Wireless Communication in the Brain: I. Evidence from Long Range Synchronization

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Abstract

In the past two decades, experiments have provided strong evidence that the brain may use extracellular electric fields to convey information, a process known as "ephaptic coupling". This paper shows that the brain may also employ electromagnetic (EM) waves for light-speed communication. In the wireless communication system, ion channels may act as the transmitting antennas while microtubules at the axon initial segment may serve as the receiving antennas. The proposed mechanism explains why some neurons are sensitive to extracellular electric fields and why distant brain areas can synchronize with zero phase lag. The ephaptic coupling may enhance neuronal excitability but usually insufficient to initiate action potentials. The final push above threshold is achieved by the EM waves to ensure precise timing of spikes in a distributed network. This notion is supported by the observation of "phase precession", namely, the spike timing precedes the peak of local field potential.

Introduction

For over a century, synaptic transmission between neurons has been thought to be the predominate method of information transfer within the brain. However, the wired communication will cause time delay, while long range synchronization with zero phase lag was commonly observed (Roelfsema et al., 1997; Neuenschwander and Singer, 1996; Volgushev et al., 2006). Its underlying mechanism remains unclear.

Brain activity is associated with ionic processes at dendrites, soma and axon, resulting in fluctuation of electric fields at the extracellular space. The extracellular electric fields give rise to electroencephalogram (EEG) when

recorded from the scalp, and to local field potential (LFP) when recorded by a microelectrode in the brain (Buzsáki et al., 2012). These electric fields act instantaneously over a long distance, capable of conveying information between distant brain areas. The cell-cell interaction mediated by extracellular electric fields is called **ephaptic coupling** which has been shown to play an important role in synchronization (Dudek et al., 1998; Anastassiou et al., 2011). However, the ephaptic coupling alone does not seem to be sufficient for zero-lag long range synchronization because the extracellular fields are weak and, at any moment, LFP may vary between distant brain areas. Indeed, the spike timing usually precedes, rather than coincides with, the LFP peak, a phenomenon known as "phase precession" (Harvey et al., 2009, Figure 1).

This paper proposes that the brain may also employ electromagnetic (EM) waves for light-speed communication. Within this framework, the ephaptic coupling is a non-radiative component of the wireless communication system. Both ephaptic coupling and radiative EM coupling could be widely used by the brain for long range synchronization. The ephaptic coupling may enhance neuronal excitability but usually insufficient to initiate action potentials. The final push above threshold is achieved by the EM waves to ensure precise timing of spikes in a distributed network.

The mechanism proposed in this paper is supported by a growing body of evidence, including long range synchronization, stimulation of spatial cells (place cells, grid cells, etc.), and pleasure principle (seek pleasure, avoid pain) which will be presented in a series of papers.

Overview

In the human-invented wireless communication systems, EM waves are radiated from transmitting antennas and absorbed by receiving antennas. The absorbed EM energy is then transformed into sound or images. In the brain, wireless communication may work in a similar manner:

- Ion channels may act as the transmitting antennas.
- Microtubules at the axon initial segment (AIS) may serve as the receiving antennas.
- The microtubule antennas may transform the absorbed EM energy into neuronal excitability.

- Tau proteins can regulate neuronal excitability by binding with microtubule antennas.
- The extracellular electric fields may interact with microtubule antennas to enhance excitability.

Details are described in the following sections.

Ion Channels as the Transmitting Antennas

Neural activity is associated with the motion of ions passing through ion channels in the nerve membrane. From the viewpoint of wireless communication, the ionic current through a channel is equivalent to the electric current in a metal antenna. According to physical laws, EM waves will be radiated whenever an electric charge is accelerated, either the electrons in metal antennas or ions in ion channels. In the metal antenna, the acceleration of electrons is caused by the electric circuit connected with the antenna. In the ion channel, ions are accelerated by the electrochemical force resulting from the differences in the electric potentials and ion concentrations on both sides of the membrane. Since ions carry charges, their accelerated motion will emit EM waves.

