Haphazard Wiring of Simple Receptive Fields and Orientation Columns in Visual Cortex

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Ringach, Dario L. Haphazard wiring of simple receptive fields and orientation columns in visual cortex. J Neurophysiol 92: 468–476, 2004. First published March 3, 2004; 10.1152/jn.01202.2003. The receptive fields of simple cells in visual cortex are composed of elongated ON and OFF subregions. This spatial arrangement is widely thought to be responsible for the generation of orientation selectivity. Neurons with similar orientation preferences cluster in “columns” that tile the cortical surface and form a map of orientation selectivity. It has been proposed that simple cell receptive fields are constructed by the selective pooling of geniculate receptive fields aligned in space. A recent analysis of monosynaptic connections between geniculate and cortical neurons appears to reveal the existence of “wiring rules” that are in accordance with the classical model. The precise origin of the orientation map is unknown, but both genetic and activity-dependent processes are thought to contribute. Here, we put forward the hypothesis that statistical sampling from the retinal ganglion cell mosaic may contribute to the generation of simple cells and provide a blueprint for the selective pooling of geniculate receptive fields aligned in space. A recent analysis of monosynaptic connections between geniculate and cortical neurons via cross-correlation analysis (Alonso et al. 2001; Reid and Alonso 1995). These findings indicated that the probability of a monosynaptic connection was greatest when the geniculate receptive field overlapped a strong simple cell subregion of the same signature (either ON or OFF), while the probability of “inappropriate” connections between receptive fields of opposite signature was much lower. These data have been interpreted as supporting the existence of precise rules of synaptic connectivity in accordance with the classic wiring scheme for simple cells.

This conclusion is warranted if, at any one point in visual space, the cortex receives afferents from a large number of ON and OFF geniculate neurons with spatially overlapping receptive fields. In this situation, random isotropic sampling of the afferents would result in largely overlapping ON and OFF inputs and round (nonoriented) receptive fields. However, the X retinal ganglion cell (X-RGC) mosaic in the cat has a relatively low coverage factor of \( \approx 3 \) for both ON- and OFF-center cells (Wässle et al. 1981) (the coverage factor represents the number of receptive fields that cover 1 point in visual space). It is known that there are about 2.5 times more cells in the lateral geniculate nucleus (LGN) than RGCs (Peters and Payne 1993), but the majority of LGN cells receive dominant input from only one RGC, and their receptive field centers are very similar (Cleland and Lee 1985; Cleland et al. 1971; Usrey et al. 1999). Thus the RGC mosaic effectively represents the pattern of visual input to primary visual cortex (Hubel and Wiesel 1961). Following the work of Soodak (1987), we reasoned that, with relatively low coverage factors, simple receptive fields may simply arise by the random sampling of a small area of the RGC mosaic. In this scenario, an ON subregion of a simple cell results in the event that more ON-center, rather than OFF-center, geniculate neurons connect to that location. The elongation of the subregion results from the chance alignment of receptive fields with the same signature or by the sampling of a pair of ON and OFF receptive fields at slightly displaced locations, which would generate a profile well approximated by the directional derivative of a two-dimensional Gaussian. Intuitively, this model may explain the probabilities of monosynaptic connection between the LGN and the cortex, because we expect a tendency for geniculate ON-center cells to connect to an overlapping ON subregion in a cortical simple cell. This is,
After all, how the ON subregion arises in the first place. Furthermore, if one accepts that the elongation of simple cell subregions may originate in local anisotropies of the RGC mosaic, it is conceivable that other neurons pooling signals from the same area will have similar orientation preferences. Thus the model may be able to predict the emergence of orientation columns as well. Our goal in this study was to assess the haphazard pooling model as an alternative hypothesis for the origin of simple cells, orientation selectivity, and orientation columns in visual cortex.

