

Information Capacity of a Stochastically Responding Neuron Assembly

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Abstract

In this work, certain aspects of the structure of the overlapping groups of neurons encoding specific signals are examined. Individual neurons are assumed to respond stochastically to input signal. Identification of a particular signal is assumed to result from the aggregate activity of a group of neurons, which we call information pathway. Conditions for definite response and for non-interference of pathways are derived. These conditions constrain the response properties of individual neurons and the allowed overlap among pathways. Under these constraints, and under the simplifying assumption that all pathways have similar structure, the information capacity of the system is derived. Furthermore, it is shown that there is a definite advantage in the information capacity if pathway neurons are not localized but rather dispersed among the neuron assembly.

1 Introduction

When we view the world around us we perceive objects free of analog noise. This suggests that somewhere in the brain there exists definitive information about the existence of a signal. Nevertheless, when one records electrophysiological data from pyramidal neurons in mice, the response of these neurons is nothing like definitive and noise free [1], [2]. Upon the repeated presentation of orientation gratings on mammals, there are two important observations that need to be stressed. One is that V1 neurons sometimes respond to a signal while some other times does not respond to the exact same signal, and the other is that the frequency of response to the preferred orientation is only marginally higher than the frequency of response to other nearby orientations, that can nevertheless be distinguished by the animal.

A reasonable question to ask is where the definitive information about a signal is located. There has been some speculation that there exist neurons (grandmother cells) that respond reliably to particular objects [3]. These cells are expected to receive unreliable input from many cells and integrate this into a definite response. However such cells have not been spotted in early visual areas like V1 and they are elusive even in higher areas. Hence one is led to believe that definitive information in V1 is carried by a group of (unreliable) neurons whose aggregate output responds reliably to a given stimulus (e.g. an orientation grating). Let us call this an information pathway.

The neurons giving rise to an information pathway are in general not adjacent. Since objects detected are in general extended, information is needed from an extended V1 area so as to be able to recognize an object. However, there are also position dependent local features that constitute signals and need to be encoded. In such features, neurons that are geometrically close should form a group that carries feature information.

The object of this work is to try to understand through the use of simplified models some basic principles underlying information encoding in groups of neurons, that are generally probabilistically responding to a stimulus and form overlapping sets. Two conditions that have to be satisfied and will be examined here are the following: i) The encoding must be definitive, that is the probability of an information pathway to be active when a stimulus is present should be close to 1 while the probability should be close to 0 when the pathway stimulus is not present. ii) There should be no significant interference among overlapping pathways.

In this work, the implications of conditions i) and ii) on the pathway overlap will be examined. Furthermore the information capacity of a group of neurons will be examined for three different architectures of the information pathways.

2 Definitely Responding Neurons

One neuron definite encoding: Let us assume that we have a set of N neurons responding definitely, and we want to encode as many signals as possible. To encode one signal S_i , $i = 1 \dots N$ on each neuron \mathcal{N}_i is rather suboptimal since at most we can encode N signals overall. Robustness is also an issue since if one neuron dies, one signal remains unencoded. Hence if we define the information loss (IL) as the number of signals that remain unencoded divided by the number of signals that was encoded originally, one neuron loss causes information loss $IL = 1/N$. The neuron firing required for a signal detection is one neuron firing. Hence the firing per bit of information (FPB) is $FPB = 1/\log_2 N$.

k neuron definite encoding: It is possible to increase the information encoded in our N neurons by using sets of k neurons to encode one signal. Since there are $\binom{N}{k}$ groups of k elements it is possible to encode $\binom{N}{k}$ signals. If $N \gg k$ this number is of order N^k which is generally much larger than N .

However there is one drawback in this encoding. To figure out the signal encoded from the firing pattern of the N neurons it is not sufficient to have access to the firing of the k neurons that respond to the signal. Information is needed about the neurons that are not firing as well so as to make sure that there is no superset of the k neurons firing that encodes a different signal. Hence a subsequent neuron that needs to process the signal information needs to have access to the full set of N neurons.

There is also an issue with robustness. If one neuron dies it gives no output and this corrupts the encoding of all signals in whose k -neuron responding set is the dead neuron. Hence $\binom{N}{k-1}$ signals are encoded incorrectly. This gives an information loss $IL = \frac{k}{N-k+1}$. For $N \gg k$ this is of order k/N . This is more information loss than in the one neuron definite encoding.

The firing necessary to detect one signal in this case is the firing of k neurons, however the detection is from a pool of N^k signals for $N \gg k$. Hence the firing per bit of information is in this case $FPB = \frac{k}{\log_2 N^k} = \frac{1}{N}$ which is the same as before.

This is not of course a realistic encoding of information in neurons, however it shows one great advantage that motivates the formation of information pathways. This is the vast number of possible subsets of the N neurons that may carry information. Nevertheless, there are issues to be resolved. One such issue is the issue of access of the subsequent neuron to the full set of N neurons. Another issue is the issue of information loss upon death of a neuron.

3 Overlapping Information Pathways with Bimodal Probabilistically Responding Neurons and No Spontaneous Firing

Two overlapping information pathways: Let us suppose that we have two information pathways of n_1, n_2 neurons each, and suppose that there are two distinct signals S_1, S_2 that activates them respectively. Here it is assumed that each neuron in pathway i has probability p_i of firing if S_i is present and probability 0 of firing if S_i is not present. The two pathways are assumed to have an overlap of n_{12} neurons. To decide whether a pathway is active or not, we need to set up a threshold K_i on the number of active neurons. If in pathway i more than K_i neurons are firing, then the pathway is considered active, otherwise it is considered inactive.

Let us now consider the condition for pathway i to be active given that S_i is present. Since the neurons are bimodal, the probability of more than K_i neurons firing is given by the binomial distribution

$$P(F_i > K_i | S_i) = \sum_{k > K_i} \binom{n_i}{k} p_i^k q_i^{n_i - k} \quad (1)$$

where $q_i = 1 - p_i$ is the probability of a neuron not firing when the corresponding signal S_i is present. This probability is the probability pathway i is active when S_i is present.

To facilitate the calculation we are going to use the De Moivre-Laplace theorem to approximate the binomial distribution with the normal distribution. This approximation is considered to be good for $n_i > 30$ and for a range of values of k in the sum that is of order n_i so as to avoid discreteness error. The De Moivre-Laplace theorem tells us that

$$P(F_i > K_i | S_i) \approx \frac{1}{\sqrt{2\pi}} \int_{\frac{K_i - n_i p_i}{\sqrt{n_i p_i q_i}}}^{\infty} e^{-x^2/2} dx \equiv 1 - \Phi\left(\frac{K_i - n_i p_i}{\sqrt{n_i p_i q_i}}\right) \quad (2)$$

where $\Phi(z)$ is the normal cumulative distribution function.

