12

Neuronal populations

The brain contains millions of neurons which are organized in different brain areas, within a brain area organized in different subregions, inside each small region into different layers, inside each layer into various cell types. The first two parts of this book focused on the mathematical description of an isolated neuron. Starting with this chapter, we shift our attention to the collective properties of groups of neurons, which we call "neuronal populations." Instead of modeling the spike times of a single neuron which belongs, for example, to the cell class "pyramidal" in layer 5 of subregion C4 in brain region S1 (the numbers here are completely arbitrary), we can ask the question: Suppose a human subject or animal receives a visual, auditory, or somatosensory stimulus – what is the activity of all the cells in this layer of this subregion that are of type "pyramidal" in response to the stimulus? What is the response of this subregion as a whole? What is the response of a brain area? In other words, at any of the scales of spatial resolution (Fig. 12.1), we may be interested in the response of the neuronal population as a whole, rather than in the spikes of individual neurons.

The general idea is presented in Fig. 12.2. A network of 10 000 neurons consisting of $N_{\mathrm{E}} = 8000$ excitatory and $N_{\mathrm{I}} = 2000$ inhibitory neurons, has been simulated while the excitatory neurons received a time-dependent input. Instead of analyzing the spike trains of one or two neurons, we count the number of spikes in a small time step (say $\Delta t = 1$ ms) across all the excitatory neurons in the network. After dividing by Δt and N_E, we arrive at the population activity $A(t)$ of the group of excitatory neurons; see Section 7.2. Analogously, we can determine the population activity of the inhibitory neurons or that of the network as a whole. The central questions of this and the following chapters are: Can we predict the population activity $A(t)$ from the properties of its neurons and the network connectivity? How does the population activity respond to a novel input?

The aim of this chapter is to provide the foundation of the notions of "neuronal population" and "population activity." In the first section we argue that the organization of cortex into columns and layers provides the biological substrate of "neuronal populations." In Section 12.2 we identify the mathematical assumptions and idealizations that will enable us to predict the population activity from single-neuron properties. The basic idea is that, for the definition of a neuronal population, we should group neurons with similar properties together. The aim of Sections 12.2 and 12.3 is to make this intuition more precise.

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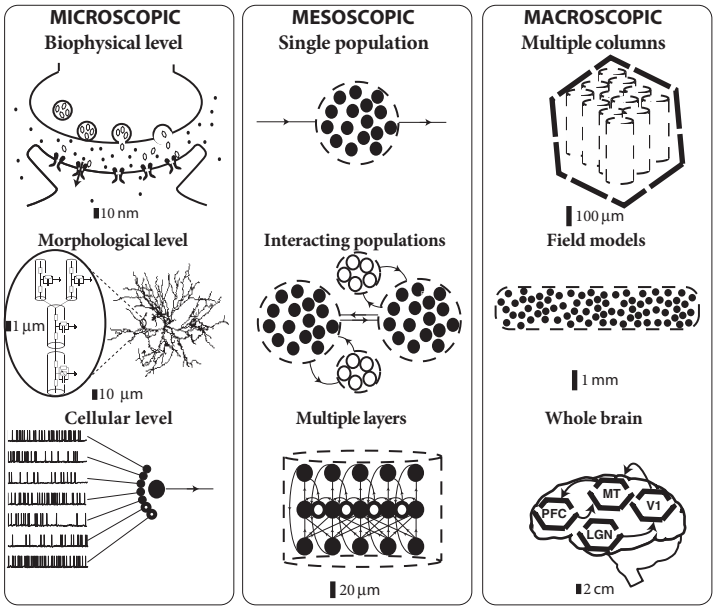

Fig. 12.1 The levels of description in neuroscience models.

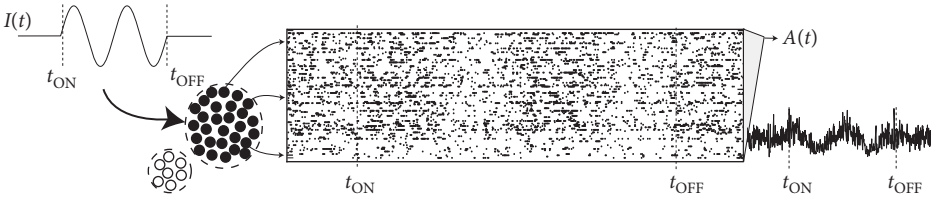

Fig. 12.2 Input and output of a population. A signal $I(t)$, represented by a sinusoidal modulation of the input starting at t_{ON} and ending at t_{OFF}, stimulates the population of 8000 excitatory neurons in a randomly coupled network of 8000 excitatory and 2000 inhibitory neurons (left). Each neuron produces a spike train (middle) illustrated here by lines of dots, each dot corresponding to a spike. Only 1% of the population is shown. The population activity $A(t)$ (right) counts spikes in time bins of 1 ms averaged over the 8000 excitatory neurons.

In Section 12.4 we give a first example of the population activity approach by analyzing stationary activity in a highly connected population. The notions developed in this chapter will be used in the next two chapters in order to analyze the dynamics of one or several connected populations.

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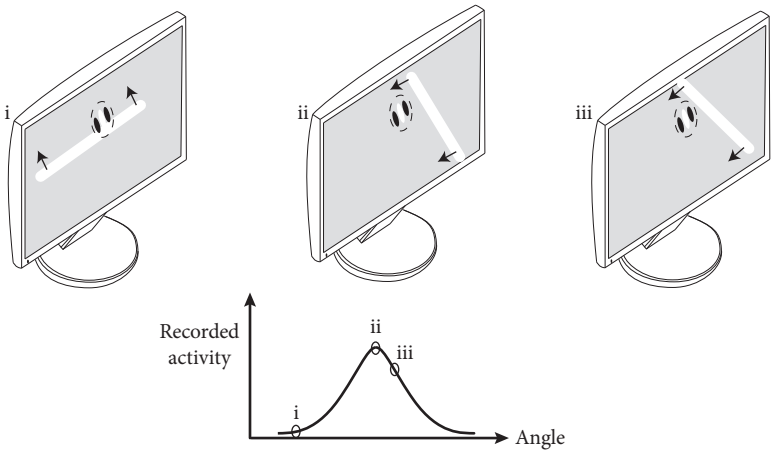

Fig. 12.3 Orientation tuning. The receptive fields of simple cells in the visual cortex have positive and negative subfields. To test orientation tuning, a light bar is slowly moved across the screen (i). The neuron responds maximally if the light bar with an orientation aligned with that of the receptive field moves into the positive subfield (ii) and responds slightly less if the orientation of the bar is not optimal (iii). The response as a function of the orientation of the bar is shown at the bottom (schematic figure).

12.1 Columnar organization

Before we turn, in Section 12.2, to the rather abstract notion of a "neuronal population," we present in this section a short introduction to the structural organization and functional characterization of the cortex. We will argue that a cortical column, or more precisely, a group of cells consisting of neurons of the same type in one layer of a cortical column, can be considered as a plausible biological candidate of a neuronal population.

12.1.1 Receptive fields

Neurons in sensory cortices can be experimentally characterized by the stimuli to which they exhibit a strong response. The receptive field of so-called simple cells in the visual cortex (see Chapter 1) has typically two or three elongated spatial subfields. The neuron responds maximally to a moving light bar with an orientation aligned with the elongation of the positive subfield. If the orientation of the stimulus changes, the activity of the cell decreases (Fig. 12.3). Thus simple cells in the visual cortex are sensitive to the orientation of a light bar (Hubel and Wiesel, 1968).

In this and the following chapters, we exploit the fact that neighboring neurons in the visual cortex have similar receptive fields. If the experimenter moves the electrode vertically down from the cortical surface to deeper layers, the location of the receptive field and its preferred orientation does not change substantially. If the electrode is moved to a neighboring location in the cortex, the location and preferred orientation of the receptive field of

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neurons at the new location changes only slightly compared to the receptive fields at the previous location. This observation has led to the idea that cortical cells can be grouped into "columns" of neurons with similar properties. Each column stretches across different cortical layers, but has only a limited extent on the cortical surface (Hubel and Wiesel, 1968). The exact size and anatomical definition of a cortical column is a matter of debate, but each column is likely to contain several thousand neurons with similar receptive fields (Lund *et al*., 2003).

In other sensory cortices, the characterization of neuronal receptive fields is analogous to that in visual cortex. For example, in the auditory cortex, neurons can be characterized by stimulation with pure tones. Each neuron has its preferred tone frequency, and neighboring neurons have similar preferences. In the somatosensory cortex, neurons that respond strongly to touch on, say, the index finger are located close to each other. The concepts of receptive field and optimal stimuli are not restricted to mammals or cortex, but are also routinely used in studies of, for example, retinal ganglion cells, thalamic cells, olfactory bulb, or insect sensory systems.

Example: Cortical maps

Neighboring neurons have similar receptive fields, but the exact characteristics of the receptive fields change slightly as one moves parallel to the cortical surface. This gives rise to cortical maps.

A famous example is the representation of the body surface in the somatosensory area of the human brain. For example, neurons which respond to touch on the thumb are located in the immediate neighborhood of neurons which are activated by touching the index finger, which in turn are positioned next to the subregion of neurons that respond to touching the middle finger. The somatosensory map therefore represents a somewhat stretched and deformed "image" of the surface of the human body projected onto the surface of the cortex. Similarly, the tonotopic map in the auditory cortex refers to the observation that the neurons' preferred pure-tone frequency changes continuously along the cortical surface.

In the visual cortex, the location of the neurons' receptive field changes along the cortical surface giving rise to the retinotopic map. At the same time, the preferred orientation of neurons changes as one moves parallel to the cortical surface giving rise to orientation maps. With modern imaging methods it is possible to visualize the orientation map by monitoring the activity of neuronal populations while the visual cortex is stimulated by the presentation of a slowly moving grating. If the grating is oriented vertically, certain subsets of neurons respond; if the grating is rotated by 60°, other groups of neurons respond. Thus we can assign to each location on the cortical surface a preferred orientation (see Fig. 12.4), except for a few singular points, called the pinwheels, where regions of different preferred orientations touch each other (Bonhoeffer and Grinvald, 1991; Kaschube *et al*., 2010).

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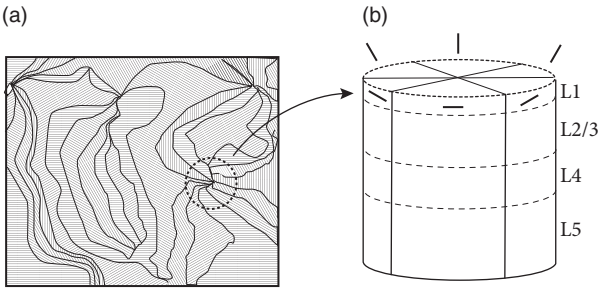

Fig. 12.4 Orientation maps and columns. (a) Top view onto the surface of the visual cortex. Neurons that are optimally activated by a moving grating with an orientation of, say, 60°, form bands. The direction of the hash-line texture in the image indicates the preferred orientation. Iso-orientation contour lines converge to form pinwheels. One of the pinwheels is highlighted by the dashed circle. (b) Side view of a pinwheel (dashed circle in (a)). Orientation selectivity is indicated by thick bars. Neurons with the same orientation form vertical columns. Schematic representation following experimental data shown in Bressloff and Cowan (2002).

12.1.2 How many populations?

The columnar organization of the cortex suggests that all neurons in the same column can be considered as a single population of neurons. Since these neurons share similar receptive fields we should be able to develop mathematical models that describe the net response of the column to a given stimulation. Indeed the development of those abstract models is one of the aims of mathematical neuroscience.