**Methods**

**General model structure**

To test if simple cells and orientation maps could emerge by statistical sampling of LGN afferents, we constructed a simplified wiring model from the retina to layer IV in the cat based on published anatomical and physiological data (Fig. 1). We provide a broad description of the model first and discuss the details next. The first layer of the model consists of two independent (noisy) hexagonal lattices of ON and OFF X-RGCs (Fig. 1, top). Receptive field centers are modeled as two-dimensional (isotropic) Gaussians functions (Fig. 1, filled disks). In the second layer, representing the LGN, there are 2.5 times more cells than RGCs (Fig. 1, middle) (Peters and Payne 1993). Each LGN neuron receives input from only one RGC, and topography is maintained (Cleland and Lee 1985; Cleland et al. 1971). This is a reasonable approximation because, even if some geniculate cells receive more than one afferent, the receptive fields of their inputs overlap almost entirely (Usrey et al. 1999). Given a location of a neuron in the cortex, we modeled the probability of connection from an LGN neuron to the cortical cell as a Gaussian function of the distance between their receptive field centers (Fig. 1, bottom) (Alonso et al. 2001). If a geniculate cell is connected to a cortical target, the synaptic strength is also assumed to be a Gaussian function of the distance between the receptive field centers (Alonso et al. 2001). The cortical receptive field linearly sums its inputs. This provides the linear component of the receptive field and forms the basis to replicate the analysis of Alonso et al. (2001), who measured the linear kernel by reverse correlation with M-sequences.

**Details of the model**

We now provide a detailed description of the model and the associated parameters. The first layer of the model is defined by two independent mosaics representing the centers of the ON- and OFF-center X-RGCs. The lattices are generated by adding two-dimensional Gaussian noise to the coordinates of a perfect hexagonal grid

\[ \mathbf{\tilde{x}} = \lambda \left[ \frac{1}{\sqrt{3}} \mathbf{j} - \frac{1}{\sqrt{3}} \mathbf{i} \right] + \mathbf{n} \]

Here, \( \mathbf{\tilde{x}} \) is a two-dimensional vector in the plane, \( 2\lambda \) is the distance from any point to the nearest neighbor in the hexagonal grid, \( \mathbf{n} \) is a two-dimensional vector drawn from a two-dimensional Gaussian distribution with SD \( \sigma_{\text{pos}} \), and \( i, j = 0, \pm 1, \pm 2, \ldots \). The noise is drawn independently for each point on the grid. In what follows, all the spatial scales in the model are defined as multiples of \( \lambda \). A value of \( \sigma_{\text{pos}} = 0.155\lambda \) was selected to match the experimental CV of the nearest neighbor distance distributions for the ON and OFF lattice, which turns out to approximate well the CV of the nearest neighbor distribution between cells irrespective of their signature (Wässle et al. 1981). The mean nearest neighbor distance within a cell class in our simulations is 0.758 ± 0.1433 (SD). This means the CV is 0.1883, which is nearly identical to 0.1889 for ON-β cells and 0.1882 for OFF-β cells (Wässle et al. 1981). The mean nearest neighbor distance between any two cells (either ON or OFF) in our simulations is 0.4 ± 0.16\( \lambda \), resulting in a CV of 0.4 compared with 0.37 in the data (Wässle et al. 1981).

A sample of a simulated RGC mosaic with ON- and OFF-center cells is shown in Fig. 2A (ON cells are shown in red, OFF cells in blue). The nearest neighbor distribution for the lattice is shown in Fig. 2B and for ease of comparison, we reproduce the original data of Wässle et al. (1981) in Fig. 2C. The top histogram in Fig. 2B shows the distribution of nearest neighbor distances between X cells independent of their signature (either ON or OFF). The histogram at the bottom of Fig. 2B shows the distribution of nearest neighbor distances between cells of the same signature. The simulation generates distributions that are similar to the empirical data, not only in terms of their CV but overall shape. It is worth noting that histogram at the top of Fig. 2B shows that the nearest neighbor of an ON-center cell type is expected to be an...
off cell (and vice versa). This is also evident by visual inspection of the sample lattice in Fig. 2A—it is as if ON and OFF cells come “in pairs” (Wässle et al. 1981). This is a direct consequence of the independence of the ON-OFF lattices. This result by itself suggest that pooling within a small area of the RGC mosaic may generate oriented receptive fields that look like the difference of two Gaussians displaced in space.