Let us now focus on the condition of definite response (i) of the pathway to signal S_i . Like in any probabilistic response system we have to set a level of certainty above which we consider the system to give a definite response. Let us call this level $1 - \epsilon$. The

condition of definite response of information pathways S_i assumes the form

$$\Phi\left(\frac{K_i - n_i p_i}{\sqrt{n_i p_i q_i}}\right) < \epsilon \quad (3)$$

Condition (3) admits the following interpretation: $n_i p_i$ is the expected number of neurons that are firing in pathway i . Condition (3) is satisfied when the expected number of firing neurons is much larger (in units of standard deviation $\sqrt{n_i p_i q_i}$) than the threshold K_i .

The condition of no interference between the overlapping pathways S_1, S_2 is a little bit more tricky since one needs to carefully set the thresholds so as to achieve maximal separability of the pathways. The probability of S_j , $j \neq i$, to cause activity in pathway i is given by

$$P(F_i > K_i | S_j) = \sum_{k > K_i} \binom{n_{ij}}{k} p_j^k q_j^{n_{ij}-k} \approx 1 - \Phi\left(\frac{K_i - n_{ij} p_j}{\sqrt{n_{ij} p_j q_j}}\right) \quad (4)$$

This probability has to be kept low at confidence interval ϵ , and this leads to the condition

$$\Phi\left(\frac{K_i - n_{ij} p_j}{\sqrt{n_{ij} p_j q_j}}\right) > 1 - \epsilon \quad (5)$$

The interpretation of this equation is that the expected number of firing neurons in the overlap ($n_{ij} p_j$) under presentation of signal S_j is much smaller than the threshold K_i of pathway i .

The threshold K_i should be such that the expected number of firing neurons upon presentation of signal S_i is well above K_i , while the expected number of firing neurons in pathway i upon presentation of signal S_j due to the pathway intersection is well below K_i . One could take K_i to be the midpoint of the two expected numbers of firing neurons, however this would ignore possible differences in the standard deviation of the number of firing neurons in the two cases. Instead one should take a weighted average of the two expected numbers by the standard deviations as threshold. This is given by

$$K_i = \frac{n_i p_i \sqrt{n_{ij} p_j q_j} + n_{ij} p_j \sqrt{n_i p_i q_i}}{\sqrt{n_{ij} p_j q_j} + \sqrt{n_i p_i q_i}}. \quad (6)$$

Suppose now that we set $\epsilon = 0.01$. Then the condition of definite response (3) can be solved by using the normal cumulative distribution table to give

$$\frac{K_i - n_i p_i}{\sqrt{n_i p_i q_i}} < -2.33. \quad (7)$$

Furthermore the condition of no interference gives similarly

$$\frac{K_i - n_{ij} p_j}{\sqrt{n_{ij} p_j q_j}} > 2.33 \quad (8)$$

Setting the threshold to the optimal value (6) the conditions (7) and (8) collapse to the single condition

$$\frac{n_i p_i - n_{ij} p_j}{\sqrt{n_{ij} p_j q_j} + \sqrt{n_i p_i q_i}} > 2.33 \quad (9)$$

Many overlapping pathways: In this case the condition of definite response is the same for each pathway, however the condition of no interference becomes more complicated since there are now many possible overlaps. This obscures the optimal choice of threshold for each pathway since now the optimal threshold of a pathway depends on firing probabilities and overlapping sets of all overlapping pathways. This suggests that there must be some uniformity in the expected number of firing neurons in the overlapping sets so as to *have* a possible choice of thresholds that avoid interference.

To proceed further, we will suppress the diversity in the neuron number, firing probabilities and overlapping set size and consider a simplified model in which all pathways have the same number (n) of neurons, and the maximum number of neurons in the overlap of two pathways is $m < n$. Furthermore the firing probability of each neuron in a pathway under presentation of the signal associated with the pathway is taken to be constant (p). In this case the optimal threshold for all pathways is given by

$$K = \frac{np\sqrt{m} + mp\sqrt{n}}{\sqrt{m} + \sqrt{n}} \quad (10)$$

The conditions of definite response and no interference (7) and (8) now collapse to the single condition

$$(\sqrt{n} - \sqrt{m})\sqrt{\frac{p}{q}} > 2.33 \quad (11)$$

To have maximum number of encoding pathways it is necessary to increase the overlap m to a maximum value m_0 , without violating condition (11). This is achieved when

$$m_0 = [(\sqrt{n} - 2.33\sqrt{q/p})^2] \quad (12)$$

where the brackets here denote integer part.

In Fig.1 we see how the value of the maximum overlap m_0 varies as a function of the probability of neuronal response p for a fixed number $n = 1000$ of neurons in a pathway. From this graph we see that already at $p = 0.06$ we have a possible 50% overlap. Hence we can say that for maximal number of noninterfering definitely responding pathways of size $n = 1000$, even for very low probabilities of response high overlaps are possible.

One important aspect of these probabilistic overlapping pathways is that a subsequent neuron does not need the full set of neurons to decode the signal, like in the case of k neuron definite encoding, since there are no super-pathways that contain smaller information pathways inside. If a subsequent neuron is connected to a pathway and thresholds the overall input at the optimal threshold then this neuron has the information the pathway carries. Hence such a reading system is implementable in real neural networks.

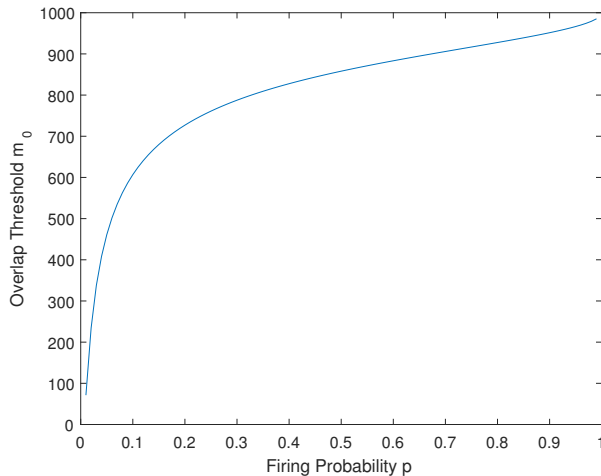


Figure 1: The pathway overlap threshold m_0 is plotted against the probability p of a neuron in a pathway responding to its signal. Here the number of neurons in the pathway is taken to be $n = 1000$.

4 Overlapping Pathways in the Presence of Spontaneous Firing

Let us now suppose that each pathway has n bimodal neurons and that a pair of pathways has m neuron overlap, as before. In this case we will assume that there is a spontaneous probability of firing p_0 for a neuron not participating in a pathway whose signal is present. There is also a stimulated probability of firing p when the neuron is in a pathway whose associated signal is present.

The spontaneous firing may arise in many ways. It may be for example built in the network so as to maintain dynamic equilibrium. After all, excess firing will generate too much input to the network and this may lead to seizure if network control fails, while not enough firing may result in too little input to the network leading to global silencing. Another source of spontaneous firing may be the parallel operation of overlapping pathways. It is quite possible that a neuron belongs not only to the pair of pathways considered here, but also to a third pathway that may or may not be active in parallel to our pair of pathways. It is quite possible that both sources of spontaneous firing mentioned exist and cooperate in the brain.

