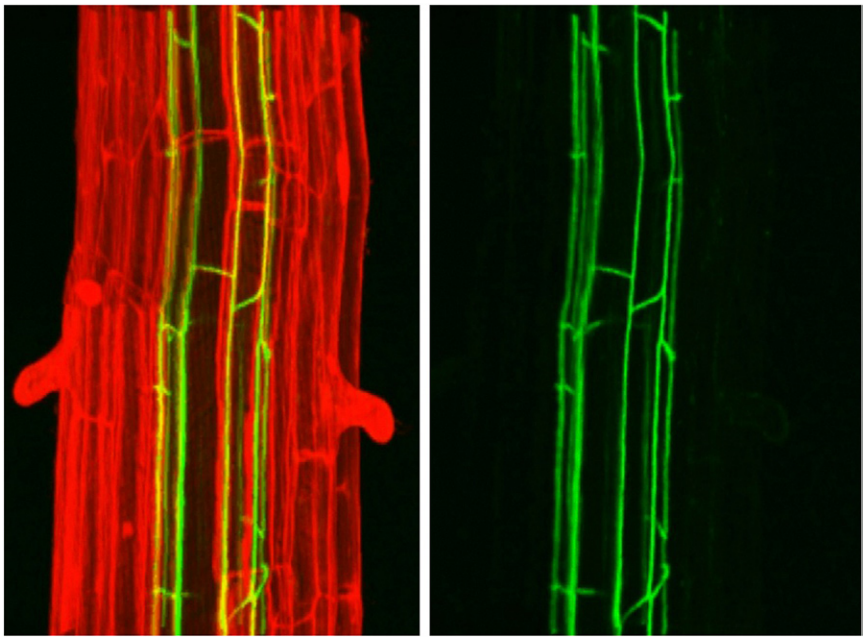

Figure 1. Casparian strips.
An *Arabidopsis* root stained for PI (red) to mark cell membranes, and CASP1-GFP (green) to show the Casparian strip membrane domain. (Photo: Julien Alassimone, University of Lausanne.)

to compare roots to 'everted guts'. They explore the soil and extract nutrients from it and must interact with many beneficial or not-so-beneficial microorganisms, and they must therefore balance uptake with protection, just like the gut. Putting the diffusion barrier deep inside the root may allow the cortex to act like a 'lobby' where many things are admitted and can be perceived and selected for uptake. Only at the point where the vasculature begins (which is a direct highway to the precious leaves) would the Casparian strips of the endodermis then put up a strict diffusion barrier.

What are Casparian strips made of? They are made of lignin, the same polymer that is used for building the xylem vessels of the vasculature and the characteristic constituent of wood. A primary cell wall impregnated with lignin makes for a very sturdy, chemically resistant structure, perfect for a protective barrier. In textbooks it is often said that the Casparian strip is made of suberin, the polymer of cork. That would also make a lot of sense, but it's wrong — let's see how long it will take for these findings to diffuse into the textbooks...

Are they structurally similar to tight junctions? No, Casparian strips are an independent invention of higher plants. The plasma membrane proteins that localize to the strips and are important for forming them (creatively called 'Casparian strip domain proteins', CASPs) do not have homology to the Claudins that form the tight junctions, although they are also small tetraspan membrane proteins. While CASPs do put up a lateral membrane diffusion barrier, CASPs from different cells do not interact, like Claudins do. Rather, the CASPs form ring-like domains that align between neighboring cells without touching (the cell wall wouldn't allow for this). The CASPs then cause polymerization of lignin in the cell wall space between two endodermal cells. CASPs probably do so by organizing a whole lignin polymerizing machinery at this place. So in some ways, Casparian strips are even more complicated to build than tight junctions.

Where can I find out more?