Next, we have to decide on the size of the RGC center. Wässle and co-workers have shown that the dendritic field radius is approximately the same as the mean nearest neighbor distance in the mosaic, which equals ~0.76A. However, the physiological receptive field center is slightly larger than the actual dendritic tree, with the amplitude decaying to about 30% of its peak at the boundary of the dendritic tree (Peichl and Wässle 1983). To match this value, we selected the center of the RGC to be a two-dimensional Gaussian function with SD equal to σon = 0.7 × λ. The schematic in Fig. 1 shows this relationship to scale.

The layer of LGN neurons was constructed by replicating the original RGC layer and adding 1.5 times more receptive fields. This was done by random sampling (with replacement) to result in a total of 2.5 times the number of original RGCs. This assumes that each LGN neuron receives dominant input from only one retinal afferent (Cleland and Lee 1985; Cleland et al. 1971).

The random pooling mechanism by a cortical cell is modeled as follows. We assume that the probability of connection between a geniculate receptive field centered at x with a cortical cell centered at y is given by a Gaussian function of their relative distance

\[ p = p_{\text{max}} \exp \left( -\frac{||x - y||^2}{2\sigma_{\text{conn}}^2} \right) \]

and independent of other geniculate cells. It is known from physiological measurements that if the dominant subregion of a simple cell and a geniculate receptive field overlap strongly and they have the same signature, their probability of connection is 0.85 (Reid and Alonso 1995). Thus we selected \( p_{\text{max}} = 0.85 \). We based our selection of \( \sigma_{\text{conn}} \) on the fact that the probability of connection begins to drop when distances get to be near the width of a subregion widths (Fig. 4 in Alonso et al. 2001) and selected \( \sigma_{\text{conn}} = 0.97 \times \sigma_{\text{on}} \) under the assumption that our subfield widths would be on the same order of magnitude as the LGN centers, which is confirmed by our simulations (Fig. 4F).

Finally, we had to select the strength of the synapse when a geniculate cell is connected to a cortical neuron. Synaptic efficacy is also known to drop as a function of the distance between the centers of the LGN and cortical receptive field subregion (Fig. 4 in Alonso et al. 2001). We assume that synaptic strength between a geniculate receptive field centered at \( x \) and a cortical cell centered at \( y \) is given by a Gaussian function of their relative distance

\[ s = s_{\text{max}} \exp \left( -\frac{||x - y||^2}{2\sigma_{\text{syn}}^2} \right) \]

The value of \( s_{\text{max}} \), which represents the maximum synaptic strength, is irrelevant in our model, because we do not include a cortical contribution to compare this value to. We chose \( s_{\text{max}} = 1 \). Based on the scatterplots in Fig. 4A of Alonso et al. (2001), we selected \( \sigma_{\text{syn}} = 1.1 \times \sigma_{\text{on}} \). The “synaptic efficacy” of one connection in the model was calculated by taking its strength and dividing by the sum of all the synaptic weights to the cortical neuron. The linear receptive field of the cortical cell is calculated as the weighted sum of the afferent receptive fields, with ON receptive fields being positive and OFF receptive fields being negative.

**Limitations of the model**

The model represents a rather simplified view of thalamocortical connectivity. First, we only model input from the X-RGCs, while it is clear that many layer IV simple cells also receive Y input (Ferster and LeVay 1978). We also assume the receptive fields of the RGCs are isotropic Gaussians, when in fact, they are slightly elongated (Hammond 1974). Finally, the model is “static” and does not include full spatio-temporal receptive fields. However, there is no reason to believe that a straightforward extension of the model to space and time would fail, as the basic principles will remain the same and one would expect a correlation between connectivity and the similarity of the entire spatio-temporal receptive fields.