However, the transition from single neurons to a whole column might be too big a challenge to be taken in a single step. Inside a column neurons are organized in different layers. Each layer contains one or several types of neurons. At a first level, we can distinguish excitatory from inhibitory neurons and at an even finer level of detail different types of inhibitory interneurons. As we shall see in the next section, the mathematical transition from single neurons to populations requires that we put only those neurons that have similar intrinsic properties into the same group. In other words, pyramidal neurons in, say, layer 5, of a cortical column are considered as one population whereas pyramidal neurons in layer 2 are a different population; fast-spiking inhibitory neurons in layer 4 form one population while non-fast-spiking interneurons in the same layer form a different group. The number of populations that a theoretician takes into account depends on the level of "coarse-graining" that he is ready to accept, as well as on the amount of information that is available from experiments.

Example: Connectivity in the barrel cortex

The somatosensory cortex of mice contains a region which is sensitive to whisker movement. Each whisker is represented in a different subregion. These subregions are,

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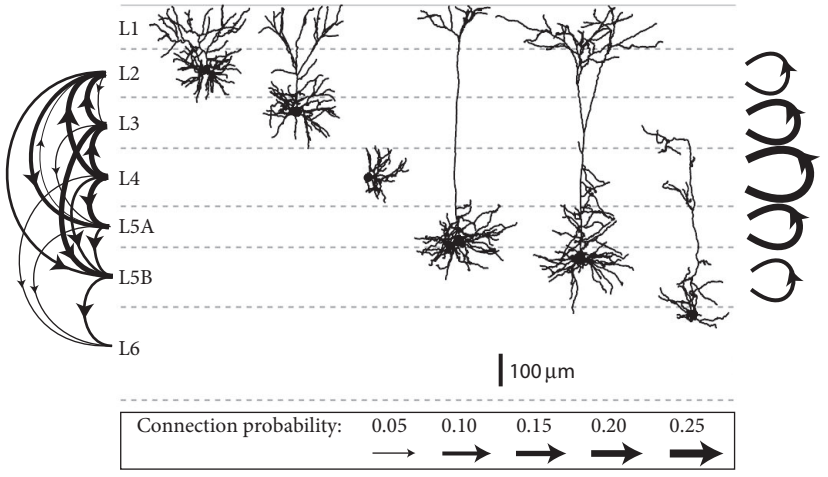

Fig. 12.5 Connectivity patterns inside one column. Examples of shapes of excitatory neurons in different layers. Arrows on the left indicate connection probabilities between excitatory neurons across layers. Arrows on the right show connection probabilities between excitatory neurons within a given layer. Data from a barrel column in the somatosensory cortex of the mouse. After Lefort *et al*. (2009).

at least in layer 4, clearly separated. Neurons are connected vertically across layers so that, in this part of cortex, cortical columns are exceptionally well identifiable. Because of the barrel-shaped subregions in layer 4, this part of the somatosensory cortex is called the barrel cortex (Woosley and Van der Loos, 1970).

Neurons in different layers of a barrel cortex column have various shapes and form connections with each other with different probabilities (Lefort *et al*., 2009), indicating that excitatory neurons in the column do not form a homogeneous population; see Fig. 12.5. However, if we increase the resolution (i.e., a less "coarse-grained" model) to that of a single layer in one barrel cortex column, then it might be possible to consider all excitatory neurons inside one layer as a rather homogeneous population.

12.1.3 Distributed assemblies

The concept of cortical columns suggests that localized populations of neurons can be grouped together into populations, where each population (e.g., the excitatory neurons in layer 4) can be considered as a homogeneous group of neurons with similar intrinsic properties and similar receptive fields. However, the mathematical notion of a population does not require that neurons need to form local groups to qualify as a homogeneous population.

Donald Hebb (1949) introduced the notion of neuronal assemblies, i.e., groups of cells which get activated together so as to represent a mental concept such as the preparation of a movement of the right arm toward the left. An assembly can be a group of neurons which

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are distributed across one or several brain areas. Hence, an assembly is not necessarily a local group of neurons. Despite the fact that neurons belonging to one assembly can be widely distributed, we can think of all neurons belonging to the assembly as a homogeneous population of neurons that is activated whenever the corresponding mental concept is evoked. Importantly, such an assignment of a neuron to a population is not fixed, but can depend on the stimulus. We will return to the concept of distributed assemblies in Chapter 17 when we discuss associative memories and the Hopfield model.

12.2 Identical neurons: a mathematical abstraction

As discussed in the previous section, there exist many brain regions where neurons are organized in populations of cells with similar properties. Prominent examples are columns in the somatosensory and visual cortex (Mountcastle, 1957; Hubel and Wiesel, 1962) and pools of motor neurons (Kandel *et al.*, 2000). Given the large number of neurons within such a column or pool it is sensible to describe the mean activity of the neuronal population rather than the spiking of individual neurons.

The definition of population activity has already been introduced earlier, but is repeated here for convenience. In a population of N neurons, we calculate the proportion of active neurons by counting the number of spikes $n_{\rm act}(t;t+\Delta t)$ in a small time interval Δt and dividing by N. Further division by Δt yields the *population activity*

$$A(t) = \lim_{\Delta t\to 0} \frac{1}{\Delta t} \frac{n_{\rm act}(t;t+\Delta t)}{N} = \frac{1}{N} \sum_{j=1}^{N} \sum_{f} \delta(t-t_j^f)\,, \qquad (12.1)$$

where δ denotes the Dirac δ-function. The double sum runs over all firing times t_j^f of all neurons in the population. In other words the activity A is defined by a population average. Even though the activity has units of a rate and indeed is often called the population rate, the population activity is quite different from the concept of a mean firing rate defined by temporal averaging in a single neuron; see Section 7.2.

Theories of population dynamics, sometimes called "neural mass models," have a long tradition (Knight, 1972; Wilson and Cowan, 1972, 1973; Amari, 1974; Abbott and van Vreeswijk, 1993; Gerstner and van Hemmen, 1992; Treves, 1993; Amit and Brunel, 1997b; Brunel and Hakim, 1999; Fusi and Mattia, 1999; Brunel, 2000; Gerstner, 2000; Nykamp and Tranchina, 2000; Omurtag *et al.*, 2000). Their aim is to predict the temporal evolution of the population activity $A(t)$ in large and homogeneous populations of spiking neurons.

Why do we restrict ourselves to large populations? If we repeatedly conduct the same experiment on a population of, say, one hundred potentially noisy neurons, the observed activity $A(t)$ defined in Eq. (12.1) will vary from one trial to the next. Therefore we cannot expect a population theory to predict the activity measurements in a single trial. Rather all population activity equations that we discuss in this chapter predict the *expected* activity. For a large and homogeneous network, the observable activity is very close to the expected

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activity. For the sake of notational simplicity, we do not distinguish the observed activity from its expectation value and in what follows we denote the expected activity by $A(t)$.

Why do we focus on homogeneous populations? Intuitively, we cannot expect to predict the activity of a population where each neuron receives a different input and has a different, and potentially unknown, set of parameters. However, if all neurons in a population have roughly the same parameters and receive roughly the same input, all neurons are more or less exchangeable and there is a realistic chance that, based on the knowledge of the parameters of a typical neuron in the population, we would be able to predict the activity of the population as a whole. The notion of a homogeneous network will be clarified in the following subsection. In Section 12.2.2 we will ask whether the requirements of homogeneous populations can be relaxed so as to include some degree of heterogeneity within a population.

12.2.1 Homogeneous networks

We study a large and homogeneous population of neurons. By homogeneous we mean that (i) all neurons $1 \le i \le N$ are identical; (ii) all neurons receive the same external input $I_i^{\rm ext}(t) = I^{\rm ext}(t)$; and (iii) the interaction strength w_{ij} for the connection between any pair j, i of pre- and postsynaptic neurons is "statistically uniform." The notion will be made precise in Section 12.3, but for the moment we can think of connections inside the population as being either absent or "roughly the same," $w_{ij} \approx w_0$, where w_0 is a parameter. For $w_0 = 0$ all neurons are independent; a value $w_0 > 0$ ($w_0 < 0$) implies excitatory (inhibitory) coupling. Not all neurons need to be coupled with each other; connections can, for example, be chosen randomly (see Section 12.3 below).

Example: Homogeneous population of integrate-and-fire neurons

In the case of leaky integrate-and-fire neurons, encountered in Chapters 1 and 5 (Section 5.1), the dynamics are

$$\tau_m \frac{\mathrm{d}}{\mathrm{d}t} u_i = -u_i + R I_i(t) \quad \text{for } u_i < \vartheta \tag{12.2}$$

combined with a reset condition: if $u_i \ge \vartheta$ then integration restarts at u_r. A homogeneous network implies that all neurons have the same input resistance R, the same membrane time constant τ_m, as well as identical thresholds ϑ and reset values u_r. Note that we have shifted the voltage scale such that the resting potential is $u_{\rm rest} = 0$, which is only possible if all neurons also have the same resting potential.

We assume that a neuron is coupled to all others as well as to itself with coupling strength $w_{ij} = w_0$. The input current I_i in Eq. (12.2) takes care of both the external drive and synaptic coupling

$$I_i = \sum_{j=1}^{N} \sum_f w_{ij} \alpha(t - t_j^f) + I^{\rm ext}(t)\,. \tag{12.3}$$

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Here we have assumed that each input spike generates a postsynaptic current with some generic time course $\alpha(t-t_j^f)$. The sum on the right-hand side of Eq. (12.3) runs over all firing times of all neurons. Because of the homogeneous all-to-all coupling, the total input current is identical for all neurons. To see this, we insert $w_{ij} = w_0$ and use the definition of the population activity, Eq. (12.1). We find a total input current,

$$I(t) = w_0 N \int_0^\infty \alpha(s) A(t-s)\,\mathrm{d}s + I^{\mathrm{ext}}(t)\,, \tag{12.4}$$

which is independent of the neuronal index i. Thus, the input current at time t depends on the past population activity and is the same for all neurons.

As an aside we note that for conductance-based synaptic input, the total input current would depend on the neuronal membrane potential which is different from one neuron to the next.

12.2.2 Heterogeneous networks

Our definition of a homogeneous network relied on three conditions: (i) identical parameters for all neurons; (ii) identical external input to all neurons; (iii) statistically homogeneous connectivity within the network. We may wonder whether all three of these are required or whether we can relax our conditions to a certain degree (Tsodyks *et al*., 1993; Chow, 1998). Potential connectivity schemes will be explored in Section 12.3. Here we focus on the first two conditions.

Let us suppose that all N neurons in the population receive the same input I, considered to be constant for the moment, but parameters vary slightly between one neuron and the next. Because of the difference in parameters, the stationary firing rate $\nu_i = g_{\theta_i}(I)$ of neuron i is different from that of another neuron j. The index θ_i refers to the set of parameters of neuron i. The mean firing rate averaged across the population is $\langle \nu \rangle = \sum_i \nu_i / N$.

Under the condition that, first, the firing rate is a smooth function of the parameters and, second, that the differences between parameters of one neuron and the next are small, we can linearize the function g around the *mean* parameter value $\bar{\theta}$ and find for the mean firing rate averaged across the population

$$\langle \nu \rangle = g_{\bar{\theta}}(I), \tag{12.5}$$

where we neglected terms $(\mathrm{d}^2 g_\theta / \mathrm{d}\theta^2)\,(\theta_i - \bar{\theta})^2$ as well as all higher-order terms. Eq. (12.5) can be phrased as saying that the mean firing rate across a heterogeneous network is equal to the firing rate of the "typical" neuron in the network, i.e., the one with the mean parameters.

