How the visual system can detect feature homogeneity from spike latencies

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Abstract

We propose that homogeneous image regions may be used to quickly segment a visual scene, when an object hypothesis is not yet available. In a cooperative model of the konio-cellular and the parvo- or magno-cellular visual pathway of primates, we demonstrate how homogeneity-selective responses can be gained from LGN neurons using a spike-latency code, and how these responses can confine edge detection to borders between mid-sized objects. We propose that spike-latency-based homogeneity detection is a general neural principle that may apply to any sensory feature and modality. The mechanism is sensitive to exact spike timing, which makes it vulnerable to noise. Nevertheless we demonstrate how the mechanism can be made robust against realistic levels of neural cross talk in the brain. We show that biphasic synaptic events do yield very sharp post synaptic currents that maintain good separation of homogeneity responses in massively noisy environments. The same technique opens up a neural method of tuning neurons to varying degrees of homogeneity. The simple neural implementation, generality, robustness, and variable tuning make homogeneity-detection an attractive scheme of computation in spiking neural networks and in the brain.

Keywords: vision; spike-latency-code; feature homogeneity; segmentation; biphasic synaptic events, PSC-shaping

1 Introduction

In early visual processing, speed matters. If an animal is confronted with a visual scene, it is important to quickly arrive at an hypothesis about its main constituents so that an appropriate reaction (e.g. escape) is possible. In order to quickly segment the visual input, the information has to be reduced to its most salient parts. After a first object hypothesis has been established
at higher processing levels, a top-down signal may then enable a refined analysis of object detail (Körner et al., 1999a; Ullman, 2000a). This poses the question, how the visual system can perform a segmentation, when an hypothesis about objects in the scene is not yet available. We propose that information on homogeneous image regions, extracted by konio-cellular neurons in the lateral geniculate nucleus (LGN), confines edge detection to borders between mid-sized objects. The neural design pattern we use for modeling this effect turns out to be so simple that it can be universally applied, not in the visual domain, but for all modalities.

1.1 Magno-, parvo-, and konio-cellular pathway of the primate visual system

Primates have two major pathways through which visual information flows from the retina via the LGN to the primary visual cortex: the parvo-cellular and the magno-cellular pathway. Magno-cells in the LGN have large receptive fields and are highly contrast sensitive. Parvo-cells have smaller receptive fields and their contrast sensitivity is comparatively low, however, many of them are sensitive to color (wavelength). Magno- and parvo-cellular neurons account for 90% of all LGN relay cells. The remaining 10% belong to a third, less well investigated pathway, the so-called konio-cellular pathway, which is thought to be a phylogenetically old part of the visual system.

Konio-cellular neurons are a very heterogeneous class of cells. Xu et al. (2001) report that many konio-cells in the LGN of owl monkeys have very large receptive fields, up to three degrees of visual angle. Of the investigated konio-cellular neurons, 34% did not respond to gratings, but were activated by flashes or moving bars. Fifty-three percent of the konio-cells showed sustained responses to a stationary stimulus in their receptive field for at least five seconds, 47% responded transiently. Only 60% had typical center-surround receptive fields. At matched eccentricities, konio-cells exhibited lower spatial and intermediate temporal resolution compared to parvo- and magno-cells.

Projections from konio-cellular neurons of the LGN to cortical area V1 terminate in the cytochrome-oxidase-blobs of the superficial layers (Lund et al., 1994; Hendry and Reid, 2000). By contrast, inputs from the magno-cellular and parvo-cellular pathways terminate in layer four. Moreover, while magno- and parvo-cellular neurons exclusively project to V1, konio-cellular inputs have been identified in higher areas of the ventral visual pathway (Hernandez-Gonzalez et al., 1994). Because of their large receptive fields, konio-cellular neurons cannot resolve fine detail, however, they can provide information about large homogeneous stimulus regions. We propose that surface information, provided by the konio-cellular pathway, interacts with the processing of oriented contrast in the primary visual cortex, and supports the fast formation of an initial stimulus hypothesis.

1.2 Latency coding in the visual system

There is convincing experimental (Nelken et al., 2005; Johansson and Birznieks, 2004; Reinagel and Reid, 2000; Golisch and Meister, 2008) and theoretical (van Rullen et al., 2005; Delorme, 2003; Reinagel and Reid, 2000; van Rullen and Thorpe, 2001; Masquelier and Thorpe, 2007; Kupper et al., 2005) evidence that the spike latency of neurons encodes their stimulation strength. Experimental evidence for spike latency coding in the visual system of primates can already be found in the classical work of Hubel and Wiesel (1968). Figure 1 shows recordings from orientation-selective cells in monkey primary visual cortex. It shows spike trains and mean firing rates for varying orientations of a bar stimulus. The panel on the right is based on an original figure from Hubel and Wiesel (1968). We have added indications of firing latency, i.e., the time
from stimulus onset to the first action potential recorded. Latency of the first response spike is large when firing rate is small, and vice versa. Both carry similar information, but the latency code can readily be assessed with the first spike of a neuron, usually after a few milliseconds, while read-out of rates requires integrating many spikes over time or space.

Recently we have shown that local luminance of a visual stimulus can be reliably mapped to the spike latency of a neuron (Kupper et al., 2005), and how this latency code can be maintained by the early visual system. We described neural mechanisms for putting ensembles of spiking neurons into consistent internal states, establishing a common temporal origin for latency coding. The brain can then use this fast and accurate time-based code to detect a variety of stimulus properties.

1.3 Homogeneity-detection based on spike coincidence

The reference to a common temporal origin enables postsynaptic neurons to detect homogeneous stimulation by coincidence detection, since homogeneous stimulation in the presynaptic neurons causes their spikes to coincide on a postsynaptic neuron (fig. 2, right). Typically, feature selective neurons that project to a single postsynaptic neuron have their receptive fields located in a small local region of the input space (e.g., a small local region in the field of view, fig. 2, left). A postsynaptic neuron detects spike coincidences and will indicate homogeneous activation across the afferent neural population – which then becomes a statement on homogeneous distribution of the respective feature in this region of the input space.

Figure 2 shows an example for luminance-encoding neurons, but in general, this principle applies to any topologically arranged set of feature-coding neurons, such as retinotopically arranged orientation-selective cells in V1, or retinotopically arranged directionally sensitive neurons in MT. Even features from other sensory modalities can be processed. The only prerequisite is
Figure 2: The principle of spike-latency based homogeneity processing; example from the visual domain. **Left:** Edge regions in the stimulus typically have high variance of luminance, while surface regions typically have low variance of luminance. **Right:** A set of visual neurons respond at latencies depending on the luminance inside their receptive field. The receiving neuron responds when the incoming action potentials coincide. This makes it selective for homogeneous luminance inside its receptive field. In the visual domain, this allows identifying surface regions.

that the respective feature can be latency-encoded. We will now formulate this idea as a general neural principle.

