Abstract

Quian Quiroga et al. [Nature 435, 1102 (2005)] have recently discovered neurons that appear to have the characteristics of grandmother (GM) cells. Here we quantitatively assess the compatibility of their data with the GM-cell hypothesis. We show that, contrary to the general impression, a GM-cell representation can be information-theoretically efficient, but that it must be accompanied by cells giving a distributed coding of the input. We present a general method to deduce the sparsity distribution of the whole neuronal population from a sample, and use it to show there are two populations of cells: a distributed-code population of less than about 5% of the cells, and a much more sparsely responding population of putative GM cells. With an allowance for the number of undetected silent cells, we find that the putative GM cells can code for $10^5$ or more categories, sufficient for them to be classic GM cells, or to be GM-like cells coding for memories. We quantify the strong biases against detection of GM cells, and show consistency of our results with previous measurements that find only distributed coding. We discuss the consequences for the architecture of neural systems and synaptic connectivity, and for the statistics of neural firing.

Key words: Neural representations; grandmother cells; distributed coding;

1. Introduction

Critical to understanding how information is processed in the brain is the form of the neural coding that underlies the storage and recall of memories. Is there a local, or gnostic (Konorski, 1967), code — colloquially called a grandmother-cell (GM-cell) representation — in which the firing of a single neuron (or group of neurons) exclusively codes recognition of a particular object, person or memory? Or is the code much more distributed?

Although it is generally accepted [e.g., Churchland and Sejnowski (1992)] that GM representations are not used in reality, experiments (Hahnloser, Kozhevnikov and Fee, 2002; Jung and McNaughton, 1993; Thompson and Best, 1989) often find localist responses by individual neurons. Most dramatically, Quian Quiroga et al. (2005) have recently found many neurons in humans that, within the limits of the measurements, behave like classic GM cells.

In this paper, we therefore quantitatively re-examine the viability of GM-cell representations, with the outcome that we refute the standard quantitative arguments against them, both theoretical and phenomenological. The information-theoretic argument is that GM representations need far too many neurons for the information coded (Rolls and Treves, 1998; Rolls, 2001; Churchland and Sejnowski, 1992). We show that this argument fails when one examines the information storage capacity of the synapses rather than the representational capacity of neurons for input stimuli. The standard efficiency argument applies only to the input representation, needed to represent any of the myriad possible stimuli. For storage, a GM representation can be optimally efficient.

The phenomenological argument is that GM cells should fire in response to a much smaller fraction of stimuli than has been deduced from measurements of neural responses (Hung et al., 2005; Abbott, Rolls and Tovee, 1996; Waydo et al., 2006). A GM cell can be regarded as a categorizer, and the data appear to imply that any apparent GM cell responds to many categories of stimuli rather than to one category.

However, our information theoretic argument shows that associated with any GM-cell population, with its ultra-low sparsity, is a more conventional population with a much higher sparsity. This two-population property, always a part of the GM-cell idea (Konorski, 1967; Page, 2000), was not allowed for in older analyses, including that (Waydo et al. 2006) by the group responsible for the new data (Quiroga et al., 2005). We devise a very general method of analyzing neural systems with multiple sparsities, and apply it to the data of Quian Quiroga et al. (2005). It enables us to quantify the biases against experimental detection of GM-like cells, most of which simply...
GM-like cells can be 10\(^5\) or more. Uncertainties are minor relative to the orders of magnitude involved. The data of Quian Quiroga et al. (2005) therefore appear in strong quantitative agreement with the GM-cell hypothesis.

The biases against detecting GM cells are enough to allow consistency with previous Abbott, Rolls and Tovee (1996), Waydo et al. (2006) measurements and analyses that use a single-population model and that quantitatively argued against GM cells. We will examine other arguments against GM representations in the Discussion section.

2. GM systems

Inappropriate or excessively rigid definitions can exclude biologically interesting cases. For example, Rolls and Treves (1998, p. 12) define a local representation as one where “all the information that a particular stimulus or event occurred is provided by the activity of one of the neurons”. The word “all” appears to exclude biologically interesting cases. Full conscious recall of one particular memory requires some extra cues and modulation. With a GM-like memory system, non-exclusive priming recall would be at a relatively low level of firing above some threshold, with full recall involving exclusive firing at a much higher level.

In this case the exclusivity is not between the actual firing of different GM groups, but between the concepts corresponding to the groups. Of course, the situation is a little more complicated for episodic memory, since episodes are happenings along a continuum in time. A memory cell for an episode corresponds to a small range in time. The exclusivity between different GM groups is between well-separated episodes, of which there are evidently a very large number.

Another natural generalization of the GM-cell concept is to local coding for output, with strong experimental evidence in the work of Hahnloser, Kozhevnikov and Fee (2002).

High-level GM systems v. low-level local coding We choose to restrict our use of the “GM-cell” terminology to the higher levels of neural processing. For lower levels, we will use the broader term “local coding”; by this we mean coding by a set of cells each of which is responsive to some patch of stimulus space (with fall off at the edges naturally). When the relevant properties of the stimulus are in a low-dimensional space — as for color or position in an environment — the collection of patches can give complete coverage. But when the relevant stimulus space is high dimensional, one can only expect local coverage of a minute fraction of possible stimuli — for example, to correspond to linguistic phonemes out of all possible auditory stimuli.
There is considerable evidence that can be interpreted as supporting some kind of local coding below the highest perceptual levels. The key question for us is whether local coding is also used at the highest levels.

Internal stimuli Input to a memory system need not correspond to actual physical events external to the brain. Some input can be completely generated internally, as with an author planning a novel. Memories of people and episodes in the novel have the same neural status as memories of real people and events.

2.2. Properties

One big computational advantage of a GM-like system is the simplicity of information flow, Fig. 1, so that the representation is easy to construct and manipulate — e.g., Gardner-Medwin and Barlow (2001); Baum, Moody and Wilczek (1988). New memories are formed with new or unallocated neurons, so that they interfere minimally with old memories (Quartz and Sejnowski 1997). The input to GM cells is from a distributed representation of the stimuli. Output to downstream neurons using the categorization can be simply taken from a single GM cell.

Above-threshold firing of a GM-cell models a property of the cause of the stimulus. Thus when firing is exclusive between different GM groups, this corresponds to exclusivity between the modeled properties of the associated external stimuli, like the identity of a person.

It is therefore tempting to use exclusivity of firing between different GM groups as the primary measurable criterion for characterizing GM-like systems. But natural generalizations to declarative memory motivate us to relax this criterion. For example, with episodic memories, a stimulus may cause a response in nodes for those episodes having important commonalities with the stimulus.

In addition, there can be multiple categorization systems, and there is no requirement of exclusivity between different systems. This is particularly clear at low levels in the processing hierarchy, where we could have separate local representations, for example, of the color and shape of an object. A collection of local representations of such relatively low-level features then forms a distributed representation of the whole object, suitable for input to the next level of hierarchical processing.

Note that we prefer to use the terminology “GM-like system” rather than “GM representation” to emphasize two aspects: The first is that if the GM-cell idea applies in its classic sense of recognition of individual people, it is likely to apply much more generally to all declarative memories.

The second is that we wish to treat the firing of a GM cell as coding the result of a recognition computation from a stimulus. But the use of the word “representation” for a GM-like system would carry the connotation of representing the stimulus, which is not generally appropriate. If nothing else, local coding typically dramatically fails to cover the stimulus space. For example, consider a distributed input representation on 100 binary neurons, a small number compared with real sensory systems. There are $2^{100} \approx 10^{30}$ distinct stimuli, many orders of magnitude larger than the total number of neurons in any brain. A practical local representation can only apply to a minute fraction of stimuli, presumably ones that are especially salient. A local representation can only provide full coverage for a stimulus space of very low dimension, like that for color.

2.3. Detection of GM-like systems

Practical experiments only involve a limited number of stimuli and cells, and definitely do not give the detailed synaptic information that determines all possible causes of a cell’s firing. Thus it is non-trivial to distinguish a GM system from a distributed representation, when the distributed representation is sparse, and when we allow natural generalizations of the GM-cell idea.

We illustrate the issues by comparing two very different computational memory models, one by Hopfield (2006), and one by Baum, Moody and Wilczek (1988) (BMW).

Hopfield’s model In Hopfield’s particular example, the input stimulus concerns properties of people, and the input representation is carried by a set of 1000 binary neurons. These are divided into 50 sets of 20 neurons. The 20 neurons in each set give a local representation of 20 possible values of a property of the stimulus. For example, to input the name of a person, one of a set of 20 name-coding neurons would be active. Binary synapses connect every neuron to every other neuron.

Each stored memory is considered as the set of 50 property values for a particular person, and is coded in the state of the synapses. The synaptic strengths are set by a Hebbian-like rule on presentation of stimuli.

Recall of a memory is caused by a stimulus that consists of partial data about an individual, i.e., values for a subset of the 50 properties. Memory retrieval results in completion of a partial stimulus to the full set of properties for the corresponding individual. There is no corresponding GM cell; the model is a fully distributed memory system.
BMW model The basic BMW model is the simplest possible form of a GM system: a feed-forward perceptron with one intermediate memory-cell layer, Fig. 2. The memory cells are arranged to respond in a GM-cell style to recognize inputs that correspond to stored memories. The model gains its power by applying it to the situation that the input forms a sparse binary representation of stimuli. This would typically be obtained from the top of a processing hierarchy, as in Fig. 1. Improvements in the model can be made, for example, by adding inhibitory interneurons to enforce a winner-take-all action in the memory-cell layer, but the simplest form of the model is sufficiently robust for our illustration.