During the opening of a channel, a train of ions will pass through the channel one by one. Each ion will generate a pulse of EM wave. Thus, the frequency of EM waves emitted by these accelerated ions can be estimated from single channel currents. For voltage-gated sodium and potassium channels, the single channel current is about 1 - 4 pA (Aldrich and Stevens, 1987; Zagotta et al., 1988), where 1 pA = 10^{-12} ampere and 1 ampere = 6 x 10^{18} electron charge per second. Hence, about 10^{7} ions can pass through a single channel within a second. The interval between two consecutive ions is then equal to 10^{-7} second, corresponding to a frequency of 10 MHz, which belongs to the radio band. This radio wave represents the "carrier signal" used in wireless communication while the low frequency waves (alpha, theta, gamma, etc.) observed by electroencephalography (EEG) are the modulating signals. Information is encoded in modulating signals, rather than the carrier signal.

Synchronization to Maximize Radiation Power

To influence a receiving brain region, the transmitting signals must be sufficiently strong. The power radiated from an accelerating charge can be obtained from the Larmor formula. In SI units, it is given by

$$P = \frac{q^2 a^2}{6\pi\varepsilon_0 c^3} \text{ (SI units)}$$

where a is the acceleration, q is the charge, ε_o is the vacuum permittivity (= 8.8 x 10^{-12} F/m), and c is the speed of light (= 3 x 10^8 m).

In free space, the acceleration can be calculated from the formula,

$$\text{Force} = Ma = eE$$

where E is the electric field (~ 1 x 10^7 V/m), e and M are the charge and mass of an ion respectively. For Na^+, e = 1.6 x 10^{-19} coulombs and M ~ 10^{-26} kg. The above equation gives a = 10^{14} m/s^2. Within the channel pore, the acceleration could be reduced by friction. Let us assume a ~ 10^{13} m/s^2. Hence,

$$P_{single} \sim 10^{-28} \text{ W}$$

which is approximately the power radiated by a single Na^+ ion passing through an open channel. What will the power be for a group of accelerating ions? By simple multiplication, the total power radiated by N ions is given by

$$P_{total} = N \times P_{single}$$

However, if in a small region a large number of neurons fire synchronously, a group of ions may be accelerated simultaneously so that N ions can be treated as a single charge. In this case, instead of using the simple multiplication, we should substitute q = Ne into the Larmor formula. The result is

$$P_{total} = N^2 \times P_{single}$$

Note that the total power is now proportional to the **square** of N. Thus, synchronization can dramatically increase the radiation power. This explains why synchronization is crucial for information transfer within the brain.

As estimated above, 10^7 ions can pass through a single channel per second. The open duration of a channel is about 1 ms. Therefore, during channel opening, roughly 10^4 ions can pass through a channel. These ions may be treated as a single charge in the Larmor formula. Suppose a network of 10^6 neurons fire synchronously, each having 1000 open channels, then $N \sim 10^{13}$. Finally, the synchronized network may radiate

$$P_{total} = N^2 \times P_{single} \sim 0.01 \text{ W}$$

This radiation power could be sufficient to influence brain activity based on two different experimental approaches. First, mobile phones have been demonstrated to affect the alpha rhythms (Valentini et al., 2007). The old 2G phones radiate 2 W, but the 3G phones radiate only 0.25 W, which can still enhance cortical reactivity (Roggeveen et al., 2015a) and cause significant effects on the alpha, beta, and gamma bands (Roggeveen et al., 2015b) when the phone was placed on the ear of a human subject. Inside the brain, the required power to influence neural activity could be even smaller as demonstrated from another approach: the electric fields.

Sensitivity of Neurons to Weak Electric Fields

The membrane potential field, as obtained from the membrane voltage and membrane thickness, is on the order of 10^7 V/m. Surprisingly, some neurons are sensitive to the external electric field as small as 1 V/m (Francis et al., 2003; Reato et al., 2010). Voltage-gated ion channels depend on the membrane potential field, which changes only a tiny fraction by superposition with the small external field. Therefore, the significant effects on neural activity must not arise from the direct interaction between the external field and ion channels. The neuron should possess certain mechanism to "amplify" the external signal, which is necessary for wireless communication because the transmitting EM waves also produce very weak electric fields.