### 1.4 Design of a homogeneity detecting circuit

Starting from a set of neurons selective for some arbitrary feature $f$, it is straightforward to create a set of cells selective for the **homogeneous appearance of** $f$. We require only two well-known architectural ingredients that can be found virtually everywhere in cortex: Topologically arranged sets of feature selective neurons, and topology-preserving convergent projections.

The design pattern for the neural detection of topological feature homogeneity is as follows (cf. fig. 3):

1. A sending neural population $N_s$ is a set of latency-coding neurons selective for feature $f$. That is, firing of a neuron in $N_s$ corresponds to the appearance of feature $f$, and latency corresponds to some gradual quality $q$ of $f$, usually its strength. However, $q$ may correspond to any feature quality that can be mapped to latency, such as size or orientation.

2. The neural population $N_s$ is arranged across the cortical surface preserving the topology of a stimulation space $T$, e.g. retinotopically, tonotopically, somatotopically, etc.

3. Neurons in a receiving population $N_r$ receive convergent input from local sub-populations of $N_s$ with a fixed diameter $d$ measured in $T$, and with a fixed transmission delay.

4. The convergent projections preserve topology. This makes $N_r$ topologically arranged according to $T$.

Then, activity of a neuron in population $N_r$ indicates the appearance of feature $f$ with homogeneous quality $q$ across a local region of diameter $d$ in the stimulation space $T$, and at the
Figure 3: Detecting feature homogeneity from spike latency in a topological arrangement of feature selective neurons. Homogeneity is detected in two network stages. $N_s$ is a topologically arranged set of neurons selective for feature $f$. They generate a spike-latency code for the quality of feature $f$ at the respective location in the input space $T$. A single $N_r$ neuron receives action potentials from a local patch of $N_s$ neurons. Its sensitivity for coincident synaptic events makes this neuron selective for homogeneous appearance of feature $f$ inside its receptive field. All $N_r$ neurons receive action potentials from topologically corresponding local patches of $N_s$. This makes $N_r$ a topologically arranged set of neurons selective for homogeneous appearance of feature $f$. In our generalized model (sec. 3), two noise pools $np1$ and $np2$ provide random spikes mimicking crosstalk from 20 000 unrelated neurons.

location corresponding to the respective neuron’s topological position. Note that the quality $q$ of feature $f$ is still encoded in the firing latency of this neuron.

This is a general design principle, independent of the class of stimuli or features. Visual, auditory and somatosensory features come to mind, like

- detection of homogeneous movement,
- detection of harmonics in an auditory signal,
- visual or tactile detection of textures.

This suggests that the same design principle may be used at various cortical locations of different sensory modalities.

1.5 Simulations

We apply this general design principle in two network models. In a model of V1, we demonstrate how visual homogeneity-selective cells can identify surfaces in natural gray-scale images. We
show that surface-detection can support stimulus processing in primate visual cortex. The model consists of two retinotopically arranged sets of spiking neurons (fig. 3). The sending network stage $N_s$ is a retinotopic set of latency-coding feature selective neurons. Here, we use a model of LGN-konio-cells that encode local luminance. The receiving network stage $N_r$ applies the principle of spike-latency based homogeneity-detection to the output of these neurons. This makes the $N_r$ neurons selective for image regions of homogeneous luminance (surfaces). Since regions of homogeneous luminance and contrast edges exclude each other, the output of surface-selective neurons can be used to suppress unsought responses in orientation-selective cells, which do not define borders between mid-sized visual objects (Gewaltig et al., 2003).

In a second model, we focus on coding properties of our latency-based principle for homogeneity-detection. We examine the robustness to neural cross-talk at levels that are to be expected in the brain. We show that the latency code can be read out flexibly: The same mechanism that improves robustness to noise (forward inhibition) can also dynamically change the threshold at which homogeneity is detected.

2 A model of cooperative feature detection in V1

Our first model is an example of one specific application of the design pattern described in sec. 1.4. We model LGN and V1 responses for the konio-cellular and the parvo- or magno-cellular pathway. The schematic model setup is shown in fig. 4. It consists of four retinotopic network stages:

1. a retinal input layer,
2. a layer of konio-cellular cells in the LGN ($N_s$, ON and OFF),
3. a layer of konio-driven surface-selective neurons in V1 ($N_r$, ON and OFF),
4. a layer of parvo- or magno-cellular orientation-selective cells in V1 (four orientations).

We apply natural gray-scale images to the model retina.
2.1 LGN-konio-cells ($N_s$)

We model the response of LGN-konio-cells in a two-step process:

1. To imitate the neural transfer properties of konio-cellular neurons, we convolve the input image $I(x,y)$ with a Gaussian kernel $G(x,y)$ (mimicking the effect of large receptive fields), and pass the result through a sigmoidal activation function $\Theta$ (Gewaltig et al., 2003):

$$A := \Theta(I * G),$$

where

$$G : (x,y) \mapsto \frac{1}{\sigma \sqrt{2\pi}} \exp\left(-\frac{x^2 + y^2}{4\sigma^2}\right)$$

and

$$\Theta : z \mapsto \frac{1}{1 + \exp(-2b(z - \theta))},$$

with $\sigma$ the spatial standard deviation of the Gaussian, and $\theta, b$ threshold and slope of the sigmoid.

2. The activation values $A(x,y)$ are then transformed into spike trains by an integrate-and-fire model.

We use a current based leaky integrate-and-fire neuron model (Lapicque, 1907; Tuckwell, 1988) with post-synaptic currents shaped like $\alpha$-functions. Its membrane dynamics is given by

$$\tau_m \frac{dV}{dt} = E_l - V(t) + R_m I(t),$$

where $\tau_m$ is the membrane time constant, $R_m$ the membrane resistance, and $E_l$ the leakage (=resting) potential. An action potential is produced when the membrane potential $V$ crosses the firing threshold $V_{th}$, after which it is reset to the resting potential $E_l$ and clamped at this value for an absolute refractory period of 2 ms.