To solve the same pattern-completion task as Hopfield’s model, we make the output cells identical to the input cells, thereby specializing from a heteroassociative memory system to a homoassociative system.

Memories are created by presenting a full stimulus to the system, and arranging for some unallocated memory neuron $\alpha$ to match its synaptic strengths to the stimulus. The chosen neuron changes to a state of being allocated, or it may be some full pattern, thereby specializing from a heteroassociative memory system to a homoassociative system.

Measurements To mimic a biological experiment, one could measure the responses of a sample of model neurons to a sample of stimuli. In Hopfield’s model if there are fewer than about 10 or 20 stimuli, the firing of many of the input and GM cells, and thereby distinguish the system from the sample to show that each neuron is responsive to multiple unrelated stimuli.

As for the BMW model, the input/output cells would have the same kind response characteristics as all the cells of the Hopfield model, responding to 5% of the stimuli. But there are also GM memory cells, which respond much more rarely. If the sample data include at least a few responsive GM neurons, one can detect the different properties of the input and GM cells, and thereby distinguish the system from the Hopfield model.

In this paper, we will construct a method to extrapolate in general from data with a sample of cells and stimuli to the whole system. The method enables us to compute which of the following properties of a set of putative GM cells is consistent with data: (a) The cells actually could code for single properties. (b) Each cell is expected to respond to
multiple unrelated properties, after an extrapolation to the full set of possible stimuli.

One confounding issue is that if one detects a response from a GM-like cell it is easy to misidentify the property corresponding to the cell’s firing. If a cell only responds, within limited data, to a particular individual person, the cell could indeed be a classic GM cell corresponding to that person. But it could also be, for example, a GM cell for an episodic memory that contains the person. In that case it would respond to stimuli containing other components of the episode.

The distinguishing feature of the most general kind of GM-like cell, but one that can be hard to test, is that apparently different stimuli that cause it to respond are in fact related. In contrast, with a purely distributed representation at the highest level, there is no high-level relation between, for example, the multiple people causing a particular cell to respond. The only relation is an identity of lower-level features in the input representation. The locality is at the feature level, not at the high level.

3. Information requirements for pattern recognition

We now quantify the information requirements for a memory or categorization system. First we distinguish the activity state and the storage state. The activity state is the pattern of firing of the neurons, and the storage state (Churchland and Sejnowski, 1992) is the pattern of synaptic connectivity and strengths.

Furthermore, within the activity state of a memory system, we distinguish an input representation and a recognition representation. The input representation is of the current input stimulus, like a visual scene, while the recognition representation concerns which stored pattern (if any) corresponds to the current input.

3.1. Input representation

The immediate input to a memory system must be able to represent the relevant features of any possible stimulus, and not just those previously encountered stimuli for which there is a memory trace. Here the standard arguments for distributed representations apply unambiguously, and a GM representation is not possible. The argument is simply that \( N \) neurons code for at most \( N \) exclusive properties in a GM system, but that they code for exponentially more with a distributed representation, for example \( 2^N \) with a simple binary code.

In general the immediate input to a memory or categorization system is not the raw sensory input but a highly processed representation of those high-level features that are relevant for the system’s particular task. For example, input for face recognition could involve neurons coding for the presence, shape and position of eyes, nose and mouth, etc; individual features could well be coded locally, but the collection of feature representations would always form a distributed representation. Each neuron has a range of distinguishable firing rates, so that the raw information capacity in the activity of \( N \) neurons is a few times \( N \) bits. But robustness requires a certain amount of redundancy, and firing is often sparse, both of which reduce the information per neuron. Measurements by Abbott, Rolls and Tovee (1996) show that in hippocampal neurons about 0.3 bits of independent information are coded per neuron and suggest that a few hundred neurons suffice for coding possible faces. For example, 300 neurons code about 100 bits, sufficient for about \( 2^{100} \approx 10^{30} \) different categories of face, an entirely satisfactory number.

Note that once an input representation is sufficiently small, pure representation efficiency is not a dominant consideration. Issues of processing speed, metabolic efficiency, and algorithmic robustness can be more important. For example, sparse distributed representations appear to be favored by Rolls and Treves (1998), since they can give weak interference between memories during synaptic plasticity.

3.2. Storage in synapses

The information capacity in the storage state, i.e., in the synapses, was estimated by Chklovskii, Mel and Svoboda (2004) and by Stepanyants, Hof and Chklovskii (2002). There are two contributions, from the synaptic topology and from the synaptic strengths, giving a total of 5 to 10 bits of raw storage capacity per synapse. We divide by 10, to provide a plausible allowance for redundancy. Thus we need about one synapse for each bit of storage information.

This calculation uses only very basic physical information about synapses and neural processing, so it is certainly accurate at the order of magnitude level. Since measures of information in units of bits are independent of the physical implementation, the numbers are directly compared with those for ordinary digital computers, and experience with data processing and storage can be used to derive minimum numbers of synapses for a task. A human brain has around \( \left( \frac{10^{11}}{10^4} \right) \) neurons and \( 10^4 \) synapses per neuron. So its synapses store about \( 10^{15} \) bits, i.e., about \( 10^9 \) GByte, up to a factor of 10 or so.

3.3. Recognition representation and total stored information

Suppose the system has a repertoire of \( R \) stored memories. Each is an arbitrary association of a category to stimulus features. So we attribute to each memory \( A \) bits of association information, which we term its semantics. This includes both input and output information. (E.g., facial structure and name for a person, and, in fact, all remembered information about the person.) It is important to include output semantics in the associations, since they are what allow the retrieval of a particular memory to cause memory-specific behavior. These are quite arbitrary asso-
associations, for example of a linguistic name to a specific person, and without such output associations there is no purpose to storing a memory. The output associations do need to be stored, and they therefore contribute to the calculation of the minimum size of the system just like the input associations.

The total association information is \( RA \) bits, so that approximately this number of synapses is needed. For example, with a repertoire of \( R = 5000 \) and an average of \( A = 10000 \) bits of information per item, we need about \( 5 \times 10^7 \) synapses. The measured number of synapses per neuron is around \( 10^4 \), thereby implicating about \( RA/10^4 \) neurons, i.e., a number proportional to the size of the system’s repertoire (and the information per memory trace).

Hence if the system uses \( R \) GM cells for a recognition representation, this is at most a constant factor beyond the neurons needed to carry the storage synapses. Moreover, if \( A \) is larger than about \( 10^4 \), there is not even any overhead at all in using GM cells. We can characterize this by saying that if the memories are semantically rich, then a GM strategy for the recognition output can be optimally efficient, as in the model of [Baum, Moody and Wilczek (1988)].

In contrast, if one ignored the storage requirement, one would assert that \( N \) recognition neurons can code exponentially more recognized categories, e.g., \( 2^N \).

It is important that in quantifying the information the term “bit” is used in the strict information theoretic sense. This means that if each memory were coded as an actual bit pattern, each of the \( 2^A \) possible patterns would be equiprobable. Thus, on a computer, the bit count refers to an optimally compressed representation. However, only certain features of the input are relevant for tasks like face identification: a minimalist line drawing often suffices for unambiguous identification. So in computer terms the association bits are with respect to a representation that is both lossy and compressed. In the opposite direction, it can be difficult to perform computations on optimally compressed representations, and it is also difficult to measure accurately the probabilities of occurrences of different kinds of stimuli, since the number of possible stimuli far exceeds the number actually experienced. Moreover redundancy (in the information theoretic sense of using more than the minimum necessary number of bits) is useful in giving robustness to a system. For all these reasons, we must expect the physical capacity of a system needed to code memories to be a substantial factor larger than a minimal physical implementation of \( RA \) bits. Nevertheless this measure is important in quantifying information in an implementation-independent way. It also enables us to estimate the information storage requirements by examining implementations of related tasks on a digital computer.

Well-known examples of simple line drawings and pictures of artificially low resolution show that the information to identify faces could be quite modest, if a suitable representation is used. But the synaptic size of the system also depends on the remaining association information, treated as output. This can be much larger in size, effectively amounting to a biography of the individual concerned in each memory.

3.4. Objections and answers

A number of objections to our bounds and ideas for evading them have been proposed, which we now answer. The general answer is simply that the information theoretic bound represents an absolute physical limit that it is impossible to exceed. All that is required is that we count the bits of information in the strictly correct information theoretic sense, and that we have identified the correct physical location of memory in the synapses.

Counting memories Does not the idea of quantifying memories as discrete items that can be counted carry the implication that we use a GM system? Are there not difficulties in counting memories in distributed memory systems? In fact the classical kinds of distributed memory, e.g., [Hopfield (2006); Treves and Rolls (1991, 1992); Amit, Brunel and Tsodyks (1994)], are regarded as storing patterns, which can be counted. What we do have in mind is declarative, or explicit memory, the kind considered as prototypically hippocampal. Here it is reasonably clear what is meant by a single memory: a picture, a scene, or the meaning of a word; all of these are discrete. Even with episodic memory, where there is a continuous time variable, we can observe that there is a correlation time within a continuous series of events. As regards storage requirements, we simply identify a single memory with the happenings within a correlation time, which is evidently of the order of seconds or minutes. Of course, only a small fraction of these are stored in long term memory. We will not require great precision here.