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Primer

Gap junctions

Kazumichi Shimizu and Mark Stopfer

In vertebrates and invertebrates, signaling among neurons is most commonly mediated by chemical synapses. At these synapses neurotransmitter released by presynaptic neurons is detected by receptors on the postsynaptic neurons, leading to an influx of ions through the receptors themselves or through channels activated by intracellular signaling downstream of the receptors. But neurons can communicate with each other in a more direct way, by passing signals composed of small molecules and ions through pores called gap junctions. Gap junctions that transmit electrical signals are called electrical synapses. Unlike most chemical synapses, electrical synapses interact through axon-to-axon or dendrite-to-dendrite contacts. Found throughout the nervous system, they are probably best known for linking the relatively few inhibitory, GABAergic, neurons into large, effective networks within vertebrate brains. They are particularly important early in development before the formation of most chemical synapses, but recent work shows gap junctions play important roles in the adult nervous system, too. Gap junctions are sometimes thought to be mere passageways between cells. But, as recent work shows, their properties can be complex and surprising. Gap junctions help generate, propagate, and regulate neural oscillations, can filter electrical signals, and can be modulated in a variety of ways. Here we discuss recent work highlighting the diversity and importance of gap junctions throughout the nervous system.

Structure and development

Gap junctions are composed of pairs of hemichannels, each consisting of a hexameric complex of connexins, embedded in closely apposed plasma membranes of neighboring cells (Figure 1). Connexins are diverse. In mammals, they are encoded by a family

containing at least 20 members. Two hemichannels with the same or different connexin composition form homotypic or heterotypic gap junctions, respectively, although heterotypic gap junctions appear to be rare. Each hemichannel can also be homomeric or heteromeric. In invertebrates, gap junctions are composed of innexins. Although the amino acid sequences of innexins lack homologies to the connexins of mammals, they are predicted to share similar secondary structures: four hydrophobic transmembrane domains connected by one cytoplasmic and two extracellular loops. Mammals also have innexin homologues called pannexins, although none has been shown to form gap junctions *in vivo*.

Gap junctions develop before chemical synapses. Early in development they serve many functions, such as allowing networks of progenitor neurons to link up, forming clusters of neurons sharing synchronized cell cycles, and later, coordinating calcium waves that help control proliferation. Gap junctions can even serve as adhesion molecules that attach migrating neurons to their radial fiber highways. Connexins have been proposed to promote neural differentiation and neurite outgrowth. Electrical coupling between pyramidal cells in rodent neocortex is extensive during the first postnatal week, and, in fact, the period during which circuits are assembled in the developing neocortex coincides with the greatest prevalence of electrical coupling. As the animal matures, though, these electrical synapses virtually disappear. Electrical synapses are critical for the establishment of normal circuitry. For example, the shared feature selectivity often observed among sister neurons can be abolished by expressing a dominant negative connexin that forms closed gap junctions. In some cases, it is not clear whether gap junctions mediate mainly electrical or biochemical effects early in cortical circuit assembly: electrical coupling was shown to facilitate synchronous spiking between sister excitatory neurons in the developing neocortex, but earlier work showed the neural activity in the columnar domains is propagated by the spread of a second messenger (inositol triphosphate) through gap junctions rather than by electrical coupling.

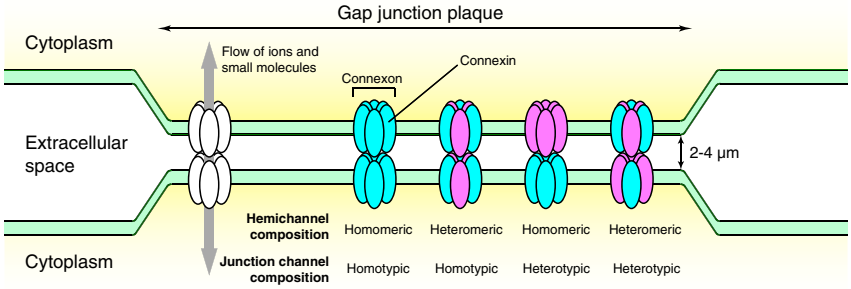

Figure 1. Molecular composition of gap junctions.
Two connexons, or hemichannels, in the apposed membranes of neighboring cells form a conduit for ions and small molecules. A cluster of these channels form a gap junction plaque which brings the membranes of neighboring cells close together (2–4 μm). Each connexon is composed of six connexin subunits. At least 20 connexin genes are found in mammalian genomes; here different connexin proteins are shown as different colors. Hemichannels can be either homomeric or heteromeric, and gap junctions can be either homotypic or heterotypic based on the composition of constituent connexons.