However, strong heterogeneity may cause effects that are not well described by the above averaging procedure. For example, suppose that the network contains two subgroups of neurons, each of size $N/2$, one with parameters θ_1 and the other with parameters θ_2. Suppose that the gain function takes a fixed value $\nu = \nu_0$ whenever the parameters

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are smaller than $\hat{\theta}$ and has some arbitrary dependence $\nu = g_\theta(I) > \nu_0$ for $\theta > \hat{\theta}$. If $\bar{\theta} = (\theta_1 + \theta_2)/2 < \hat{\theta}$, then Eq. (12.5) would predict a mean firing rate of ν_0 averaged across the population, which is not correct. The problem can be solved if we split the population into two populations, one containing all neurons with parameters θ_1 and the other containing all neurons with parameters θ_2. In other words, a strongly heterogeneous population should be split until (nearly) homogeneous groups remain.

The same argument also applies to a population of N neurons with identical parameters, but different inputs I_i. If the differences in the input are small and neuronal output is a continuous function of the input, then we can hope to treat the slight differences in input by a perturbation theory around the homogeneous network, i.e., a generalization of the Taylor expansion used in the previous subsection. If the differences in the input are large, for example, if only a third of the group is strongly stimulated, then the best approach is to split the population into two smaller ones. The first group contains all those that are stimulated, and the second group all the other ones.

12.3 Connectivity schemes

The real connectivity between cortical neurons of different types and different layers, or within groups of neurons of the same type and the same layer, is still partially unknown, because experimental data is limited. At most, some plausible estimates of connection probabilities exist. In some cases the connection probability is considered distance-dependent, in other experimental estimates it is considered uniform in the restricted neighborhood of a cortical column.

In simulations of spiking neurons, there are a few coupling schemes that are frequently adopted (Fig. 12.6). Most of these assume random connectivity within and between populations. In what follows we discuss these schemes with a special focus on the scaling behavior induced by each choice of coupling scheme. Here, scaling behavior refers to a change in the number N of neurons that participate in the population.

An understanding of the scaling behavior is important not only for simulations, but also for the mathematical analysis of the network behavior. However, it should be kept in mind that real populations of neurons have a fixed size because, for example, the number of neurons in a given cortical column is given and, at least in adulthood, does not change dramatically from one day to the next. Typical numbers, counted in one column of mouse somatosensory cortex (barrel cortex, C2), are 5750 excitatory and 750 inhibitory neurons (Lefort *et al.*, 2009). Thus numbers are finite and considerations of the behavior for the number N going to infinity are of little relevance to biology.

We note that numbers are even smaller if we break them down per layer. The estimated mean ($\pm$ standard error of the mean) number of excitatory neurons in each layer (L1 to L6) are as follows: L2, 546 $\pm$ 49; L3, 1145 $\pm$ 132; L4, 1656 $\pm$ 83; L5A, 454 $\pm$ 46; L5B, 641 $\pm$ 50; L6, 1288 $\pm$ 84; and for inhibitory neurons: L1, 26 $\pm$ 8; L2, 107 $\pm$ 7; L3, 123 $\pm$ 19; L4, 140 $\pm$ 9; L5A, 90 $\pm$ 14; L5B, 131 $\pm$ 6; L6, 127 $\pm$ 9 (Lefort *et al.*, 2009).

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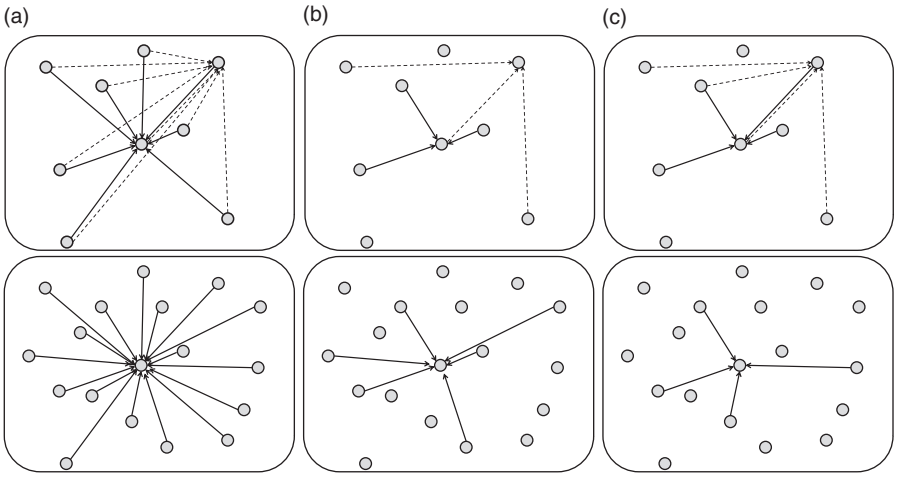

Fig. 12.6 Coupling schemes. (a) Full connectivity: Top: A network of 9 neurons with all-to-all coupling. The input links are shown for two representative neurons. Self-couplings are not indicated. Bottom: The number of input links (indicated for one representative neuron) increases, if the size of the network is doubled. (b) Random coupling with fixed connection probability. In a network of 18 neurons (bottom) the number of input links is larger than in a network of 9 neurons (top). (c) Random coupling with fixed number of inputs. The number of links from presynaptic neurons (top: input links to two representative neurons) does not change when the size of the network is increased (bottom: input links to one representative neuron).

Example: Scaling of interaction strength in networks of different sizes

Suppose we simulate a fully connected network of 1000 noisy spiking neurons. Spikes of each neuron in the population generate in all other neurons an inhibitory input current of strength $w < 0$ which lasts for 20 ms. In addition to the inhibitory inputs, each neuron also receives a fixed constant current $I_0^{\rm ext}$ so that each neuron in the network fires at 5 Hz. Since each neuron receives input from itself and from 999 partner neurons, the total rate of inhibitory input is 5 kHz. Because each input exerts an effect over 20 ms, a neuron is, at each moment in time, under the influence of about 100 inhibitory inputs – generating a total input $I \approx 100\,w + I_0^{\rm ext}$.

We now get access to a bigger computer which enables us to simulate a network of 2000 neurons instead of 1000. In the new network each neuron therefore receives inhibitory inputs at a rate of 10 kHz and is, at each moment in time, under the influence of a total input current $I \approx 200\,w + I_0^{\rm ext}$. Scaling the synaptic weights w by a factor of $\frac{1}{2}$ leads us back to the same total input as before.

Why should we be interested in changing the size of the network? As mentioned before, in biology the network size is fixed. An experimenter might tell us that the system he studies contains 20 000 neurons, connected with each other with strength $w_{ij} = 1$ (in some arbitrary units) and connection probability of 10%. Running a simulation of the

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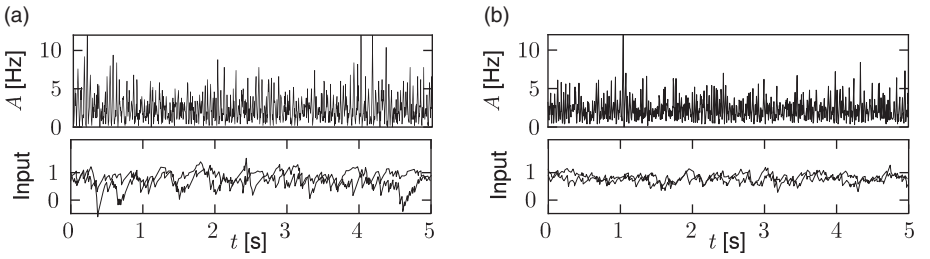

Fig. 12.7 Simulation of a model network with a fixed connection probability $p = 0.1$. (a) Top: Population activity $A(t)$ averaged over all neurons in a network of 4000 excitatory and 1000 inhibitory neurons. Bottom: Total input current $I_i(t)$ into two randomly chosen neurons. (b) Same as (a), but for a network with 8000 excitatory and 2000 inhibitory neurons. The synaptic weights have been rescaled with a factor of $\frac{1}{2}$ and a common input current I^{ext} is given to all neurons to ensure that the same population activity is obtained. All neurons are leaky integrate-and-fire units with identical parameters interacting by short current pulses.

full network of 20 000 neurons is possible, but will take a certain amount of time. We may want to speed up the simulation by simulating a network of 4000 neurons instead. The question arises whether we should increase the interaction strength in the smaller network compared to the value $w_{ij} = 1$ in the big network.

12.3.1 Full connectivity

The simplest coupling scheme is all-to-all connectivity within a population. All connections have the same strength. If we want to change the number N of neurons in the simulation of a population, an appropriate scaling law is

$$w_{ij} = \frac{J_0}{N} . \tag{12.6}$$

This scaling law is a mathematical abstraction that enables us to formally take the limit of $N \to \infty$ while keeping fixed the expected input that a neuron receives from its partners in the population. In the limit of $N \to \infty$, the fluctuations disappear and the expected input can be considered as the actual input to any of the N neurons. Of course, real populations are of finite size, so that some fluctuations always remain. But as N increases the fluctuations decrease.

A slightly more intricate all-to-all coupling scheme is the following: weights w_{ij} are drawn from a Gaussian distribution with mean J_0/N and standard deviation $\sigma_0/\sqrt{N}$. In this case, each neuron in the population sees a somewhat different input, so that fluctuations of the membrane potential are of the order σ_0 even in the limit of large N (Faugeras *et al.*, 2009).

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12.3.2 Random coupling: fixed coupling probability

Experimentally the probability p that a neuron inside a cortical column makes a functional connection to another neuron in the same column is in the range of 10%, but varies across layers; see Fig. 12.5.

In simulations, we can fix a connection probability p and choose connections randomly with probability p among all the possible N^2 connections. In this case, the number of presynaptic input links C_j to a postsynaptic neuron j has a mean value of $\langle C_j \rangle = pN$, but fluctuates between one neuron and the next with variance $p(1-p)N$.

Alternatively, we can take one model neuron $j = 1, 2, 3, \ldots, N$ after the other and choose randomly $C = pN$ presynaptic partners for it (each neuron can be picked only once as a presynaptic partner for a given postsynaptic neuron j). In this case all neurons have, by construction, the same number of input links $C_j = C$. By an analogous selection scheme, we could also fix the number of output links to exactly pN as opposed to simply imposing pN as the average value.

Whatever the exact scheme to construct random connectivity, the number of input connections per neuron increases linearly with the size N of the population; see Fig. 12.6b. It is therefore useful to scale the strength of the connections as

$$w_{ij} = \frac{J_0}{C} = \frac{J_0}{pN}, \tag{12.7}$$

so that the mean input to a typical neuron does not change if the number of model neurons in the simulated population is increased. Since in a bigger network individual inputs have smaller effects with the scaling according to Eq. (12.7), the amount of fluctuation in the input decreases with population size; compare Fig. 12.7.

12.3.3 Random coupling: fixed number of presynaptic partners

The number of synapses onto the dendrites of a single pyramidal neuron is estimated to lie in the range of a few thousand (Kandel *et al.*, 2000). Thus, when one simulates networks of a hundred thousand neurons or millions of neurons, a modeling approach based on a fixed connection probability in the range of 10% cannot be correct. Moreover, in an animal participating in an experiment, not all neurons will be active at the same time. Rather only a few subgroups will be active, the composition of which depends on the stimulation conditions and the task. In other words, the number of inputs converging onto a single neuron may be much smaller than a thousand.

We can construct a random network with a fixed number C of inputs by the following procedure. We pick one model neuron $j = 1, 2, 3, \ldots, N$ after the other and choose randomly its C presynaptic partners; see Fig. 12.6c. Whenever the network size N is much bigger than C, the inputs to a given neuron can be thought of as random samples from the current network activity. No scaling of the connections with the population size N is necessary; see Fig. 12.8.