The EM wave consists of oscillating electric and magnetic fields. Given the radiation power, it is possible to estimate the electric field strength at certain distance from the source. A formula is available on this website. Although the formula is accurate only for large distance, our purpose is to get a rough estimate. For power = 0.01 W and distance = 5 cm, the formula gives E = 10 V/m. This field strength is negligible compared with the membrane potential field, but is sufficient to influence neural activity as demonstrated experimentally. The underlying mechanism for signal amplification could involve microtubules which contain high density of negative charges.

Signal Amplification by Microtubules

In most cell types, a microtubule consists of 13 protofilaments, which form a hollow tube with a diameter of 25 nanometers (nm). Each protofilament is made up of tubulin dimers: α and β. The α subunit of one dimer is attached to the β subunit of the next dimer. Thus, in a protofilament, one end (called "minus end") has the α subunit exposed while another end (called "plus" end) has the β subunit exposed. It is important to note that the definition of "+" and "-" on both ends does not mean that the microtubule is an electric dipole with the plus end dominated by positive charges. In fact, the entire microtubule is highly negatively charged (Baker et al., 2001), because tubulin dimers are enriched with acidic residues (aspartate and glutamate). In a solution at the physiological pH value (~ 7), these amino acids become negatively charged. Another amino acid, histidine, also has significant probability to become negatively charged at pH = 7. From its amino acid sequence, the net charge on a tubulin dimer can be calculated to be 50.9 e^- at pH = 6.7 (Minoura and Muto, 2006).

With this electric property, the microtubule will experience a strong force exerted by electric fields. Thus, applied electric fields can direct microtubules moving toward the anode (Kim et al., 2007). In a solution, microtubules are surrounded by counterions and polar water molecules which may reduce the electrostatic interaction between microtubules and external fields. The effective charge on a tubulin dimer was estimated to be 12 - 20 e^- (van den Heuvel et al., 2006; Minoura and Muto, 2006).

Neuronal excitability is fundamentally governed by the opening and closing of ion channels, which in turn depend on the membrane voltage. By definition, the voltage between two points is given by the integration of

electric fields from one point to another. In a nerve membrane, the electric fields may arise from various sources, including ions in the intracellular and extracellular solutions, charges on surface molecules and the microtubules. The effects of surface charges on channel gating and excitability have been reported (Cukierman et al., 1988; Genet and Cohen, 1996), but the contribution from microtubules was largely ignored. As shown below, a microtubule can significantly modulate the membrane potential field (and thus excitability) when it localizes near the membrane (Figure 1).

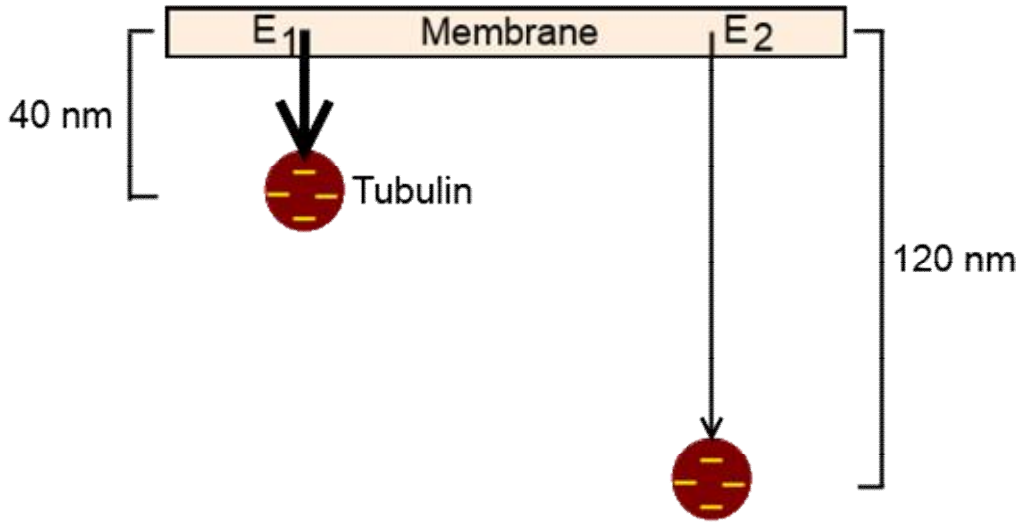

Figure 1. The contribution of a tubulin dimer to the membrane potential field. At the distance of 40 nm, the electric field (E_1) produced at the membrane by the tubulin dimer is about 10^7 V/m. When the tubulin dimer translocates to a distance of 120 nm, the produced field (E_2) will decrease to 10^6 V/m.