Electrical properties and regulation

Electrical synapses can have interesting properties. They are generally characterized by injecting depolarizing or hyperpolarizing current into one of the coupled cells and measuring the resulting change in the membrane potential of its coupled neighbor. The strength of an electrical synapse is usually given as a coupling coefficient (CC), the ratio of the voltage change in the coupled cell over the change in the stimulated cell at the steady state (Figure 2). This measure combines the conductance of the uninjected cell with the junction conductance between the coupled cells: $CC = g_j/(g_j + g_{uninjected})$, where g_j is the junction conductance and $g_{uninjected}$ is the membrane conductance of the uninjected cell. Although most electrical synapses pass current in both directions (a CC can be determined for each), one-way (rectified) transmission has been observed in both vertebrates and invertebrates.

Although gap junctions are sometimes thought of as simple openings between neurons, the strength of the electrical connection can be regulated in several ways. The most common connexin in the mammalian nervous system (connexin 36) and its fish homologue are known to have at least two phosphorylation sites for protein kinase A (PKA). Phosphorylation of these amino acid residues is dynamically regulated and positively correlated with gap junction coupling. Protein kinase G (PKG) and Ca^{2+}/calmodulin-dependent protein kinase II (CaMKII) have been shown to phosphorylate connexin 36, and the phosphorylation of connexins has been shown to change the unitary conductance and open probability of gap junctions. Phosphorylation may also influence underlying processes that can affect junction conductance, such as the assembly, trafficking or internalization of gap junctions. Intracellular pH and Ca^{2+} concentration have also been shown to regulate gap junction conductance.

A well-studied example of the regulation of electrical synapses by neuromodulators is found in the mammalian retina, where visual input arrives with a wide range of light intensity. The retina's ability to respond flexibly is mediated in part by adjusting the conductance of gap junctions that interconnect all of the main types of retinal neurons (photoreceptor, horizontal, bipolar, amacrine, and ganglion cells). Changes in light intensity have been shown to regulate electrical synapses in at least three places, between rods and cones, between horizontal cells, and between AII amacrine cells.

Electrical synapses joining rods and cones provide one pathway for visual information. Signals from rods are directed to the cones through electrical synapses, then to cone bipolar and cone ganglion