We transform the activation values of the LGN-konio-cells into neural spike responses by injecting currents of corresponding magnitude into a retinotopically arranged layer of these model neurons. At each retinotopic position, an ON- and an OFF-cell are located. The interval of activation values $[0,a_{max}]$ is linearly mapped to an interval of injection currents $[I_0,I_1]$: $[0,a_{max}] \mapsto [I_0,I_1]$,

$$a \mapsto I_0 + \left(I_1 - I_0\right) \frac{a}{a_{max}}$$

with

$$[I_0,I_1] = \begin{cases} [400,750] \text{ pA for ON-cells} \\ [750,400] \text{ pA for OFF-cells} \end{cases}$$

In the leaky integrate-and-fire neuron model, for a constant injected current $I$, the time of the first spike after current onset is (Dayan and Abbott, 2001b):

$$t_{cross} = -\tau_m \ln\left(\frac{R_m I + E_l - V_{th}}{R_m I + E_l - V_{start}}\right),$$
Figure 5: Current/latency relation (9) for injection of a constant current $I$ into a leaky integrate-and-fire model neuron ($\tau_m=10$ ms, $E_l=-70$ mV, $V_{th}=-55$ mV, $R_m=40$ M$\Omega$). $t_{\text{cross}}$, time of the first spike after stimulation onset. Gray scales on abscissa, mapping of pixel luminance to injection current; the range black–white maps to currents of 400–750 pA; luminance-to-current mapping is sigmoidal (transfer function of LGN-konio-cells), but symmetric. Dotted lines, current-to-latency mapping is asymmetric: a current range of 50 pA maps to decreasing latency ranges; this motivates the use of ON- and OFF-channels (sec. 2.6.1).

provided that the current is strong enough to drive the neuron above threshold. $V_{\text{start}}$ is the membrane potential at which the neuron starts integrating the stimulus current. We will later consider its effect on the latency code of neurons. Here, we assume the neurons are relaxed to their resting potential at the time of stimulation onset ($V_{\text{start}} = E_l$). Thus, relation (8) becomes

$$t_{\text{cross}} = -\tau_m \ln \left( 1 - \frac{V_{\text{th}} - E_l}{R_m I} \right). \quad (9)$$

Figure 5 shows this relation for the used parameter set.

As a result of this process, the simulated LGN-konio-cells form a retinotopically arranged set of latency-coding feature selective neurons. The feature $f$ encoded in the firing latency of these neurons is the mean luminance inside their large receptive fields. We will now use this layer of LGN-konio-cells as the sending population $N_s$ in the aforementioned design pattern (fig. 3).

2.2 V1 surface-selective neurons ($N_r$)

The konio-driven V1 neurons of our model correspond to neurons in the cytochrome-oxidase-blobs of the superficial layers 2/3 in the primary visual cortex. These cells receive direct input from the konio-layers of the LGN. We model the konio-driven V1 neurons as a retinotopic layer of leaky integrate-and-fire model neurons. They play the role of the receiving population $N_r$ in the aforementioned design pattern. Konio-driven V1 neurons receive spike input from locally restricted populations of luminance-selective LGN-konio-cells, according to fig. 3.

We use the same neuron model as for the LGN cells. Incoming action potentials cause $\alpha$-shaped excitatory postsynaptic currents in the V1 neurons. If enough EPSCs coincide, the receiving neuron’s membrane potential crosses threshold, and the neuron produces an action potential. Given that the LGN-neurons encode the luminance in their receptive fields, this property
makes the konio-driven V1 neurons selective for image regions of homogeneous luminance. Regions of homogeneous luminance can typically be attributed to the surfaces of mid- to large-sized objects. The konio-driven V1 neurons can thus be described as surface-selective (Gewaltig et al., 2003).

### 2.3 V1 orientation-selective neurons

We model the response of parvo- or magno-cellular orientation-selective cells in V1 in a two-step process similar to the one used for LGN-konio-cells:

1. We compute activation values for four classes of orientation-selective neurons by convolving the input image with Gabor-filters of four orientations, spaced at 45 degree intervals.
2. The activation values are then transformed into spike trains by current-injection into a leaky integrate-and-fire model, as described before.

At each position of the retinotopic layer, four orientation-selective neurons are located (preferring contrast orientations of 0, 45, 90, 135 degrees visual angle).

### 2.4 Cooperative features in V1

In the initial phase of visual scene interpretation, the information provided by the surface-selective neurons may be very helpful in a quick segmentation of the visual input. To obtain a good hypothesis on the main contents of the scene “at a first glance”, the rich information in the stimulus must be reduced to its most salient parts.

The different input pathways to the CO blobs of V1 use different strategies to contribute to processing of visual information (Ding and Casagrande, 1998). Solomon et al. (1999) report that K-cells in marmosets have temporal properties intermediate between those of parvo- and magno-cells. Electric field potentials evoked in human V1 by S-cone isolating stimuli appear earlier (latencies of about 40 ms) than the common luminance-defined motion-specific potentials (Morand et al., 2000). This indicates that konio-cellular responses in V1 are present at least as early as the responses of orientation selective neurons. Since edges and surfaces of physical objects exclude each other, the output of surface-selective konio-neurons can be used to suppress those responses in orientation-selective cells that are unsought in this initial phase of scene analysis (Gewaltig et al., 2003), i.e., those orientation responses which do not define borders between mid-sized visual objects.

We model this process by inhibitory coupling from surface-selective konio-cells to the retinotopically corresponding orientation-selective parvo- or magno-cells in V1. Activity in a surface-selective neuron suppresses responses of all orientation-selective cells that have their receptive field located inside the receptive field of the surface-selective neuron.

### 2.5 Simulator

We use the NEST simulator developed in collaboration with the Neural Simulation Technology Initiative (Diesmann and Gewaltig, 2003) for simulating the structured neural network. The simulation has a temporal resolution of 0.1 ms. All neuronal stimulation enters through current injection into the model neurons, according to the procedures described above. The neurons do not show spontaneous activity without stimulation.
2.6 Results

We applied natural gray-scale images to our model retina and recorded the spike responses of all model neurons. From these data we derived topologically arranged response maps for the different layers of model neurons. In these retinotopic maps, bright pixels indicate neurons that produced at least one action potential in response to the stimulus.

Figures 6c–d show response maps for the input image in fig. 6a. Surface-selective koniocellular neurons inhibit orientation-selective cells in homogeneous image regions, confining their responses to regions in between potential objects.

2.6.1 ON- and OFF-channels

The LGN-konio-cells of our model map a range of luminance values inside their receptive field to a range of firing latencies. However, the variance of produced latencies does not only depend on the variance of luminances in the receptive field. It depends on their absolute values, too. The relationship (9) between stimulation current and spike latency is not linear, but decays logarithmically (fig. 5). Current intervals of fixed widths yield latency intervals of different widths, depending on the absolute values of currents, as illustrated in fig. 5. As a consequence, a luminance change of a certain magnitude causes a bigger change in spike latency when it appears inside a dark image region, than in a bright image region. This means, the LGN-konio-cells \(N_s\) in our model are more sensitive to luminance changes in dark regions than in bright regions. As a result, dark regions need to be more homogeneous to trigger responses in surface-selective cells \(N_r\). Accordingly, we experienced that with homogeneity thresholds that yield good results for bright image regions, surface-selective neurons would seldom respond to dark image regions at all.