Let r_m be the distance between the center of a tubulin dimer and the middle of the membrane. The electric field produced at the membrane by the tubulin dimer is given by

$$E = kQ/r_m^2$$

where k is the Coulomb's constant and Q represents the effective charge on a tubulin dimer. Assuming Q = 12 e^-, at r_m = 40 nm, we have $E \sim 10^7$ N/C = 10^7 V/m, which is on the same order of magnitude as the total membrane potential field. As the tubulin (or microtubule) moves away from the membrane, it should have significant impact on the membrane potential field, and thus the channel gating. Generally speaking,

association of the microtubule with the membrane is equivalent to membrane hyperpolarization while dissociation from the membrane has the same effects as depolarization. Since a microtubule is highly negatively charged, a small external field may be able to exert a strong force to modulate its movement within the cell.

In non-neurons and most neuronal compartments, microtubules are tightly packed as lattices. They do not have much space to move around. Nor can a small field cause significant translocation. The only exception is the microtubules at the axon initial segment (AIS). Interestingly, AIS contains high density of voltage gated ion channels for the initiation of action potentials (Buffington and Rasband, 2011). It is the ideal place for microtubules to amplify the effects of external electric fields on neuronal excitability.

Microtubule Fascicles at AIS

At AIS, microtubules are organized as "fascicles", each is a bundle of several individual microtubules that are parallel with each another and cross linked. An AIS may contain 1 - 7 fascicles and the number of microtubules in each fascicle varies between 2 and 25. Its average number depends on neuronal types. In the motor neurons of the spinal cord, the number of microtubules per fascicle ranges from three to five, but in the pyramidal neurons of the cerebral cortex, the number can reach 22. Single or isolated microtubules are rarely observed in AIS (Palay et al., 1968).

The bundling of microtubules into fascicles is a unique feature of the AIS. This special structure was not found in the nodes of Ranvier, even though they resembles AIS in many aspects. Since AIS is the initiation site of nerve impulses and enriched with voltage-gated ion channels, one may expect the microtubule fascicles to play a role in neuronal excitability, as already did by Palay and colleagues. In 1968, they speculated that " the regulated contraction of the microtubules could change the shape of the initial segment and thus alter the configuration of the plasmalemma in this region, and consequently its permeability, with a resultant change in excitability".

In the past few years, experimental studies have provided great insights into the interaction between microtubules and Ankyrin-G, which allows for a more detailed description of the underlying mechanism (Figure 2). It turns out that the microtubule end-binding protein and the microtubule-associated protein Tau are also involved. The Tau protein is a central player in Alzheimer's disease and other neurodegenerative disorders. Its involvement in excitability opens the door for the fundamental understanding of neurodegeneration (Lee, 2015).

Microtubules as the Receiving Antennas

The main purpose of receiving antennas is to convert the received EM waves into other physical forms that can directly affect the receiving system. For instance, the EM waves cannot interact directly with the electric circuit in a radio or television. They must be converted into electric currents by the metal antenna. In the brain, neuronal excitability depends on channel gating and membrane potential, which cannot be directly modulated by EM waves. Some kind of "antenna" is required to transform EM energy into neuronal excitability. The highly negatively charged microtubules are well suited for serving as an antenna in a neuron, especially if they are localized to AIS.

The AIS can be divided into three layers: the plasma membrane, submembrane coat, and inner AIS shaft, each having AIS-specific features (Jones and Svitkina, 2016, Figure 1). The submembrane coat consists of Ankyrin-G, βIV-spectrin, and actin filaments. Microtubules, which usually exist in the form of fascicles, are located in the inner AIS shaft. During early development, the thickness of the submembrane coat varies in the range 3–11 nm (Jones et al., 2014). From electron micrographs (Palay et al., 1968), the coat thickness is about the same as the diameter of a microtubule (25 nm). Therefore, the distance between the center of a microtubule and the middle of the membrane cannot be shorter than 40 nm. At this minimum distance, the electric field produced at the membrane by a tubulin dimer is on the same order of magnitude as the resting membrane potential field (see above). The diameter of AIS is about 1500 nm while translocation from 40 nm to 120 nm is sufficient to reduce the hyperpolarizing field from a microtubule by an order of magnitude. Thus, translocation of a microtubule within the AIS can have

significant impact on channel gating. This property could play a crucial role in the modulation of excitability by EM waves and extracellular electric fields.