A third source for the "spontaneous" probability of firing p_0 may be simply the signal to the second pathway that is causing partial response to the first pathway. This may happen for example if the signals are close orientation gratings. In this case both orientation gratings generate response to the neurons of a pathway, however the preferred orientation generates response with higher probability p than the nearby orientation which generates a response with probability p_0 . In this case we will refer to the pathways as close signal pathways and to the difference $dp = p - p_0$ as the probability resolution of the two pathways.

As before, there is a threshold K for the number of active neurons in a pathway,

above which the pathway is considered active. The condition of definite response again assumes the form

$$\Phi\left(\frac{K - np}{\sqrt{npq}}\right) < \epsilon, \quad (13)$$

however in this case there is one further condition, the condition of non spontaneous response which tells us that

$$P(F_i > K | NoSignal) \approx 1 - \Phi\left(\frac{K - np_0}{\sqrt{np_0q_0}}\right) < \epsilon. \quad (14)$$

Fortunately, this condition is weaker than the non-interference condition hence it is automatically imposed.

The non-interference condition in this case assumes the form

$$P(F_i > K | S_j) = \sum_{k,l,k+l > K} \binom{m}{l} p^l q^{m-l} \binom{n-m}{k} p_0^k q_0^{n-m-k} < \epsilon. \quad (15)$$

It is not too difficult to show, following a proof similar to the De Moivre-Laplace theorem, that in the limit $n \gg m$, $m \gg 1$, $P(F_i > K | S_j)$ can be approximated by

$$P(F_i > K | S_j) \approx 1 - \Phi\left(\frac{K - (n-m)p_0 - mp}{\sqrt{(n-m)p_0q_0 + mpq}}\right). \quad (16)$$

This leads to the non-interference condition

$$\Phi\left(\frac{K - (n-m)p_0 - mp}{\sqrt{(n-m)p_0q_0 + mpq}}\right) > 1 - \epsilon \quad (17)$$

In this case, the optimal choice of threshold is

$$K = \frac{np\sqrt{(n-m)p_0q_0 + mpq} + ((n-m)p_0 + mp)\sqrt{npq}}{\sqrt{(n-m)p_0q_0 + mpq} + \sqrt{npq}} \quad (18)$$

As before, setting the confidence limit $\epsilon = 0.01$ the two conditions collapse to the condition

$$\frac{(n-m)(p-p_0)}{\sqrt{(n-m)p_0q_0 + mpq} + \sqrt{npq}} > 2.33. \quad (19)$$

It is this condition that determines the maximum allowed overlap m_0 of distinct pathways in the presence of spontaneous firing.

This is also the condition that determines the maximal allowed overlap of two close signal pathways at probability resolution $dp = p - p_0$. The maximum possible value of m , which we call m_0 , for which condition (19) is satisfied is shown in Fig.2 as a function of p for various values of dp . From this graph we see that higher overlaps are favored for both high and low response probability p . Since higher overlaps translate to higher information capacity, one would expect that if information capacity is an important

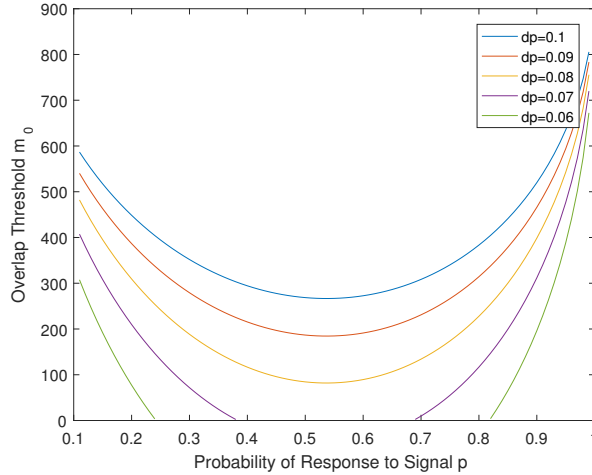


Figure 2: The overlap threshold m_0 is plotted against the probability of response to signal p for various values of the probability resolution $dp = p - p_0$. Here the pathway neuron number is $n = 1000$

parameter in the operation of information pathways, then these would operate at either $p \approx 0$ or at $p \approx 1$.

The mode of operation $p \approx 0$ appears to be problematic, even though it has a definite energetic advantage over higher response probabilities. First of all p has a low bound $p_0 + dp$ determined by the spontaneous firing probability p_0 and the necessary probability resolution. If p_0 is large due to network steady state requirements (e.g. non-silencing of the network) or due to parallel operation of many overlapping pathways, then it is not possible to operate in this regime. Another problem is that all pathways have to operate at low p to avoid pathway interference. It may be true that in this model we have assumed, for simplicity, that all pathways have a common response probability, but in real brain networks this is not likely to be the case.

The mode of operation $p \approx 1$ seems to be more feasible, however it is more energetically demanding. This mode of operation is favored if information capacity is a major concern, at least in the case all pathways have the same p, p_0 . However, in real brain networks p, p_0 are not expected to be constant since the demand of information processing as well as the specific nature of the signals encoded may exercise control over the response probabilities. This suggests that the allowed pathway overlaps m_0 have to be such that condition (19) is satisfied for all p, p_0 at a given probability resolution dp . This happens at the minima of the curves in Fig.2.

5 Number of Signals Encoded

Let us now turn more closely to the question of how many signals can be encoded independently on a set of N neurons given that the pathway size is n and the maximal overlap allowed is $m_0 < n$.

5.1 2D Dense Neighbourhood Pathway Model

In this model we are going to assume that the neurons are irregularly and randomly placed on a surface modeling a cortex layer. We will furthermore assume that pathways are constructed by geometrically adjacent neurons placed closest together (i.e. they form circles), hence the number of neurons in this model is proportional to the surface area that they occupy. We will assign a surface density d to the number of neurons per unit area. Hence the areas associated to N , n and to the overlap m are $A_N = N/d$, $A_n = n/d$ and $A_m = m/d$. Since the pathways are circular, we can associate a radius $R_n = \sqrt{A_n/\pi} = \sqrt{n/\pi d}$. The overlap area A_m is the overlap of two circles, and the size of this area is fully determined by the radius of the pathway circles and the distance of the centers of the pathways. In fact it is easy to show that

$$A_m = 2R_n^2 \sin^{-1}\left(\frac{\sqrt{R_n^2 - D^2/4}}{R_n}\right) - D\sqrt{R_n^2 - D^2/4}. \quad (20)$$

Inverting relation (20) it is possible to determine the minimum distance D_{m_0} allowed for a maximum overlap A_{m_0} that corresponds to m_0 neurons. Defining the regularized distances with respect to the density $\hat{R}_n = R_n\sqrt{d}$, $\hat{D} = D\sqrt{d}$, (20) assumes the form

$$m = 2\frac{n}{\pi} \sin^{-1}\left(\frac{\sqrt{n/\pi - \hat{D}^2/4}}{\sqrt{n/\pi}}\right) - \hat{D}\sqrt{n/\pi - \hat{D}^2/4}. \quad (21)$$

Consider now the question of how many pathways of size n fit in N neurons if the maximal overlap allowed is m_0 neurons. This question corresponds to the question of how many circles of radius R_n fit within an area A_N of neurons if the nearest distance of their centers allowed is D_{m_0} .