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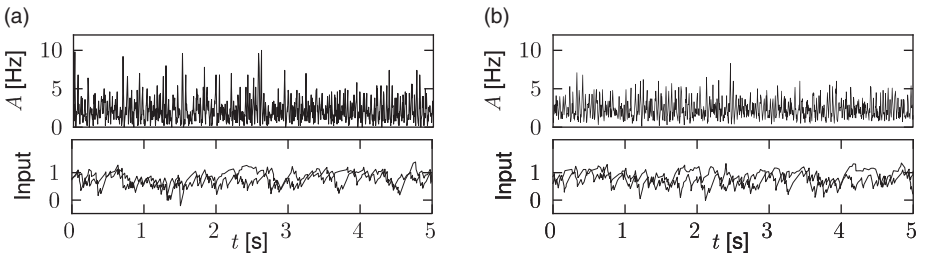

Fig. 12.8 Simulation of a model network with a fixed number of presynaptic partners (400 excitatory and 100 inhibitory cells) for each postsynaptic neuron. (a) Top: Population activity $A(t)$ averaged over all neurons in a network of 4000 excitatory and 1000 inhibitory neurons. Bottom: Total input current $I_i(t)$ into two randomly chosen neurons. (b) Same as (a), but for a network with 8000 excitatory and 2000 inhibitory neurons. The synaptic weights have not been rescaled. While the fluctuations of the population activity $A(t)$ decrease compared to the smaller network (top), the mean and variance of the synaptic input do not change with the size of the network (bottom). All neurons are leaky integrate-and-fire units with identical parameters interacting by current pulses; see Brunel (2000).

12.3.4 Balanced excitation and inhibition

In the simulations of Fig. 12.2, we have assumed a network of excitatory and inhibitory neurons. In other words, our network consists of two interacting populations. The combination of one excitatory and one inhibitory population can be exploited for the scaling of synaptic weights if the effects of excitation and inhibition are "balanced."

In the discussion of scaling in the previous subsections, it was mentioned that a fixed connection probability of p and a scaling of weights $w_{ij} = J_0/(pN)$ leads to a mean neuronal input current that is insensitive to the size of the simulation; however, fluctuations decrease with increasing N. Is there a possibility of working with a fixed connection probability p and yet control the size of the fluctuations while changing N?

In a network of two populations, one excitatory and one inhibitory, it is possible to adjust parameters such that the mean input current into a typical neuron vanishes. The condition is that the total amount of excitation and inhibition cancel each other, so that excitation and inhibition are "balanced." The resulting network is called a balanced network or a population with balanced excitation and inhibition (van Vreeswijk and Sompolinsky, 1996; Vogels *et al.*, 2011).

If the network has balanced excitation and inhibition the mean input current to a typical neuron is automatically zero and we do not have to apply a weight rescaling scheme to control the mean. Instead, we can scale synaptic weights so as to control specifically the amount of fluctuation of the input current around zero. An appropriate choice is

$$w_{ij} = \frac{J_0}{\sqrt{C}} = \frac{J_0}{\sqrt{pN}} \,. \tag{12.8}$$

With this choice, a change in the size of the network hardly affects the mean and variance of the input current into a typical neuron; see Fig. 12.9. Note that in simulations of

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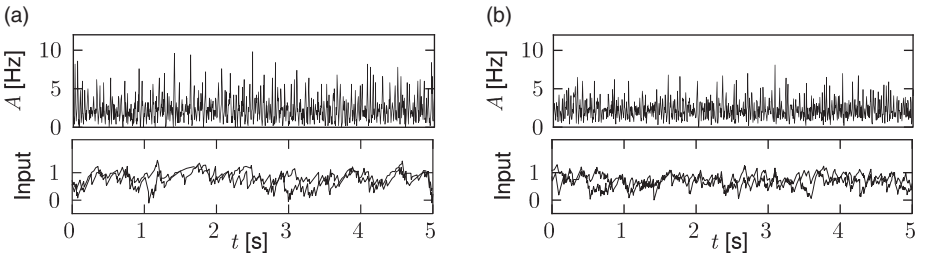

Fig. 12.9 Simulation of a model network with balanced excitation and inhibition and fixed connectivity $p = 0.1$. (a) Top: Population activity $A(t)$ averaged over all neurons in a network of 4000 excitatory and 1000 inhibitory neurons. Bottom: Total input current $I_i(t)$ into two randomly chosen neurons. (b) Same as (a), but for a network with 8000 excitatory and 2000 inhibitory neurons. The synaptic weights have been rescaled by a factor $1/\sqrt{2}$ and the common constant input has been adjusted. All neurons are leaky integrate-and-fire units with identical parameters coupled interacting by short current pulses.

networks of integrate-and-fire neurons, the mean input current to the model neurons is in practice often controlled, and adjusted, through a common constant input to all neurons. In Figs. 12.7–12.9 we simply report the main effects of network size on the population activity and synaptic currents; the analysis of the observed phenomena will be postponed to Section 12.4.

12.3.5 Interacting populations

In the previous subsections we considered two populations, one of them excitatory and the other one inhibitory. Let us generalize the arguments to a network consisting of several populations; see Fig. 12.10. It is convenient to visualize the neurons as being arranged in spatially separate pools, but this is not necessary. All neurons could, for example, be physically localized in the same column of the visual cortex, but of three different types: excitatory, fast-spiking inhibitory, and non-fast-spiking interneurons.

We assume that neurons are homogeneous within each pool. The activity of neurons in pool n is

$$A_n(t) = \frac{1}{N_n} \sum_{j \in \Gamma_n} \sum_f \delta(t - t_j^f)\,, \tag{12.9}$$

where N_n is the number of neurons in pool n and Γ_n denotes the set of neurons that belong to pool n. We assume that each neuron i in pool n receives input from all neurons j in pool m with strength $w_{ij} = J_{nm}/N_m$; see Fig. 12.10. The time course $\alpha_{ij}(s)$ caused by a spike of a presynaptic neuron j may depend on the synapse type, i.e., a connection from a neuron in pool m to a neuron in pool n, but not on the identity of the two neurons. The input current to a neuron i in group Γ_n is generated by the spikes of all neurons in the network,

$$I_{i,n} = \sum_j \sum_f w_{ij}\, \alpha_{ij}(t - t_j^f)$$

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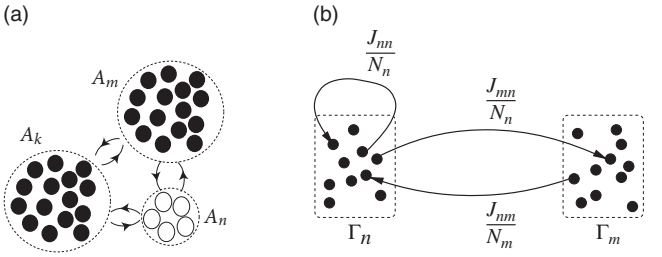

Fig. 12.10 Several interacting populations of neurons. (a) A neuron in population Γ_k is driven by the population activity A_m and A_n of other groups, as well as by the activity A_k of its own population. (b) All neurons in group Γ_n are coupled with synaptic efficacy $w_{ij} = J_{nn}/N_n$. Each pair of neurons i, j with the presynaptic j in groups Γ_m and the postsynaptic neuron i in Γ_n is coupled via $w_{ij} = J_{nm}/N_m$.

$$= \sum_m J_{nm} \int_0^\infty \alpha_{nm}(s) \sum_{j \in \Gamma_m} \sum_f \frac{\delta(t - t_j^f - s)}{N_m} ds\,, \tag{12.10}$$

where $\alpha_{nm}(t - t_j^f)$ denotes the time course of a postsynaptic current caused by spike firing at time t_j^f of the presynaptic neuron j which is part of population m. We use Eq. (12.9) to replace the sum on the right-hand side of Eq. (12.10) and obtain

$$I_n = \sum_m J_{nm} \int_0^\infty \alpha(s) A_m(t - s)\, \mathrm{d}s\,. \tag{12.11}$$

We have dropped the index i since the input current is the same for all neurons in pool n.

Thus, we conclude that the interaction between neurons of different pools can be summarized by the population activity $A(t)$ of the respective pools. Note that Eq. (12.11) is a straightforward generalization of Eq. (12.4) and could have been "guessed" immediately; external input I^{ext} could be added as in Eq. (12.4).

12.3.6 Distance-dependent connectivity

The cortex is a rather thin sheet of cells. Cortical columns extend vertically across the sheet. As we have seen before, the connection probability within a column depends on the layer where pre- and postsynaptic neurons are located. In addition to this vertical connectivity, neurons make many horizontal connections to neurons in other, cortical columns in the same, but also in other, areas of the brain. Within the same brain area the probability of making a connection is often modeled as distance dependent. Note that distance dependence is a rather coarse feature, because the actual connectivity depends also on the function of the pre- and postsynaptic cell. In the primary visual area, for example, it has been found that pyramidal neurons with a preferred orientation for horizontal bars are more likely to make connections to other columns with a similar preferred orientation (Angelucci and Bressloff, 2006).

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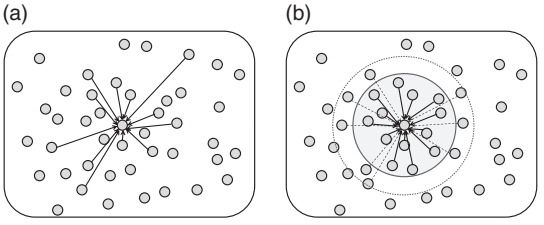

Fig. 12.11 Distance-dependent connectivity. (a) Random coupling where the probability of making a connection falls off with distance. Incoming connections to a single selected neuron are shown. (b) Full connectivity in a local neighborhood, but the connection strength falls off as a function of distance. Incoming connections to a single selected neuron are strong (thick solid arrow among close neighbors), weak (dashed) or non-existent (for distant neurons).

For models of distance-dependent connectivity it is necessary to assign to each model neuron i a location $x(i)$ on the two-dimensional cortical sheet. Two different algorithmic procedures can be used to assign distance-dependent connectivity (Fig. 12.11). The first one assumes full connectivity with a strength w_{ij} which falls off with distance

$$w_{ij} = w(|x(i) - x(j)|), \tag{12.12}$$

where $x(i)$ and $x(j)$ denote the location of post- and presynaptic neurons, respectively, and w is a function into the real numbers. For convenience, one may assume finite support so that w vanishes for distances $|x(i) - x(j)| > d$.

The second alternative is to give all connections the same weight, but to assume that the probability P of forming a connection depends on the distance

$$\text{Prob}(w_{ij} = 1) = P(|x(i) - x(j)|)\,, \tag{12.13}$$

where $x(i)$ and $x(j)$ denote the location of post- and presynaptic neurons, respectively and P is a function into the interval $[0, 1]$.

Example: Expected number of connections

Let us assume that the density of cortical neurons at location y is $\rho(y)$. The expected number of connections that a single neuron i located at position $x(i)$ receives is then $C_i = \int P(|x(i) - y|)\,\rho(y)\,\mathrm{d}y$. If the density is constant, $\rho(y) = \rho_0$, then the expected number C of input synapses is the same for all neurons and controlled by the integral of the connection probability, i.e., $C_i = \rho_0 \int P(|x(i) - y|)\,\mathrm{d}y$.

12.3.7 Spatial continuum limit (*)

The physical location of a neuron in a population often reflects the task of a neuron. In the auditory system, for example, neurons are organized along an axis that reflects the

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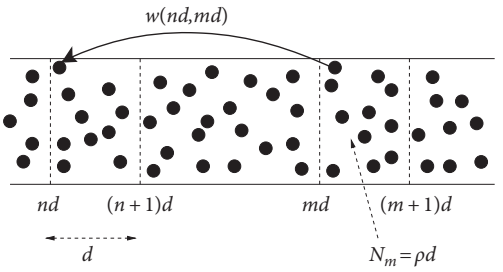

Fig. 12.12 In a spatially continuous ensemble of neurons, the number of neurons in a segment of size d is $N = \rho\, d$. The efficacy w_{ij} between two neurons depends on their location. The coupling strength between a presynaptic neuron j at position $x(j) \approx md$ and a postsynaptic neuron i at location $x(i) \approx nd$ is $w_{ij} \approx w(nd, md)$.

neurons' preferred frequency. A neuron at one end of the axis will respond maximally to low-frequency tones; a neuron at the other end to high frequencies. As we move along the axis the preferred frequency changes gradually. In the visual cortex, the preferred orientation changes gradually as one moves along the cortical sheet. For neurons organized in a spatially extended multi-dimensional network, a description by discrete pools does not seem appropriate. We will indicate in this section that a transition from discrete pools to a continuous population is possible. Here we give a short heuristic motivation of the equations. A thorough derivation along a slightly different line of arguments will be performed in Chapter 18.