We accounted for this by using two classes of luminance-selective LGN-konio-cells, ON- and OFF-cells, with inverse characteristics for luminance (7). They project separately to two corresponding classes of surface-selective V1 neurons: ON-cells, whose firing threshold was set to yield good results in bright image regions, and OFF-cells, which have the same firing threshold, and consequently yield good results for dark image regions. Results from ON- and OFF-neurons are complementary. Figure 7 shows separate response maps for surface-selective ON- and OFF-neurons, and the superposition of the two.
2.6.2 Low-pass filtering by LGN neurons

Spatial low-pass filtering by large LGN receptive fields shifts the focus of surface-detection toward mid- or large-sized structures in the visual field. This is in accordance with the assumed purpose of surface-processing, i.e., to direct visual processing to large and salient objects in the initial phase of scene processing, until a first hypothesis on scene content has been established. Figure 8 shows responses of surface-selective neurons that were obtained with and without spatial low-pass filtering in the LGN-konio-cell model.

3 Generalized model and noise resistance

In our model of V1, we showed how the natural property of neurons, to encode stimulation strength in firing latency, gives rise to an efficient detecting algorithm for homogeneous image regions. We have proposed, that the konio-cellular pathway of the visual system extracts this image property, and that it is used to restrict orientation-selective responses in V1. Parallel extraction of mutually exclusive features in two pathways supports the fast formation of a stimulus hypothesis.

It is worth noting that the mechanism we use for homogeneity detection is not bound to the substrate of V1. It merely detects temporal coherence in a packet of spikes. These spikes can originate from visual feature detectors like in our model, but they can just the same originate from neurons of other modalities. In that sense, latency-based homogeneity-detection is a generic mechanism. It can operate at various cortical sites, and detect homogeneous appearance of whatever features they process. It is not important what the features are, only that they translate into a latency code, where identical (or similar) features map to identical (or similar) latencies.

In addition the process requires that the coherence of the spike package must not been lost during transmission, and that the spike packet elicits a reliable response in the receiving neuron. The latter is not a trivial assumption, given likely levels of cross talk in the brain. Among a neuron’s ten thousand synapses (Braitenberg and Schüz, 1991) the ones transmitting the “signal” (spikes from a local set of feature detectors) may make one percent or less. The majority of post synaptic potentials will be caused by spike trains that are unrelated to the “signal” in question. Our model of V1 can serve as a proof-of-concept for the mechanism of homogeneity detection. It did however not account for the background activity from thousands of functionally unrelated neurons that is to be expected in the real brain. It is common practice to regard and model this unrelated background activity as a noise component in the input signals to neural detectors. (In the way we use the word “noise”, it refers to input components such as cross-talk from other...
neural systems that are statistically unrelated to the “signal” in question. It does not imply that these inputs are useless noise that could be removed."

In the following, we will apply this technique to test the noise-resistance of our mechanism for homogeneity detection. Specifically, we investigate whether operation degrades slowly (gracefully) with increasing noise.

We use a generalized network architecture similar to the one of our V1 model. However, we apply a simpler stimulation scheme. Since we are interested in the fundamental properties of homogeneity detection, independent from the specific features, we drop the parallel OFF-channel, because it is functionally identical to the ON-channel, and we drop the preprocessing stage (low-pass filtering and contrast enhancement), which was used to mimic the konio-cellular properties of the primate LGN.

3.1 Noise pools

To account for the neural crosstalk that is to be expected in the brain, we add two sources of massive spike noise:

**Poissonian noise pool np1** provides each connected neuron with independent spike trains. Each spike train mimics the background activity of 16,000 independent excitatory neurons firing at a mean rate of 2 Hz each. A single spike elicits a peak excitatory current of 15 pA in the receiving neuron.

**Poissonian noise pool np2** does the same, but for each connection mimics the spikes of 4,000 independently firing inhibitory neurons at a mean rate of 0.787 Hz each. A single spike elicits a peak inhibitory current of 150 pA in the receiving neuron.

The rates of the two noise pools have been calibrated using a “balanced neuron” paradigm. In this paradigm, a single model neuron is connected to both the excitatory and inhibitory noise...
pool. The rates and synaptic weights of the two pools are then chosen such that the model neuron fires as if it were a part of the excitatory pool. The target rate of the model neuron (and thus the excitatory pool), as well as the synaptic weights are free parameters in this setup. We chose a mean rate of 2 Hz resting activity, and peak synaptic currents of 15 pA and 150 pA respectively. These values are well in the physiological range (see e.g. Thomson and Lamy, 2007).

We connect the two noise pools to every neuron of the \(N_r\) population of our model (fig. 3). This means that these neurons will be subject to background stimulation from 20000 virtual neurons each, maintaining them at a spontaneous rate of 2 Hz in the absence of stimuli.

### 3.2 Stimuli

For stimuli we extract patches of 100\(\times\)100 pixels from natural gray-scale images. They represent sections of 10\(\times\)10 degrees in the visual field. We transform pixel luminances into neural spike responses by injecting currents into a layer of 100\(\times\)100 integrate-and-fire neurons (\(N_s\)). Here, pixel luminances are linearly mapped to injection currents without preprocessing:

\[
\text{[black, white]} \mapsto [I_0, I_1]
\]

\[
l \mapsto I_0 + l(I_1 - I_0)
\]

with

\[
[I_0, I_1] = [376, 800] \text{ pA.}
\]

The spatial mapping of image pixels to stimulated neurons is retinotopic.

### 3.3 Neural reset

The stimulation procedure maps the pixel luminances of the stimulus patches to firing latencies of the corresponding \(N_s\) neurons. Bright pixels map to large currents (small response latencies).

In the leaky integrate-and-fire neuron model, for a constant injected current \(I\), the time of the first spike after stimulus onset was described by (8). The crossing time \(t_{\text{cross}}\), i.e., the neuron’s spike latency, depends on the membrane potential \(V_{\text{start}}\) at which the neuron starts integrating the stimulus current. This starting state will naturally vary across a neural population, depending on the individual neurons’ stimulation history. Figure 9 shows the current/latency relation for different starting values \(V_{\text{start}}\). Small variations in the starting membrane potential cause strong variations in firing latency, especially in the physiologically relevant excitation regime below 600 pA (corresponding to 100 Hz of tonic firing, i.e., a latency of 10 ms starting from resting potential, see thick curve). As a consequence, to generate a coherent latency code across the \(N_s\) population, the neurons need to be reset to identical starting membrane potentials.