Ankyrin-G, end-binding proteins and Tau proteins have been demonstrated to regulate the association between microtubules and the membrane. In the absence of external forces, a microtubule fascicle may be anchored to the membrane by Ankyrin-G and the microtubule end-binding protein, EB1 or EB3 (denoted by EB1/3). The membrane-bound microtubule fascicle has inhibitory effects on neuronal firing. Similar to the interaction with metal antenna, the EM wave may exert a force on the negatively charged microtubules. Its oscillating electric field may cause the membrane-bound microtubules to vibrate in the transverse direction, resulting in dissociation from the membrane (Figure 2). This should increase excitability.

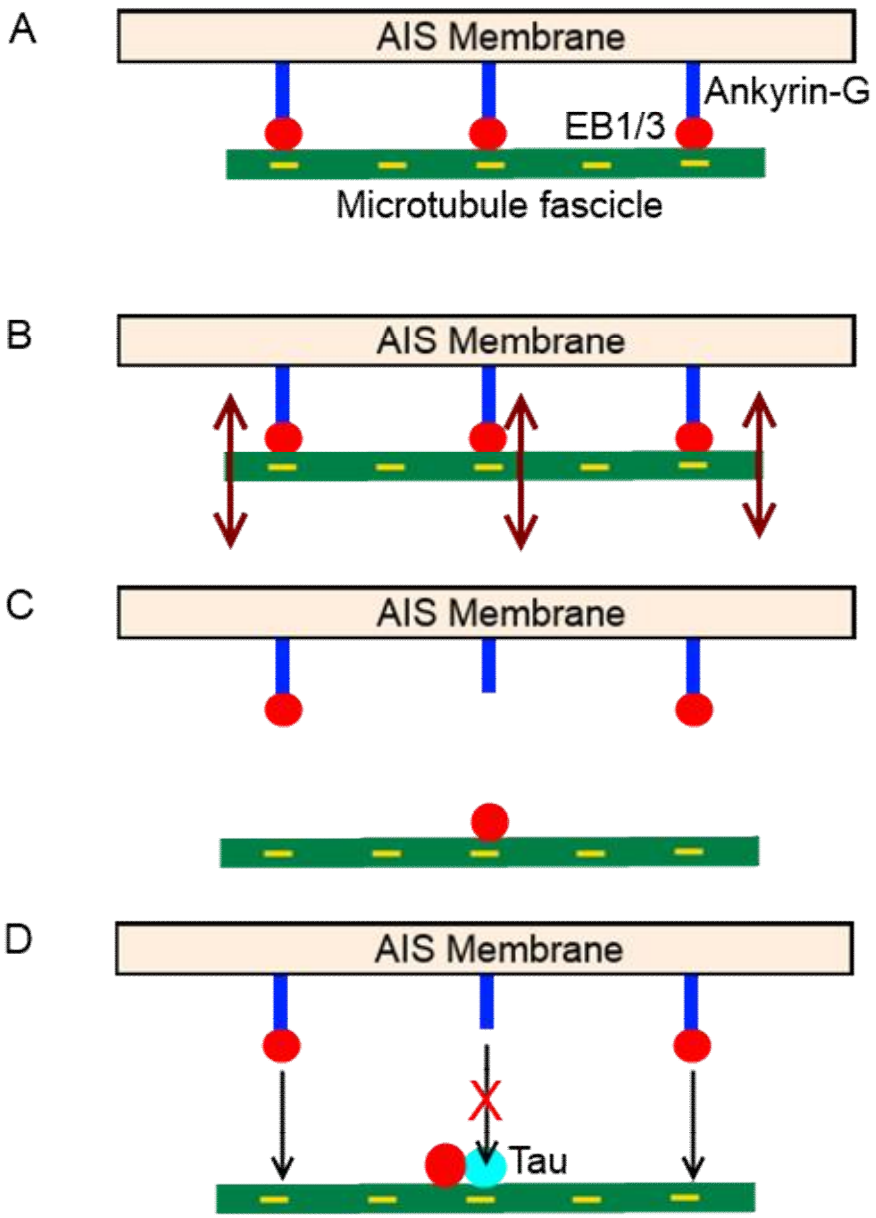

Figure 2. A model for the regulation of excitability at AIS.
(A) The association of the negatively charged microtubule fascicle with the membrane is mediated by Ankyrin-G and EB1/3. This should reduce excitability.
(B) The oscillating electric field in the EM wave may cause microtubules to vibrate in the transverse direction.
(C) The vibration of microtubules may result in dissociation from the membrane, thereby increasing excitability. EB1/3 may attach to either Ankyrin-G or microtubule.
(D) The Tau protein may enhance excitability by preventing the association between the microtubule fascicle and the membrane.

The Role of Ankyrin-G

Ankyrin-G, encoded by the ANK3 gene, plays a pivotal role in anchoring various proteins to the membrane, including microtubules (Figure 2). The "giant anchor" is located predominately at AIS and nodes of Ranvier (Iqbal et al., 2013). In experiments using TsA201 cells, it was found that Ankyrin-G could suppress the sodium current through Nav1.6 (Shirahata et al., 2006), which is a type of sodium channels that do not inactivate (O'Brien and Meisler, 2013). This observation supports the view that anchoring of microtubules to the membrane by Ankyrin-G has the same effects as hyperpolarization.

The persistent (non-inactivating) sodium channel, Nav1.6, is enriched in AIS, with a crucial role for resonance amplification, namely, enhancing membrane potential oscillation without shifting resonance frequency (Hutcheon and Yarom, 2000). Many cortical neurons exhibit spontaneous membrane potential oscillation below threshold at the resonance frequency that depends on the intrinsic membrane properties. Then a slight enhancement by the opening of