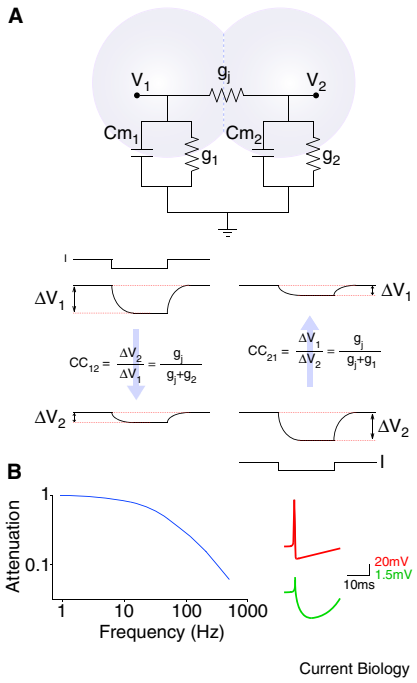

Figure 2. Electrical properties of gap junctions.
(A) Equivalent circuit and characterization of the coupling coefficient (CC) of two neurons connected through gap junctions. Parallel circuits composed of the conductance (g_1 and g_2) and capacitance (C_{m1} and C_{m2}) of two neurons are connected through a gap junction conductance (g_j). CC can be measured in both directions. To characterize CC from cell1 to cell2 (CC_{12}), a step current is injected into cell1 and the resulting voltage deflections in cell1 (ΔV_1) and cell2 (ΔV_2) are measured at their steady state. CC_{12} corresponds to the ratio of ΔV_2 to ΔV_1, which equals $g_j/(g_j+g_2)$. CC_{12} and CC_{21} are not necessarily equal because different cells have different membrane conductances. (B) Electrical synapses work as low-pass filters because membrane capacitance takes time to charge. To measure the filter properties of an electrical synapse, sinusoidal currents of different frequencies are injected into one cell and signal attenuation is measured in the other cell. Signal attenuation is normalized to measurements made for low frequency. A result of such filtering is that at some electrical synapses, fast components in the presynaptic signal (red), such as spikes, are greatly attenuated in the postsynaptic signal (green), while slower components, such as the hyperpolarization that follows a spike, are less affected.

cells. In goldfish and mouse, this rod–cone coupling is controlled by a circadian clock. During the day, the retinal clock increases the release of dopamine from interplexiform cells to the extracellular space, which activates the D2-like dopamine receptor on rods and cones, and then reduces intracellular cAMP and PKA activity, leading to a reduction in the conductance of gap junctions between rods and cones. At night, by contrast, decreased dopamine levels allow the activation of PKA that increases the junction conductance of rod-cone electrical synapses. This retinal clock mechanism ensures the rod–cone pathway operates only at night; in bright light, signals from cones would otherwise leak through the gap junctions and saturate the responses of rods, compromising daylight acuity.

Well-regulated gap junctions play an important role in distributing information throughout the retina. Horizontal cells, which extend processes laterally in the outer layer, receive input from photoreceptors, and distribute their responses through electrical synapses to other horizontal cells. Thus, each horizontal cell has a much larger receptive field than the area covered by its dendritic arbor. Horizontal cells feed inhibition back to the surrounding photoreceptors and forward to bipolar cells, ensuring the center-surround organization of responses in bipolar cells. The conductances of gap junctions joining horizontal cells are also regulated by luminance-dependent extracellular dopamine and nitric oxide levels, which act through cAMP/PKA- and cGMP/PKG-dependent intracellular mechanisms, respectively. The phosphorylation of connexins by PKA or PKG appears to decrease the junction conductance between horizontal cells. Interestingly, the extent of coupling is greatest at intermediate light intensity and decreases under both dark- and light-adapted conditions. When dark adapted, coupling among horizontal cells decreases, attenuating the surround signal, thus increasing sensitivity to light at the cost of contrast detection. In bright light, decreased coupling among horizontal cells enhances local contrast detection.

Because the main rod pathway relies on AII amacrine cells, regulation of electrical coupling among these cells is critical for keeping the retinal circuit sensitive through a wide range of brightness. As in horizontal cells, the modulation of electrical coupling among AII amacrine cells is also triphasic. When dark-adapted, the array of AII amacrine cells becomes relatively decoupled and the main rod pathway becomes more sensitive to single photons by preventing its signals from dissipating through surrounding AII amacrine cells. As luminance increases, coupling among AII amacrine cells increases, leading to an improved signal-to-noise ratio achieved by averaging inputs across the coupled amacrine cell population. When light adapted, AII amacrine cells are decoupled to a level comparable to the dark-adapted condition, which decreases the extent of 'crossover inhibition', a mechanism allowing ON bipolar cells to inhibit OFF bipolar cells and OFF ganglion cells through AII amacrine cells, which impairs acuity. The strength of the gap junction coupling is modulated by dopamine released from a subtype of amacrine cells, which signals through the D1 receptor and a downstream cAMP-mediated PKA cascade.

In other neurons the strength of an electrical synapse can be regulated by the activity of glutamatergic synapses. The activity-dependent regulation of gap junction conductance has been studied in the teleost auditory system, where afferent input makes mixed electrical and chemical synapses in each terminal (known as a club ending) onto Mauthner cells (Figure 3A). Depending on the stimulus protocol, tetanic stimulation of the afferent input can lead to long-term potentiation, short-term potentiation, or long-term depression. After afferent nerves are stimulated with a brief burst of high frequency (500 Hz) stimulation, both electrical and chemical components of the postsynaptic potential become potentiated over both the short- and long-term.