This is a question that can be answered easily if we ignore insignificant edge effects associated with the exact shape of the area A_N . For closest packing, the centers of the pathway circles form a triangular lattice of edge D_{m_0} , and the area of the triangular cell of the lattice is $A_c = D_{m_0}^2\sqrt{3}/4$. Hence we get that the number of pathways N_p is

$$N_p = \frac{A_N}{A_c} = \frac{2N}{\sqrt{3}dD_{m_0}^2} = \frac{2N}{\sqrt{3}\hat{D}_{m_0}^2} \quad (22)$$

From equation (22) we see that the number of pathways N_p increases linearly with N , but with a proportionality coefficient that depends on both n, m_0 .

The graph of this proportionality coefficient for maximum overlap m_0 is depicted in Fig.3 for $n = 1000$ pathway neurons. As can be seen, N_p/N increases with increasing m_0 , however only for very large overlaps it becomes comparable to 1.

5.2 3D Dense Neighbourhood Pathway Model

In this model we are going to assume that the neurons are irregularly and randomly placed in three dimensions, essentially considering the cortical layer to be thick, hence

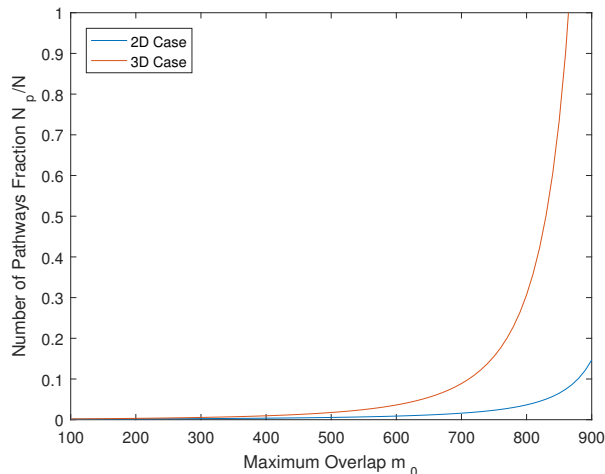


Figure 3: The pathway fraction N_p/N is plotted against the maximum number of overlap neurons m_0 . Here the number of neurons in the pathway is taken to be $n = 1000$.

allowing three dimensional structure. We will also assume, as in the 2D model, that pathways are constructed by adjacent neurons placed closest together, forming overlapping spheres that are closest packed at the overlap permitted. Hence the centers of the pathways form a closest packed sphere lattice (hexagonal close packed or cubic close packed lattice). In this case a volume neuron density d associates volumes with neuron numbers, giving $V_N = N/d$ for the volume of the aggregate of neurons, $V_n = n/d$ for the volume of the neurons in a pathway and $V_m = m/d$ for the volume of the neurons in the overlap of two pathways. The radius of the pathway sphere is $R_n = \sqrt[3]{3V_n/4\pi} = \sqrt[3]{3n/4\pi d}$. The overlap volume of two overlapping spheres distance D apart is given by

$$V_m = \frac{1}{12}\pi(4R_n + D)(2R_n - D)^2. \quad (23)$$

Regularizing by setting $\hat{R}_n = R_n \sqrt[3]{d}$, $\hat{D} = D \sqrt[3]{d}$, (23) assumes the form

$$m = \frac{1}{12}\pi(4\sqrt[3]{3n/4\pi} + \hat{D})(2\sqrt[3]{3n/4\pi} - \hat{D})^2. \quad (24)$$

Suppose now that m_0 is the maximum allowed overlap, so that pathways do not confuse each other. Inverting numerically equation (24) it is possible to obtain the minimum distance D_{m_0} allowed between the centers of the pathways. The maximum number of pathways that can be packed at this minimum distance is equal to the number of auxiliary hard spheres of radius $D_{m_0}/2$ that can be packed in the volume V_N . A theorem of Gauss tells us that the maximum fraction of volume that can be occupied by closely packed hard spheres is $\pi/3\sqrt{2}$. Hence the volume of the auxiliary spheres is $V_{aux} = \frac{\pi V_N}{3\sqrt{2}}$ and the number of them is the quotient of V_{aux} by the hard sphere volume

$V_{HS} = \frac{\pi D_{m_0}^3}{6}$. Since this is the number of pathways we get that

$$N_p = \frac{V_{aux}}{V_{HS}} = \frac{V_N \sqrt{2}}{D_{m_0}^3} = \frac{N \sqrt{2}}{\hat{D}_{m_0}^3} \quad (25)$$

Equations (24,25) implicitly determine the number of pathways in terms of the number of pathway neurons n and the maximum allowed number of overlap neurons m_0 . Again, the number of pathways increases linearly in the number of neurons N , but with a higher proportionality coefficient than in the 2d case, as can be seen in Fig.3.

5.3 Random Selection Model

In the previous models described the information pathways formed were *local* in the sense that neurons in a pathway were neighbouring neurons. They were also *dense* in the sense that all neurons within a radius from the pathway center are in the pathway. Both these restrictions limit severely the number of pathways that are non interfering. To understand better these limitations suppose the pathway neurons are chosen from the full aggregate. Suppose that we are given a set of N neurons, and that each neuron is chosen at random with probability $p = n/N$ to participate in a particular pathway. This does not quite fix the pathway neurons to be n , but rather demands the expectation value of the number of pathway neurons to be n . Lets say that the non-interference condition allows m_0 neuron overlaps among distinct pathways and that activation of a pathway by coactivation of two other pathways is a rare event and can be ignored.

Since the overlap neuron number m determines whether there exists interference among pathways we need to calculate the overlap probability $P(O = m)$ where O is the two pathway overlap random variable. To do this let us suppose that we have fixed the n neurons of pathway P_i , and that we count the ways we can choose the neurons of pathway P_j so as to have m neuron overlap. This number of ways is $\binom{n}{m} \binom{N-m}{n-m}$. Hence the probability of m overlap in the pathways P_i, P_j is

$$P(O = m) = \frac{\binom{n}{m} \binom{N-m}{n-m}}{\binom{N}{n}} \quad (26)$$

Suppose now that we work in the large N limit, that is assume $N \gg n$. Then we can apply Stirling's formula to get the N dependence of the overlap probability. Doing this we get that

$$P(O = m) = \frac{1}{m!} \left(\frac{n!}{(n-m)!} \right)^2 N^{-m}. \quad (27)$$

The importance of this formula is that $P(O = m) \sim N^{-m}$. Suppose now that the condition of no interference is $O < m_0$. Then the probability of interference of the two pathways is

$$P(O \geq m_0) = \sum_{m=m_0}^n P(O = m) \sim \frac{1}{m_0!} \left(\frac{n!}{(n-m_0)!} \right)^2 N^{-m_0}. \quad (28)$$

To determine the number of non-interfering pathways N_p in this model we have to set a level of tolerance since the pathway overlap is stochastic. A rather strict tolerance level, that guarantees that the pathways are operating properly, is to demand that it is unlikely to have one interfering pair per pathway. This is equivalent to saying that the expected number of interfering pairs is a small ϵ fraction of the number of pathways.