**RESULTS**

Results from the simulations show that the low coverage ratio of X cells, the independence of the ON and OFF RGC mosaics, and random sampling of the LGN afferents can generate spatial kernels that resemble simple cell receptive fields (Fig. 3). Superimposed on the kernels, we indicate the location of inputs from ON (○) and OFF (△) geniculate receptive fields that are connected to the simulated cortical cell (unconnected geniculate cells are not shown). The size of the symbols is proportional to the synaptic strength of their connections. In some cases, one observes receptive fields with only one subregion: either ON or OFF (Fig. 3, A and B) (Kato et al. 1978). In other cases, the random statistics of ON and OFF inputs generate two (Fig. 3, C–G), and sometimes even three (Fig. 3H), elongated subregions. It can be seen that the effective number of RGC centers (with high synaptic strength) that generate a simple cell receptive field can be small.
To make a quantitative comparison with the empirical data, we first calculated the distribution of cell pairs and their relative probability of monosynaptic connection with respect to the receptive field sign (Fig. 4A; cf. with Fig. 4A in Alonso et al. 2001). The probability of connection in the model depends strongly on the agreement between the receptive field signatures. Monosynaptic connections are often found when the geniculate center overlaps with a subregion of the same sign and are rarely encountered when the signs differ. If the receptive fields had different signs, the probability of a connection was only 18.5% compared with the reported value of 18% in the experimental data of Alonso et al. (2001). The probability of finding a monosynaptic connection when the receptive fields had the same sign was 42% compared with 44% in the data. In addition, probability that a geniculate and cortical cell with overlapping receptive fields would be connected (independent of their sign) is 32% (obtained as the sum of the cases depicted by the black bars divided by the sum of all bars in Fig. 3A) compared with 33% reported experimentally. Thus the simulated and experimental data agree well on the probability of connection.

Second, we analyzed the distribution of synaptic strength, or efficacy, with respect to receptive field sign (Fig. 4B). As in the empirical data, we find that connections form LGN cells with similar sign have a higher efficacy compared with connections from LGN centers of opposite sign. The main difference between the distributions of connected and unconnected pairs is in the tails, which is also a feature observed in the empirical data (Fig. 4A, right, in Alonso et al. 2001). The absolute values of efficacy in the model are higher than the actual measurements. This is because the model does not include a cortical contribution to the response. Including such a contribution would make the absolute efficacy values smaller without changing the shape of the distributions in Fig. 4B. Thus the model is in good agreement with the dependence of synaptic efficacy with respect to receptive field sign.

Third, we analyzed the probability of connection as a function of a measure of the overlap between the geniculate and cortical receptive field in the model (Fig. 4C). Overlap was defined as the cross-correlation coefficient between the receptive fields (Alonso et al. 2001). The simulations show that monosynaptic connections are found among receptive fields with higher degrees of overlap, replicating the experimental result (Fig. 5B in Alonso et al. 2001).

Interestingly, both the model and the data show that the overlap for pairs that are not connected is, on average, positive. This means that Alonso et al. (2001) rarely encountered geniculate cells with large anti-correlated values of overlap that were not connected to the cortical receptive field. One interpretation is that these geniculate cells were just not present, and that, as in the model, the (average) positive correlation between receptive fields in the unconnected pairs results from the local dominance of a single (on- or off-center) geniculate RF.

Fourth, we investigated how the efficacy for each connection related to spatial overlap (Fig. 4D). The joint distribution of overlap and efficacy from our simulated data shows that the two are positively correlated \( r = 0.47 \); the higher the overlap, the stronger the synaptic strength. This relationship is also consistent with the experimental findings (Fig. 5B, right, in Alonso et al. 2001).

Fifth, we computed the relative size of the LGN center with respect to the subregion width (Fig. 4F). The distribution shows that the majority of subregions have widths that are slightly smaller than the geniculate centers, in agreement with the experimental data (Fig. 14 in Alonso et al. 2001).

Sixth, we computed the aspect ratio of the dominant subregion in the simulated receptive fields (Fig. 4E). The range of the aspect ratio is between 1 and 3, with a mean of \( 1.36 \pm 0.26 \) (SD). This is roughly one-half the mean value of \( 2.5 \pm 0.8 \) measured from the first-order kernels of layer IV simple cells (Fig. 4D in Alonso et al. 2001). This means that the model falls short in accounting for the full elongation of the subregions as measured by the first-order kernels. If we compare, instead, the simulated data with the average aspect ratio of the afferent geniculate input inferred from the population measurements (Fig. 4, A and B, in Alonso et al. 2001), we find a reasonable match. Across the population, the data show the probability of connection decays to half-height at a distance of \( \approx 0.65 \) subregion widths when measured across the subregion of the receptive field, while the probability of connection decays to half-height at a distance of about \( \approx 1.88 \) subregion widths when measured along the length of the receptive field (Alonso et al. 2001). Thus the average aspect ratio of the afferents would be \( \approx 1.35 \), which is close to the value of 1.36 that resulted from the simulation.