We initiate latency coding by resetting the neurons’ membrane potential to resting potential each 100 ms, a period roughly corresponding to a short saccade-interval. Stimulation is constantly applied. This procedure makes the \(N_s\) population a retinotopically arranged set of latency-coding neurons selective for local luminance (luminance, in this example, is the feature \(f\)). The neurons re-generate the latency-code after each reset (every 100 ms).

### 3.4 Homogeneity-selective cells

Sub-populations of diameter \(d = 11\) neurons of \(N_s\) (representing \(d = 1.1\) degree visual angle) project convergently onto a layer of 100\(\times\)100 model neurons (\(N_r\)), while preserving topology (fig. 3). Transmission delays and synaptic weights are identical for all connections. According to our design principle, this makes the \(N_r\) population a retinotopically arranged set of neurons selective for spatially homogeneous luminance across distances of \(d = 1.1\) degree visual angle.
Figure 9: Current/latency relation (8) for different starting membrane potentials $V_{\text{start}}$. **Thick curve**, starting from resting state ($V_{\text{start}} = E_l$, corresponds to fig. 5); **thin curves**, starting from depolarized states ($V_{\text{start}} < E_l$); **grey curves**, starting from hyperpolarized states ($V_{\text{start}} < E_l$).

### 3.5 Results

We recorded spike responses of all neurons over the complete time-course of simulation. From this data, we retrieved topological latency maps of $N_s$ and $N_r$ neurons, as well as topological maps of response probability. Over the course of the whole simulation, the $N_r$ neurons received synaptic input from the two noise pools that imitate neural background activity. This massive rain of spikes from the excitatory and inhibitory pool drove the $N_r$ neurons close to firing threshold and caused them to fire at a spontaneous rate of 2 Hz in the absence of a visual stimulus, due to fluctuations in the membrane potential. We then applied a static image for stimulation of the $N_s$ neurons and performed multiple resets of the $N_s$ membrane potentials, so that latency codes for the applied stimulus are repeatedly generated. After each reset, the response latency of a neuron was defined as the time span between the reset and the neuron’s first spike thereafter.

In order to test the noise resistance of our model, we conducted all experiments with varying strengths of input from the background noise pools, 0%, 50%, and 100%.

#### 3.5.1 Single trial response latencies

Figures 10 and 11 show typical spike-trains recorded from $N_s$ and $N_r$ neurons. $N_s$ neurons were excited according to the visual stimulus shown in panel 10A. At $t = 0$, latency coding was initiated by resetting all neurons to consistent membrane potentials. Regions of homogeneous luminance in the stimulus patch cause groups of neighboring $N_r$ neurons to respond with similar latencies. Their action potentials show up as “spike-fronts” in the plots. These coincident spikes, in turn, cause spike responses in the $N_r$ neurons in corresponding locations. The **right column** of figure 11, shows the response latencies on a gray-level scale. (Blue color indicates that the respective neuron did not produce an action potential.)

Panels 11A, B and C depict response latencies obtained with different strengths of the noise pools imitating background activity. Panel A shows the obtained spike latencies in a “silent” network that is completely undisturbed from neural crosstalk. The locations of active $N_r$ neurons correspond to regions of homogeneous luminance in the stimulus patch — the $N_r$ neurons act as **homogeneity-selective neurons**. Note that by relying on the first action potentials of feature
Figure 10: A, stimulus patch; small circle, receptive field of an $N_s$ neuron; big circle, receptive field of an $N_r$ neuron. B, typical single-trial spike trains of 100 $N_s$ neurons with receptive field positions along the marked line in A; gray scale on latency axis, corresponding pixel luminance by (10–12).

detectors, homogeneity processing is very fast, with first components signaled already after 10 ms. Panels B and C show the same results for 50% and 100% noise levels, respectively. It is obvious that the background noise disturbs homogeneity detection, and triggers responses in neurons that were previously silent (blue area).

3.5.2 Ensemble responses

In order to generate a behavioral response from a stimulus, the neural measurement involved must be made in a single trial. In contrast to simulation or laboratory conditions, a living being cannot to repeat the same measurement one hundred times and compute the mean result. The measurement must be good, or the individual might be dead. In our case this means that the classification of homogeneous vs. non-homogeneous must be successfully made in a single trial. There are generally two possibilities to do this:

1. Make the single detectors work reliably. We have seen in the last section, that this is not the case in presence of neural cross talk. In particular, the noise caused single detectors to wrongly fire in non-homogeneous regions.

2. Duplicate the individual detectors several times, and compute the ensemble mean. In contrast to time averages, this can be computed instantly, so that behavioral responses can be based on the measurement.

Figure 12 shows the mean response latency, and the spike probability in ensembles of 100 neurons located at each position in the visual field. Spike probability is defined as the fraction of $N_r$ neurons in each ensemble that produced a spike in response to the stimulus patch. The probability equals 1, if all neurons of the ensemble produced a spike in response to the stimulus, and 0, if no neuron in the ensemble fired. If homogeneity-detection was robust against neural cross talk, spike probabilities should turn into a binary relation (values 0.0 and 1.0 only): An action potential should either be produced by all or by none of the neurons that encode a particular spot in the visual field. Panel 12A shows that in a “quiet” network, this is the case: Spike probability has a strictly bimodal distribution, with contributions only at 0.0 and 1.0 (right column).
Figure 11: Typical single-trial homogeneity responses. Left, spike trains of 100 $N_r$ neurons with receptive field positions along the marked line in fig. 10; right, latency map of all $N_r$ neurons. A–C, with increasing spike-noise from the background population, spike-times scatter. Moreover, noise triggers spikes in neurons which previously not responded (marked regions). D, additional forward inhibition counteracts the effect of background noise.
Figure 12: Mean $N_r$ spike latency, spike probability, and histogram of spike probabilities in ensembles of 100 neurons at each position in the view field. $A$–$C$, with increasing cross talk from background neurons, separation between homogeneous and non-homogeneous image regions breaks down; a level of 100% corresponds to the amount of crosstalk expected in the brain. $D$, additional forward inhibition reestablishes good separation.
Panels 12B and C in contrast show that spike responses of the $N_r$ neurons are sensitive to neural cross talk. Here, the strength of the background populations was set to 50% and 100% of the predefined strength respectively. This massive background activity constantly drives the $N_r$ neurons close to threshold, and frequent fluctuations in the membrane potential cause the neurons to falsely respond to non-homogeneous stimuli. However, panels 12B and C show that the quality of homogeneity processing degrades gracefully with increasing noise. In non-homogeneous regions (regions that “should be quiet”, see panel A), spike probabilities are very low for the 50% noise case, while homogeneous regions have a generally high spike probability. Although the spike probability histogram is not strictly bimodal, a good separation of homogeneous from non-homogeneous regions can be made by setting a threshold at 0.5 in ensemble activity (half of the neurons active, see histogram in panel B).