To keep the notation simple we consider a population of neurons that extends along a one-dimensional axis; see Fig. 12.12. We assume that the interaction between a pair of neurons i, j depends only on their location x or y on the line. If the location of the presynaptic neuron is y and that of the postsynaptic neuron is x, then $w_{ij} = w(x, y)$. In other words we assume full, but spatially dependent, connectivity and neglect potential random components in the connectivity pattern.

In order to use the notion of population activity as defined in Eq. (12.11), we start by discretizing space in segments of size d. The number of neurons in the interval $[n\,d, (n+1)\,d]$ is $N_n = \rho\, d$ where ρ is the spatial density. Neurons in that interval form the group Γ_n. We now change our notation with respect to the discrete population and replace the subscript m in the population activity A_m with the spatial position of the neurons in that group

$$A_m(t) \longrightarrow A(md, t) = A(y, t)\,. \tag{12.14}$$

Since the efficacy of a pair of neurons with $i \in \Gamma_n$ and $j \in \Gamma_m$ is by definition $w_{ij} = J_{nm}/N_m$ with $N_m = \rho\, d$, we have $J_{nm} = \rho\, d\, w(nd, md)$. We use this in Eq. (12.11) and find for the input current

$$I(nd, t) = \rho \sum_m d\, w(nd, md) \int_0^\infty \alpha(s) A(md, t-s)\, \mathrm{d}s\,, \tag{12.15}$$

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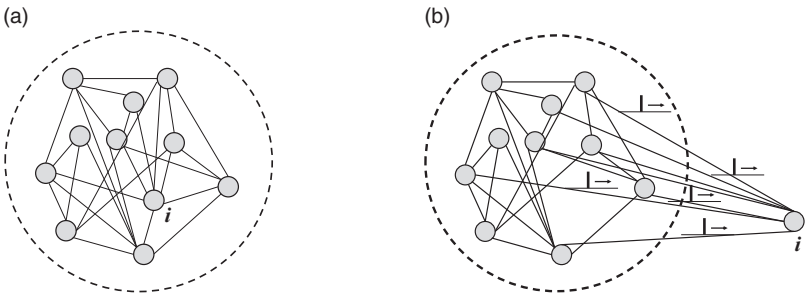

Fig. 12.13 The essence of a mean-field argument. (a) A fully connected population of neurons (not all connections are shown). An arbitrary neuron in the network is marked as i. (b) Neuron i has been pulled out of the network to show that it receives input spikes from the whole population. Hence it is driven by the population activity $A(t)$. The same is true for all other neurons.

where $\alpha(s)$ describes the time course of the postsynaptic current caused by spike firing in one of the presynaptic neurons. For $d \to 0$, the summation on the right-hand side can be replaced by an integral and we arrive at

$$I(x,t) = \rho \int w(x,y) \int_0^\infty \alpha(s) A(y,t-s)\, \mathrm{d}s\, \mathrm{d}y\,, \tag{12.16}$$

which is the final result. To rephrase Eq. (12.16) in words, the input to neurons at location x depends on the spatial distribution of the population activity convolved with the spatial coupling filter $w(x,y)$ and the temporal filter $\alpha(s)$. The population activity $A(y,t-s)\Delta s$ is the number of spikes in a short interval Δs summed across neurons in the neighborhood around y normalized by the number of neurons in that neighborhood.

12.4 From microscopic to macroscopic

In this section we will give a first example of how to make the transition from the properties of single spiking neurons to the population activity in a homogeneous group of neurons. We focus here on stationary activity.

In order to understand the dynamic response of a population of neurons to a changing stimulus, and to analyze the stability of the dynamics with respect to oscillations or perturbations, we will need further mathematical tools to be developed in the next two chapters. As we have see in Chapters 13 and 14, the dynamics depends, apart from the coupling, also on the specific choice of neuron model. However, if we want to predict the level of *stationary activity* in a large network of neurons, i.e., if we do not worry about the temporal aspects of population activity, then knowledge of the single-neuron gain function (f–I curve, or frequency–current relation) is completely sufficient to predict the population activity.

The basic argument is as follows (Fig. 12.13). In a homogeneous population, each

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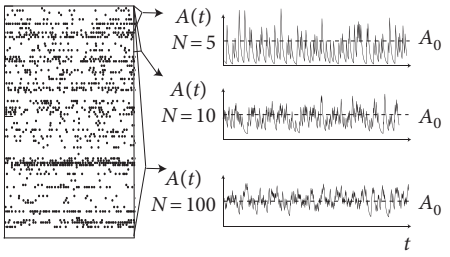

Fig. 12.14 Asynchronous firing. The empirical population activity $A(t)$, defined as an average over the spikes across a group of N neurons, can be plotted after smoothing spikes with a filter $\gamma(s)$ (here the filter is exponential). In the state of stationary asynchronous activity, the filtered population activity converges toward a constant value A_0 if the size N of the group is increased (top: $N = 5$; middle $N = 10$; bottom $N = 100$).

neuron receives input from *many* others, either from the same population, or from other populations, or both. Thus, a single neuron takes as its input a large (and in the case of a fully connected network even a *complete*) sample of the momentary population activity $A(t)$. This has been made explicit in Eq. (12.4) for a single population and in Eq. (12.11) for multiple populations. To keep the arguments simple, in what follows we focus on a single fully connected population. In a homogeneous population, no neuron is different from any other one, so all neurons in the network receive the same input.

Moreover, under the assumption of stationary network activity, the neurons can be characterized by a constant mean firing rate. In this case, the population activity $A(t)$ must be directly related to the constant single-neuron firing rate ν. We show in Section 12.4.2 that, in a homogeneous population, the two are in fact equal: $A(t) = \nu$. We emphasize that the argument sketched here and in the next subsections is completely independent of the choice of neuron model and holds for detailed biophysical models of the Hodgkin–Huxley type just as well as for an adaptive exponential integrate-and-fire model or a spike response model with escape noise. The argument for the stationary activity will now be made more precise.

12.4.1 Stationary activity and asynchronous firing

We define asynchronous firing of a neuronal population as a macroscopic firing state with constant activity $A(t) = A_0$. In this section we show that in a homogeneous population such asynchronous firing states exist and derive the value A_0 from the properties of a single neuron. In fact, we shall see that the only relevant single-neuron property is its gain function, i.e., its mean firing rate as a function of input. More specifically, we will show that the knowledge of the gain function $g(I_0)$ of a *single* neuron and the coupling parameter J_0 is sufficient to determine the activity A_0 during asynchronous firing.

At first glance it might look absurd to search for a constant activity $A(t) = A_0$, because the population activity has been defined in Eq. (12.1) as a sum over δ-functions. Empirically the population activity is determined as the spike count across the population in a

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finite time interval Δt or, more generally, after smoothing the δ-functions of the spikes with some filter. If the filter is kept fixed, while the population size is increased, the population activity in the stationary state of asynchronous firing approaches the constant value A_0 (Fig. 12.14). This argument will be made more precise below.

12.4.2 Stationary activity as single-neuron firing rate

The population activity A_0 is equal to the mean firing rate ν_i of a single neuron in the population. This result follows from a trivial counting argument and can best be explained by a simple example. Suppose that in a homogeneous population of $N = 1000$ neurons we observe over a time $T = 10\,\mathrm{s}$ a total number of 25 000 spikes. Under the assumption of stationary activity $A(t) = A_0$ the total number of spikes is $A_0 N T$ so that the population firing rate is $A_0 = 2.5\,\mathrm{Hz}$. Since all 1000 neurons are identical and receive the same input, the total number of 25 000 spikes corresponds to 25 spikes per neuron, so that the firing rate (spike count divided by measurement time) of a single neuron i is $\nu_i = 2.5\,\mathrm{Hz}$. Thus $A_0 = \nu_i$.

More generally, the assumption of stationarity implies that averaging over time yields, for each single neuron, a good estimate of the firing rate ν_0. The assumption of homogeneity implies that all neurons in the population have the same parameters and are statistically indistinguishable. Therefore a spatial average across the population and the temporal average give the same result:

$$A_0 = \nu_i \,, \tag{12.17}$$

where the index i refers to the firing rate of a single, but arbitrary neuron.

For an infinitely large population, Eq. (12.17) gives a formula to predict the population activity in the stationary state. However, real populations have a finite size N and each neuron in the population fires at moments determined by its intrinsic dynamics and possibly some intrinsic noise. The population activity $A(t)$ has been defined in Eq. (12.1) as an *empirically observable* quantity. In a finite population, the empirical activity fluctuates and we can, with the above arguments, only predict its expectation value

$$\langle A_0 \rangle = \nu_i \,. \tag{12.18}$$

The neuron models discussed in Parts I and II enable us to calculate the mean firing rate ν_i for a stationary input, characterized by a mean I_0 and, potentially, fluctuations or noise of amplitude σ. The mean firing rate is given by the gain function

$$\nu_i = g_\sigma(I_0) \,, \tag{12.19}$$

where the subscript σ is intended to remind the reader that the shape of the gain function depends on the level of noise. Thus, considering the pair of equations (12.18) and (12.19), we may conclude that the expected population activity in the stationary state can be predicted from the properties of single neurons.

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Example: Theory vs. simulation, expectation vs. observation

How can we compare the population activity $\langle A_0 \rangle$ calculated in Eq. (12.18) with simulation results? How can we check whether a population is in a stationary state of asynchronous firing? In a simulation of a population containing a finite number N of spiking neurons, the observed activity fluctuates. Formally, the (observable) activity $A(t)$ has been defined in Eq. (12.1) as a sum over δ-functions. The activity $\langle A_0 \rangle$ predicted by the theory is the *expectation* value of the observed activity. Mathematically speaking, the observed activity A converges for $N \to \infty$ in the weak topology to its expectation value. More practically this implies that we should convolve the observed activity with a continuous test function $\gamma(s)$ before comparing with A_0. We take a function γ with the normalization $\int_0^{s^{\max}} \gamma(s)\,\mathrm{d}s = 1$. For the sake of simplicity we assume furthermore that γ has finite support so that $\gamma(s) = 0$ for $s < 0$ or $s > s^{\max}$. We define

$$\overline{A}(t) = \int_0^{s^{\max}} \gamma(s) A(t-s)\,\mathrm{d}s\,. \tag{12.20}$$

The firing is asynchronous if the averaged fluctuations $\langle |\overline{A}(t) - A_0|^2 \rangle$ decrease with increasing N; see Fig. 12.14.

To keep the notation light, we normally write simply $A(t)$ here, even in places where it would be more precise to write $\langle A(t) \rangle$ (the expected population activity at time t, calculated by theory) or $\overline{A}(t)$ (the filtered population activity, derived from empirical measurement in a simulation or experiment). Only in places where the distinction between A, $\overline{A}$, and $\langle A \rangle$ is crucial do we use the explicit notation with bars or angle brackets.

12.4.3 Activity of a fully connected network

The gain function of a neuron is the firing rate ν as a function of its input current I. In the previous subsection, we have seen that the firing rate is equivalent to the expected value of the population activity A_0 in the state of asynchronous firing. We thus have

$$\langle A_0 \rangle = g_\sigma(I)\,. \tag{12.21}$$

The gain function in the absence of any noise (fluctuation amplitude $\sigma = 0$) will be denoted by g_0.