An important point here is that the measured distributions of afferent inputs into the subregions of simple cells appear insufficient to account for their high aspect ratio in the subregions of the first-order kernels. This point is reinforced by Fig.
4C in Alonso et al. (2001), which shows that the distribution of distances of the geniculate inputs along the length of the subregion rarely exceed one-half its size. The mismatch between the aspect ratio of the afferents and those of the receptive field subregions might be explained by a cortical contribution to the (linear) receptive field (Sompolinsky and Shapley 1997). This hypothesis is weakened by experiments showing an invariance of orientation tuning in layer IV cells when the cortex is functionally deactivated (Chung and Ferster 1998; Ferster et al. 1996). More investigations are required to understand better the reason for the discrepancy between the inferred aspect ratio of the afferents and those of the first-order kernels.

Seventh, we asked if independent sampling from the retinal mosaic could generate a bias for the preference of orientation in cells located in the same cortical column. We found that this was indeed the case. Two examples where the local organization of the RGC mosaic clearly biased the orientation tuning of the cells to a particular orientation angle are shown in Fig. 5. The panel on the top left in Fig. 5A shows the local distribution of on (red) and off (blue) RGCs, with their size indicating the probability of connection to the cortical neurons (note that synaptic efficacy is not shown in this diagram). The panels on the right show the outcome of 16 receptive fields independently sampled from the RGC mosaic. Visual inspection reveals that the receptive fields tend to be elongated along similar orientation angles. Intuitively, this results because there are two pairs of on-off ganglion cells with high probability of connection arranged parallel one to the other (Fig. 5A, top left). A summary of the preferred orientation angle is shown in the polar histogram in the bottom left of Fig. 5A. A second example
is shown in Fig. 5B. We conclude the model is capable of generating a local bias for a preferred orientation in the cortex.

Eighth, we asked if a given RGC mosaic could induce an orientation map across the cortical surface. An example of the orientation map generated by the model is shown in Fig. 6A. In this simulation, a local histogram of orientation preferences was computed from 100 independent cells at a fixed cortical location. The mean orientation is shown at each location using a pseudo-color map. The orientation map shows linear regions, fractures, and pinwheels, which resemble those observed in empirical data (Bonhoeffer and Grinvald 1991; Obermayer and Blasdel 1993; Shmuel and Grinvald 2000). We do not want to imply that the maps generated by the model match, in quantitative terms, the statistical features of real maps. Instead, we propose that cortical columns generated by random pooling of afferents may provide a blueprint for the development of an orientation map that is going to be further refined by activity-dependent processes (Erwin and Miller 1998; Miller 1994; Swindale 1996).

Finally, we investigated the orientation scatter at each location in the orientation map assessed by an orientation bias index of the preferred orientations of neurons within a “column.” The index was defined as one minus the circular variance of the angular distribution of preferred angles (Mardia 1972). An index close to zero means that all the orientations are uniformly distributed; an index close to one means that they are tightly distributed around one specific angle. Figure 6B shows that in the majority of cortical locations the orientation bias is higher than that expected by chance (the probability of obtaining an index larger than 0.2 by chance, given the number of simulated points is $<10^{-4}$).

**DISCUSSION**

Wässle et al. (1981) have previously emphasized that the RGC mosaic serves an important constraint on the generation of cortical receptive fields and discussed some of its relevance for orientation tuning. Soodak (1987) first proposed that sampling from the retinal ganglion mosaic may result in a tangential organization of orientation columns in the cortex and showed that the size of the simulated orientation columns agrees with the data. His model was deterministic in the sense
that, at each point in space, a single receptive field was constructed by weighting all the geniculate inputs with a Gaussian distance function. Here, we extended this model by including a function that determines the probability of monosynaptic connectivity between a geniculate neuron and a cortical target. This random component to the model means that not all cortical cells at the same cortical location need to have identical receptive fields. The stochastic nature of the connectivity allows us to evaluate the probability of monosynaptic connection and compare with the data of Alonso et al. (2001), which cannot be done with a deterministic model.