At the full level of crosstalk however (panel 12C), all regions have a spike probability of more than 0.4, and the probability distribution is close to unimodal (see histogram in panel C). This must be seen as a definite failure of classification, since the ensemble responses in homogeneous and non-homogeneous regions get indistinguishable. We must conclude, that homogeneity detection in the proposed way is not feasible in a network with realistic background noise.

4 Shaping of post-synaptic potentials by forward inhibition

The breakdown of homogeneity processing in presence of realistic background noise that we have observed in sec. 3 seems to render this method unfeasible for other than controlled laboratory conditions. Certainly, it cannot be of any use for the real brain, which has a high degree of neural crosstalk especially in the lower sensory areas, where this method might have its most obvious applications. Do we have to conclude, then, that this method is but an interesting thought experiment with no practical relevance? In this section we will show that this is not the case. Indeed, with little effort the mechanism can be altered in a way that makes it conceivably more robust against noise.

4.1 Uni- vs. biphasic post synaptic potentials

Figure 13 shows an $\alpha$-function (black line). This function is a good approximation to the shape of the typical post synaptic current that is caused by a single action potential at the synapse. We use this approximation for our integrate-and-fire neuron model (Lapicque, 1907; Tuckwell, 1988). The synaptic current $I(t)$ has a relatively short rise time of 2 ms followed by a much longer “excitatory tail” that keeps having a positive (hyperpolarizing) contribution to the membrane potential for at least 10 ms. The overall charge delivered to the neuron is positive.

Since the hyperpolarization caused by a single spike is small, a lot of spikes must coincide at one neuron in a time window that is roughly this 12 ms. Only then will their excitatory currents add up and deliver enough charge to raise the membrane potential above firing threshold. It is obvious that controlling the width of this time window is a way of altering the neuron’s sensitivity to spike noise, since stochastic spikes are less likely to add up when the time window for integration is short. However, controlling the synaptic time constant $\tau$ through biologically plausible network effects is difficult. In most cases, the speed of synaptic dynamics is fixed, or depends upon chemical processes which cannot be directly influenced for the means of neural computation.

In the complex network of the brain, the synapses of sensory neurons undergo massive bombardment during stimulation, and in this operating regime synaptically induced conductance
Inhibitory PSC delayed by **1ms**

Inhibitory PSC delayed by **2ms**

Inhibitory PSC delayed by **4ms**

Figure 13: Shaping an excitatory PSC by delayed feed-forward inhibition. The excitatory PSC is followed by an inhibitory PSC of the same shape and magnitude. The plots show superpositions for different delays of the inhibitory PSC ($\Delta t = 1$, 2, or 4 ms). **Black line**, excitatory PSC with time constant $\tau = 2$ ms; **grey line**, inhibitory PSC with $\tau = 2$ ms and delay $\Delta t$; **thin line**, effective PSC resulting from superposition. If spikes converge on a homogeneity-selective neuron of this type, the positive parts of their resulting PSCs need to overlap in order to effectively hyperpolarize the neuron to the firing threshold. Note how the positive part of the effective PSC changes width with $\Delta t$: Shorter delays require spikes to arrive closer in time, i.e., shorter delays require increased homogeneity for the neuron to respond.

Changes indeed affect the membrane time constants (see Kuhn et al., 2004, for a model). Purposefully controlling synaptic integration via this mechanism may however be difficult. But there is a means of shaping the effective PSC of a single synaptic event: This is achieved by pairing an excitatory action potential with an inhibitory action potential transmitted shortly afterwards to the same neuron (forward inhibition). Figure 13 shows the effect of an inhibitory PSC delivered shortly after an excitatory PSC of the same magnitude and time constant. The currents superimpose, and the resulting effective PSC is much narrower than the original curve. The net charge delivered by the paired pulse is zero, which prevents the postsynaptic potential from accumulating over a longer time. Both effects raise the temporal constraint for constructive superposition of subsequent synaptic events. Spikes have to follow each other more closely to raise the neuron above firing threshold.

In the brain, the delay at which inhibitory action potentials are produced can be influenced through network effects. Diffuse background stimulation can change the effective firing threshold of neurons (Chance et al., 2002). The neurophysiological effects of selective attention can promote neural processing (Müller and Kleinschmidt, 2004; Hochstein and Ahissar, 2002). It is hence conceivable that the criterion for homogeneity-detection, the “homogeneity threshold”, may be dynamically changed according to the momentary cognitive requirements or interests.

### 4.2 Homogeneity threshold

We include PSC shaping into our network for the processing of spatial homogeneous luminance. We duplicate the connections from the sending $N_s$ layer to the receiving $N_r$ layer. Action potentials transmitted along the duplicated connections cause inhibitory currents in the postsynaptic $N_r$ neurons (mediated by inhibitory interneurons, which we did not model explicitly). Inhibitory spikes have a fixed time delay relative to the excitatory spike. We now systematically vary the time delay between excitatory and inhibitory action potentials in otherwise identical simulation runs.

Figure 14 shows spike probabilities of the homogeneity processing $N_r$ neurons under these conditions. Inhibitory delays increase from upper left to lower right in reading order. The area reliably classified as homogeneous (high probability of an action potential) changes monotonically...
with the inhibition delay. For short delays, fewer regions of the stimulus patch are classified as homogeneous, due to the much sharper effective PSCs caused by these delays. The delay of the paired feed-forward inhibition effectively determines the threshold, at which a local region is classified as homogeneous by the \( N_r \) neurons.

### 4.3 Response separation

Chopping off the excitatory part of the postsynaptic current after a few milliseconds not only shifts the homogeneity threshold, it can even enable sensible homogeneity detection, where this would not otherwise have been possible. Going back to fig. 12 panel C on page 17, we remember that a realistic level of cross-talk rendered our mechanism for homogeneity detection useless, since the high amount of unrelated spike noise caused the detectors to spike with a more than 50% probability, regardless of the image content.

In comparison, panel D of fig. 12 shows the same setting applying shaped PSCs. Actually, this is the same result as shown in the upper right panel of figure 14 (8 ms inhibitory delay). Obviously, the biphasic synaptic events help to better separate positive from negative detector responses: The spike probability histogram in fig. 12 panel D is clearly bi-modal, meaning that detectors tend to either respond with a high or a low probability to the stimulus. Thus, in a local ensemble of neurons, good separation of homogeneous from inhomogeneous image regions can be achieved by setting a threshold, e.g. at 50% of firing cells, as shown in panel D. The separation of homogeneous from inhomogeneous image parts is then comparable to the only moderately disturbed case in panel B.