$$0.5N_p(N_p - 1)P(O \geq m_0) < \epsilon N_p. \quad (29)$$

This means that the allowed number of non-interfering pathways is

$$N_p \sim 2\epsilon m_0! \left(\frac{(n - m_0)!}{n!} \right)^2 N^{m_0} \quad (30)$$

Observe that the situation here is very different from the situation in either the 2D or the 3D dense neighbourhood pathway models. Here the number of non-interfering pathways increases like a power of the number of neurons, $N_p \sim N^{m_0}$, while in either of the geometric models it increases *linearly* in the number of neurons, $N_p \sim N$. Hence the random selection model can store much more information than the dense neighbourhood pathway models, suggesting that the brain architecture may drop locality when there are many distinct signals to be encoded. Nevertheless there is also a penalty to pay. This model for pathway construction is not appropriate when topographical mapping has to be maintained. Since this happens in the early visual areas, this model is not appropriate for pathways in V1. What we need in V1 is a model that will take the advantages of dilute pathways and combine them with the locality of the pathways in the dense neighbourhood pathway models. Such a model is the Sharp Cutoff Constant Density Pathway Model.

6 Sharp Cutoff Constant Density Pathway Model

6.1 2D case

In this model we will assume that the n pathway neurons are uniformly distributed within a distance R_n from the pathway center. Two densities are associated to this model. One is the neuron density $d = N/Area$, and the other is the pathway neuron density $d_n = n/\pi R_n^2$. In terms of these densities the number N_n of neurons within radius R_n from the pathway center is $N_n = \pi R_n^2 d > n$. The probability p that a neuron within radius R_n from the pathway center belongs to the pathway is $p = d_n/d = n/N_n$. We will also assume that the maximum overlap permitted between pathways is m_0 neurons, which is determined by the response properties of the neurons.

If the pathways form a closest packed lattice as in the 2D geometric model, then the adjacent pathway overlap area is again given by (20). However the expected number of overlap neurons m is modified by the ratio of the two densities:

$$m = \left[2 \frac{n}{\pi} \sin^{-1} \left(\frac{\sqrt{n/\pi - \hat{D}^2/4}}{\sqrt{n/\pi}} \right) - \hat{D} \sqrt{n/\pi - \hat{D}^2/4} \right] \frac{n}{N_n} \quad (31)$$

Here $\hat{R}_n = R_n\sqrt{d_n}$, and $\hat{D} = D\sqrt{d_n}$ are again the appropriate dimensionless radius and pathway center distance. Solving implicitly (31) for \hat{D} after substituting the maximum overlap m_0 for m , gives us the minimum pathway distance \hat{D}_{m_0} .

If \hat{D}_{m_0} is different from zero, this is sufficient to give us the maximum number of pathways that can be packed in our neuron area to be

$$N_P = Area/2A_{triang.cell} = 2N/\sqrt{3}\hat{D}_{m_0}^2. \quad (32)$$

In this case the maximum number of pathways N_P increases linearly with the number of neurons present.

The situation can change when $\hat{D}_{m_0} = 0$. From (31) it is easy to see that this happens when

$$m_0 \geq n^2/N_n. \quad (33)$$

In this case two pathways can operate without interference at any distance D . However if too many overlapping pathways are present some pairs will interfere. In this case it is unlikely to maintain closest packed structure for the pathway centers. Let us denote by \hat{N}_I the number of interfering pairs. As in the Random Selection Model, we will demand that the expected number of interfering pairs is small compared to the number of pathways,

$$E\hat{N}_I < N_P\epsilon. \quad (34)$$

Let us consider now two adjacent pathways. The probability p_b that a random neuron belongs to the overlap of these pathways is given by

$$p_b = \frac{A_m}{Area}p^2 = \frac{m}{N}p^2 \quad (35)$$

where A_m is the overlap area and $Area$ is the area of all the neurons. The ratio $A_m/Area$ represents the probability that the neuron picked is in the overlap area, and p^2 represents the probability that it belongs to both pathways.

Consider the interference probability $P_{m_0}(D)$ which is the probability that the overlap of two adjacent pathways is greater than or equal to m_0 . Then

$$P_{m_0}(D) = \sum_{m \geq m_0} \binom{N}{m} p_b^m q_b^{N-m}. \quad (36)$$

Since in the regime we are working p_b is small, we can apply the Poisson approximation to the binomial distribution and we have

$$P_{m_0}(D) \approx \sum_{m \geq m_0} \frac{(Np_b)^m}{m!} e^{-Np_b}. \quad (37)$$

The ratio of two successive terms in this sum is given by

$$ratio = \frac{\frac{(Np_b)^{m+1}}{(m+1)!} e^{-Np_b}}{\frac{(Np_b)^m}{m!} e^{-Np_b}} = \frac{Np_b}{(m+1)}. \quad (38)$$

Recall that we are in the regime where the expected number of overlap neurons Np_b , when two pathways overlap completely, is less than m_0 , so as to have $\hat{D}_{m_0} = 0$. In our case we do not have complete overlap, hence the condition $Np_b \ll m_0$ is rather a mild condition to impose. Hence in $P_{m_0}(D)$ we can keep only the first term to get

$$P_{m_0}(D) \approx \frac{(Np_b)^{m_0}}{m_0!} e^{-Np_b} \quad (39)$$

Using now (36) and (20) it is easy to show that

$$Np_b = \frac{2}{\pi} \left[\sin^{-1} \sqrt{1 - \left(\frac{D}{2R_n} \right)^2} - \left(\frac{D}{2R_n} \right) \sqrt{1 - \left(\frac{D}{2R_n} \right)^2} \right] \frac{n^2}{N_n} \equiv f(D) \frac{n^2}{N_n}. \quad (40)$$

Here $f(D) = A_m/\pi R_n^2$ is a geometric factor that is valued in the interval $[0, 1]$ and is zero when $D > 2R_n$. Applying Stirling's formula on the factorial in (39) we get

$$P_{m_0}(D) \approx \frac{1}{\sqrt{2\pi m_0}} e^{-m_0(r(D) - \ln(r(D)) - 1)} \quad (41)$$

where $r(D) = n^2 f(D)/N_n m_0 < 1$ in our regime of interest. Noticing that the function $g(r) = r - \ln(r) - 1$ is decreasing for $r < 1$ and $r(1) = 0$ we get that $g(r(D)) > 0$. Hence we have a negative exponent of m_0 in the pathway interference probability.

