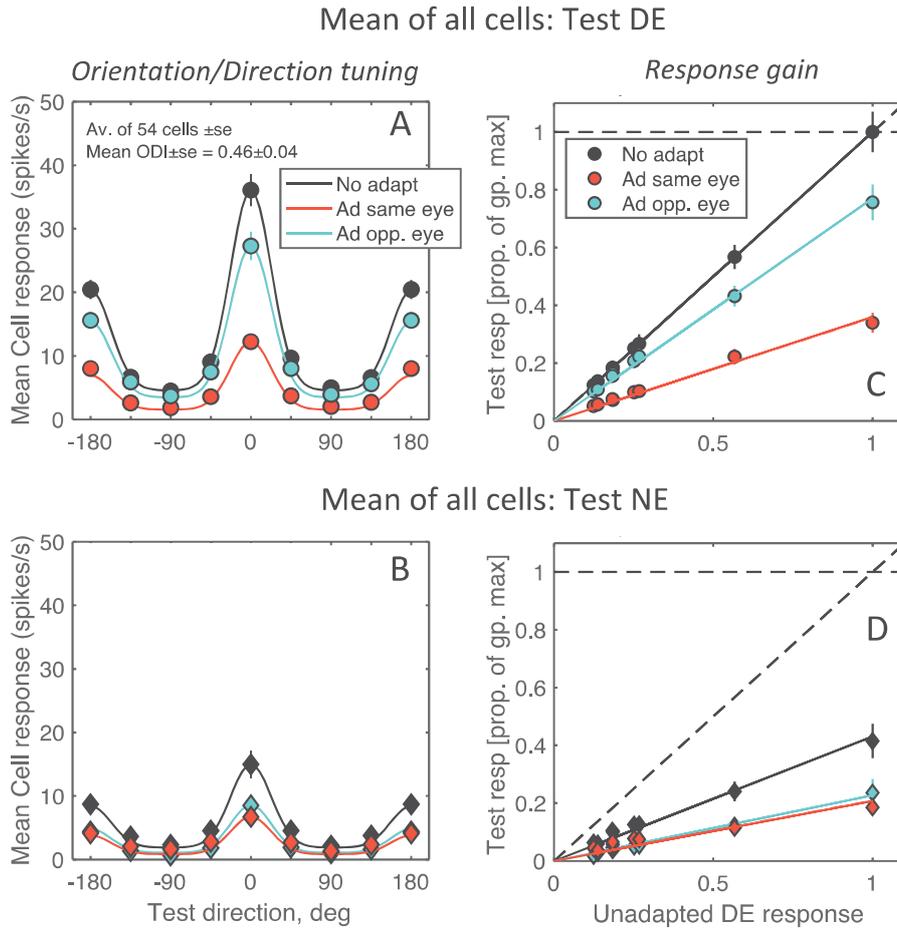
$$R = R_{max} \cdot \{(c_L d_L M_L + c_R d_R M_R) \cdot S(\square) \cdot B\}^p$$

**Figure 1.** Schematic model of functional architecture for a binocular cell in V1 (elaborated from Fig. 1B of Truchard *et al*, 2000). We aim here to account for the responses of cat cortical cells to sinewave gratings shown to one eye, with or without prior adaptation to a grating in the same eye or the opposite eye. Monocular and binocular gains ( $M, B$ ) vary with exposure to the current contrast and any prior (adapting) contrast, while the orientation/direction tuning curve  $S$  and ocular dominance gain factors  $d_L, d_R$  are taken to be fixed ( $0 \leq d_R \leq d_L$ ). The ratio  $d = d_R/d_L$  varies with the degree of ocular dominance (OD) exhibited by the cell. A purely monocular cell would have  $d=0$ ; a perfectly balanced binocular cell would have  $d=1$ . Output values are raised to power  $p$  in the conversion to firing rate  $R$ .