persistent sodium channels can elicit a train of spikes with the original resonance frequency. This feature is important for long range synchronization. Whenever spikes are generated synchronously in one brain region, the emitted EM waves will immediately be received by any neurons in the brain, but only those with the same resonance frequency (e.g., alpha band, theta band, etc.) can join the synchronization.

The Role of End-Binding Proteins

The microtubule end-binding protein EB1/3 is required for the binding between the microtubule and Ankyrin-G (Leterrier et al., 2011; Fréal et al., 2016). Normally, EB1/3 binds to the microtubule's plus end, regulating its dynamic growth. However, at the AIS, the microtubule fascicles are decorated by EB1/3 over the entire molecule. This is because the microtubule also possesses other binding sites for EB1/3, albeit weaker. At low concentration, EB1/3 binds preferentially at the plus end which has stronger binding affinity. The weaker binding sites may be occupied only when the EB1/3 concentration is high (Bu and Su, 2001).

Ankyrin-G may also bind with EB1/3 through its tail domain (Fréal et al., 2016), which can extend into the intracellular AIS shaft at a maximum depth

of ~140 nm, with an average of only 26 nm below the submembrane coat (Jones and Svitkina, 2016). The diameter of the AIS is about 1500 nm. Therefore, most region within the AIS shaft is beyond the reach of Ankyrin-G. The fascicle has ample room to move randomly and exert little influence on channel gating. As the fascicle moves toward the membrane and within the reach of Ankyrin-G, it will be anchored to the membrane, thereby reducing excitability. On the other hand, the EM force may disrupt the interaction among Ankyrin-G, EB1/3, and the microtubule fascicle, causing the fascicle to dissociate from the membrane, thus increasing excitability (Figure 2).

The Role of Tau Protein

The Tau protein is a central player in Alzheimer's disease and other neurodegenerative disorders. It can directly interact with and recruit EB1/3 to the microtubule bundle (Sayas et al., 2015). Thus Tau may interfere with the interaction among Ankyrin-G, EB1/3, and the microtubule fascicle, preventing the fascicle association with the membrane, and consequently enhancing excitability. In animal models, Tau reduction or knockout has been demonstrated to attenuate hyperexcitability (Holth et al., 2013; DeVos et al., 2013; Li et al., 2014).