Recall that the total input I to a neuron of a fully connected population consists of the external input $I^{\mathrm{ext}}(t)$ and a component that is due to the interaction of the neurons within the population. We copy Eq. (12.4) to have the explicit expression of the input current

$$I(t) = w_0 N \int_0^\infty \alpha(s) A(t-s)\,\mathrm{d}s + I^{\mathrm{ext}}(t)\,. \tag{12.22}$$

Since the overall strength of the interaction is set by w_0, we can impose a normalization $\int_0^\infty \alpha(s)\,\mathrm{d}s = 1$. We now exploit the assumption of stationarity and set $\int_0^\infty \alpha(s) A(t-s)\,\mathrm{d}s = A_0$. The left-hand side is the filtered observed quantity which is in reality never exactly

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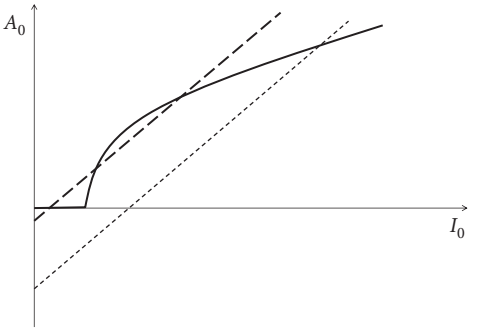

Fig. 12.15 Graphical solution for the fixed point A_0 of the activity in a population of spiking neurons. The intersection of the gain function $A_0 = g_0(I_0)$ (solid line) with the straight line $A_0 = [I_0 - I_0^{\rm ext}]/J_0$ (dotted) gives the value of the activity A_0. Depending on the parameters, several solutions may coexist (dashed line).

constant, but if the number N of neurons in the network is sufficiently large, we do not have to worry about small fluctuations around A_0. Note that α here plays the role of the test function introduced in the previous example.

Therefore, the assumption of stationary activity A_0 combined with the assumption of constant external input $I^{\rm ext}(t) = I_0^{\rm ext}$ yields a constant total driving current

$$I_0 = w_0 N A_0 + I_0^{\rm ext} . \tag{12.23}$$

Together with Eq. (12.21) we arrive at an *implicit* equation for the population activity A_0,

$$A_0 = g_0 \left(J_0 A_0 + I_0^{\rm ext} \right) , \tag{12.24}$$

where g_0 is the noise-free gain function of single neurons and $J_0 = w_0 N$. In words, the population activity in a homogeneous network of neurons with all-to-all connectivity can be calculated if we know the single-neuron gain function g_0 and the coupling strength J_0. This is the central result of this section, which is independent of any specific assumption about the neuron model.

A graphical solution of Eq. (12.24) is indicated in Fig. 12.15 where two functions are plotted: first, the mean firing rate $\nu = g_0(I_0)$ as a function of the input I_0 (i.e., the gain function); second, the population activity A_0 as a function of the total input I_0 (i.e., $A_0 = [I_0 - I_0^{\rm ext}]/J_0$; see Eq. (12.23)). The intersections of the two functions yield fixed points of the activity A_0.

As an aside we note that the graphical construction is identical to that of the Curie–Weiss theory of ferromagnetism which can be found in any physics textbook. More generally, the structure of the equations corresponds to the mean-field solution of a system with feedback. As shown in Fig. 12.15, several solutions may coexist. We cannot conclude from the figure whether one or several solutions are stable. In fact, it is possible that *all* solutions are unstable. In the last case, the network leaves the state of asynchronous firing and evolves toward

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an oscillatory state. The stability analysis of the asynchronous state requires equations for the population *dynamics*, which will be discussed in Chapters 13 and 14.

The parameter J_0 introduced above in Eq. (12.24) implies, at least implicitly, a scaling of weights $w_{ij} = J_0/N$ – as suggested earlier during the discussion of fully connected networks; see Eq. (12.6). The scaling with $1/N$ enables us to consider the limit of a large number of neurons: if we keep J_0 fixed, the equation remains the same, even if N increases. Because fluctuations of the observed population activity $A(t)$ around A_0 decrease as N increases, Eq. (12.24) becomes exact in the limit of $N \to \infty$.

Example: Leaky integrate-and-fire model with diffusive noise

We consider a large and fully connected network of identical leaky integrate-and-fire neurons with homogeneous coupling $w_{ij} = J_0/N$ and normalized postsynaptic currents ($\int_0^\infty \alpha(s)\mathrm{d}s = 1$). In the state of asynchronous firing, the total input current driving a typical neuron of the network is then

$$I_0 = I_0^{\text{ext}} + J_0 A_0 \,. \tag{12.25}$$

In addition, each neuron receives individual diffusive noise of variance σ^2 that could represent spike arrival from other populations. The single-neuron gain function (Siegert, 1951) in the presence of diffusive noise has already been stated in Chapter 8; see Eq. (8.54). We use the formula of the gain function to calculate the population activity

$$A_0 = g_\sigma(I_0) = \left\{ \tau_m \sqrt{\pi} \int_{\frac{u_r - RI_0}{\sigma}}^{\frac{\vartheta - RI_0}{\sigma}} \mathrm{d}u \, \exp\left(u^2\right) \left[1 + \mathrm{erf}(u)\right] \right\}^{-1} , \tag{12.26}$$

where σ with units of voltage measures the amplitude of the noise. The fixed points for the population activity are once more determined by the intersections of these two functions; see Fig. 12.16.

12.4.4 Activity of a randomly connected network

In the preceding subsections we have studied the stationary state of a large population of neurons for a given noise level. In Fig. 12.16 the noise was modeled explicitly as diffusive noise and can be interpreted as the effect of stochastic spike arrival from other populations or some intrinsic noise source inside each neuron. In other words, noise was added *explicitly* to the model while the input current $I_i(t)$ to neuron i arising from other neurons in the population was constant and the same for all neurons: $I_i = I_0$.

In a randomly connected network (and similarly in a fully connected network of finite size), the summed synaptic input current arising from other neurons in the population is, however, not constant but fluctuates around a mean value I_0, even if the population is in a stationary state of asynchronous activity. In this subsection, we discuss how to mathematically treat the additional noise arising from the network.

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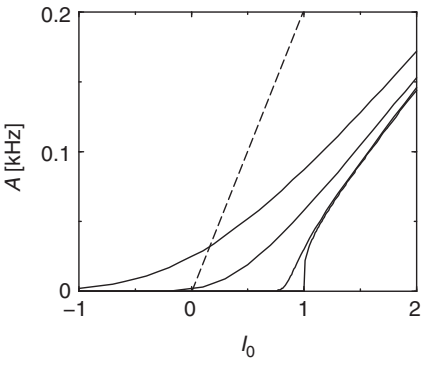

Fig. 12.16 Graphical solution for the fixed point A_0 in the case of a fully connected network of leaky integrate-and-fire neurons. The solid lines show the single-neuron firing rate as a function of the constant input current I_0 for four different noise levels, namely, $\sigma = 1.0, \sigma = 0.5$, $\sigma = 0.1, \sigma = 0.0$ (from top to bottom). The intersection of the gain function with the dashed line with slope $1/J_0$ gives solutions for the stationary activity A_0 in a population with excitatory coupling J_0. Other parameters: $\vartheta = 1$, $R = 1$, $\tau = 10\,\text{ms}$.

We assume that the network is in a stationary state where each neuron fires stochastically, independently, and at a constant rate ν, so that the firing of different neurons exhibits only chance coincidences. Suppose that we have a randomly connected network of N neurons where each neuron receives input from C_{pre} presynaptic partners. All weights are set equal to $w_{ij} = w$.

We are going to determine the firing rate $\nu = A_0$ of a typical neuron in the network self-consistently as follows. If all neurons fire at a rate ν then the mean input current to neuron i generated by its C_{pre} presynaptic partners is

$$\langle I_0 \rangle = C_{\text{pre}}\, q\, w\, \nu + I_0^{\text{ext}}\,, \tag{12.27}$$

where $q = \int_0^\infty \alpha(s)\,\text{d}s$ denotes the integral over the postsynaptic current and can be interpreted as the total electric charge delivered by a single input spike; see Section 8.2.

The input current is not constant but fluctuates with a variance σ_I^2 given by

$$\sigma_I^2 = C_{\text{pre}}\, w^2\, q_2\, \nu\,, \tag{12.28}$$

where $q_2 = \int_0^\infty \alpha^2(s)\,\text{d}s$; see Section 8.2.

Thus, if neurons fire at constant rate ν, we know the mean input current and its variance. In order to close the argument we use the single-neuron gain function

$$\nu = g_\sigma(I_0), \tag{12.29}$$

which is supposed to be known for arbitrary noise levels σ_I. If we insert the mean I_0 from Eq. (12.27) and its standard deviation σ_I from Eq. (12.28), we arrive at an implicit equation for the firing rate ν which we need to solve numerically. The mean population activity is then $\langle A_0 \rangle = \nu$.

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We emphasize that the above argument does not require any specific neuron model. In fact, it holds for biophysical neuron models of the Hodgkin–Huxley type as well as for integrate-and-fire models. The advantage of a leaky integrate-and-fire model is that an explicit mathematical formula for the gain function $g_\sigma(I_0)$ is available. An example will be given below. But we can use Eqs. (12.27)–(12.29) just as well for a homogeneous population of biophysical neuron models. The only difference is that we have to numerically determine the single-neuron gain function $g_\sigma(I_0)$ for different noise levels (with noise of the appropriate autocorrelation) before starting to solve the network equations.

Note also that the above argument is not restricted to a network consisting of a single population. It can be extended to several interacting populations. In this case, the expressions for the mean and variance of the input current contain contributions from the other populations, as well as from the self-interaction in the network. An example with interacting excitatory and inhibitory populations is given below.

The arguments that have been developed above for networks with a fixed number of presynaptic partners $C_{\rm pre}$ can also be generalized to networks with *asymmetric random* connectivity of fixed connection probability p and synaptic scaling $w_{ij} = J_0/\sqrt{N}$ (Amari, 1972; Sompolinksy *et al*., 1988; Cessac *et al*., 1994; van Vreeswijk and Sompolinsky, 1996; Ben Arous and Guionnet, 1995).

Brunel network: excitatory and inhibitory populations

The self-consistency argument will now be applied to the case of two interacting populations, an excitatory population with $N_{\rm E}$ neurons and an inhibitory population with $N_{\rm I}$ neurons. The neurons in both populations are modeled by leaky integrate-and-fire neurons. For the sake of convenience, we set the resting potential to zero ($u_{\rm rest} = 0$). We have seen in Chapter 8 that leaky integrate-and-fire neurons with diffusive noise generate spike trains with a broad distribution of interspike intervals when they are driven in the subthreshold regime. We will use this observation to construct a self-consistent solution for the stationary states of asynchronous firing.