Another form of potentiation between club endings and the Mauthner cell has also been reported. Low frequency tetanizing stimulation of the presynaptic nerve induces the release of endocannabinoids from the lateral dendrites of the Mauthner cell, which in turn activates the release of dopamine from varicosities that surround the club endings. Local dopamine application has been shown to induce long-term potentiation of both chemical and electrical components of these synapses.

Interactions between glutamatergic synapses and electrical synapses

may provide a common mechanism for activity-dependent modulation of electrical synapses; as in teleost Mauthner cells, glutamate receptors and gap junctions have been observed in close proximity in the inferior olive. Also, long-term depression of electrical synapses between thalamic reticular nucleus (TRN) neurons has been reported. Tetanization of glutamatergic corticothalamic input to TRN neurons or pharmacological activation of metabotropic glutamate receptors (mGluRs) can cause a decrease in the coupling coefficient between TRN neurons; pretreatment with mGluR antagonist blocks this decrease. Electrical synapses between TRN neuron pairs are also known to robustly synchronize their spiking; reducing the electrical coupling between pairs of TRN neurons after mGluR agonist application reduces spike synchrony between these pairs. A reduction of coupling can also be induced by causing one coupled TRN neuron to fire a burst of spikes that elicits depolarizing responses below the firing threshold of the other coupled TRN neuron. Interestingly, these neurons undergo an asymmetric modulation of electrical coupling: the coupling coefficient is reduced less from the cell in which spiking is induced than in the opposite direction, suggesting TRN neurons fine-tune the relative strengths of signals they send and receive through electrical synapses.

As described by the equation described above (Figure 2), coupling through gap junctions can be adjusted by changing the passive membrane conductance of the postsynaptic neuron. Increasing or decreasing passive conductance decreases or enhances, respectively, the strength of electrical coupling. The response size can also be affected by active subthreshold conductances in the postsynaptic neuron. When the postsynaptic neuron is depolarized beforehand, for example, signals conducted by gap junctions are amplified and may be more likely to elicit spiking (Figure 3). This type of voltage-dependent amplification of the response appears to play an important role in the gold fish auditory system and in the mammalian cerebellar Golgi cell network.

Electrical synapses can give rise to complex physiological responses.