In the contrasting case of implicit procedural memory, it is much less obvious what should be defined as a single memory item. But we are not concerned with this case.

Multiplexing Could not one gain by allowing a neuron to respond to multiple different stimuli? Could not a single face-identification cell respond to either George Bush, Jennifer Aniston or John Hopfield, for example? This multiplexing is just going in the direction of a distributed representation. Our argument so far does not rule that out; all it says is that this does not provide a way of reducing the synapse count. It is our statistical argument in later sections that enables us to estimate the degree of multiplexing.

Now if a neuron responds to multiple categories, then there is interference at the neural level between different memory traces. An unambiguous categorization then requires the use of the firing information from more than one neuron. In the case of lightweight memories, i.e., with substantially less than 10000 bits of associations per memory, multiplexing of memories can indeed reduce the neuron count, and is allowed by our general argument. But with richer memories, there is no gain.
Representation v. memory The memories we have discussed are typified by hippocampal memories. Consider instead visual area V1; the number $P$ of distinguishable activation patterns is exponential in the number $N$ of neurons, e.g., $P = 2^N$. An outside observer could identify different faces from the different activation patterns. Why should we not regard this as memory, if we are to regard observation of (much simpler) activation patterns in GM cells as identifying faces, and as in fact part of a memory system?

The difference is not in the outside observations, but in the use the organism itself makes of the information. A memory is not useful unless it produces some consequence when recalled. What we mean, for example, is that seeing John Hopfield on the other side of a street might induce us to cross the street and greet him by name. For the billions of other possible people, there would be either be either a different response or no response at all.

This is why we defined the associations for a memory to include output as well as input. There must be sufficient information stored to enable the relevant computation to be done from the activation pattern corresponding to the stimulus.

Without being concerned with storage, it is perfectly possible to compare one activation pattern with a previous one, to identify whether or not it has changed. But to compare it with patterns of activation for all the people one remembers, and to take appropriate actions, one needs appropriate storage. It is simply not possible to evade the fundamental physical necessities given by information theory.

Coding commonalities Many remembered faces have features in common. Cannot this be used to reduce the number of synapses and neurons needed, by coding the common features in a special subsystem for the common features? Suppose we had a set of faces characterized by very short, dark, and curly hair. Would there not be a gain by allocating a neuron to this combination of characteristics and using it instead of separate neurons for hair length, color and curliness?

If the different hair features were equiprobable and uncorrelated, the general argument would prevent any gain. As an example, suppose each of the three hair characteristics has 8 equiprobable values, for $8^3 = 512$ distinguishable combinations. If all the combinations were equiprobable, we would allocate 9 bits for the information content. Probability here refers to a prior probability of occurrence, i.e., before the creation of a memory trace.

But if instead all the characteristics were perfectly correlated, then there would only be 8 combinations, which could be represented in 3 bits. When we construct memories, this gives a gain of a factor of 3 in storage if we only represent the combinations that actually occur in the input representation rather than all possible combinations.

But this is exactly what is meant by using a correct measure of information to compute the minimum number of synapses. Note that the task carried out by a memory system is not merely to identify the best fit to a current stimulus among stored memories; for that very few bits are needed. For example, if a stimulus is represented by 100 bits, but only 8 memories are stored, then only $\log_2 8 = 3$ suitably coded bits are needed to identify the stimulus, if it is assumed that the stimulus corresponds to one of the memories. But it is also necessary to identify the case that the stimulus fails to correspond to a stored memory, so that it is a candidate for a new memory. It is for this that the other 97 bits are needed.

3.5. Comparison with models

Although our argument was used to show that GM coding can be optimally efficient in the use of synapses and neurons, the bounds on synapse and neuron number are independent of the coding method. It is therefore useful to verify that the bounds are obeyed by the polar opposite of GM systems, i.e., by conventional distributed memory systems. Calculations of the capacity of such systems have been made, e.g., [Treves and Rolls (1991, 1992); Amit, Brunel and Tsodyks (1994)], and it can be checked that they do obey our bounds. But what appears to have been missed is that the capacity limit also removes the argument against using GM cells.

Hopfield’s recent model (Hopfield, 2006) provides an excellent example. In this model, each stored memory corresponds to the values of each of 50 categories, with 20 possible values per category. This gives a total of $50 \log_2 20 = 216$ bits of information. Retrieval of a memory results in a neural representation of this information in the firing of 50 out of 1000 binary neurons, for a biologically realistic sparsity of 5%.

Storage is in binary synapses with all-to-all connectivity on the 1000 neurons, for a total of slightly under $10^8$ bits of storage capacity. This implies that the system can store at most $10^8/216 \approx 4500$ separate memories. In fact, the simulations in Hopfield (2006) show that, with the algorithms used in that paper, performance noticeably degrades when about 250 memories, i.e., a factor of 20 below the physical limit. Thus the information-theoretic bounds are obeyed. The problem is that the memories are stored on the synapses connecting the active neurons in a particular memory. Synapses overlap between memory traces, which causes interference if too many memories are stored.

Consider in contrast the BMW model (Baum, Moody and Wilczek, 1988), which, as observed by its authors, is optimal in its number of synapses. Suppose for input and output we use the same 1000 neurons as in the distributed model. Then to store $N$ memories we add $N$ memory neurons and 2000 $N$ synapses (for input and output). We also may use a relatively few extra interneurons and synapses to implement winner-take-all dynamics.

The number of synapses is double our minimum estimate, because we treat input and output semantics separately: they could be different. This is much more efficient than the distributed model. There is also an extra neurons for each
memory. But we can readily increase the capacity for stored memories by increasing the number of memory neurons. With \( N = 500 \), we would have the same number of synapses as in Hopfield’s model, 50% more neurons, but double the capacity.

With the distributed model, the capacity can be increased only either by simply duplicating the system, which is always a possibility, or by a change in architecture. An appropriate change in architecture would be to use the original input neurons to feed a separate layer which codes the same information more sparsely, after the style of a support vector machine.

Note that the BMW model reliably performs the same pattern completion task as Hopfield’s model. That is, a stimulus consisting of a few values is completed to the values of all categories for the corresponding memory. The BMW model essentially performs a comparison of the active bits in the sparse input pattern with the on-bits in the stored pattern. Because of the 5% sparseness of the input, the probability that an on-bit in the stimulus coincides with an on-bit in an unrelated stored pattern is also 5%. Once more than a few bits are examined, the probability of a chance coincidence is extremely small, with a correspondingly small misidentification probability.

4. Statistics of neural responses

4.1. Two-population property of GM systems

We have seen that the efficiency argument against GM cells disappears, especially for semantically rich objects, like most people’s grandmothers. But the efficiency analysis for the input representation shows that the GM cell population is necessarily accompanied by a population of cells carrying a distributed code.

Experimental characterizations of the different kinds of coding can be made by measuring the sparsity of neural responses to stimuli.

By sparsity we mean, for each cell, the fraction of stimuli to which it responds. We assume that, as in Quian Quiroga et al. [2005], some threshold criterion is defined cell-by-cell as to whether a cell responds or not. Thus the neuron is treated as binary. Other definitions involving analog firing rates are possible, but we will not use them. Note that with our definition and when all cells have the same sparsity, both the population and the lifetime sparsity are equal, unlike the case (Willmore and Tolhurst, 2001) with other definitions of sparsity.

For a system with fully distributed coding, we expect to measure sparsities characteristic of the input and output representations. For example, in Hopfield’s model, the sparsity is exactly 5%. More realistically, there will be a range of sparsity.

The input and output cells of a GM system will have naturally have similar sparsities to those of all the cells in a distributed-memory system. But the GM cells must respond much more rarely. So with a GM system we expect there to be two very different populations of cells distinguished by one population having a dramatically smaller sparsity than the other. Whether or not the two populations are in the same area of the brain is not determined by general arguments. But we will find that, in fact, the putative GM cells of Quian Quiroga et al. [2005] do have an accompanying distributed-code population. For our purposes it will be unimportant whether the detected distributed-code population is the one that provides the actual input and output for the detected GM-like cells.

Expectations for the sparsity of GM cells can be provided in terms of the repertoire size of a system, which has a connection to behavioral data.

There are two somewhat different kinds of memory system we will consider. One is typified by face recognition, where a recognized input is categorized into one of \( R \) categories, corresponding to distinct persons; recognition is exclusive between categories. Then for a random sample of faces in the repertoire of the system the sparsity of the GM cells is \( 1/R \); if we assume that each person is allocated the same number of cells.

The second case is for declarative memory (episodes, facts, etc). Recall is by a stimulus containing a few components of the original memory. Since the same components could be part of other memories, the pattern of recognition firing need not be exclusive between memories. Let us regard these patterns as priming the recall of the memories. Full conscious recall of one particular memory requires some extra cues and modulation. With a GM system, non-exclusive priming recall would be at a relatively low level of firing above some threshold, with full recall involving exclusive firing at a much higher level.