Previously, the long excitatory tails of the PSCs added up, even if spikes were wide apart, raising the mean membrane potential of the receiving neuron closer to the threshold. Since the distance to threshold is very small in the presence of heavy spike noise, this would increase the neuron’s sensitivity to fluctuations, eventually causing the neuron to fire, even for a very dispersed input spike packet. In contrast, the biphasic PSCs have a short excitatory part, followed by a long inhibitory tail. For a dispersed input spike packet, the inhibitory tails add up – bringing the mean membrane potential even further from the threshold, which lowers the probability of a false spike. For narrow input spike packets, biphasic PSCs overlap to a short and intense excitatory part, followed by a considerable inhibitory tail, i.e., an additional relative refractory period of the neuron. Both effects increase the separation of narrow from dispersed spike packets. We observe
this as a separable bi-modal firing probability distribution, well suited for basing a decision.

5 Discussion

We have presented a simple neural mechanism for the detection of homogeneous stimuli. In a set of neural feature detectors (visual or other modality), it naturally extracts local populations that are equally stimulated. The mechanism exploits that synaptic currents caused by action potentials are short (temporally restricted) events. Many of those events need to superimpose to make the post-synaptic neuron fire. Since spike latency is a monotonous function of stimulation strength, equally stimulated pre-synaptic neurons fire coincident spike waves, which are easily detected in a receiving neuron. (Obviously, the pre-synaptic neurons need to have coherent temporal reference frames, which can be supplied through coincident stimulus onset, e.g. saccades or other adequate methods, Kupper et al., 2005)

The usefulness of this concept for the brain is evident, since homogeneously stimulated regions in the sensor space will often constitute relevant stimuli, like uniformly colored objects, uniform texture, uniform movement. The simple circuitry needed to implement it (local convergence) makes it a ubiquitous candidate for all sensory cortices.

5.1 Surface detection as a means for information reduction

We have implemented an example simulation from the visual domain (V1). In the initial phase of visual scene interpretation, information provided by homogeneity (here: surface) selective neurons can help generate a good starting hypothesis on scene contents “at a first glance”. In fact, besides features like color, orientation, etc (Itti and Koch, 2000), homogeneity might constitute an important component of a visual saliency map. Since edges and surfaces of physical objects exclude each other, the output of surface-selective neurons can be used to suppress responses in orientation-selective cells that are unsought in this initial phase of scene analysis (Gewaltig et al., 2003).

We demonstrated in our model of V1 that this function can be implemented in a simple feed-forward network of parallel feature-extraction (parvo-/magno- and konio-cellular pathway).

Parallel extraction of possibly redundant features (edges and surfaces) comes at increased metabolic costs. However, homogeneity-selective neurons have large receptive fields, since they integrate from a whole region of the input space. In order to sample the space, they need to be much less densely packed than the original detectors. Moreover, since the mechanisms for detecting edges and homogeneous regions differ, both signals are independent and can be combined to a signal that is more reliable than either one. Thus, the increased metabolic costs are justified by the improved reliability and processing speed.

If response times suggest only one spike per processing stage, as in the works of Thorpe et al. (1996), there is no time for lateral interaction. Forward processing in parallel pathways is then the only alternative, if different aspects of the stimulus shall be determined at all.

After a first hypothesis about the content of the visual scene has been established at higher processing levels, a top-down signal can then enable a refined analysis of object detail (see Körner et al., 1999b; Ullman, 2000b). This would simply involve inhibiting surface-selective neurons of the konio-cellular pathway, thereby dis-inhibiting the previously suppressed orientation-selective neurons in V1. Information on oriented contrast at fine detail will then be channeled into an already pre-adjusted system. This process seems to define a natural time-course of processing from coarse to fine, without unconditionally blurring the stimulus by low-pass-filtering in the way conventional models of resolution pyramids do. By contrast, orientation responses for presumed
object boundaries are relayed in full fidelity from the start, and can take part in the formation of a reliable hypothesis.

5.2  Detection of general topological feature homogeneity

The latency-coding property of visual neurons (fig. 1) makes them natural candidates for spike-based homogeneity processing. But detecting homogeneous luminance in a retinal image is not the only example for the processing of topological feature homogeneity. The type of homogeneity-features that can be processed is only limited by the features \( f \) that can be extracted from the stimulus and latency coded. This may happen at any phase in visual processing as well as other modalities like auditory or somatosensory.

In the visual domain, neurons processing topological homogeneity can detect homogeneous appearances of complex geometrical features like oriented contrast edges, lines, or directions of movement. Possible applications include the detection of surfaces that are not homogeneous in luminance, but of homogeneous texture, like table surface, carpet, sand, pebbles, treetops, water surface, etc. If \( f \) is a feature that is spatio-temporally defined, spatially homogeneous changes or spatially homogeneous motion can be detected. Temporally coherent change of extended regions in the stimulus can form the basis of newness-detection, like appearing or disappearing of objects in the visual or auditory scene. Homogeneity-selective neurons operating on directionally sensitive cells in the parietal pathway could detect coherently moving extended regions in the visual or auditory stimulus, such as moving objects or the effects of ego-motion.

5.3  Biphasic synaptic events

To be applicable in a dense and complex neural environment like the brain, a neural mechanism must be robust to noise and crosstalk from millions of neighboring neurons. We have observed that our mechanism was not robust enough to base a reliable decision upon. At a level of crosstalk that is to be expected in the brain, homogeneity responses were basically indistinguishable from the noise baseline (fig. 12 panel C, page 17).

To improve performance, we introduced forward inhibition which creates biphasic synaptic currents with short excitatory parts, and zero net current into the receiving neuron. Both effects decrease the sensitivity to crosstalk and allow for better separation of homogeneous stimuli. At a realistic level of crosstalk, homogeneity responses were substantially more probable than the baseline of false events (bimodal probability distribution in fig. 12 panel D). This means that reliable decisions can be based on the pooling of homogeneity responses, even in a noisy brain.

5.4  Number of converging neurons

Our basic circuit is a population of sending neurons which form a population-latency-code received by a single neuron. The receiving neuron acts as a homogeneity detector.

In general, the number of sending neurons participating in the population-latency-code will influence the processing of feature homogeneity. Typically, homogeneity-selective neurons will have a high firing threshold, requiring all or most of their afferent neurons to fire synchronously. Obviously, there exists a lower bound on the number of coincident action potentials that can at all raise the receiving neuron’s membrane potential above firing threshold. The receiving neuron will not be activated by a lower number of spikes, even if they coincide perfectly. A possible example are “grating cells”, found in the upper layers of monkey V1 and V2 (von der Heydt et al., 1992; Brunner et al., 1998). These cells respond vigorously to gratings of a preferred spatial frequency and orientation, but not to single bars or edges. Their response depends critically on
the number of cycles of the gratings. This suggests that grating cells could be homogeneity selective neurons, processing homogeneous activity in a local set of visual simple or complex cells.