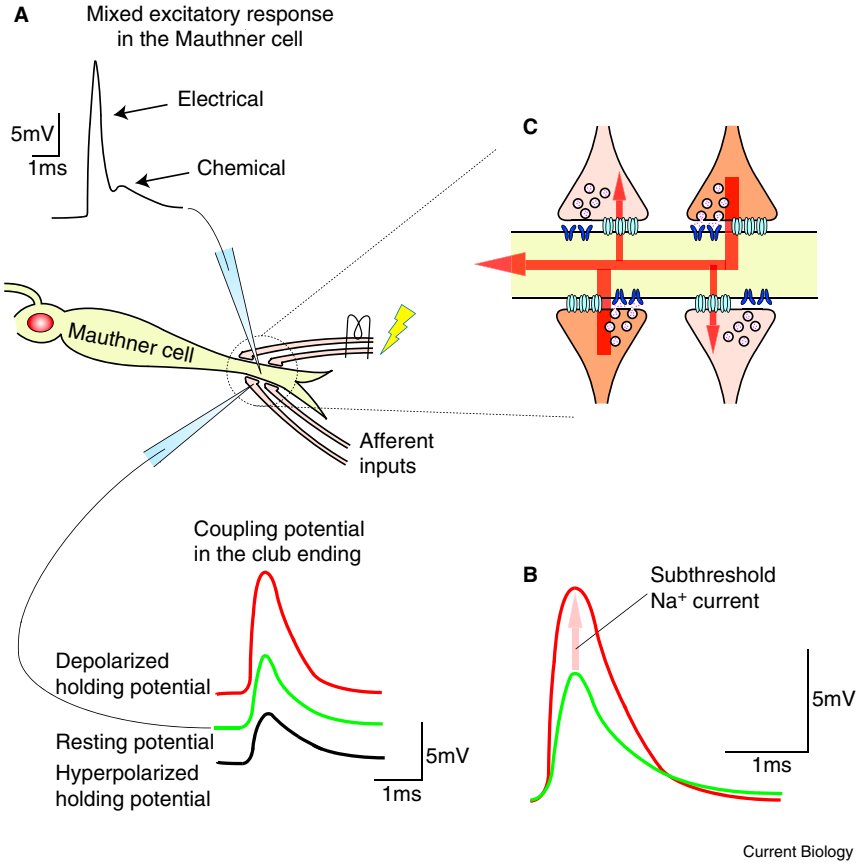

Figure 3. Lateral excitation among auditory afferent inputs in teleosts.
(A) Experimental arrangement: delivering an electric shock to afferent inputs induces a mixed electrical and chemical response in the lateral dendrite of the Mauthner cell, which in turn induces coupling potentials in the terminals of the afferent inputs (club endings). Bottom: traces show coupling potentials recorded when club endings were held at different voltage levels. (B) Superimposed coupling potentials at depolarized (red) and resting (green) potentials. Coupling potentials are enhanced at depolarized holding potentials by a subthreshold Na^+ current. (C) Terminals of afferent inputs that are activated strongly above the threshold (dark orange) induce mixed responses in the lateral dendrite of a Mauthner cell. The electrical component can be unusually large because the fast time constant of the Mauthner cell allows minimal low-pass filtering, and can therefore retrogradely induce strong depolarizing responses in the closely positioned neighboring afferent terminals. Depolarizing responses in afferent terminals that are weakly activated (pale orange) can boost the membrane potential above spiking threshold in a voltage dependent manner, enhancing the cooperative activation of afferent inputs.

Mauthner cells receive auditory afferent input consisting of both chemical and electrical synapses. Activation of afferent inputs elicits strong responses through electrical synapses in a postsynaptic Mauthner cell lateral dendrite, which can then retrogradely induce additional depolarizing responses through electrical synapses in neighboring auditory afferent terminals that didn't reach the spiking threshold (Figure 3). These responses are larger when the membrane potential of the club endings are more depolarized. This mechanism is thought to drive lateral excitation among afferent inputs, increasing their cooperativity.

Filtering properties of electrical synapses

Electrical synapses can alter the frequency characteristics of the signals they transmit, and can thus serve as electrical filters. Electrical synapses work as filters because the conductance and the capacitance of the postsynaptic cell are linked in parallel, forming a circuit in which the membrane's capacitance takes time to charge. Thus, electrical synapses function as low-pass filters: they attenuate the high frequency components of a presynaptic signal, such as action potentials, and preferentially transmit low frequency signals, such as the relatively

lengthy hyperpolarizations that can follow an action potential (Figure 2). The filtering characteristics of an electrical synapse can vary greatly depending upon the membrane properties of the post-synaptic cell.

The varying membrane properties and geometries of particular coupled cells, the gap junctions themselves, and the waveforms of spikes, all strongly influence whether depolarizing or hyperpolarizing features will control a given postsynaptic response. Many mammalian neurons have relatively long time constants, so electrical synapses among them preferentially pass slower signal features like after-hyperpolarizations while attenuating fast signal features like depolarizing spikes. In neurons with short time constants, the low-pass filtering of signals by electrical synapses is not significant. Some neurons, such as the Mauthner cells and the club endings, which in some species help mediate rapid escapes, have quick time constants on the order of hundreds of microseconds. Electrical synapses upon neurons with such brief postsynaptic time constants do not attenuate spikes and can therefore induce large depolarizing responses in the postsynaptic neurons, which can therefore propagate excitation laterally. Interestingly, it has recently been suggested that asymmetric expression of two different connexins in the presynaptic and postsynaptic neurons can lead to the biased flow of electrical current between the cells. Because the junction conductance is greater from Mauthner cell to club endings than in the opposite direction, and because club endings have lower membrane conductance than Mauthner cells, current flows more readily from Mauthner cells to club endings, promoting lateral excitation among the afferent inputs.