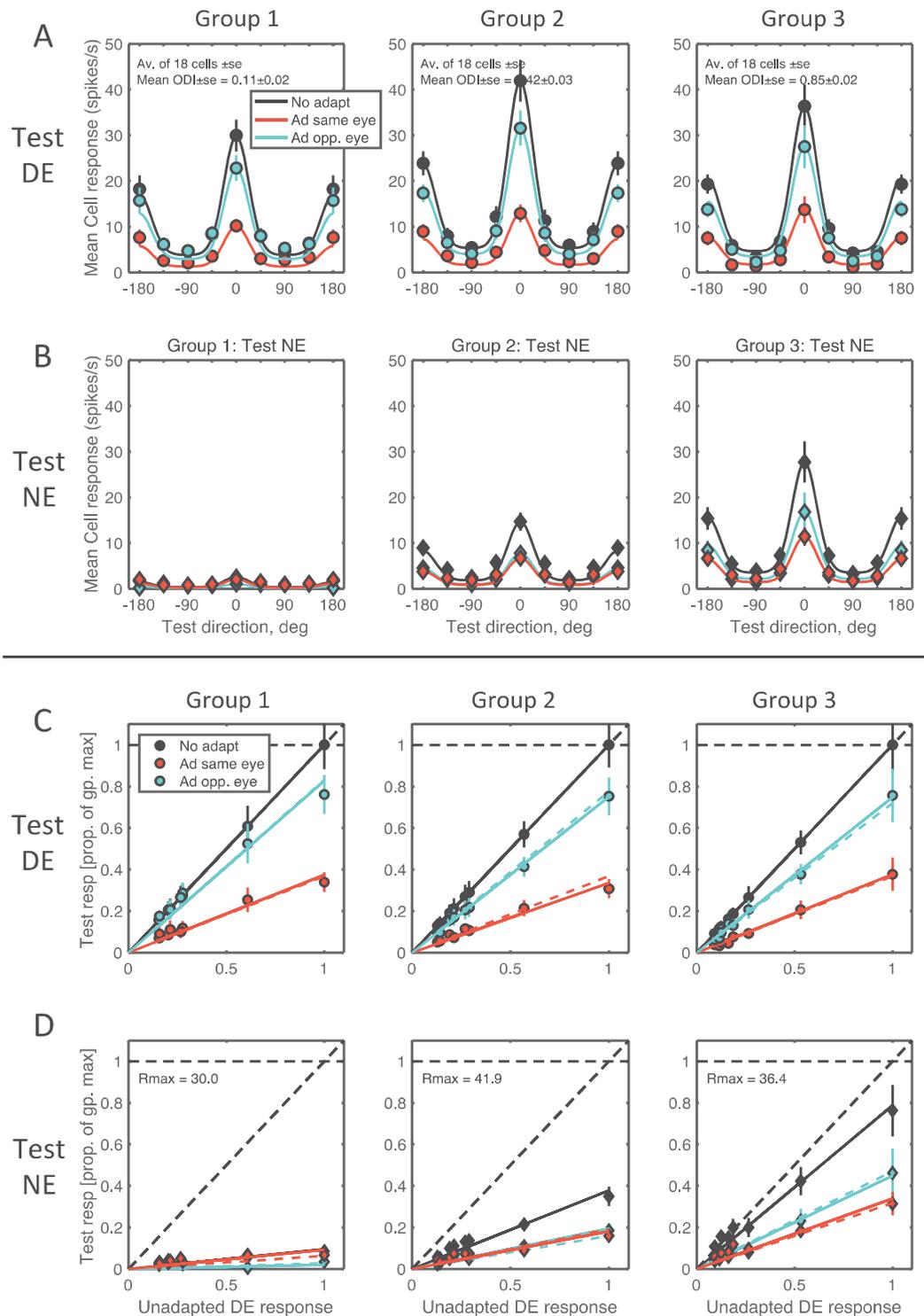




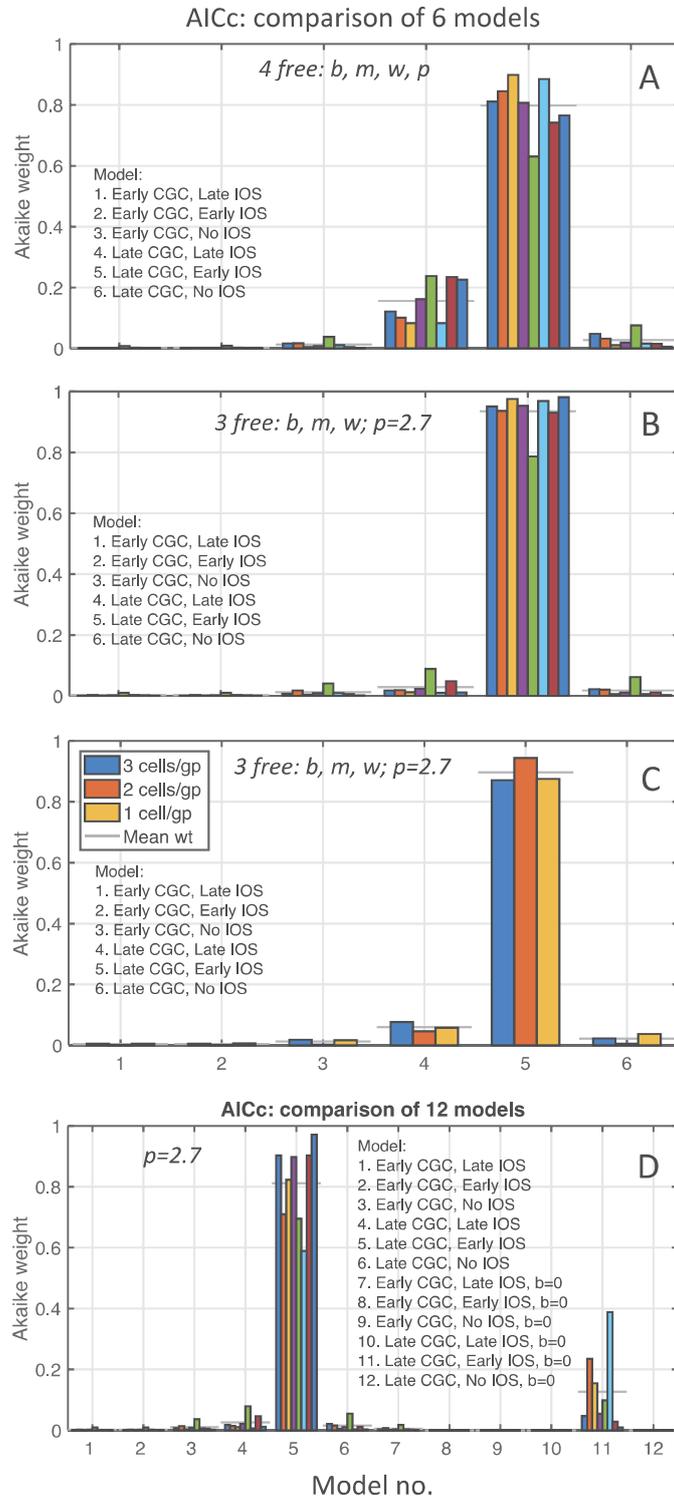
**Figure 3.** Contrast gain data used to constrain model parameters  $s$ ,  $z$ . **A:** Symbols show monocular gains ( $M$ , red) and binocular gains ( $B$ , blue) derived from cat V1 cell responses by Truchard *et al* (2000) at each level of R eye contrast when L eye contrast was fixed at 0.5. Highly significant increase of monocular contrast gain (red), but not binocular gain (blue), occurred with decreasing test contrast in one eye. Gains expressed relative to the gain at 0.5 contrast; mean  $\pm$  s.d. over 23 cells. Solid curves are model gain values for one example model (M5) from eqns. 7.5, 8.5, with  $s=0.06$  chosen to fit the observed  $M$  gains,  $z=0$  for the  $B$  gains and with ocular balance ( $d=1$ ). Agreement between model gains and cat gains was good. All four models with IOS (M1,2,4,5) gave gains similar to those shown, while in the absence of IOS (M3, M6) monocular gains were a bit higher (not shown). Increasing IOS or increasing ocular imbalance ( $d<1$ ) led to lower monocular gains (not shown). **B:** Model gain values for monocular (R eye) testing, rather than dichoptic. Gains expressed relative to the gain at 1.0 test contrast, as used in the Howarth *et al* (2009) experiments. **A,B:** Dashed lines show predicted impact on gain values of prior adaptation to contrast ( $a_R$ ). Note that model monocular gains were strongly influenced by test contrast and prior adaptation to contrast, while binocular gains were only slightly altered, if at all.



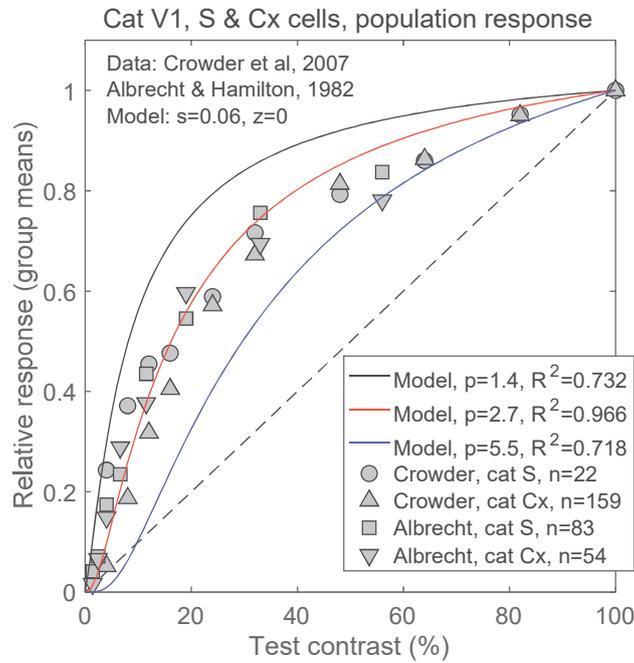
**Figure 4.** Data overview: mean responses across all 54 cells, when testing the DE (top row) and the NE (lower row). Colours represent unadapted control condition (black); adapting the same eye as the tested eye (red); adapting the opposite eye (cyan). **A,B:** Mean cell responses as a function of test orientation/direction. To describe these data we fitted a curve (see Appendix, Eqn. 14) to the unadapted DE responses (black circles in A), then used this as a template to fit the other 5 conditions simply by scaling the template amplitude to match the peak response in each case. The template shape fitted these mean data strikingly well for both DE and NE, in all three adapting conditions. This implies that, on average, both adaptation and ocular dominance scaled responses down by a common factor across all test orientation/directions. We refer to this scaling factor as the *response gain*,  $G$ , and computed it as follows. **C,D:** Data re-plotted from A,B show group mean spike rates normalized to a max. of 1 (i.e. dividing all data by the peak unadapted firing rate seen in panel A). Lines are best-fitting regression lines passing through the origin. Different data points along each line arise from the 8 different test directions (the highest being  $\theta=0$ , next highest  $\theta=180$ , and so on). Slopes of these lines are our best estimate of population response gain for a given condition, expressed relative to the unadapted DE gain (panel C, black line).