In a memory system, a typical stimulus can evoke multiple memories. If we let \( n_m \) typify the number of memories evoked, then a given GM neuron is caused to fire by a fraction \( n_m / R \) of stimuli. It is therefore convenient to define an effective repertoire size \( R_{\text{eff}} = R / n_m \), so that the typical sparsity is \( 1/R_{\text{eff}} \).

From standard psychological data, we envisage that \( R_{\text{eff}} \) is thousands to at least millions for interesting cases.

In contrast, the distributed-code cells fire much more frequently; this is known from data, and is necessary in order that this population can represent a sufficiently large number of stimuli.

4.2. General form of distribution of neural responses

Measurements of single-cell responses concern only a small fraction of cells and of all possible stimuli. So we will treat data as being from a sample over cells and stimuli, and deduce properties of the whole system: e.g., the relative sizes of the cell populations and their sparsities, and hence the number of categories coded for by the GM population. In doing this, we will quantify, and hence compensate for, the strong biases against detection of GM cells.
Suppose we present a sample of $p$ stimuli that are randomly chosen from some broad class (e.g., pictures of famous people, images concerning movies that the subject has watched, pictures of buildings). Any particular cell $i$ responds to some fraction of these, called the cell’s (lifetime) sparsity $\alpha_i$. The number $n_i$ of stimuli that evoke a response by the cell is taken from a binomial distribution of mean $\alpha_i n_i$:

$$P(n_i) = \binom{n_i}{p \alpha_i} (1 - \alpha_i)^{n_i - p \alpha_i} \frac{p^i}{n! (p - n)!}.$$  

This simply corresponds to the probability that $n$ of the stimuli are in the response-causing class and $p - n$ are in the non-response-causing class, as regards cell number $i$. These two classes of stimuli form fractions $\alpha_i$ and $1 - \alpha_i$ of the whole set of stimuli.

We now consider a sample of cells, thereby sampling the distribution of sparsity over cells, $D(\alpha)$. This means that the fraction of cells with sparsity $\alpha$ to $\alpha + \,d\alpha$ is $D(\alpha)\,d\alpha$. Then the probability of getting $n$ responses to the $p$ stimuli in some random chosen cell is obtained by integrating the single cell response with the sparsity distribution:

$$P(n|p) = \int_0^n d\alpha \; D(\alpha) \alpha^n (1 - \alpha)^{p-n} \frac{p^i}{n! (p - n)!}.$$  

This is a general result. The only necessary assumption is that the cells are randomly chosen out of some more global set of neurons (e.g., hippocampus) and that the stimuli are randomly chosen out of some global class.

The value of $\alpha$ for a cell and the distribution $D(\alpha)$ depend both on the choice of stimulus class and on the choice of the threshold for a response. Changing either will naturally affect the distribution. For the data we analyze, the response criterion is given in Quian Quiroga et al. (2003).

If multiple sessions and multiple subjects are considered, Eq. (3) continues to apply, with $D(\alpha)$ being the distribution averaged over subjects. So this form is amenable for the analysis of aggregated data.

Observe also that the derivation of the formula does not require any assumption about the independence of the firing of different neurons: the formula is simply an average over all neurons in whatever area is being sampled. This allows the formula to be completely general, in contrast to the model of Wavdo et al. (2006), which requires that neuron-neuron correlations be neglected.

A common ansatz, as in Wavdo et al. (2006), is to assume a fixed sparsity $\alpha_i$, i.e., to set $D(\alpha) = \delta(\alpha - \alpha_i)$. Such a model we term a single-population model. But for an analysis of a possible GM population, we must allow for at least two populations.

From a mathematical point of view, Eq. (3) expands $P(n_i)$ in basis functions, with expansion coefficients $D(\alpha)$. The significance to its use is four fold: (1) It relates the distributions for different pattern numbers $p$ via a common set of expansion coefficients $D(\alpha)$, (2) The expansion coefficients are non-negative, (3) For a distributed-code population to represent all possible stimuli, efficiency is important, i.e., using the minimum of neurons. This will tend to maximize the sparsity subject to other constraints like keeping relatively low the metabolic costs (Lennie, 2003) of action-potential generation. Thus we should expect the sparsity of the distributed-code population to vary over a fairly narrow range. (4) Any GM population has an extremely small sparsity, so that it populates just the bins with $n = 0$ and $n = 1$ responses to the $p$ stimuli.

Therefore the two population property leads to the qualitative expectation for $D(\alpha)$ that is shown in Fig. 3(a). The distribution of responses $P(n_i)$ can be regarded as a smeared version of the sparsity distribution $D(\alpha)$ with $\alpha = n/p$. Given this smearing, a useful approximation is to replace the distributed-code peak by a delta function at some fixed typical sparsity, Fig. 3(b).

4.3. Useful approximations

Although numerical work can always be done with the linear combination of binomial distributions Eq. (3), we find it convenient to use one of two approximations. First, they allow simple analytic calculations, with a consequent ease of understanding what features of the data are important in determining particular parameters in a model of the sparsity distribution $D(\alpha)$. Second, they also exhibit that for sufficiently small sparsity there is a degeneracy in fitting $D(\alpha)$: only certain combinations of model parameters are determined. We verify that whenever we use these approximations in our fits, they agree sufficiently accurately with the underlying binomial distribution.

When the sparsity is small, the binomial distribution for a cell’s responses is approximately Poisson:

$$P(n_i, cell_i) \simeq (p\alpha_i)^n e^{-p\alpha_i} \frac{1}{n!}.$$  

This is derived by the use of Stirling’s approximation, and is valid when $\alpha_i \ll 1$ and $p \gg 1$, which is true in all the cases we treat. The approximation depends only on the product $p\alpha_i$ and not on $p$ and $\alpha_i$ separately.

When the sparsity is so small that we can neglect the probability of getting two or more responses, we can use what we call the GM-cell approximation:
\[ P(n, \text{cell}) \simeq \begin{cases} 
1 - p/R_{\text{eff}} & \text{if } n = 0, \\
p/R_{\text{eff}} & \text{if } n = 1, \\
0 & \text{otherwise,} 
\end{cases} \tag{5} \]

where we have replaced the ultra-small sparsity \( \alpha \) by \( 1/R_{\text{eff}} \) to relate it to our expectations for the sparsity of GM cell responses.

5. Data analysis

We now analyze the measurements by Quian Quiroga et al. (2003). For each of their experimental subjects, there was first a screening session in which a large number of disparate images were presented. This was sufficient to detect responsive cells, but not to measure their selectivity. Then there was a testing session that probed the selectivity by using many different images of the same people and objects to which responses were found in the screening session. We will find useful population information from the screening session data alone.

It would be a big mistake to include the testing session data in our analysis, since the images for a testing session were systematically chosen to concern people and objects whose images evoked a response in the previous screening session. The distribution \( D(\alpha) \) would be different in the two sessions. For example, suppose that in the screening session three images of Brad Pitt, Jennifer Aniston and Halle Berry evoked responses from three classic GM cells. These cells have a very small sparsity \( 1/R \) with respect to images of people, with \( R \) being the subject’s repertoire for recognizing faces. Then in a testing session that uses equal numbers of images for just these three people, each of the three GM cells would respond to one third of the images. Thus with respect to the new, specially chosen stimulus class, these cells have sparsity \( 1/3 \).

We use the following data:

- Recordings were made from 343 single units and 650 multi-units. Given the substantial number of single units, we propose that the multi-units on average correspond to 5 neurons, to give a total of approximately 2000 cells, 250 in each of 8 patients.

The number 2000 is for cells that produced some detectable signal. However there are many more cells that are within range of detection by the extracellular electrodes used that failed to give any identified action potentials (Quian Quiroga, private communication and Waydo et al. [2000]; Henze et al. [2000]; Buzsáki [2004]). Thus we should increase the number of cells by some factor \( K \), whose value we will estimate later. Then the total number of cells available for detection is 2000\( K \); any of these would have been detected if it had given action potentials at rates comparable to that of the actually detected cells. Our numerical results will have a very simple scaling with \( K \).

- There were on average \( p = 93.9 \) stimuli in each screening session.

- A total of 132 units produced a response above threshold.

- Of these, 51 were candidate GM cells, i.e., they responded to a single image within the screening session.

- The remaining 81 were not so highly selective.

- On average, the responsive units responded to 3.1% of the presented images, i.e., to 2.9 images.

Given the low fraction of responsive units, we assume that an above-threshold response from a multi-unit is a response from one particular cell.

We will analyze the data with the aid of our general expansion, (3). Since we wish to test compatibility with the GM cell hypothesis, we arrange our analysis without any initial assumption about the necessary existence of GM cells:

(i) First we attempt to make a fit with a conventional distributed-code model with a fixed sparsity.

(ii) When we find this fails to be a good fit, we add a second component of different sparsity, as a minimal model to fit the data.

(iii) The second component turns out to have such small sparsity that only its responses for \( n = 1 \) are significant.

(iv) This is suggestive that there are indeed cells that approximate GM cells. So we reanalyze the data in terms of a model of GM-like cells together with a distributed-code population, so as to determine appropriate properties of the GM-like population. This makes it easy to allow for issues like the stimulus being or not being in the system’s repertoire.