Lateral excitation and synchrony

Gap junctions contribute to lateral excitation in the olfactory systems of vertebrates and invertebrates. In the rodent, axons of olfactory sensory neurons expressing the same olfactory receptor gene converge onto the same glomerulus in the olfactory bulb where their axons synapse onto mitral and tufted cells. When stimulated, mitral cells release vesicular glutamate from their dendritic tufts, thereby activating NMDA and AMPA autoreceptors on their own dendrites. This autoreceptor-mediated potential is distributed to neighboring mitral cell dendrites within the glomerulus through gap junctions, which interconnect the dendrites of mitral cells inhabiting the same glomerulus. This mechanism, in conjunction with glutamate spillover, boosts the excitability of mitral cells in each glomerulus. Some evidence suggests gap junctions also synchronize the spiking of mitral cells sharing the same glomerulus: a knockout mouse lacking connexin 36, thus lacking electrical coupling between mitral cells, shows impaired spike synchrony. However, other evidence from another study using different stimulus conditions suggested both intra-glomerular and inter-glomerular spike synchrony is mediated mainly by synchronized inhibitory post-synaptic potentials (IPSPs) imposed by inhibitory GABAergic granule cells.

The *Drosophila* antennal lobe is configured much like the rodent olfactory bulb, with axons of olfactory receptor neurons expressing the same olfactory receptor gene converging upon the same glomerulus in the antennal lobe, where they synapse on uniglomerular projection neurons. However, lateral excitation observed in the *Drosophila* antennal lobe occurs between rather than, as in the rodent olfactory bulb, within glomeruli. Postsynaptic projection neurons in each glomerulus respond to more odors than their corresponding presynaptic olfactory receptor neurons; that is, projection neurons are more broadly tuned to odors than are olfactory receptor neurons. Some studies suggest the broader tuning is mediated by lateral excitation in the *Drosophila* antennal lobe through electrical synapses between projection neurons in each glomerulus and local interneurons that send neurites throughout the antennal lobe.

Because most electrical synapses operate bidirectionally, they tend to equilibrate the membrane potentials of connected neurons. Consequently, electrical synapses can help to synchronize populations of cells. Indeed, electrical synapses are known to play important roles in synchronized spiking activity in neocortex, cerebellum, hippocampus, inferior olive, suprachiasmatic nucleus, retina and olfactory bulb. In many of these areas, neurons joined through electrical synapses fire synchronously at oscillatory frequencies determined by the intrinsic properties of the neurons or the broader circuit properties of the excitatory and inhibitory networks they form. The presence of electrical synapses does not seem to influence oscillation frequency, but can enhance the extent of synchronization.

An interesting example of an oscillation-generating network is one formed by cerebellar Golgi cells. Golgi cells receive input from mossy fibers and parallel fibers and make inhibitory synapses onto granule cells, but are also densely interconnected to each other exclusively through electrical synapses. When Golgi cells are activated uniformly by pharmacological means, they begin to fire synchronously. The after-hyperpolarizations of spikes, which are preferentially transmitted through their electrical synapses and lead to strong postsynaptic hyperpolarizing responses, do the main work of recruiting the Golgi cell network into common oscillatory dynamics on the time scale of oscillatory frequency (on the order of tens of milliseconds to a few hundred milliseconds). Computational models of the Golgi cell population show that electrical synapses can establish oscillatory synchronization of Golgi cells in the absence of chemical synaptic transmission or intrinsic resonance properties, a result consistent with experiments showing that rhythmic spikelets observed in Golgi cells are abolished in knockout mice lacking connexin 36.

The mechanism used by the Golgi cell network stands in contrast to that of many other systems in which electrical synapses enhance synchrony but are not necessary for generating oscillations. In the Golgi networks, spikes arriving through electrical synapses from other Golgi cells can induce excitatory, depolarizing responses ('spikelets') that ordinarily contribute little to generating network oscillations. However, when enhanced by sub-threshold sodium conductances, the

excitatory responses are amplified. Then, excitation mediated by gap junctions can contribute significantly to very precise spike synchronization on the millisecond scale through a mechanism similar to that observed at the electrical synapses between the club endings and the Mauthner cell.