We assume that excitatory and inhibitory neurons have the same parameters ϑ, τ_m, R, and u_r. In addition, all neurons are driven by a common external current $I^{\rm ext}$. Each neuron in the population receives $C_{\rm E}$ synapses from excitatory neurons with weight $w_{\rm E} > 0$ and $C_{\rm I}$ synapses from inhibitory neurons with weight $w_{\rm I} < 0$. If an input spike arrives at the synapses of neuron i from a presynaptic neuron j, its membrane potential changes by an amount $\Delta u_E = w_E\, qR/\tau_m$ if j is excitatory and $\Delta u_I = \Delta u_E\, w_I/w_E$ if j is inhibitory. Here q has units of electric charge. We set

$$\gamma = \frac{C_I}{C_E} \quad \text{and} \quad g = -\frac{w_I}{w_E} = -\frac{\Delta u_I}{\Delta u_E}\,. \tag{12.30}$$

Since excitatory and inhibitory neurons receive the same number of input connections in our model, we assume that they fire with a common firing rate ν. The total input current

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generated by the external current and by the lateral couplings is

$$\begin{aligned} I_0 &= I^{\text{ext}} + q \sum_j \nu_j w_j \\ &= I_0^{\text{ext}} + q\,\nu\, w_E C_E\,[1-\gamma g]\,. \end{aligned} \tag{12.31}$$

Because each input spike causes a jump of the membrane potential, it is convenient to measure the noise strength by the variance σ_u^2 of the membrane potential (as opposed to the variance σ_I^2 of the input). With the definitions of Chapter 8, we set $\sigma_u^2 = 0.5\sigma^2$ where, from Eq. (8.42),

$$\begin{aligned} \sigma^2 &= \sum_j \nu_j\, \tau\, (\Delta u_j^2) \\ &= \nu\, \tau\, (\Delta u_E)^2 C_E\,[1+\gamma g^2]\,. \end{aligned} \tag{12.32}$$

The stationary firing rate A_0 of the population with mean input I_0 and variance σ is copied from Eq. (12.26) and repeated here for convenience

$$A_0 = \nu = g_\sigma(I_0) = \frac{1}{\tau_m} \left\{ \sqrt{\pi} \int_{\frac{u_r - RI_0}{\sigma}}^{\frac{\vartheta - RI_0}{\sigma}} \exp\left(x^2\right)\,[1+\text{erf}(x)]\,\text{d}x \right\}^{-1}. \tag{12.33}$$

In a stationary state we must have $A_0 = \nu$. To get the value of A_0 we must therefore solve Eqs. (12.31)–(12.33) simultaneously for ν and σ. Since the gain function, i.e., the firing rate as a function of the input I_0, depends on the noise level σ, a simple graphical solution as in Fig. 12.15 is no longer possible. Numerical solutions of Eqs. (12.31)–(12.33) have been obtained by Amit and Brunel (1997a,b). For a mixed graphical-numerical approach see Mascaro and Amit (1999).

Below we give some examples of how to construct self-consistent solutions. For convenience we always set $\vartheta = 1$, $q = 1$, $R = 1$ and $\tau_m = 10\,\text{ms}$ and work with a unit-free current $I \to h$. Our aim is to find connectivity parameters such that the mean input to each neuron is $h = 0.8$ and its variance $\sigma = 0.2$.

Figure 12.17a shows that $h_0 = 0.8$ and $\sigma = 0.2$ correspond to a firing rate of $A_0 = \nu \approx 16\,\text{Hz}$. We set $\Delta u_E = 0.025$, i.e., 40 simultaneous spikes are necessary to make a neuron fire. Inhibition has the same strength $w_I = -w_E$ so that $g = 1$. We constrain our search to solutions with $C_E = C_I$ so that $\gamma = 1$. Thus, on the average, excitation and inhibition balance each other. To get an average input potential of $h_0 = 0.8$ we therefore need a constant driving current $I^{\text{ext}} = 0.8$.

To arrive at $\sigma = 0.2$ we solve Eq. (12.32) for C_E and find $C_E = C_I = 200$. Thus for this choice of the parameters the network generates enough noise to allow a stationary solution of asynchronous firing at 16 Hz.

Note that, for the same parameters, the inactive state where all neurons are silent is also a solution. Using the methods discussed in this section we cannot say anything about the stability of these states. For the stability analysis see Chapter 13.

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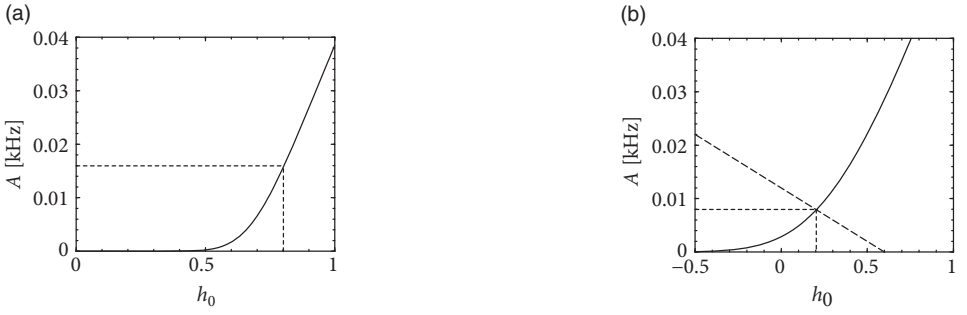

Fig. 12.17 (a) Mean activity of a population of integrate-and-fire neurons with diffusive noise amplitude of $\sigma = 0.2$ as a function of $h_0 = RI_0$. For $h_0 = 0.8$ the population rate is $\nu \approx 16\,\text{Hz}$ (dotted line). (b) Mean activity of a population of integrate-and-fire neurons with diffusive noise of $\sigma = 0.54$ as a function of $h_0 = RI_0$. For $h_0 = 0.2$ the population rate is $\nu = 8\,\text{Hz}$ (dotted line). The dashed line shows $A_0 = [h_0 - h_0^{\text{ext}}]/J^{\text{eff}}$ with an effective coupling $J^{\text{eff}} < 0$.

Example: Inhibition dominated network

About 80–90% of the neurons in the cerebral cortex are excitatory and the remaining 10–20% inhibitory. Let us suppose that we have $N_E = 8000$ excitatory and $N_I = 2000$ inhibitory neurons in a cortical column. We assume random connectivity with a connection probability of 10% and take $C_E = 800$, $C_I = 200$ so that $\gamma = 1/4$. As before, spikes arriving at excitatory synapses cause a voltage jump $\Delta u_E = 0.025$, i.e., an action potential can be triggered by the simultaneous arrival of 40 presynaptic spikes at excitatory synapses. If neurons are driven in the regime close to threshold, inhibition is rather strong and we take $\Delta u_I = -0.125$ so that $g = 5$. Even though we have fewer inhibitory than excitatory neurons, the mean feedback is then dominated by inhibition since $\gamma g > 1$. We search for a consistent solution of Eqs. (12.31)–(12.33) with a spontaneous activity of $\nu = 8\,\text{Hz}$.

Given the above parameters, the variance is $\sigma \approx 0.54$; see Eq. (12.32). The gain function of integrate-and-fire neurons gives us for $\nu = 8\,\text{Hz}$ a corresponding total potential of $h_0 \approx 0.2$; see Fig. 12.17b. To attain h_0 we have to apply an external stimulus $h_0^{\text{ext}} = RI^{\text{ext}}$ which is slightly larger than h_0 since the net effect of the lateral coupling is inhibitory. Let us introduce the effective coupling $J^{\text{eff}} = \tau C_E \Delta u_E (1 - \gamma g)$. Using the above parameters we find from Eq. (12.31) that $h_0^{\text{ext}} = h_0 - J^{\text{eff}} A_0 \approx 0.6$.

The external input could, of course, be provided by (stochastic) spike arrival from other columns in the same or other areas of the brain. In this case Eq. (12.31) is to be replaced by

$$h_0 = \tau_m \nu \Delta u_E C_E [1 - \gamma g] + \tau_m \nu_{\text{ext}} \Delta u_{\text{ext}} C_{\text{ext}}\,, \tag{12.34}$$

with C_{ext} the number of connections that a neuron receives from neurons outside the population, Δu_{ext} their typical coupling strength characterized by the amplitude of the

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voltage jump, and $\nu_{\rm ext}$ their spike arrival rate (Amit and Brunel, 1997a,b). Owing to the extra stochasticity in the input, the variance σ_u^2 of the membrane voltage is larger

$$\sigma_u^2 = 0.5\sigma^2 = 0.5\tau_m\,\nu\,(\Delta u_E)^2\,C_E\,[1+\gamma g^2] + 0.5\tau_m\,\nu_{\rm ext}(\Delta u_{\rm ext})^2\,C_{\rm ext}. \qquad (12.35)$$

The equations (12.33), (12.34) and (12.35) can be solved numerically (Amit and Brunel, 1997a,b). The analysis of the stability of the solution is slightly more involved (Brunel and Hakim, 1999; Brunel, 2000), and will be considered in Chapter 13.

Example: Vogels–Abbott network

The structure of the network studied by Vogels and Abbott (Vogels and Abbott, 2005, 2009; Brette *et al.*, 2007) is the same as that for the Brunel network: excitatory and inhibitory model neurons have the same parameters and are connected with the same probability p within and across the two sub-populations. Therefore inhibitory and excitatory neurons fire with the same mean firing rate (see Section 12.4.4) and with hardly any correlations above chance level (Fig. 12.18). The two main differences to the Brunel network are: (i) the choice of random connectivity in the Vogels–Abbott network does not preserve the number of presynaptic partners per neuron so that some neurons receive more and others less than pN connections; (ii) neurons in the Vogels–Abbott network communicate with each other by conductance-based synapses. A spike fired at time t_j^f causes a change in conductance

$$\tau_g \frac{{\rm d}g}{{\rm d}t} = -g + \tau_g \Delta g \sum_f \delta(t - t_j^f)\,. \qquad (12.36)$$

Thus, a synaptic input causes for $t > t_j^f$ a contribution to the conductance $g(t) = \Delta g \exp[-(t-t_j^f)/\tau_g]$. The neurons are leaky integrate-and-fire units.

As will be discussed in more detail in Section 13.6.3, the dominant effect of conductance-based input is a decrease of the effective membrane time constant. In other words, if we consider a network of leaky integrate-and-fire neurons (with resting potential $u_{\rm rest} = 0$), we may use again the Siegert formula of Eq. (12.26)

$$A_0 = g_\sigma(I_0) = \left\{ \tau_{\rm eff}(I_0,\sigma)\sqrt{\pi} \int_{\frac{u_r - RI_0}{\sigma}}^{\frac{\vartheta - RI_0}{\sigma}} {\rm d}u\, \exp\left(u^2\right)\,[1+{\rm erf}(u)] \right\}^{-1} \qquad (12.37)$$

in order to calculate the population activity A_0. The main difference to the current-based model is that the mean input current I_0 and the fluctuations σ of the membrane voltage now also enter into the time constant $\tau_{\rm eff}$. The effective membrane time constant $\tau_{\rm eff}$ in simulations of conductance-based integrate-and-fire neurons is sometimes four or five times shorter than the raw membrane time constant τ_m (Destexhe *et al.*, 2003; Vogels and Abbott, 2005, 2009).

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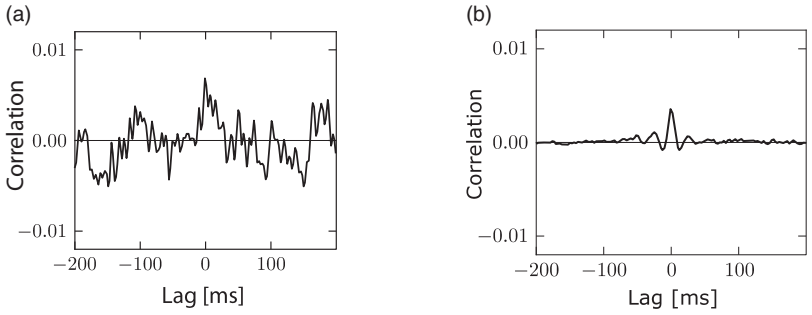

Fig. 12.18 Pairwise correlation of neurons in the Vogels–Abbott network. (a) Excess probability of observing a spike in a neuron i at time t and a spike in neuron j at time t' for various time lags $t - t'$, after subtraction of chance coincidences. Normalization such that two identical spike trains would give a value of 1 at zero time lag. (b) As in (a), but averaged across 171 randomly chosen pairs. The pairwise correlations are extremely small in this randomly connected network of 8000 excitatory and 2000 inhibitory neurons with connection probability $p = 0.02$ and conductance-based synapses; see Vogels *et al*. (2011) for details. The mean firing rate is $A_0 = 5$ Hz.