5.1. Single distributed-code population

We first try a model of a single distributed-code population with a single sparsity \( \alpha \). This is the model (Rolls and Treves, 1998) normally used in theoretical work on autoassociative networks. It corresponds to a term \( f_D \delta(\alpha - \alpha) \) in the general formula (3). Here \( f_D \) is the fraction of cells in this population, with the remaining neurons being silent. (The most conventional versions, as in Waydo et al. [2000], assume \( f_D = 1 \).) In the Poisson approximation, the probability that a particular neuron is in the distributed-code population and that it fires in response to \( n \) out of \( p \) presented images is

\[ P(n & D) \simeq f_D (pa)^n e^{-pa} \frac{1}{n!}. \tag{6} \]

In view of a possible GM-cell population, which would appear almost entirely at \( n = 1 \) and \( n = 0 \), we fit the two parameters of the population with data from those cells that give \( n \geq 2 \) responses. In App. A we give more details of the model including its later elaboration to include a GM population, and obtain formulae for two measurable quantities. One, \( P(n \geq 2) \), is the probability of getting 2 or more responses from a cell; its experimental value is 81/(2000\( K \)). The second quantity is the mean value of \( n \) for these cells, which we write as \( \langle n \rangle_{n \geq 2} \); its experimental value is obtained from
The measured number of cells in the $n = 1$ bin is 51, far in excess of the extrapolation. Even if there is a distribution of sparsity for different cells in the distributed-code population, this cannot change this deduction greatly: a cell that fires in response to at least several percent of stimuli is likely to be detected, and relatively few distributed-code cells give just $n = 1$ and $n = 0$ responses to the $\sim$ 100 presented stimuli. We also note that only around 2% of the distributed-code cells fail to get detected; these are the cells in the $n = 0$ bin. When they are in range of the electrodes, detection of the distributed-code cells is almost unbiased.

We deduce from the excess at $n = 1$ that there is strong evidence for a second population of ultra-sparingly firing cells, just as predicted by general considerations if there is a GM system. The null model, with only a distributed population of cells together with completely silent cells, appears to be ruled out. Fig. 4 and our conclusion about the $n = 1$ excess are independent of the number of undetected silent cells. They are also independent of any assumption that the cells in the second population are actual GM cells.

Similar evidence for an excess of sparsely firing cells has perhaps been found by Barnes et al. (1970). Their Fig. 8 shows an excess for some but not all hippocampal-related areas in the rat.

Of course, a better extrapolation could be made if experimental values of $P(n)$ as a function of $n$ were available. We could imagine several populations of input cells, activated by different kinds of image, so an improved model is a combination of several distributions of the form (6), as in Eq. (3).

It has been said that in order to deduce the two population property from a plot such as Fig. 4, the distribution must necessarily be bimodal. Obviously, if we have a bimodal distribution, the inference would be cleaner and without theoretical prejudice. We illustrate this in Fig. 5, where we apply a two-population model described below to predict the responses to $p = 500$ stimuli. There is a GM-cell response that remains at $n = 1$. With a more limited set of stimuli, we need a theoretical expectation for the distributed-code population to extrapolate to $n = 1$ from the data, so as to quantify a possible excess at $n = 1$. Note however that if the distributed-code population had a distribution of sparsity rather than one fixed sparsity, the peak in Fig. 5 would be spread out.
5.2. Initial two-population model

To fit the data we need a minimum of one more population of cells, evidently of much lower sparsity than the first population. Without making any initial hypothesis about its GM-like nature we try the following ansatz for the sparsity distribution:

\[ D(\alpha) = (1 - f_D) \delta(\alpha - a') + f_D \delta(\alpha - a). \]  \hspace{1cm} (12)

where we preserve notation from the previous section, and resolve the ambiguity between exchanging the definitions of the two populations by requiring \( a' < a \). This model has three parameters to be fit to three available measured quantities: the fraction of cells with 1 response, the fraction of cells with 2 or more responses, and the mean number of responses. Within the Poisson approximation, which is always good, and with the restriction to the 2000 detected cells, we need to solve the following equations

\[ \frac{N_1}{N} = (1 - f_D) x e^{-x} + f_D y e^{-y}, \]  \hspace{1cm} (13)

\[ \frac{N_1 + N_{ \geq 2}}{N} = 1 - (1 - f_D) e^{-x} - f_D e^{-y}, \]  \hspace{1cm} (14)

\[ \langle n \rangle_{n \geq 1} = (1 - f_D) x + f_D y. \]  \hspace{1cm} (15)

From the data, we use \( N = 2000, N_1 = 51, N_{ \geq 2} = 81, \) and \( \langle n \rangle_{n \geq 1} = 2.9 \). The fit parameters are \( f_D \) and the combinations \( x = pa' \) and \( y = pa \). From this we find

\[ x = 0.0224, \quad y = 3.717, \quad f_D = 0.95, \]  \hspace{1cm} (16)

so that the sparsities are

\[ a' = 2.3 \times 10^{-4}, \quad a = 0.039. \]  \hspace{1cm} (17)

The sparsity of the extra population is so low that it populates only the \( n = 1 \) (and \( n = 0 \)) bins to a good approximation, even though it concerns 95% of the neurons. Thus the properties of the higher sparsity population are essentially unchanged from the single-population fit, which confirms our original choice to fit it to the data concerning cells with 2 or more responses to stimuli.

It now becomes useful to analyze the lower sparsity population in terms of a GM-cell approximation (5). This will give us a simple way of determining whether the population’s properties are appropriate for true GM cells. Very importantly, it will also give us a simple way of treating certain variations in the population’s properties that are appropriate in the GM-cell context, and of allowing for the silent-cell correction factor \( K \).

5.3. Detailed two-population model

We therefore model the responses of the cells by a population that uses a conventional distributed code supplemented by a possible GM-cell population, as illustrated in Fig. 6, these comprising fractions \( f_D \) and \( f_{GM} \) of the total number of cells. The remaining cells do not respond to any stimuli at all in the class used (which we label as “faces”, even though some stimuli used in Quian Quiroga et al. (2005) were of other kinds); this fraction \( 1 - f_D - f_{GM} \) is completely silent for the purposes of the experiment. We let \( k \) be the fraction of the stimuli used which correspond to memory in the GM population.

Our model corresponds to a case of the general expansion Eq. (3), in which we use just two delta-functions in \( D(\alpha) \), as in Fig. 3, with the GM-cell approximation (5) used for the low sparsity population. To this we add a possible population of absolutely silent cells, i.e., a term in \( D(\alpha) \) at exactly \( \alpha = 0 \).

As explained earlier, we let \( R_{eff} \) be the effective size of the population’s repertoire, i.e., the repertoire \( R \) divided by the number of simultaneously evoked memories. For classic GM cells \( R_{eff} = R \), of course. Then a randomly chosen GM cell has a sparsity \( k/R_{eff} \), i.e., the probability for the image to be in the system’s repertoire times the probability to respond to an image in the repertoire. When 2000K cells are each presented with (an average of) \( p = 93.9 \) images of different people or objects, the expected number of GM-like responses is therefore \( 2000 \times K \times 93.9 \times f_{GM} \times k/R_{eff} \). This number we found to be 43. In fact the choice of images was made after interviews with the subjects (Quian Quiroga, private communication), to put them in the repertoires of the subjects, so we now set \( k = 1 \), to find

\[ R_{eff} \approx 4400 f_{GM} K. \]  \hspace{1cm} (18)

Now \( f_{GM} \) is less than \( 1 - f_D = 0.95 \) to 1, depending on the value of \( K \). It is useful to define \( \hat{R} = R_{eff}/(K f_{GM}) \), which is the combination of parameters actually determined by data. Then \( R_{eff} < (1 - f_D) \hat{R} = 4400 K - 200 \).

If we ignored the silent-cell issue and set \( K = 1 \) we would find that at most 4200 categories are coded for. Now the detected cells are in areas like the hippocampus that obviously perform additional functions besides face recognition. Also, the repertoire of humans for faces and many other categories is very much larger, as measured behaviorally (Dudai, 1997). This would appear to imply that the detected GM-like cells are not classic GM cells. However:

– The hippocampus is not the ultimate store of long-term memory, so that the repertoire should be perhaps only a
year’s worth.

- The fraction of GM cells, \( f_{GM} \) for the chosen stimulus class could be less than \( 1 - f_D \). The remaining cells would not respond to any stimuli in the class (e.g., images of faces); they might be distributed-code cells or GM cells for other classes of stimuli. Extrapolating results on images of faces to other stimuli is sensible, so we would expect the vast majority of cells, up to a few percent corrections to be in whatever GM-like population is appropriate, so that the upper limit on \( R_{\text{eff}} \) found above is appropriate to be applied to the whole GM population and not just those of the facially-responsive kind.

- Very familiar people might have more neurons allocated to them, as required by the storage requirements, and some of these could be GM cells. So the assumption of equal GM-cell populations for each category could easily be false, so that the estimate of categories (e.g., \( 4200K \)) is only about those categories which are highly familiar to the subjects, which brings improved plausibility.

- There could be an experimental bias in cell selection. We made a basic assumption that the cells probed were a random sample in these areas. But the properties of epilepsy and the selection of suitable subjects for study might be such that the electrodes are in areas preferentially responsive for visual images of people. In that case, the category count refers to a restricted set of stimuli, improving the plausibility.