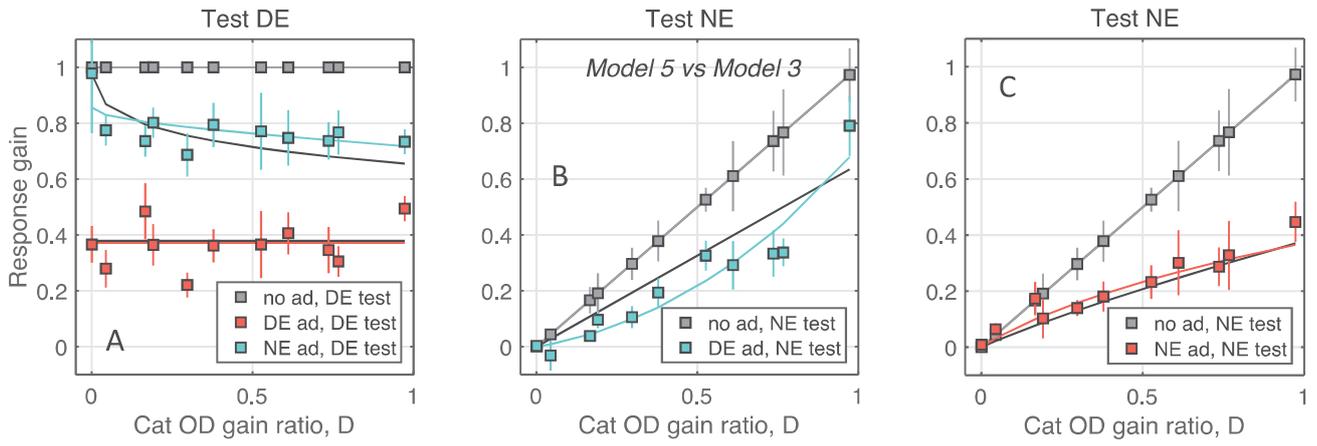
**Figure 5.** Response tuning curves and derivation of response gains for three cell groups that divided the population according to ODI. Group 1 contains the most ocularly unbalanced cells (mean ODI  $\pm$  s.e. =  $0.11 \pm 0.02$ ;  $n=18$ ); group 3 is the most balanced (mean ODI  $\pm$  s.e. =  $0.85 \pm 0.02$ ;  $n=18$ ). **A,B:** Mean responses to test grating as a function of test orientation/direction, for the three OD groupings. **A:** test DE; **B:** test NE. Curves show that the same descriptive template (Fig. A1, blue) was a fairly close fit to all the tuning curves for each OD group. Black: no adaptation; red: adapting the tested eye; cyan: adapting the other eye. **C,D:** Response gains for the three OD groups. Data from A,B re-plotted in the same format as Fig. 4C,D. Symbols: group-average cell responses. Solid lines: best-fitting linear regression passing through the origin. The control (unadapted DE) condition has a gain (slope) of 1 by definition (black, row C) and all other conditions had gains  $< 1$ . Observed response gains  $G$  defined in this way can be compared with gains predicted by the fitted model(s) (e.g. dashed lines: model 5).