If we assume that we have random positioning of the pathways, then the probability that two pathways being in $[D, D + dD]$ apart is

$$P_p(D)dD = \frac{2\pi D dD}{Area}. \quad (42)$$

This means that the confusion probability P_{m_0} which is now independent of the pathway distance D is

$$P_{m_0} = \int_0^{2R_n} P_{m_0}(D) P_p(D) dD \quad (43)$$

This is expected to retain the exponential behaviour in m_0 with an effective coefficient $g(r_{max})$ where $r_{max} = \max\{r(D)\} = n^2/N_n m_0$. In fact it is easy to see that

$$P_{m_0}(D) < \frac{1}{\sqrt{2\pi m_0}} e^{-m_0(r_{max} - \ln(r_{max}) - 1)}, \quad (44)$$

hence we get the bound

$$P_{m_0} < \frac{1}{\sqrt{2\pi m_0}} e^{-m_0(r_{max} - \ln(r_{max}) - 1)} \frac{4N_n}{N}. \quad (45)$$

In this case the tolerance condition (34) assumes the form

$$\frac{N_p(N_p - 1)}{2} P_{m_0} < \epsilon N_p \approx \epsilon(N_p - 1) \quad (46)$$

This is guaranteed if

$$N_p \frac{1}{\sqrt{2\pi m_0}} e^{-m_0(r_{max}-\ln(r_{max})-1)} \frac{2N_n}{N} < \epsilon, \quad (47)$$

hence

$$N_p < \epsilon \frac{N}{2N_n} \sqrt{2\pi m_0} e^{m_0(r_{max}-\ln(r_{max})-1)} \quad (48)$$

This model has a linear dependence of the number of pathways N_p on the number of neurons N , however there is an exponential dependence of the maximum pathway number on the maximum overlap m_0 . This means that once we have dilute enough pathways, so that the condition $n \leq \sqrt{m_0 N_n}$ is satisfied, then the number of allowed, non-interfering pathways, increases rapidly. Furthermore, once the diluteness of the pathways is regulated in the brain, pathways can be randomly placed in almost arbitrarily large numbers without significant interference. In practice however there probably are other restrictions, not to be studied here, that limit the number of pathways allowed, like for example the ability of the brain to address these pathways.

6.2 3D Case

The situation in 3D differs only by the geometric factors. In this case

$$m = \left[\frac{1}{12} \pi (4\sqrt[3]{3n/4\pi} + \hat{D})(2\sqrt[3]{3n/4\pi} - \hat{D})^2 \right] \frac{n}{N_n} \quad (49)$$

Here $\hat{R}_n = R_n \sqrt[3]{d_n}$, and $C = D \sqrt[3]{d_n}$ are again the appropriate dimensionless radius and pathway center distance. Solving implicitly (49) for \hat{D} after substituting m_0 for m gives us \hat{D}_{min} . As in the 3D Dense Neighbourhood Pathway Model, the number of pathways that can be packed at this closest distance is

$$N_p = \frac{N\sqrt{2}}{\hat{D}_{min}^3} \quad (50)$$

The situation is again expected to change when $\hat{D}_{min} = 0$. This happens, as in the 2D model, when $m_0 > n^2/N_n$. In this case

$$Np_b = N \frac{V_m}{V_n} p^2 = (1 + \frac{D}{4R_n})(1 - \frac{D}{2R_n})^2 \frac{n^2}{N_n} \equiv f(D) \frac{n^2}{N_n} \quad (51)$$

Again the bound for $P_{m_0}(D)$ is given by (44), as in the 2D case. There is a difference however in the bound of P_{m_0} for disordered pathways because now

$$P_p(D)dD = \frac{4\pi D^2 dD}{Area}. \quad (52)$$

The new 3D bound is given by

$$N_p < \epsilon \frac{N}{4N_n} \sqrt{2\pi m_0} e^{m_0(r_{max}-\ln(r_{max})-1)}, \quad (53)$$

where r_{max} is as in the 2D case.

7 Interneuron Functionally Connected Groups in Mice V1

Let us try to make contact of the above calculations with two photon data collected from the V1 area of adult mice [5]. We will make the assumption that the set of pyramidal neurons connected to an interneuron forms an information pathway. This is not a universally accepted assumption, since there are many possibilities on the way the interneurons exert control over the network. One possibility, for example, is that the activity created on a pathway through a signal presentation is dispersed in the whole network and then the interneurons exert control locally over the network to quench the extra activity. Nevertheless, we believe that this type of control is very non-specific for information processing. If quenching of activity is demanded on one pathway and not on another, this cannot be done. There is furthermore evidence that neurons connected to a particular interneuron tend to have similar tuning properties [5], which is compatible with the fact that they encode similar information. Hence we will proceed based on this assumption, which we will check anyway for internal consistency.

Since the data that we have are limited in size and it seems that there is no universal profile for the decay of pathway neuron density with distance from the interneuron (taken to be the pathway center), we will utilize the sharp cutoff constant density pathway model. Although the data we have come from a planar recording, of thickness $d_t \approx 20\mu m$ (determined by the size of neurons and not by the optical properties of the microscope), we will use the 3D case, since there is no reason to believe that there is a planar structure in the neuron functional connections. To do this we will extrapolate the planar data that we have from the recording area of thickness d_t to a sphere around the interneuron of radius $R_n = 250\mu m$ which is going to be our pathway cutoff radius. This value of the cutoff radius is motivated by the decay profile of the planar interneuron connectivity with distance, and it refers to functional connectivity only. It is certainly a rough estimate, however it is expected to improve in the future by the inclusion of more extensive data sets. Note that the interneuron-connected pyramidal density d_n inside the cutoff radius R_n is taken to be the same as in the thickened planar recording.

In our dataset the field of view allows the analysis of 5 interneurons. The real overlap matrix is the following: Since each interneuron is placed in a different position in our

Table 1: Interneuron Pathway Overlap Data

Int. No.	1	2	3	4	5
1	73	32	21	42	13
2	32	72	42	52	31
3	21	42	50	44	27
4	42	52	44	81	34
5	13	31	27	34	42

planar recording it is not fair to compare directly these intersection numbers. Instead we will extrapolate these data on spheres with center the interneuron position and radius

the cutoff radius R_n , assuming constant pyramidal neuron density within R_n .

The way this extrapolation is done is the following: First two interneurons i, j are selected. With the interneuron positions as pathway centers, the area of the intersection, A_{ij} , of the circles of radius R_n that is within our recording area, is marked. The pathway intersection neurons that are within A_{ij} are counted. This count is a number less than or equal to the intersection number in Table 1. This number is associated to the volume $V_{ij} = A_{ij} * d_t$ of the thick slice. Then, by proportionality, we extrapolate this number to the overlap volume of the two spheres of radius R_n . The results of this extrapolation are given in Table 2. The diagonal elements of Table 2 are the extrapolated neuron numbers of each pathway on the sphere of radius R_n , and the last column is the number of pyramidal neurons that are in our sphere, no matter whether they belong to the corresponding pathway or not.

Table 2: Interneuron Pathway R_n Projected Overlap Data

Int. No.	1	2	3	4	5	N_n
1	1972	642	267	468	178	3694
2	642	2014	657	756	365	3842
3	267	657	1653	1359	506	3202
4	468	756	1359	2274	714	3282
5	178	365	506	714	1188	3593

Next we examine whether the extrapolated intersection numbers n_{ij} are compatible with the definite response-non interference condition (19). Hence we plot the LHS of (19) for every pair of pathways (interneurons) and we check whether this is greater or not than our confidence limit threshold which for confidence limit $\epsilon = 0.01$ is 2.33, for $\epsilon = 0.05$ is 1.65 and for $\epsilon = 0.1$ is 1.29. This is displayed in fig.4 Note that the choice of the firing probabilities $p = 0.5$, $p_0 = 0.4$ is close to a worst case senario for probability resolution $dp = 0.1$, as can be seen from fig.2

Similar graphs to fig. 4 have been constructed for three more datasets from [5] corresponding to three different adult mice. The results are displayed in fig. 5. Again most of the pairs of pathways satisfy the non-interference and definite response condition (19) at the $\epsilon = 0.05$ confidence limit.