Our findings indicate that the basic organization of simple cell receptive fields and orientation maps in the cortex can potentially result from the haphazard pooling of LGN afferents within a small topographic window. The numerical results are in reasonably good agreement with experimental data on the probabilities and synaptic efficacy of monosynaptic connections between the LGN and the cortex. Nevertheless, the proposed mechanism is unlikely to be the only factor determining the receptive field structure of simple cells or the fine structure of the orientation maps. Instead, the proposal is that haphazard pooling of LGN afferents seeds the structure of simple cells and orientation maps onto which developmental processes act on.

One difficulty that needs to be addressed is how the proposed mechanism can ensure that inputs from the left and right eye result in similar orientation maps (Hubel and Wiesel 1962), given that is highly unlikely the RGC mosaics of the two eyes are correlated. It is known that pattern vision plays no role in the establishment of orientation columns, which emerge during the second postnatal week (Chapman et al. 1999; Godecke et al. 1997; Sengpiel et al. 1998). By this time, visual input is heavily dominated by the contralateral eye (Crair et al. 1998; Fregnac and Imbert 1978). Crair et al. (1998) have suggested that earlier development of the contralateral input may establish the pattern of orientation columns in the cortex and that the input from the ipsilateral eye may “just go for the ride.” However, these authors did not offer a specific mechanism that would explain how the contralateral input sets up the orientation map.

Here we suggest that orientation maps may be seeded by haphazard pooling of thalamic input from the contralateral eye. We speculate that some type of activity-dependent plasticity (such as a Hebbian-like mechanism) acts on both contra- and ipsilateral synapses. When the contralateral signals arrive to a “blank” cortex, they are statistically sampled by the neurons. As cells start responding, the resulting activity will be positively correlated with the input, and a Hebbian-like mechanism would work to reinforce contralateral synapses to establish a blueprint of RF and orientation columns in the cortex. In contrast, when the ipsilateral signals arrive at their targets, they find an already established pattern of RFs and orientation columns. Activity-dependent plasticity may now help to match the input from the ipsilateral eye to the structure generated by the contralateral input. Note that in this scenario, both contralateral synapses play by the same developmental rules, but it is the differential time of arrival to the cortex that allows the contralateral input to seed RF structure and orientation columns in the cortex. If this scheme is correct, one may expect differences in the probability of monosynaptic connections between the contralateral and ipsilateral input. Unfortunately, this analysis cannot be done at the present because the data in Alonso et al. (2001) represents input from the contralateral eye only (as they sampled from layer A in the geniculate).

Within the framework of the haphazard connectivity model, one would attribute the robustness of the orientation map to manipulations during the critical period to the inherent stability of the RGC mosaic and a robust topographic map (Chapman et al. 1996, 1999; Godecke et al. 1997; Kim and Bonhoeffer 1994; Sengpiel et al. 1998). As a consequence, the model predicts that disruptions in the spatial arrangement of the RGC mosaic during development may result in abnormal receptive fields and interfere with the emergence of orientation maps. One such example is the failure of orientation selectivity in ferret cortex to develop in the absence of activity of on-center retinal ganglion cells (Chapman and Godecke 2000).

Another prediction of the model is that the structure of the orientation map should be partially determined by the RGC mosaic of the contralateral eye. One way to test this hypothesis is to optically image the structure of orientation columns and reconstruct the mosaic of X cells in the retina of the contralateral eye at the same visuotopic location. The RGC mosaic can be taken as the input to the haphazard model, which will yield a simulated orientation map as a result. The next step is to match the simulated and measured maps as best as possible, allowing for translation, rotation, and scaling of the simulated map. The statistical significance of the max-
imal cross-correlation thus achieved can be compared with the distribution of values attained using areas of the mosaic in other regions of the visual field or from the retinas of other subjects as control cases. A positive result would imply that the structure of the RGC mosaic contributes to the establishment of the orientation map.