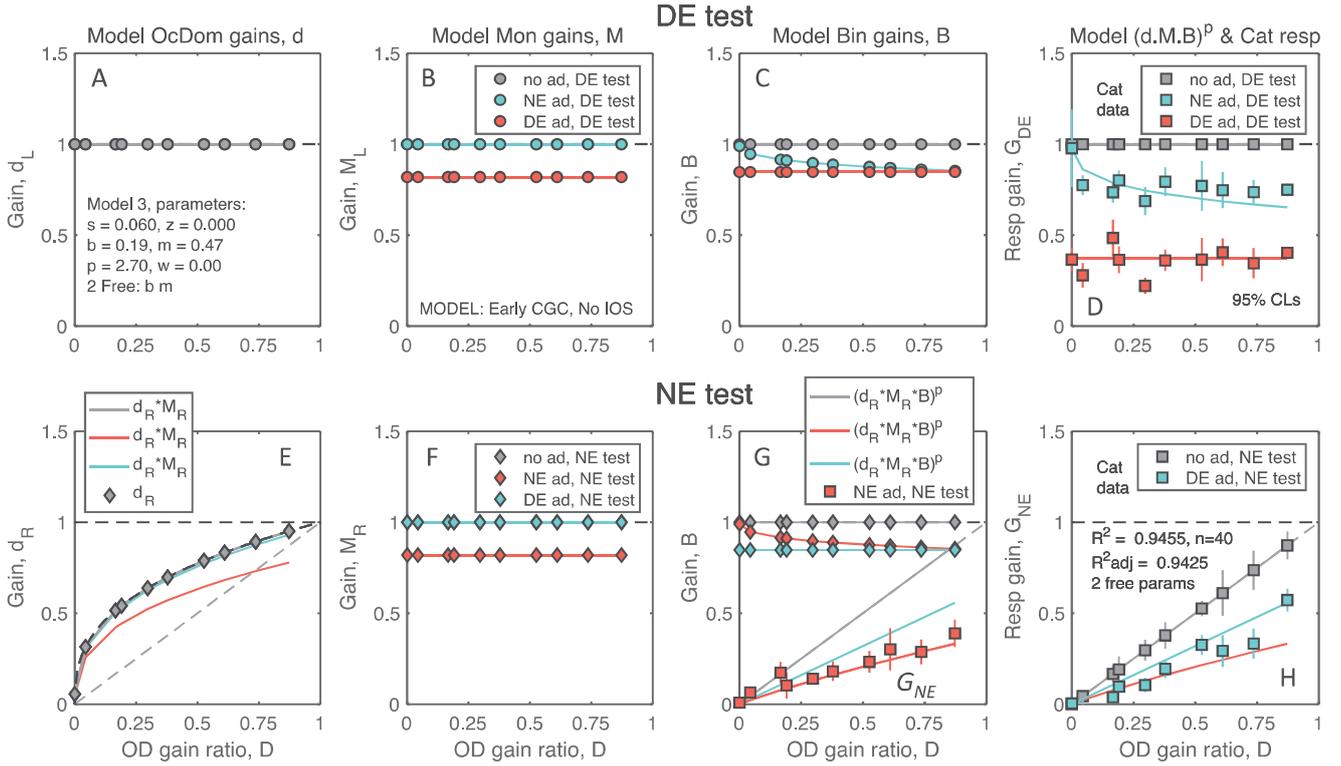
**Figure 6.** Relative strength of evidence favouring each model (Fig. 2) is compared using the Akaike Information Criterion (AIC), with a standard correction for small sample sizes (AICc). The model with the highest Akaike weight can be interpreted as most likely to be the 'best' model, given the present data (Burnham & Anderson, 2002). Of those models considered in the AIC analysis, the best model is the one closest in information-theoretic terms to an (unknown) true model. **A, B:** Barchart colours represent the number  $N$  of OD groups into which the cells were divided, ranging from 3 to 10 (left to right respectively). Horizontal grey bars represent mean Akaike weights over these 8 groupings. **A:** with 4 free parameters, the Akaike weights favoured model 5 (late CGC, early IOS), but also lent some support to model 4 (late CGC, late IOS). **B:** When we used two other studies to constrain the output exponent  $p$  to a fixed value ( $p = 2.7$ ; Fig. 7), then model 5 was uniquely favoured (mean weight = 0.935) while support for model 4 evaporated (mean weight = 0.029). **C:** Model 5 remained the strongly favoured model even with little or no averaging across cells (ie. with 54, 27 or 18 groups, hence 1, 2 or 3 cells per group). **D:** Comparison of 12 models, comprising models 1-6, and models 7-12 which were the same as 1-6, except binocular adaptation parameter  $b=0$ . Model 5 remained the strongly favoured model, with slight support for model 11 (ie. model 5 with  $b=0$ ).