The Siegert formula holds in the limit of short synaptic time constants ($\tau_E \to 0$ and $\tau_I \to 0$). The assumption of short time constants for the conductances is necessary, because the Siegert formula is valid for white noise, corresponding to short pulses. However, the gain function of integrate-and-fire neurons for colored diffusive noise can also be determined (Fourcaud and Brunel, 2002); see Section 13.6.4.

12.4.5 Apparent stochasticity and chaos in a deterministic network

In this section we discuss how a network of *deterministic* neurons with fixed random connectivity can generate its own noise. In particular, we will focus on spontaneous activity and argue that there exist stationary states of asynchronous firing at low firing rates which have broad distributions of interspike intervals (Fig. 12.19) even though individual neurons are deterministic. The arguments made here have tacitly been used throughout Section 12.4.

Van Vreeswijk and Sompolinsky (1996, 1998) used a network of binary neurons to demonstrate broad interval distribution in deterministic networks. Amit and Brunel (1997a,b) were the first to analyze a network of integrate-and-fire neurons with fixed random connectivity. While they allowed for an additional fluctuating input current, the major part of the fluctuations were in fact generated by the network itself. The theory of randomly connected integrate-and-fire neurons has been further developed by Brunel and Hakim (1999). In a later study, Brunel (2000) confirmed that asynchronous highly irregular firing can be a stable solution of the network dynamics in a completely deterministic network consisting of excitatory and inhibitory integrate-and-fire neurons. Work of Tim Vogels and Larry Abbott has shown that asynchronous activity at low firing rates can indeed be observed reliably

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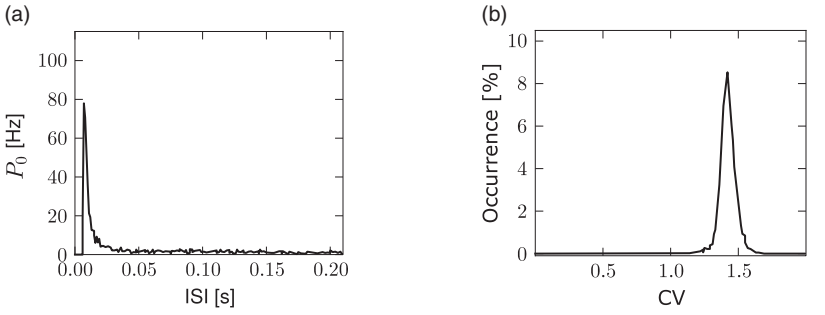

Fig. 12.19 Interspike interval distributions in the Vogels–Abbott network. (a) Interspike interval distribution of a randomly chosen neuron. Note the long tail of the distribution. The width of the distribution can be characterized by a coefficient of variation of $CV = 1.9$. (b) Distribution of the CV index across all 10 000 neurons of the network. Bin width of horizontal axis is 0.01.

in networks of leaky integrate-and-fire neurons with random coupling via conductance-based synapses (Vogels and Abbott, 2005, 2009; Brette *et al*., 2007). The analysis of randomly connected networks of integrate-and-fire neurons (Brunel, 2000) is closely related to earlier theories for random nets of formal analog or binary neurons (Amari, 1972, 1974, 1977; Kree and Zippelius, 1991; Nützel, 1991; Crisanti and Sompolinsky, 1988; Cessac *et al*., 1994). However, the reset of neurons after each spike can be the cause of additional instabilities that have been absent in these earlier networks with analog or binary neurons.

Random connectivity of the network plays a central role in the arguments. We focus on randomness with a fixed number C of presynaptic partners. Sparse connectivity means that the ratio

$$\delta = \frac{C}{N} \ll 1 \tag{12.38}$$

is a small number. Formally, we may take the limit as $N \to \infty$ while keeping C fixed. As a consequence of the sparse random network connectivity two neurons i and j share only a small number of common inputs. In the limit of $C/N \to 0$ the probability that neurons i and j have a common presynaptic neuron vanishes. Thus, *if* the presynaptic neurons fire stochastically, then the input spike trains that arrive at neuron i and j are independent (Derrida *et al*., 1987; Kree and Zippelius, 1991). In that case, the input of neurons i and j can be described as an uncorrelated stochastic spike arrival which in turn can be approximated by a diffusive noise model; see Chapter 8. Therefore, in a large and suitably constructed random network, correlations between spiking neurons can be arbitrarily low (Renart *et al*., 2010); see Fig. 12.18.

Note that this is in stark contrast to a fully connected network of finite size where neurons receive highly correlated input, but the correlations are completely described by the time course of the population activity.

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12.5 Summary

Neurons do not work in isolation, but are embedded in networks of neurons with similar properties. Such networks of similar neurons can be organized as distributed assemblies or as local pools of neurons. Groups of neurons with similar properties can be approximated as homogeneous or weakly heterogeneous populations of neurons. In mathematical models, the connectivity within the population is typically all-to-all or random.

The population activity is defined as the number of spikes fired in a short instant of time, averaged across the population. Since each neuron in a population receives input from many others (from the same and/or from other populations) its total input at each moment in time depends on the activity of the presynaptic population(s). Hence the population activity $A(t)$ controls the mean drive of a postsynaptic neuron.

If the population in a self-connected network is in a state of stationary activity, the expected value $\langle A_0 \rangle$ of the population activity can be determined self-consistently. To do so, we approximate the mean drive of a neuron by $\langle A_0 \rangle$ and exploit that the firing rate of the population must be equal to that of a single neuron. In the stationary state of asynchronous activity the population activity is therefore fully determined by the gain function of a single neuron (i.e., its frequency–current curve) and the strength of feedback connections. This result, which is an example of a (stationary) mean-field theory, is independent of any neuron model. The mean-field solution is exact for a fully connected network in the limit of a large number of neurons ($N \to \infty$), and a good approximation for large randomly connected networks.

The assumption of a stationary state is, of course, a strong limitation. In reality, the activity of populations in the brain responds to external input and may also show non-trivial intrinsic activity changes. In other words, the population activity is in most situations time-dependent. The mathematical description of the dynamics of the population activity is the topic of the next three chapters.

Literature

The development of population equations, also called "neural mass" equations, had a first boom around 1972 with several papers by different researchers (Wilson and Cowan, 1972; Knight, 1972; Amari, 1972). Equations very similar to the population equations have sometimes also been used as effective rate model neurons (Grossberg, 1973). The transition from stationary activity to dynamics of population in the early papers is often ad hoc (Wilson and Cowan, 1972).

The study of randomly connected networks has a long tradition in the mathematical sciences. Random networks of formal neurons have been studied by numerous researchers (e.g., Amari 1972, 1974, 1977; Sompolinksy *et al*. 1988; Cessac *et al*. 1994; van Vreeswijk and Sompolinsky 1996, 1998), and a mathematically precise formulation of mean-field theories for random nets is possible (Faugeras *et al*., 2009).

The theory for randomly connected integrate-and-fire neurons (Amit and Brunel, 1997a;

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Brunel and Hakim, 1999; Brunel, 2000; Renart *et al*., 2010) builds on earlier studies of formal random networks. The Siegert formula for the gain function of a leaky integrate-and-fire model with diffusive noise appears in several classic papers (Siegert, 1951; Amit and Brunel, 1997a; Brunel and Hakim, 1999; Brunel, 2000). In arbitrarily connected integrate-and-fire networks, the dynamics is highly complex (Cessac, 2008).

Exercises

1. **Fully connected network.** *Assume a fully connected network of N Poisson neurons with firing rate $\nu_i(t) = g(I_i(t)) > 0$. Each neuron sends its output spikes to all other neurons as well as back to itself. When a spike arrives at the synapse from a presynaptic neuron j to where a postsynaptic neuron i is, it generates a postsynaptic current*

$$I_i^{\text{syn}} = w_{ij} \exp[-(t - t_j^f)/\tau_s] \quad \text{for } t > t_j^f \,, \tag{12.39}$$

where t_j^f is the moment when the presynaptic neuron j fired a spike and τ_s is the synaptic time constant.

(a) Assume that each neuron in the network fires at the same rate ν. Calculate the mean and the variance of the input current to neuron i.

Hint: Use the methods of Chapter 8.

(b) Assume that all weights are of equal weight $w_{ij} = J_0/N$. Show that the mean input to neuron i is independent of N and that the variance decreases with N.

(c) Evaluate the mean and variance under the assumption that the neuron receives 4000 inputs at a rate of 5 Hz. *The synaptic time constant is 5* ms *and $J_0 = 1$* μA.

2. **Stochastically connected network.** *Consider a network analogous to that discussed in the previous exercise, but with a synaptic coupling current*

$$I_i^{\text{syn}} = w_{ij} \left\{ \left(\frac{1}{\tau_1} \right) \exp[-(t - t_j^f)/\tau_1] - \left(\frac{1}{\tau_2} \right) \exp[-(t - t_j^f)/\tau_2] \right\} \quad \text{for } t > t_j^f \,, \tag{12.40}$$

which contains both an excitatory and an inhibitory component.

(a) Calculate the mean synaptic current and its variance assuming arbitrary coupling weights w_{ij}. How do the mean and variance depend upon the number of neurons N?

(b) Assume that the weights have a value $J_0/\sqrt{N}$. How do the mean and variance of the synaptic input current scale as a function of N?

3. **Mean-field model.** *Consider a network of N neurons with all-to-all connectivity and scaled synaptic weights $w_{ij} = J_0/N$. The transfer function (rate as a function of input potential) is piecewise linear:*

$$f = g(h) = \frac{h - h_1}{h_2 - h_1} \quad \text{for } h_1 \le h \le h_2 \,. \tag{12.41}$$

The rate vanishes for $h < h_1$ and is constant $f = 1$ (in units of the maximal rate) for $h > h_2$.

The dynamics of the input potential h_i of a neuron i are given by

$$\tau \frac{\mathrm{d}h_i}{\mathrm{d}t} = -h_i + RI_i(t) \,, \tag{12.42}$$

with

$$I(t) = I^{\text{ext}} + \sum_j w_{ij} \alpha(t - t_j^f) \,. \tag{12.43}$$

(a) Find graphically the fixed points of the population activity in the network with connections as described above.

(b) Determine the solutions analytically.

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4. **Mean field in a network of two populations.** *We study a network of excitatory and inhibitory neurons. Each excitatory neuron has, in the stationary state, a firing rate*

$$e = f(I) = \gamma I \quad \text{for } I > 0 \quad \text{and } f(I) = 0 \text{ otherwise.} \tag{12.44}$$

Inhibitory neurons have a firing rate

$$s = g(I) = I^2 \quad \text{for } I > 0 \quad \text{and } g(I) = 0 \text{ otherwise.} \tag{12.45}$$

Assume that we have a large network of N excitatory and N inhibitory neurons, where $N \gg 1$. The input to an excitatory neuron i is

$$I_i(t) = I_0 + \sum_{k=1}^{N} \frac{w}{N} e_k - \sum_{n=1}^{N} \frac{1}{N} s_n, \tag{12.46}$$

where e_k is the rate of excitatory neuron k and s_n the rate of inhibitory neuron n. The input to an inhibitory neuron n is

$$I_i(t) = \sum_{k=1}^{N} \frac{w}{N} e_k. \tag{12.47}$$

(a) Give the analytical solution for the steady state of the network. If there are several solutions, indicate the stability of each of these.

Hint: Introduce the parameter $A = \sum_{k=1}^{N} \frac{1}{N} e_k$ for the excitatory population activity; express the activity of the inhibitory population by A and insert the result into the excitatory equation.

(b) Solve graphically for the stationary state of the activity in the network, for two qualitatively different *regimes which you choose. Free parameters are the coupling strength w and the external input I.*

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