Electrical synapses also mediate a surprising behavior in the Golgi cell network: the arrival of sparse and coincident feed-forward input from mossy fibers can actually desynchronize the Golgi network. This results from the filtering properties and broad interconnectivity afforded by electrical synapses. Spiking input from mossy fibers can induce spikes including an after-hyperpolarization in a Golgi cell directly postsynaptic to it. These spikes, in turn, filtered through electrical synapses, can induce attenuated spikelets but strong hyperpolarizations in neighboring Golgi cells that do not receive direct synaptic input from the spiking mossy fibers. The strong hyperpolarizing component of the gap junction potential then briefly inhibits spike generation in the neighboring cells, desynchronizing the coupled cells.

Synchronization mechanisms in some other cell populations appear to work differently from those of the Golgi cell network. In the hippocampus and neocortex, spikelets carried through electrical synapses between pyramidal cells seem to play an important role in synchronizing these neurons. Electrical synapses in the hippocampus and neocortical areas between pyramidal cells are thought to underlie ultrafast oscillations (~200 Hz), whereas those between interneurons are important for oscillations at gamma frequencies (20~80 Hz). Many interesting questions remain about the existence and roles of gap junctions between pyramidal cells, though. The connexins that underlie the electrical synapses between pyramidal cells remain to be elucidated: ultrafast oscillations are intact in knockout mice lacking connexin 36, in which gamma frequency oscillations are impaired, while pharmacological gap junction blockers abolish high frequency oscillations.

Outlook

Gap junctions and the electrical synapses they form play important and varying roles throughout the brain. Electrical synapses form before chemical synapses and help shape the brain's development. But, throughout life, they filter, distribute, and coordinate neural activity in complex and well-regulated ways that have only recently come into focus. Exploring the computational properties of gap junctions is an active area of research; many questions remain about the diverse contributions they make to establish and regulate neural synchrony. Also, to date, most studies on electrical synapses have focused on connexin 36, which is the most prevalent; but other connexins and pannexins are also expressed in neurons, and their contributions to shaping neural activity in the brain remain largely unknown. New molecular and computational tools will no doubt add to our knowledge of gap junctions and electrical synapses.

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Microplastic ingestion decreases energy reserves in marine worms

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The indiscriminate disposal of plastic to the environment is of concern. Microscopic plastic litter (<5 mm diameter; 'microplastic') is increasing in abundance in the marine environment, originating from the fragmentation of plastic items and from industry and personal-care products [1]. On highly impacted beaches, microplastic concentrations (<1mm) can reach 3% by weight, presenting a global conservation issue [2]. Microplastics are a novel substrate for the adherence of hydrophobic contaminants [1], deposition of eggs [3], and colonization by unique bacterial assemblages [4]. Ingestion by indiscriminate deposit-feeders has been reported, yet physical impacts remain understudied [1]. Here, we show that deposit-feeding marine worms maintained in sediments spiked with microscopic unplasticised polyvinylchloride (UPVC) at concentrations overlapping those in the environment had significantly depleted energy reserves by up to 50% (Figure 1). Our results suggest that depleted energy reserves arise from a combination of reduced feeding activity, longer gut residence times of ingested material and inflammation.

Seabeds worldwide are composed of a range of organic and inorganic sediments that sustain a vast range of marine species. The polychaete worm *Arenicola marina* (lugworm) of the globally distributed family Arenicolidae is a keystone species inhabiting intertidal sediments in Northern Europe; it bioturbates and irrigates the sediment and is an important secondary producer, as a prey species for fish and wading birds. Using a laboratory mesocosm, we performed chronic (four weeks) and short-term (48 hours) experiments, exposing *A. marina* to natural sediments containing clean, chemically-inert UPVC ranging from 0–5% by weight. PVC is denser than