8 Conclusion

Although in this work we have made a number of simplifications there are certain conclusions that can be drawn. The most important simplifications we have made are the following: a) Definitive information is carried by pathways that either are active or inactive, according to the presence of a signal (maybe object) or not. These we will call bimodal pathways. This is a strong assumption since there is also analog information that is encoded in the brain, like for example orientation or light intensity. However, such bimodal pathways seem to be well fitted for object recognition, especially parallel

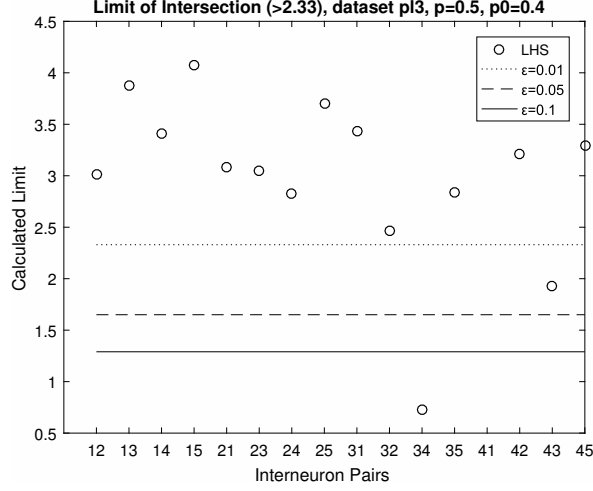


Figure 4: The LHS of (19) is plotted for every pathway (interneuron) pair. The value of this LHS should be above the cutoff line for every pair of pathways to avoid interference. The three cutoff lines correspond to the indicated confidence limits. Interneuron pair (3,4) appears to have interference. Note that the two interneurons are also very close together.

(pop-up) object recognition. b) All pathways have equal size. We make this assumption to avoid complications that would arise from a distribution in pathway size. We hope that this assumption will not make us miss any important features of the pathway assembly. c) Neurons are randomly placed in the physical region where the pathway lies. This is supported by physiological data once we stay within a particular layer and region. However even for many-layer regions, horizontally randomness is maintained. d) The response properties of each neuron are the same for all neurons in a given pathway, are probabilistic and are signal dependent. Indeed, neurons in mice V1 behave stochastically. Even when a neuron responds almost definitely to a given orientation, which is unlikely unless the signal is very strong, the same neuron will respond with high probability to a nearby orientation, hence it is difficult to tell the orientation presented to the mouse. Again, it is evident that in real mice not all neurons have the same response properties, however we chose to make this assumption for the sake of simplicity. e) The neuron assembly that eventually reads the pathway information has the ability to adjust the threshold imposed on the pathway, so as to achieve maximal discriminability of signals.

The conclusions drawn are the following: a) Information capacity of the neuron assembly is severely limited if the pathways are dense. Hence it is not favored to have pathways that include all neurons within a region around the pathway center. The reason for this is that pathways cannot come close together without causing confusion, limiting in this way the number of pathways in the neuron assembly. Maximization of the information capacity in this case forces the pathways to maintain a lattice geometric structure which is not observed in mice V1. This pathway assembly is said to be in the ordered phase. On the contrary, dilute pathways allow random placement of pathway

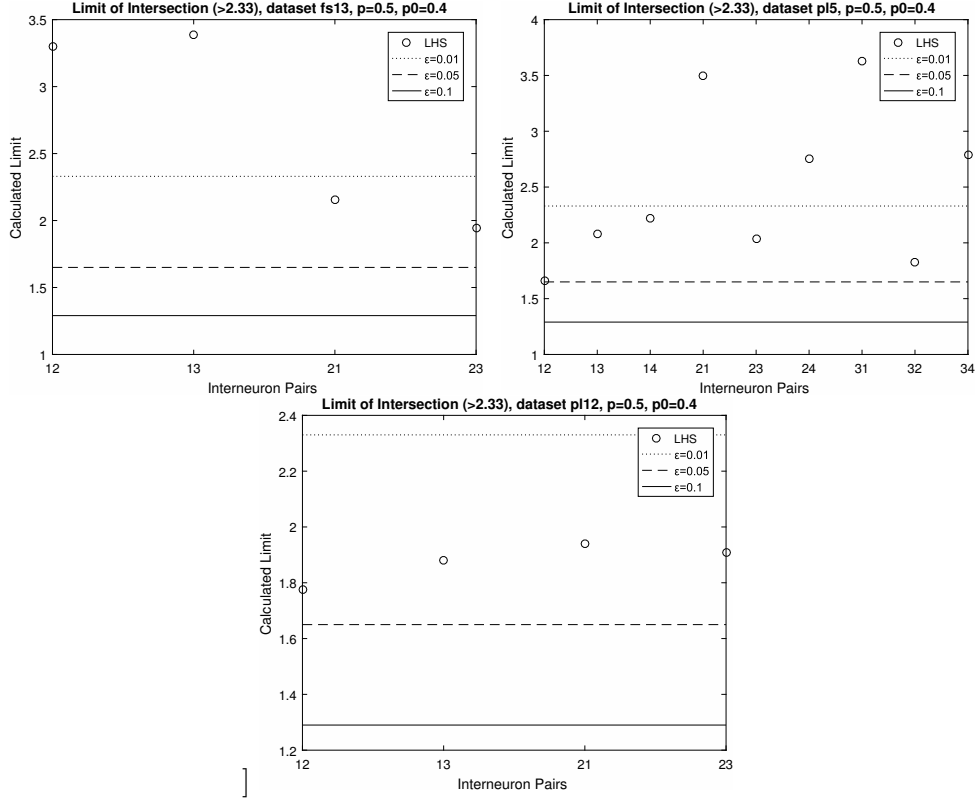


Figure 5: The LHS of (19) is plotted for every pathway (interneuron) pair for three more datasets corresponding to different mice. The results are not very different .

centers in the neuron assembly region, without severely limiting the information capacity. This has the advantage that no complicated organization of pathways is needed in space, allowing pretty much pathway formation without worrying about its placement in space. This pathway assembly is said to be in the disordered phase. b) For a pathway assembly that is in the ordered phase and only pairwise overlaps cause confusion, the number of pathways that can be fitted in an N -neuron assembly increases linearly in N . This linear behavior changes in the random selection model to a power law behavior N^{m_0} where m_0 is the maximum allowed overlap between two pathways. However, in the random selection model locality is lost, since the pathways are formed by the random selection of neurons from the whole neuron assembly. A hybrid model that maintains locality of pathways and allows more dilute pathways is the sharp cutoff constant density model. Once the density is low enough, the pathway system finds itself in the disordered phase. In this phase, although the number of pathways increases linearly in N , the number of pathways increases exponentially in m_0 , allowing a huge number of non-interfering pathways to coexist. c) A behaviour similar to the one in the sharp cutoff constant density model is expected to hold for the distance related selection model (see Appendix),

where the probability that a neuron belongs to a pathway depends on the distance from the pathway center. If this probability is small enough to cause dilute pathways then the system is expected to come to the disordered phase where the number of pathways increases exponentially in the allowed overlap m_0 .