The number of action potentials that can in principle coincide at a homogeneity-detector is bounded by the number of afferent neurons – the homogeneity-detector’s fan-in. A typical homogeneity-selective cell with a low fan-in operates as a logical AND gate. In this case, the single action potential has a large influence on the resulting membrane potential, so that typically all of them are required to cross the firing threshold. That means, the homogeneity-selective neuron will only become active, when all necessary stimulus features are present (and when all of them are present in equal quality $q$, the value mapped to latency by the sending neurons). The homogeneity threshold can be changed by PSC shaping to allow more or less jitter in latencies. But, typically, the AND characteristics will persist, since a missing spike will make a large relative change in the resulting membrane potential.

By contrast, in a typical homogeneity-selective cell with a high fan-in, the single action potential has only a small influence on the resulting membrane potential. The small change caused by a missing spike can be counteracted by an increased overlap of the remaining PSCs, allowing the homogeneity-selective neuron to be activated by a large variety of spike distributions. The cell will then collect global evidence for the presence of homogeneous features from its receptive field, without requiring the activation of a distinct set of local features. It can be used to find textures that appear globally homogeneous, but have locally varying structure.

5.5 Pulse-packet description

Homogeneity processing depends on a number of factors that are determined by neuronal and network properties. First, the neurons must be able to detect coincident spikes. Second, the network between the input and the coincidence-detecting neurons must not distort the variance of the input layer. In our model, the variance of a given feature is translated into a variance of spike latencies. For a neuron to function as homogeneity detector, its response must be determined by the variance of its input spike times, rather than their exact temporal configuration. Diesmann et al. (1996, 1999) presented the concept of pulse packets to describe spike volleys by two parameters: the number of spikes $n_{\text{spikes}}$ in the pulse packet and their temporal dispersion, measured by the standard deviation of the spike times $\sigma_t$. The authors report that the response probability of a cortical neuron to a pulse packet critically depends on the two parameters $n_{\text{spikes}}$ and $\sigma_t$. Thus, the degree to which a neuron detects coincidences or just integrates spikes depends on the temporal structure of the input.

For our purpose, we interpret coincidence detection as a threshold for input variance. The luminance distribution of an image patch is translated into a spike latency distribution with a standard deviation $\sigma_t$ that depends on the luminance variance in the receptive field. Generally, the response probability depends on both the number of spikes and their variance. But for a dedicated homogeneity processing network we can always find a mapping of stimulation to latency that assures that each sending $N_s$ neuron produces exactly one spike in the given observation interval such that $n_{\text{spikes}} = \text{const}$. The response probability then becomes a one-dimensional function of $\sigma_t$ with $n_{\text{spikes}} = \text{const}$ as parameter. This is illustrated in fig. 15. For small $\sigma_t$, the response probability is large and decreases monotonically with increasing $\sigma_t$. We can use the inflection point as a measure for the variance threshold. The slope and position of this point are determined by the rise-time of the post-synaptic potential, because larger rise-times allow the neuron to integrate incoming spikes over longer periods of time. The larger the rise-time of a PSC, the more the threshold will move to larger variances. Changing the PSC amplitude will have similar effects (not shown).
Figure 15: Response probability as function of the standard deviation $\sigma_t$ of the latency distribution for different rise-times $\tau$ of the post-synaptic potential and for a fixed number of input spikes. Here, $n_{\text{spikes}} = 100$. The response probability of a neuron decreases with increasing standard deviation of the spike-latency distribution of its input. The lower the rise-time of the post-synaptic potential, the lower the variance threshold. The curves are obtained from the waiting-time distributions of an inhomogeneous Poisson-process (Gewaltig, 2000).

5.6 Homogeneity-selective neurons as neural filters

The spike response of a homogeneity-selective neuron carries two components of information:

1. The fact that an action potential was produced at all indicates that the incoming spikes arrived close enough in time for their PSCs to superimpose constructively. This indicates that the feature homogeneity in the respective stimulus region exceeds a given threshold. This is a binary statement on topological feature homogeneity (yes/no).

2. The response latency of the homogeneity-selective neuron reflects the latency of the coincident input spikes. The response will have an additional, constant delay, given by the spike transmission time from the sending population $N_s$ to the receiving population $N_r$, and the neuron’s internal dynamics.

   This is a gradual analog value, describing the quality $q$ of the feature (the value that was mapped to firing latency by the sending $N_s$ neurons).

As a result, the homogeneity-processing neurons do not only detect regions of homogeneous feature activation in the stimulus. They are also transparent to the latency code of the afferent neurons, encoding how strongly activated this feature is. Hence, we can look upon the homogeneity-processing neurons as a kind of neural filters: They block spike responses from non-homogeneous regions, and they let the latency code from homogeneous regions pass.

In doing so, the single $N_r$ neuron transforms the incoming pulse packet to a single spike, reflecting the homogeneously encoded feature quality in its receptive field. The spike times of all spikes leaving the $N_r$ layer do thus preserve the relative spike times of $N_s$ neurons in the homogeneous stimulus regions. The $N_r$ layer is transparent in homogeneous regions in the sense that it preserves the relative spike times. It is, thus, a filter element that can be inserted into the spike stream of latency-coding neurons at any level of processing.
5.7 Spike-time and rate coding in a single network

The principle of PSC shaping opens up an attractive opportunity for dynamically controlling the mode of operation in a spike-coding network. Stimulus-related information can be differently conveyed in the train of action potentials produced by a neuron:

1. in the instantaneous rate of firing, and

2. in the latency of individual spikes, relative to a given temporal origin.

Processing of this information in post-synaptic neurons calls for two different mechanisms. For the extraction of latency, the exact timing and separability of single synaptic events is crucial, while the extraction of rate requires integrating large numbers of synaptic events over space or time. In general, PSC shaping, as a means of controlling the overlap of synaptic events in the post-synaptic neuron, holds the opportunity to select, which of the two components of information the neuron extracts. Its operation can be locally switched through a neural process (e.g., forward inhibition). It requires no change in network structure, and no change in processing of the pre-synaptic neurons.

Information on sensory stimuli is encoded in both firing rate and temporal structure of spike-trains (Tsodyks and Markram, 1997; Reinagel and Reid, 2000; van Rullen and Thorpe, 2001; Nelken et al., 2005). Dynamic transition from latency to rate processing in the very same neurons can unify the processing of spike-time and rate codes in a single network, and hence forms an attractive scheme of computation in neural networks.

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