The model also generates predictions about the spatial structure of receptive fields derived from the contralateral geniculate input. A difficulty in testing these predictions is that there are no available data on the two-dimensional spatial structure of simple cell receptive fields when the cortex is inactivated. Nevertheless, it may be worth noting the following. It is apparent from the examples provided in Figs. 3 and 5 that the model generates some receptive fields that have effectively one subregion. If we require the amplitude of the nondominant subregion to be \(\geq 10\%\) of the dominant region, the model generates about 38% cells with only one subfield (data not shown). Some investigators define simple cells as having more than one subregion, so they would not consider these receptive fields as being “simple.” Regardless of their classification, cells with only one subregion have been described in the past (Kato et al. 1978), but appear to be rare in layer IV (Hirsch 2003; Hirsch et al. 2002; Martinez et al. 2002). One possibility is that the surround of the LGN neurons, which were not included in the model, contribute to the creation of subregions flanking a central one. When simulations were performed including a surround with size and magnitude typical of geniculate cells (Cai et al. 1997), we found the proportion one subregion receptive fields reduced to 18%. It is worth mentioning here that Alonso et al. (2001) also assessed the connectivity to weak subregions (<30% of the amplitude of the dominant subregion) in a few cases and could not find any evidence of direct monosynaptic connections. Thus some of the weak subregions may result from the LGN surround or a cortical contribution to the receptive field.

Another implication of the model is that simple receptive fields are constructed by summing of a handful of spatially displaced ON- and OFF-center receptive fields. Heggelund (1981, 1986) arrived at a similar conclusion by analyzing the interaction effects between two stationary flashing light stimuli on simple cells. Alonso et al. (2001) estimated the number of LGN inputs to a simple cell to be \(\approx 30\), which, at first sight, seems to be higher than the numbers generated by the model (Fig. 3). We must recall, however, that the geniculate receptive fields are nearly identical to their retinal counterparts. In other words, no “new” receptive fields are created in the geniculate, and the cortex can be considered to effectively sample from a replica of the RGC mosaic. If we ask, instead, what is the number of different ON-OFF receptive fields a simple cell receives input from (which is what Fig. 3 is actually showing), the number will be \(30/2.5 = 12\). Furthermore, this figure does not reflect the fact that synaptic strength is highest in the dominant subregion and decreases significantly in the nondominant subregions (Alonso et al. 2001). Thus the effective number of different receptive fields (the ones with large synaptic drive) may be a fraction of 12. These numbers are not that dissimilar from those generated by the model (Fig. 3).

There are also some anatomical data that could be used to estimate the number of inputs to layer IV cells (Alonso et al. 2001; Peters and Payne 1993). It is currently estimated that there are about 125 geniculo-cortical synapses per layer IV neuron (Peters and Payne 1993) and that most layer IV cells receive one to two synapses per geniculate axon (Freund et al. 1985). This would make the average number of different geniculate receptive fields arriving at a layer IV cell about 80 (125 synapses per neuron/1.5 synapses per axon). Those 80 axons would represent an average of 32 different ON-OFF centers (80 axons/2.5 coverage ratio in LGN). This number, however, does not take into account the synaptic strength of the connections and how they decay with distance. If we assume that about one-third of these receptive fields provide relatively strong inputs to the cell, we obtain an effective number of about 11 receptive fields. Thus the anatomical figure is within a factor of 2 from the predictions of the model.

Overall, the results suggest that we should consider haphazard pooling a potential contributor to the establishment of orientation maps and study its interactions with other possible mechanisms such as cortical scaffolding (Ernst et al. 2001; Shouval et al. 2000) or wire-minimization principles (Koulakov and Chklovskii 2001). If the present hypothesis turns out to be correct, it would suggest that we may have underestimated the role of statistical connectivity in the wiring of the brain (Braitenberg and Schutz 1991). Nature could be taking advantage of the properties of statistical connectivity to minimize the amount of developmental “rules” required to put the circuitry together. For example, in a system where retinal coverage factors are very high, it would be necessary to stipulate exactly how the cortical neurons are going to sample from LGN afferents to generate orientation tuning and simple-like receptive fields, as well as a method to ensure that all orientations are sampled evenly across a cortical area. By keeping coverage factors relatively low, simple cells and orientation tuning orientation columns emerge naturally out of the properties of the retinal mosaic and its topographic connection to the cortex. Only a developmental “recipe” for a topographic mapping is required to achieve the basic scaffolding of visual cortex. Thus connectivity by statistical sampling does not necessarily have to be detrimental in the wiring of the nervous system.

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