One important remark is due. Although the information capacity increases drastically in the disordered phase, the control and the adressibility of this information can be problematic. Let us make the assumption, for example, that there is a direct control on pathways by interneurons. The number of interneurons is much smaller than the number of neurons N , hence this limits probably too deverely the information capacity. Nevertheless, there should be some direct control over the pathways. If a human or mouse sees two objects and only one is relevant then the other has to fade out quickly and selectively from memory. Hence there must be a mechanism to adress the irrelevant object. Furthermore if one has to recall an object from memory, one has to find a way to activate the relevant pathway. In this work we do not attempt to answer this rather important question of pathway adresssing. It seems likely however that there is interneuron control not over single pathways but rather over specific root pathways that control whole threads of pathways. We hope to adress this question in future work.

Finally, under the assumption that interneurons are directly connected to pathways in mice, we analyze real mice data. Here the assumption is made that the pathway center is the interneuron position. The group of neurons that belong to the interneuron controlled pathway is determined by the functional connectivity of the interneuron to the field of view neurons. Once the interneuron pathways are determined their overlaps are extrapolated from the field of view assuming the sharp cutoff constant density model. The signal presented to the mice consists of 12 orientation moving gratings. Since the probability of firing difference in neurons among consecutive orientations is $p - p_0 \approx 0.1$, we examined whether under this response probability difference the pathways we identified confused each other. The answer is that they do not, except in one pair of pathways where the interneurons happen to be unusually close together.

9 Appendix: Distance Related Selection Model

9.1 2D Case

Lets now suppose that there is a probability $P(|r - r_A|)$ for a neuron at position r to belong to the pathway A, whose center is located at r_A . Still each neuron will be assumed to have uniform probability to be anywhere in the area considered. Since in this model the number of neurons in a pathway can vary, we will normalize the number of such neurons by the expectation value

$$\frac{N}{Area} \int P(|\mathbf{r}|) d^2\mathbf{r} = n. \quad (54)$$

This gives us that the expected value of the distance of the neurons in a pathway is given by $Er = \frac{n}{2\pi} \frac{Area}{N}$. The probability that a neuron belongs to both pathways A and

B is given by

$$p_b = \frac{1}{Area} \int P(|\mathbf{r} - \mathbf{r}_A|)P(|\mathbf{r} - \mathbf{r}_B|)d^2\mathbf{r}. \quad (55)$$

This probability is naturally expressed in elliptical coordinates since it involves distance from two poles. If we set the distance of the two pathway centers $|\mathbf{r}_A - \mathbf{r}_B| = 2a$ then (55) becomes

$$p_b = \frac{a^2}{Area} \int_0^{2\pi} \int_0^\infty P(a \cosh u + a \cos v)P(a \cosh u - a \cos v)(\sinh^2 u + \sin^2 v)dudv \quad (56)$$

The probability of m neuron overlap is given by

$$P(O = m) = \binom{N}{m} p_b^m q_b^{N-m} \quad (57)$$

where $q_b = 1 - p_b$.

The probability of interference of two pathways is given by

$$P_{m_0}(|\mathbf{r}_A - \mathbf{r}_B|) = P_{m_0}(2a) = \sum_{m \geq m_0} P(O = m) \approx \frac{1}{\sqrt{2\pi}} \int_{\frac{(m_0 - N p_b)}{\sqrt{N p_b q_b}}}^\infty e^{-z^2/2} dz \quad (58)$$

where m_0 is really determined by the neuron probability of firing given pathway signal and the probability of firing spontaneously for the two pathways. Here, for the last equality we have used the normal approximation of the binomial distribution.

In the 3D case the pathways are assumed to have spherical shape with a neuron density that varies with distance from the center. Since the probability that a neuron is located in $d^3\mathbf{r}$ is $d^3\mathbf{r}/V$, we have that the probability that a given neuron belongs to pathway A is $\frac{1}{V} \int P(|\mathbf{r} - \mathbf{r}_A|)d^3\mathbf{r}$. This leads to the neuron number expectation value normalization

$$\frac{N}{V} \int P(|\mathbf{r}|)d^3\mathbf{r} = n \quad (59)$$

As in the 2D case, the probability for a neuron to belong to both pathways A, located at \mathbf{r}_A , and pathway B located at \mathbf{r}_B is given by

$$p_b = \frac{1}{V} \int P(|\mathbf{r} - \mathbf{r}_A|)P(|\mathbf{r} - \mathbf{r}_B|)d^3\mathbf{r} \quad (60)$$

This overlap probability simplifies again if we use elliptical coordinates on a plane through the two pathway centers and then we rotate on the axes of the two pathway centers. In this way we get

$$\begin{aligned} p_b &= \frac{a^2}{V} \int_0^{2\pi} \int_0^\infty P(a \cosh u + a \cos v)P(a \cosh u - a \cos v)(\sinh^2 u + \sin^2 v)2\pi|y|dudv \\ &= \frac{2\pi a^3}{V} \int_0^{2\pi} \int_0^\infty P(a \cosh u + a \cos v)P(a \cosh u - a \cos v)(\sinh^2 u + \sin^2 v) \sinh u \sin v dudv \\ &= \frac{4\pi a^3}{V} \int_{-1}^1 \int_1^\infty P(a(w_1 + w_2))P(a(w_1 - w_2))(w_1^2 - w_2^2)dw_1 dw_2, \end{aligned} \quad (61)$$

where y in the above formula stands for the cartesian y coordinate and $w_1 = \cosh u$ while $w_2 = \cos v$. Again the probability of interference $P_{m_0}(|\mathbf{r}_A - \mathbf{r}_B|)$ of two pathways is given in terms of this 3D overlap probability p_b by (58).

As in the sharp cutoff constant density model, we expect that we have two phases assuming maximum pathway number. One is the ordered phase where pathway centers are not allowed to overlap, since in this phase two overlapping pathways share enough neurons to cause interference in their operation. This phase occurs when the pathways are 'dense'. The other phase is the disordered phase. In this case even when the pathway centers overlap, the number of common neurons in the two pathways is small and it is not sufficient to cause confusion. In this case the lattice structure is difficult to maintain, since two pathways can, on their own, come as close as necessary. In this phase, pathways can be randomly placed on the plane or 3D space up to a certain density of pathways that makes confusion likely. This second phase occurs when pathways are 'dilute' in the sense that within a neighbourhood of the pathway center a small fraction of the neurons belong to the pathway. As has become clear from the sharp cutoff constant density model, in this phase the information capacity is very large, increasing exponentially in the number of overlapping neurons.

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