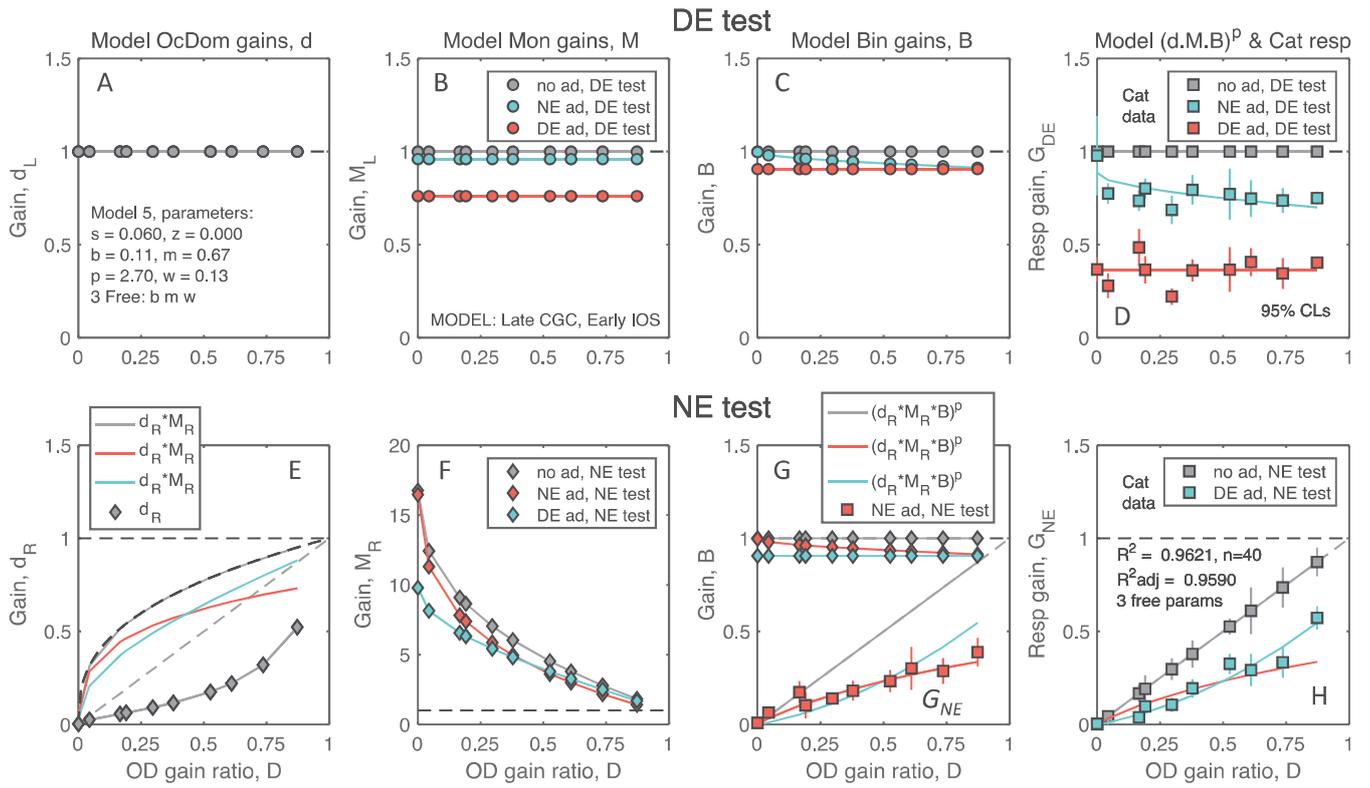
**Figure 7.** Monocular contrast-responses of cat cortical cell populations. Symbols are mean responses of cat V1 cells, either simple (S) or complex (Cx) from two previous studies; number of neurons in each cell group is shown in legend. Response values were normalized to 1 at 100% contrast. Curves show the DE response of our gain-control model as a function of contrast; all 6 variants were equivalent for this purpose. As expected, the model contrast-response curves varied with output exponent  $p$ . Given a fixed value for  $s$  ( $s=0.06$ ) the best-fitting value of  $p$  (highest  $R^2=0.97$ ) was  $p=2.7$  (red curve). Response curves for two sub-optimal values of  $p$  are also shown. Fig. 6B showed that constraining all 6 models to have a plausible contrast response ( $p=2.7$ ) increased the weight of evidence in favour of model 5 and against model 4.



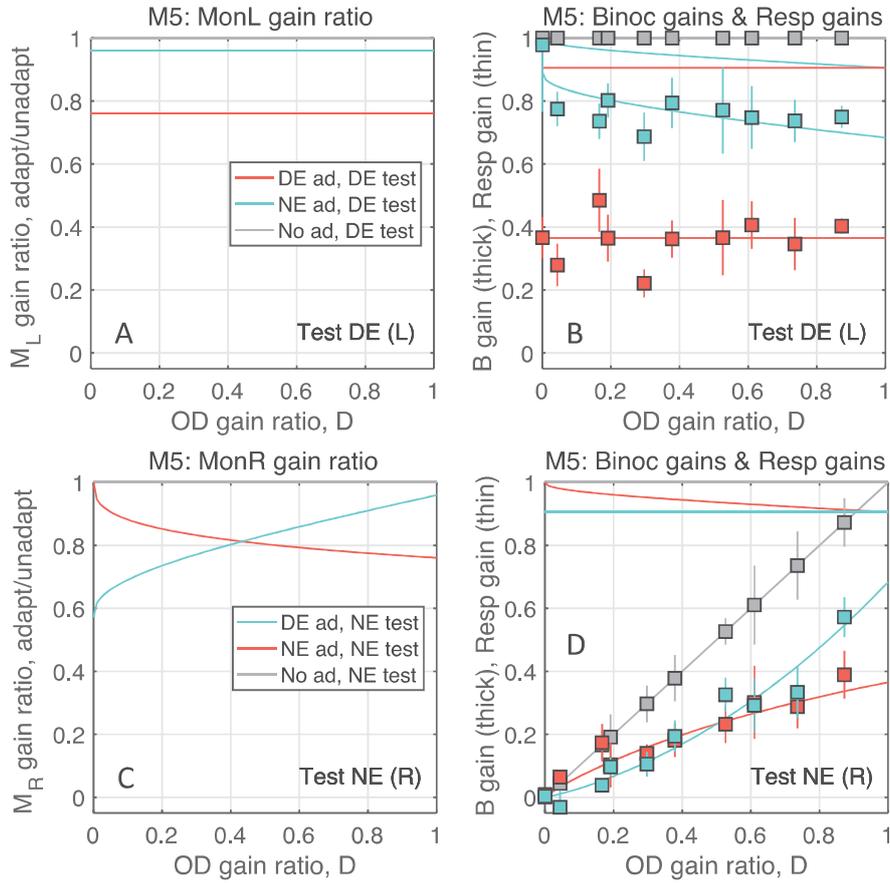
**Figure 8.** Cat response gains (symbols) and best-fitting model gains (curves) compared for  $N=11$  OD groups, chosen here to cover the full range of  $D$ . Each panel compares two models: the best-fitting one (M5, coloured curves) and the simplest one (M3, black curves). [Models 1,2,3 gave essentially identical results]. The  $x$ -axis identifies the cell groups by their OD gain ratio ( $D$ ) – the ratio of unadapted response gains (NE/DE) for each cell group.  $D = 1$  implies perfect ocular balance; lower values imply greater imbalance. There were three adapting conditions: blank adaptation (grey), adapt & test the same eye (red), adapt & test opposite eyes (cyan). Error bars are 95% confidence limits on the cat response gain values. **A:** Test DE, adapt DE or NE; **B:** adapt DE, test NE; **C:** adapt NE, test NE. Same-eye conditions (red) did not distinguish the models, but the interocular conditions (cyan) revealed that M5 fits more closely than M3, and captures better the trends of adapted gain across OD. Parameter values here for M5 were  $s=0.06$ ,  $z=0$ ,  $b=0.075$ ,  $m=0.74$ ,  $p=2.7$ ,  $w=0.16$ ,  $R^2=0.95$ ,  $n \text{ data}=44$ . Only 3 parameters were adjusted to fit this model ( $b, m, w$ ; same for all cell groups and stimulus conditions). See supplementary Fig. S8 for similar comparisons between M5 and M4, or M5 and M6.