- An interesting possibility is that the cells are not classic GM cells but code for memories in a GM style, as in the model of Baum, Moody and Wilczek (1988). This is of course a natural hypothesis for the hippocampus, if we grant the GM cell idea at all.

Then \( 1/R_{\text{eff}} \), as estimated above, is the average fraction of recent memories that concern the people pictured in the images. \( R_{\text{eff}} \) will be biased to relatively low values since the images were chosen to be of people well-known to the subjects.

If the cells were classic GM-cells, we can estimate the number of cells per exclusive category. There are roughly \( 10^7 \) cells in one region of the human hippocampus. Dividing by \( 4200K \) gives the number of cells per category, about \( 2500/K \). With \( K = 1 \) this appears rather large. But with the large value of \( K \) that we will calculate later, the number is quite modest.

Finally, from the fraction \( f_D = 4.6\% / K \) of non-GM cells, we deduce that they number about \( 5 \times 10^5 / K \). An undoubtedly excessively simple idea is to identify them with the input representation. This number could obviously much higher than the few hundred that are perhaps needed at a minimum for coding features relevant for face identification. But there must obviously be many other specialized and less specialized kinds of distributed input representation, as well as distributed output representations.

Beyond approximating the general sparsity distribution \( D(\alpha) \) by a sum of a small number delta functions, our calculations used the Poisson approximation for the distributed-code population and the GM approximation for the ultra-low sparsity population. We have verified that using the full binomial distribution does not significantly affect the results.

5.4. Silent-cell correction

The experimental estimates of the numbers of single and multiple units in Quian Quiroga et al. (2005) depended on detection of spikes from the neurons, even if the spike numbers never passed the limit for the defined threshold for a responsive neuron. However, within detection range of the extracellular electrodes used are many cells that never give a detected signal. Waydo et al. (2006) state that in the data 1–5 units are identified per electrode and they cite Henze et al. (2000) for an estimate that that 120–140 neurons are within detection range. To get a first estimate we can say that on average 2 units are detected per electrode out of 130 possible neurons, so that the neuronal population is about 65 times the number of units.

We already estimated that about the number of neurons detected in Quian Quiroga et al. (2005) was about twice the number of units, to give a total of about 2000 detected neurons. So we should further multiply the number of neurons by \( K = 65/2 \approx 30 \), for a total of 60000 neurons in range of detectability.

This increase does not affect our estimate of the sparsity of the response of the distributed-code neurons; that stays at 4%. It also does not affect our estimate of the relative numbers of detected cells in our two populations (distributed-code v. GM-like). But it does drastically decrease the fraction of the distributed-code population to \( 4.6\% / K \approx 0.015\% \).

Most importantly it increases our estimate of the number of categories coded. The basic quantity here is \( R_{\text{eff}} \approx 4400K \approx 10^5 \). Given the roughness of our calculations, this is probably accurate to a factor of 2 or so. The key issues concern the orders of magnitude.

6. Comparison with previous measurements of distributed representations

6.1. Waydo et al.

Waydo et al. (2006) work with data that is evidently a superset of the data that the same group published in Quian Quiroga et al. (2005) and to which we made a two-population fit. They make a fit with a one-population model that is the same as ours in the special case that all detected cells are in the distributed-code population: \( f_{GM} = 0, f_D = 1 \). One superficial difference is that they use the exact binomial distribution instead of the analytically more tractable Poisson approximation; this makes a negligible difference for the small sparsities in question. Another difference is that they normalize to units rather than neurons; but this only results in trivial scalings of certain parameters.
They performed a Bayesian fit to the session-by-session data on the joint probability of measuring $N_i$ responsive units and $S_i$ evocative stimuli in a set of $N$ units with $S$ presented stimuli. The probabilities in the model can be derived from the underlying probability distribution (3), with one further critical assumption, that firing in the different neurons is independent.

As can be seen from the on-line supplementary material for Waydo et al. (2006), not only is it quite difficult to derive the distribution from the underlying distribution for the response of one neuron, but the resulting formula involves a delicate cancellation of opposite-sign terms. The formula therefore needs extreme care in numerical work. However certain averaged quantities considered in Waydo et al. (2006) are easier to derive, and in App. B we present derivations that apply also to our two-population model, given only the extra assumption of independence of different neurons.

The main result of the analysis in Waydo et al. (2006) is a distribution for sparsity, which is to be interpreted as a posterior distribution in the Bayesian sense for the single sparsity $a_1$ of the neural population. We use the symbol $a_1$ for the sparsity to avoid confusion with the sparsity parameter of the distributed-code population in our fits. 

When the same criterion for a neural response as in Quian Quiroga et al. (2005) is used, the peak of the distribution is at $a_1 = 0.23\%$, which is therefore the best fit according to the usual maximum likelihood criterion. The distribution has a long asymmetric tail to large $a_1$ and the average value of the posterior distribution, at $a_1 = 0.54\%$, is also a useful estimate of the value of $a_1$.

These values are much lower than the value of $a$ in our two-population model, as is natural if the fit is to be a compromise between matching an ultra-low sparsity GM-like population and a higher sparsity distributed-code population. We see this quantitatively in Table 1, where we show data and the results of their model fit and the predictions of our model. The data are from Waydo et al. (2006) and are an average over 34 sessions. One datum is the average number of units $N_i$ in one session that respond to at least one stimulus, out of an average of $S = 88$ stimuli. Another datum is the average number of stimuli $S_i$ to which at least one unit responds in a session, out of an average of $N = 42$ detected.

The one-population model evidently has a choice, to have a relatively low sparsity to get the correct number of responsive units, or to have a relatively high sparsity to get the correct number of evocative stimuli. Our two-population model does considerably better. We can improve its results by increasing the number of neurons a bit more relative to the number of units than we originally supposed. Note that from the first line of the table, the fraction of responsive units is measured to be $7.9/42 = 19\%$. This is substantially higher than the measurement for the same fraction $132/993 = 13\%$ given in Quian Quiroga et al. (2005). So the data are not completely consistent.

A final piece of session-averaged data given in Waydo et al. (2006) is the fraction of stimuli that produced a (simultaneous) response in at least two neurons. In App. B, we derive a formula for this quantity. As with the number of evocative stimuli, a neglect of neuron-neuron correlations is needed. The results are shown in the last line of Table 1. As already observed in Waydo et al. (2006), the one-population model with their preferred value $a_1 = 0.54\%$ gives a fraction $2.2\%$ that is rather below the data (4.1%). A comparably bad fit is obtained by our two-population model.

In fact, the bad fit happens quite generally. The formulae for both $S_r$ and $S_{r,n_i \geq 2}$ given them in terms of a single property of the model, the cell-averaged sparsity $\bar{a}$. We show in App. B, that when $\bar{a} N$ is not too large, the two quantities obey an approximate relation

$$
\frac{S_r}{S} \approx 1 + \frac{1}{2} \left( \frac{S_r}{S} \right)^2.
$$

This relation is obeyed to useful accuracy in the model calculations in the last two lines in Table 1, but it is violated by a factor of two by the data. The derivation is not affected by adding yet more populations of different sparsities, but only by including neuron-neuron correlations.

There are in fact two simple ways to overcome this problem. One is simply that one fraction is proportional to the number of detected units in a session, and the other is proportional to its square. Since this number varied quite widely between sessions (18 to 74), the session average of $N^2$ cannot be replaced by the square of the average of $N$. This could easily account for the factor of two mismatch. In contrast, the fraction of evocative

| $N = 42$ units | Waydo et al. (2006) | Our fit |
|--------------|------------------|--------|
| $S = 88$ stimuli | Data | $a_1 = 0.23\%$ | $a_1 = 0.54\%$ | Best | 84 neurons | 105 neurons | 126 neurons |
| $N_i$ | Responsive units | 7.9 | 7.7 | 15.9 | 5.5 | **6.9** | 8.3 |
| $S_i$ | Evocative stimuli | 16.4 | 8.1 | 17.9 | 14 | **17** | 20 |
| $S_{r,n_i \geq 2}$ | Fraction of stimuli with $N_i \geq 2$ | 4.1% | 0.44% | 2.2% | 1.4% | **2.1%** | 2.9% |

Table 1

Comparison of the per session data in Waydo et al. (2006) with the results of their one-population model, and with the predictions of our two-population model, using its already-determined population parameters. In the two-population model, the number of neurons corresponding to 42 units is adjustable, with the best result in the middle column. The data as well as the fit in the column headed $a_1 = 0.54\%$ are those in Waydo et al. (2006). For comments on the predictions in the last line, see the text.
The second possibility is from neuron-neuron correlations, to which \( S_{n,n \geq 2} \) is much more sensitive than the other observables. If there were a small fraction of nearby neurons that always fired in pairs, these would disproportionately contribute to \( S_{n,n \geq 2} \), but not nearly as much to \( S_r \).

Of course, both these suggestions can be tested by a closer examination of the data.