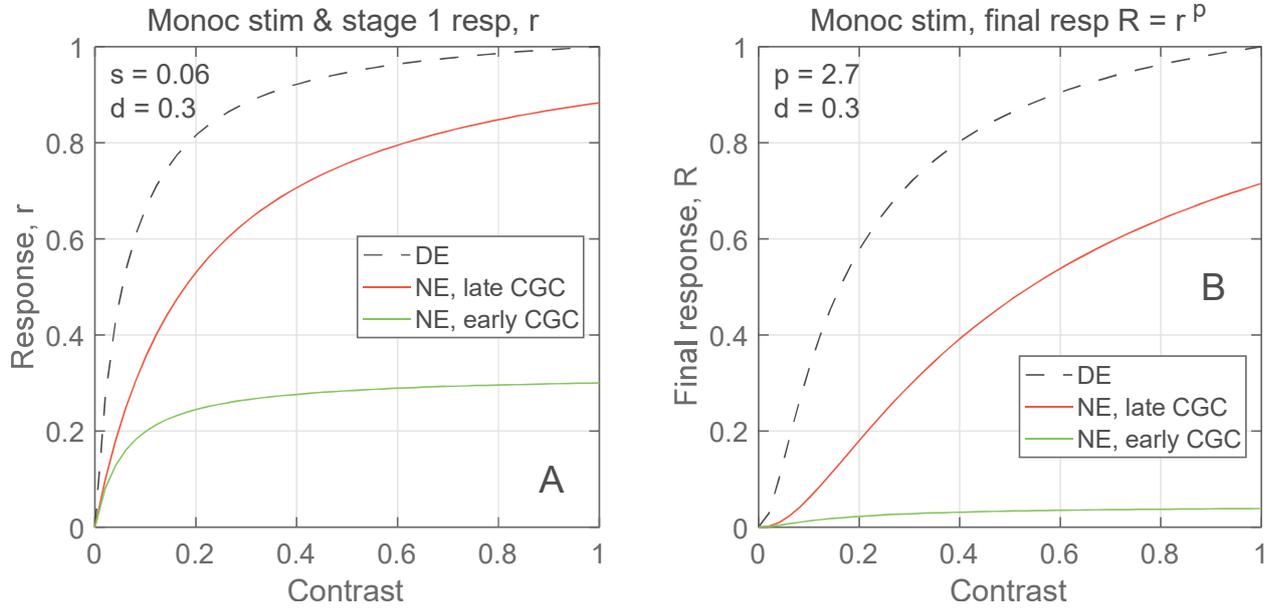
**Figure 9.** Sources of adaptation and OD in the simplest model, M3: Early CGC, no IOS, applied to  $N=10$  cell groups. **First 3 columns:** attenuation of cell responses after adaptation arises from a sequence of three sources: (i) attenuation ( $d_R$ ) of the non-dominant input, (ii) reduction of gain ( $M_L, M_R$ ) at the monocular stage, and (iii) variation of gain ( $B$ ) at the binocular stage. Subscripts  $L, R$  refer to the *tested* eye and for simplicity of presentation we presumed  $L = DE, R = NE$ . Responses in the model were scaled so that the gains  $d_L, M_L, B$  had a value of 1 for the DE in the no-adapt condition (top row, grey; hidden behind cyan in B,F). Effects of adaptation and ocular dominance are seen where the  $d, M$ , or  $B$  terms deviate from 1. **Fourth column:** model response gains are the product of the three gain factors ( $d.M.B$ , subscripted by  $L$  or  $R$  as appropriate; eqn. 6) raised to the power  $p$ . Solid lines plot this prediction ( $d.M.B$ ) <sup>$p$</sup> , when testing the DE (top row) or NE (lower row), to be compared with the physiological results (filled squares) expressed as cell response gains for each OD group and adapting condition. Error bars are 95% confidence limits on cat response gains. Least-squares model fitting and  $R^2$  goodness-of-fit (inset) were computed only over the adapted conditions. To avoid confusing overlap, cat gain data for DE adapt, NE test (red squares) are plotted in the lower part of panel G (instead of panel H), along with copies of the model curves. Best-fitting parameter values are in panel A.