We also examine how well the one-population model, with the parameters from [Waydo et al. (2006)], agrees with the data we used from the earlier paper [Quian Quiroga et al. (2005)]. This is shown in Table 2. It can be seen that the observables we used are particularly sensitive to the differences between the models. A low average sparsity is necessary to keep the number of responsive neurons down to the experimental value. But with a one-population model this also implies that neurons with \( n \geq 2 \) responses are many fewer than those with \( n = 1 \) responses. Moreover the number of cells with even more responses than 2 is minute, so that \( \langle n \rangle_{n \geq 2} \) is close to its minimum value of 2, whereas the data is much higher. This is a clear indication of the need for two populations of cells with very different sparsities.

6.2. Abbott, Rolls, and Tovee

Other analyses of data, e.g., [Abbott, Rolls and Tovee (1996)], have reported that hippocampal facially responsive cells carry a distributed code as opposed to a GM-type code. See also the recent work of [Hung et al. (2005)]. These analyses might appear to contradict our calculation that distributed-code cells are a very small fraction, perhaps less than 0.2% of the total. However, there is a strong bias against actually detecting GM-like cells. In this section, we use our fit to the more recent data to quantify this bias, at least roughly, to determine whether there is consistency between our results and the earlier data.

The primary issue is that experiments typically only report those cells that are actually detected to respond to at least one of the stimuli used. For example, in a paper documenting place cell [Thompson and Best (1989)] state “the electrode assemblies were advanced until one or more hippocampal complex-spike cells were isolated extracellularly.” Then they observe that cells that do not produce any detectable spikes “are excluded from analysis here due to our lack of ability to detect them”. Since GM-like cells respond to a very small fraction of stimuli, the ones that respond to no presented stimulus, i.e., the vast majority, are typically omitted from an analysis.

The resulting bias can be seen in the data that we analyzed earlier. A minority of the detected cells in [Quian Quiroga et al. (2005)] are in the GM-like class (43 out of 132), even though we have shown that the GM-like cells can be in the vast majority (99% or more).

With fewer stimuli, the bias becomes even stronger, as in the data used by [Abbott, Rolls and Tovee (1996)]. They used 20 face stimuli, and the total number of facially responsive neurons was 14. A rather higher sparsity was reported than our result. But this is partly because a different definition of sparsity was used, applied to the spike numbers rather than to a binary response criterion. Furthermore the cells have considerably larger background firing rates than those in the new data.

Despite the differences in cells and species, we blindly apply our model to give a rough test of consistency. In our two-population model, the fraction of GM cells in the detected cells is

\[
\frac{p/\hat{R}}{p/\hat{R} + f_D (1 - e^{-ps})} = \frac{1}{1 + \frac{202}{p} (1 - e^{-0.04p})}.
\]

This is the probability that a cell is a GM-like cell conditional on the cell producing a detected response to one or more of \( p \) stimuli. Notice that the silent-cell ratio \( K \) cancels in this formula; we have a relation between numbers of different kinds of detected cell under different experimental conditions. We have estimates for the parameters of the model, so substituting \( p = 20 \) predicts a de-
ected GM-cell fraction of 0.15, i.e., about 2 cells, in the set of cells investigated in [Abbott, Rolls and Tovee (1996)]. In fact, Abbott, Rolls and Tovee (1996) did report that two of their cells had a GM-like response. There is, of course, no significance to the fact that this number is exactly the value predicted: there are expected statistical fluctuations, and the measurements were done with different methods and in a different species than in Quian Quiroga et al. (2003).

Nevertheless it is very important that the previous report, viewed as evidence in favor of distributed representations, is completely compatible with GM cells being in the vast majority, with the parameters we have determined. The undetected GM cells simply appear to be silent within the experiment and are therefore classed as not facially responsive. Statements about the neural representation being distributed apply only to (most of) those cells that the measurements actually detected, not to all cells in the relevant region of the brain.

7. Discussion

The results of Quian Quiroga et al. (2005) clearly suggest the detection of grandmother cells in the classic sense. Many other experiments have detected individual cells with strikingly specific responses (e.g., Hahnloser, Kozhevnikov and Pest (2002); Jung and McNaughton (1993); Thompson and Best (1989)). Therefore it is useful to hypothesize that some of these cells are indeed GM-like cells, even though the concept of GM-cell may need to be extended and modified.

A purely experimental direct test of the idea needs too many stimuli to be practical, cf. Churchland and Sejnowski (1992, p. 179). So other arguments must be brought in, of which we have provided two. One uses an estimate of the actual storage requirements for a memory system. We showed that GM systems can be optimally efficient in the use of synapses and neurons. The usual efficiency argument applies only to the input representation, but now carries the implication that in a GM-like system there must be two populations of cells with widely different sparsities.

Our second argument is a method to analyze neural responses. A particular aim is to measure whether they are quantitatively consistent there being separate neurons coding for each recognized person, or, alternatively, for each individual declarative memory. Our method enables one to determine whether or not individual cells necessarily code for multiple persons or memories. We derived a general formula Eq. (3) for the neural responses in terms of an underlying distribution of sparsity. Our expansion is a new result and is applicable independently of any detailed theory or model of neural function.

In effect, the formula enables us to extrapolate from limited data to obtain the fraction to stimuli to which cells respond. It also allows us to compensate for the strong biases involved in detecting cells when sparsities differ by very large factors. Thus we obtain valid estimates of the numbers of cells of different kinds. We thereby solve some of the issues raised by Olshausen and Field (2005) concerning the publication of data only about responsive cells. One primary remaining bias is that different neurons may have different electrical characteristics, with a consequent different maximum distance from the electrodes for detectability of spikes. But this is presumably a milder effect than that caused by orders of magnitude differences in sparsities.

7.1. Two populations essential

From the data we find indeed that the two-population property is obeyed. Not only does the ultra-low-sparisty population comprise the vast majority of cells in the brain regions concerned (hippocampus, etc.), but its sparsity can be in a range compatible with the hypothesis of a GM-like system: Roughly $10^{-3\%-5\%}$ with a repertoire of $10^3$.

An important role is played by the many silent cells. It is obviously unreasonable to assume they have no function. But on the GM-cell hypothesis they naturally are to be interpreted as the majority of GM cells that are not relevant to the particular stimuli used in an experiment. The large number of these cells is what enables one to overcome the strong biases against detecting a response of any one GM cell to a limited set of stimuli.

Now the group responsible for the analyzed data argue that their data do not support the GM cell idea. In Waydo et al. (2006), they say “if we assume that a typical adult recognizes between 10,000 and 30,000 discrete objects (Biederman, 1987), $a = 0.54\%$ implies that each neuron fires in response to 50 – 150 distinct representations.” [a should be replaced by $a_1$ in the notation of the present paper.]

However their analysis assumed a single value of sparsity. While this is a suitable approximation for conventional mechanisms of distributed memory, it is very bad for GM-like systems. Even though the explicit aim of Waydo et al. (2006) was to test the GM-cell hypothesis, the use of a single sparsity in effect imposed an assumption that the hypothesis is wrong.

We showed that the single-population hypothesis is a bad fit to the data. Since our expansion (3) is very general, the fault is in the single-population hypothesis not in any assumption about neural properties. The rather low value of sparsity given by Waydo et al. is merely a compromise between the widely different sparsities of the two populations. Our results are consistent with an even higher number of recognized objects than in the estimates of Biederman (1987). Indeed, even without allowing for the silent cell correction, our fits allow a GM-cell population with a sparsity of $1/4200 = 0.024\%$ corresponding to a number of objects not far from the lower edge of Biederman’s range.

Note that our basic estimate of the number of categories, $10^3$, assumes that the cells are classic GM cells, each responding to a single individual person. But the number of categories could be substantially higher. If the
cells are general memory cells, in the style of the model of [Baum, Moody and Wilczek 1988], they could respond to images of several people. It could also be that more familiar stimuli, with richer associations, have more cells. In that case measurements with familiar stimuli, as is the case in the data, would be biased towards these memories with unusually large numbers of cells, with a corresponding reduction in our estimate of the number of categories compared with the true number.

7.2. Anatomy

In GM systems, like the model of [Baum, Moody and Wilczek 1988], the number of GM cells is very much larger than that of the input cells, as is consistent with the numbers we have deduced. An immediate implication is that each GM cell receives input from a modest number of input cells, but that each input cell sends output to a much larger number of memory cells. Given also our finding that the GM cells in the relevant regions are in the vast majority, there are some striking anatomical implications.

In fact striking disparities in synapse number are well known in the hippocampus ([Amaral, Ishizuka and Claiborne 1970]): For example, each CA3 pyramidal cell gets about 50 input synapses from dentate granule cells, while other connections have tens of thousands of synapses. Note that hippocampal neurogenesis results in dentate granule cells, highly appropriate if they are GM-like. However, general-purpose memories need a wider variety of (processed) input than does a face recognition system, and hippocampal-related regions are sufficiently complex that the real picture is undoubtedly much more complicated. Even so, a careful analysis of the disparities in synapse number should provide critical information on neural function and the viability of GM-like systems.

7.3. Other arguments against GM systems

Other less quantitative arguments have been advanced against the reality of GM systems, e.g., [Churchland and Sejnowski 1992], [Rolls and Treves 1998].