**Figure 10.** Sources of adaptation and OD in the best-fitting model, M5: Late CGC, Early IOS with  $N=10$  OD groups. Figure layout is the same as Fig. 9. With late CGC, monocular gain  $M_R$  increases greatly (panel F) when the suppressive signal is attenuated by OD (at low values of  $D$ ). This loss of suppression and rise in gain does not occur with early CGC (Fig. 9F), where the divisive suppression is independent of OD. The effective monocular gain for each adapting condition is given by the product  $d_R M_R$  (upper curves in panel E), and this allows easier comparison between early and late CGC models (Fig. 9E vs Fig. 10E). Compared with M3, the best-fitting model M5 shows greater adaptation at the monocular level (panel B) and less at the binocular level (panel C).

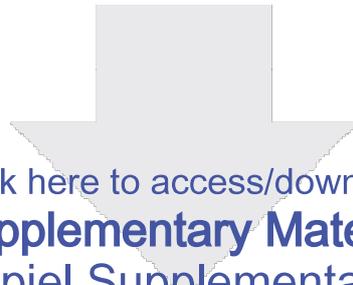


**Figure 11.** A closer look at the  $M$  and  $B$  gain changes for model 5 (late CGC, early IOS), as a function of the OD gain ratio  $D$ . **A,C:** ratio of adapted model gain to unadapted. Baseline value of 1 means no adaptation effect; lower values imply increasing loss of gain after adaptation. **B,D:**  $B$  gain (thick curves) or final output response gain (thin curves). Symbols: cat data. Upper legend applies to A, B (test DE); lower legend applies to C, D (test NE). **A:** Gains  $M_L$  for the dominant eye (DE) are reduced uniformly after DE adaptation (red line) because DE's input to the CGC is unaffected by OD. After adaptation of the other eye (NE) the model gains  $M_L$  are more modestly reduced – by IOS – and these are also uniform (cyan line) because, with *early* IOS, the suppressive signal escapes the substantial attenuation  $d_R$  that comes with late IOS. **B:** Gains  $B$  after DE adaptation are also uniformly reduced (thick red line) for the same reason as in A. The net result is a large, uniform drop in response gain  $[(d_L M_L B)^p$ , thin red line] after DE adaptation. But after NE adaptation,  $B$  gains fall smoothly with increasing  $D$  (thick cyan curve) because NE's input to the  $B$ -gain suppressive mechanism increases with  $D$ . The result is a much smaller overall loss of gain after NE adaptation (thin cyan curve) that shows a shallow decline with increasing  $D$ . **C:** Gain changes for the non-dominant eye (test NE) show a *crossover* effect that is characteristic of model M5 (and M4), but not M1,2,3,6. The effect of adaptation on  $M$  gain either increases with  $D$ , or decreases, depending on which eye is adapted. **D:** The crossover effect carries through to the final response gain (thin curves), and is seen fairly clearly in the data. See also Fig S7, lower row.



**Figure 12.** Late CGC (red) compensates for input attenuation by ocular dominance, but early CGC (green) does not. **A:** Model contrast responses  $r$  at the monocular processing stage, for monocular stimuli to the dominant eye (DE, dashed curve), or the non-dominant eye (NE) with early CGC (green) or late CGC (red). Equations for these curves are:  $r_{DE} = k \cdot c / (s + c)$ ,  $r_{NE} = k \cdot d \cdot c / (s + c)$  [early CGC],  $r_{NE} = k \cdot d \cdot c / (s + d \cdot c)$  [late CGC], where  $k = 1 + s$  and  $c$  is stimulus contrast. **B:** final 'spike-rate' responses  $R$  to the same monocular stimuli. Attenuation due to OD (green) is exaggerated by the output nonlinearity ( $p$ ), but the relative compensation afforded by late CGC (red) is even greater than at the earlier stage (A).

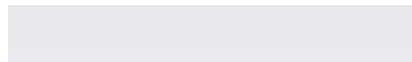
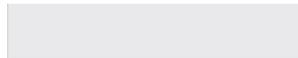




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**Supplementary Material**

Georgeson & Sengpiel Supplementary material R1.docx



## Credit Authorship Statement

**Mark Georgeson:** Conceptualization, Methodology, Software, Formal analysis, Writing - Original Draft, Writing - Review & Editing, Visualization, Funding acquisition.

**Frank Sengpiel:** Conceptualization, Methodology, Investigation, Resources, Writing - Review & Editing, Funding acquisition.