For example, distributed memory systems are said to be robust against partial destruction, since there is no single location for a single memories. But we do know that memories disappear. If there are multiple GM cells for a memory in different places, then we can overcome the robustness ambiguity by simple redundancy. Moreover memories form a network of knowledge, so that individual items of semantic memory can be readily reconstructed from other knowledge. Episodic memory is really an ordered sequence of individual episodes, not necessarily remembered at all precisely. Any one episode that disappears can be approximately filled in from neighboring episodes.

Distributed memory systems are also said to be good at filling in missing parts of input data, as in reconstructing a full remembered image from a stimulus containing only a part of the image. But this property can also be true for GM-like systems. For example, when the BMW architecture [Baum, Moody and Wilczek 1988] is used with a sparse input representation and suitable dynamics for its GM cells, it also performs pattern completion; the completion property is actually associated with properties of sparse representations used for input data.

It has been said that new memories are harder to construct in GM systems than in distributed-memory systems. But now that adult neurogenesis in the hippocampus is well established, it may well be that there is actually a pool of new neurons available for at least some uses that could include being GM-like cells for new memories. The new neuron rate may however be excessively small. In addition, it is possible that the GM nodes are on dendritic tree rather than being whole neurons. It is known that there can be substantial changes in dendritic topology, which could easily include the formation of new nodes. Here the fundamental mode of operation is of a GM-like system while the neural code of memory neurons takes on some of the aspects of distributed memory. In any case, there are potential realistic mechanisms for the formation of new GM nodes, so that there is no insuperable obstacle here.

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Appendix A. Model of neural firing

Our model for the statistics of neural firing has two cell populations: one that uses a conventional distributed code with a single sparsity $\alpha$ and a second GM-cell population, as illustrated in Fig. 6. These from fractions $f_D$ and $f_{GM}$ of the total number of cells. A remaining population of cells does not respond to any stimuli at all in the class used in the experiment. We let $k$ be the fraction of the images used which have stored representations in the GM population, we let $R$ be the repertoire of the GM cells, and we let $n_m$ be the typical number of categories (or GM groups) evoked by a stimulus in the system’s repertoire. As before, we let $R_{eff} = R / n_m$.

Suppose first we record from some random cell known to be in the distributed population. We have seen that when we present $p$ images, the probability of getting $n$ responses is approximately the Poisson distribution in Eq. (2) with $\alpha = \alpha$.

If, instead, we pick a GM cell, then for each individual image it has a probability $kn_m / R$ of responding. Therefore
over a set of $p \ll R_{\text{eff}}$ unrelated images it has a probability $kp/R_{\text{eff}}$ of responding exactly once. There is a negligible probability of $n \geq 2$ for such a cell.

Finally, if the cell is outside the above two populations, it is silent in the experiment and always gives $n$.

Summing over the distributions of $n$ for cells of the different kinds, weighted by their fractional population size, gives

$$
P(n) \simeq \begin{cases} 
1 - \frac{f_{\text{GM}} kp}{R_{\text{eff}}} - f_D(1 - e^{-pa}) & \text{if } n = 0, \\
\frac{f_{\text{GM}} kp}{R_{\text{eff}}} + f_D pa e^{-pa} & \text{if } n = 1, \\
f_D(pa)^n e^{-pa} \frac{1}{n!} & \text{if } n \geq 2.
\end{cases} \tag{A.1}
$$

This is of the form of the general distribution Eq. (3) with

$$
D(\alpha) = f_D \delta(\alpha - a) + f_{\text{GM}} k \delta(\alpha - 1/R_{\text{eff}}) + (1 - f_D - f_{\text{GM}} k) \delta(\alpha), \tag{A.2}
$$

and with the Poisson approximation approximation for the distributed-code population and with the GM approximation that the GM-like cells fire so rarely that their responses for $n \geq 2$ can be neglected.

There are several meaningful parameters for the GM population, but only one combination affects the distribution $P(n)$. So we define $\hat{R} = R_{\text{eff}}/(k f_{\text{GM}}) = R/(n_m k f_{\text{GM}})$, to find

$$
P(n) \simeq \begin{cases} 
1 - \frac{p}{\hat{R}} - f_D(1 - e^{-pa}) & \text{if } n = 0, \\
\frac{p}{\hat{R}} + f_D pa e^{-pa} & \text{if } n = 1, \\
f_D(pa)^n e^{-pa} \frac{1}{n!} & \text{if } n \geq 2.
\end{cases} \tag{A.3}
$$

The parameter $\hat{R}$ has the meaning that if a cell is outside the distributed-code population then it responds to a stimulus in the chosen global class with probability $1/\hat{R} (1 - f_D)$, where the last approximate equality applies in the realistic case that $f_D$ is small, according to our fit.

We wish to extract the properties of the distributed-code cells without contamination from the GM cells. For that we need properties of the distribution for $n \geq 2$, for which we use the probability and the mean number of responses. The probability of $n \geq 2$ is

$$
P(n \geq 2) = f_D [1 - (1 + pa)e^{-pa}]. \tag{A.4}
$$

The mean number of responses, in cells with $n \geq 2$, is

$$
\langle n \rangle_{n \geq 2} = \frac{\sum_{n \geq 2} n P(n)}{P(n \geq 2)} = \frac{pa}{1 - (1 + pa)e^{-pa}}. \tag{A.5}
$$

These last two equations suffice to determine $a$ and $f_D$ from the data in [Quian Quiroga et al. (2003)] — see Eqs. (9) and (10).

### Appendix B. Further applications of model

Several further observables are considered by [Wavdo et al. (2006)](Wavdo et al. (2006)). These observables refer to a session in which $S$ stimuli are presented to $N_c$ cells or $N$ units.

One observable is the number of neurons $N_t$ that respond to at least one stimulus. Its average is just the number of cells or units times the probability that one cell or unit responds, which in our notation is $P(n \geq 1|S)$, in the notation of Eq. (3). Hence the number of responsive units in one-population model of [Wavdo et al. (2006)] is

$$
P(n \geq 1|S) \times \# \text{ units} = (1 - e^{-S\alpha}) N. \tag{B.1}
$$

In our two-population model it is

$$
P(n \geq 1|S) \times N_c = \left( \frac{SK}{R} + K f_D(1 - e^{-Sa}) \right) N_c. \tag{B.2}
$$

We are not quite sure how many cells correspond to each unit in the new data, so in Table 1 we gave results for several choices of the ratio of cells to units, $N_c/N$: 2 (as we estimated for the earlier data), 2.5, and 3.

A second observable is the number $S_t$ of stimuli that evoked a response in at least one neuron in the session. To derive this from the response distributions requires a further assumption that correlation between the firing of different detected neurons can be neglected.

Now the probability of one stimulus evoking no response in any of $N$ independent cells (or units) is $P(0|1)^N$, where $P(0|1)$ is the probability of no response in one cell/unit on presentation of 1 stimulus. Hence the average number of evocative stimuli in a session is

$$
S_t = S \left[ 1 - P(0|1)^N \right]. \tag{B.3}
$$

From Eq. (3), we find that in general $P(0|1) = 1 - \bar{\alpha}$, where $\bar{\alpha}$ is the sparsity averaged over cells. In the one-population model of [Wavdo et al. (2006)] we therefore get

$$
S_t = S \left[ 1 - (1 - a)^N \right], \tag{B.4}
$$

while in our two-population model it is

$$
S_t = S \left[ 1 - \left( 1 - \frac{K}{\hat{R}} - K f_D a \right)^{N_c} \right]. \tag{B.5}
$$

The appearance of $K$ in this last formula is misleading: the dependence on $K$ of $\hat{R}$ and $f_D$ in our fit cancels the explicit factor of $K$. We have used the number of cells $N_c$ in this formula rather than the number of units $N$, since our fit is made with respect to cells.

A final observable we consider is the number of stimuli in a session that evoked responses in 2 or more cells/units. This quantity, denoted $S_{r_t}$, can be obtained from the distribution of the number of neurone responding to a single stimulus:

$$
P(n_t \text{ for 1 stim.}) = \bar{\alpha}^{n_t} (1 - \bar{\alpha})^{N-n_t} \frac{N!}{n_t!(N-n_t)!} e^{-N(1-\bar{\alpha})}, \tag{B.6}
$$

$$
\simeq \left( \frac{N (1 - \bar{\alpha})^{n_t}}{n_t} \right) e^{-N(1-\bar{\alpha})}, \tag{B.6}
$$
It follows that on average
\[ S_{r, n > 2} = S \left[ 1 - (1 - \bar{\alpha})^N - N\bar{\alpha}(1 - \bar{\alpha})^{N-1} \right] \tag{B.7} \]

From Eqs. (B.5) and (B.7), we get a relation between \( S_r \) and \( S_{r, n > 2} \) valid when \( N\bar{\alpha} \) is less than about unity and \( N \) is substantially larger than unity. We expand the powers of \( 1 - \bar{\alpha} \) for small \( \bar{\alpha} \) to obtain
\[ \frac{S_r}{S} \simeq N\bar{\alpha} \quad \text{if} \quad N\bar{\alpha} \lesssim 1, \tag{B.8} \]

and then
\[ \frac{S_r(N_r > 2)}{S} \simeq \frac{(N\bar{\alpha})^2}{2} \simeq \frac{1}{2} \left( \frac{S_r}{S} \right)^2 \quad \text{if} \quad N\bar{\alpha} \lesssim 1. \tag{B.9} \]

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