Near Field Communication

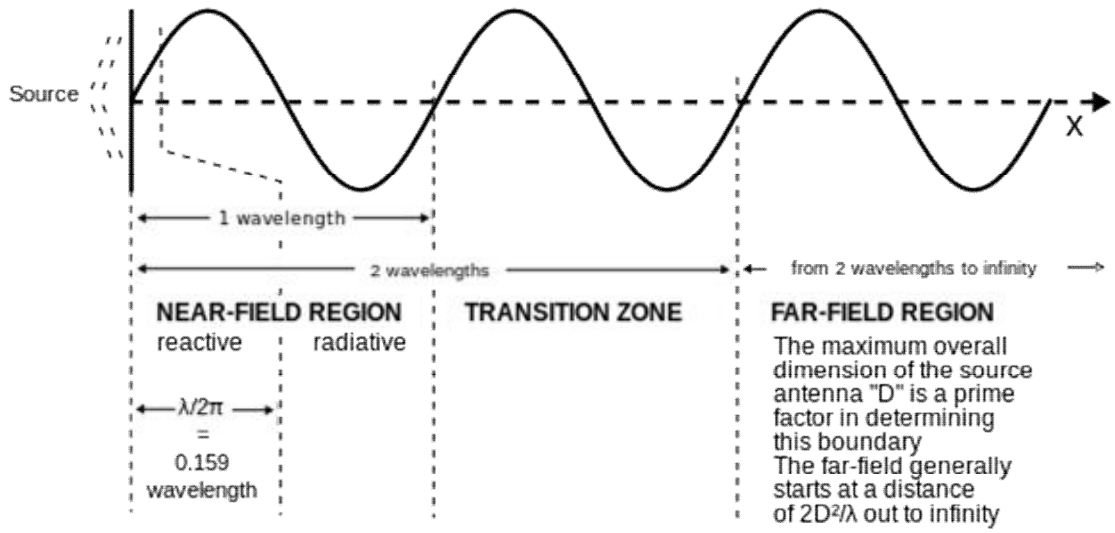

Figure 3. Classification of regions based on their distances from the transmitting antenna. The nearest region, within 0.159 wavelength, is called the reactive region which contains both radiative and non-radiative components. The entire brain is within the reactive region, where the non-radiative component is the extracellular electric fields that mediate the ephaptic coupling. [Image source: US Department of Labor]

The electric and magnetic fields near a transmitting antenna are very complex. This "reactive region" consists of not only the radiative EM fields, but also static electric and magnetic fields. For instance, the electrons moving along a metal antenna will generate non-radiative magnetic fields. If the antenna is a coil, rather than a straight wire, the magnetic field will be produced at the center of the coil, which may induce currents in a nearby coil (receiving antenna) through Faraday induction. Such "near field communication" has been widely used in payment systems and access to restricted areas. It may also be applied to wireless power transfer (Kurs et al., 2007).

From the viewpoint of antenna communication, the ephaptic coupling via extracellular electric fields is a special case of near field communication. The reactive region covers the distance of 0.159 wavelength (λ) from the transmitting antenna. The carrier frequency used in the brain is estimated to be about 10 MHz, corresponding to a wavelength of 30 m. Thus, the entire brain is within the reactive zone.

While static magnetic fields can be generated by the ionic current through channels, they will be canceled out by both inward and outward transmembrane currents. Hence, the magnetic fields measured by magnetoencephalography (MEG) originate mainly from the ionic currents along the axon and dendrites. These magnetic fields do not seem to play an active role in brain functions since the microtubules react predominantly to electric fields.

The amplitude of extracellular electric fields may be as large as 6 V/m (Anastassiou et al., 2011), capable of modulating microtubule antennas to influence neural activities. The extracellular fields have been shown to induce changes in the somatic membrane potential of cortical neurons (Anastassiou et al., 2011). Although they are insufficient to elicit action

potentials from the resting membrane potential, they could represent the physical "memory traces" at the time scale of seconds, as local field potential is correlated with working memory (Bigelow et al., 2016; Huang et al., 2016).

Long Range Synchronization

A brain consists of many functionally specialized areas. Synchronization among relevant areas is critical for efficient performance of a specific task. For instance, in the awake cat, a sudden change of a visual pattern induces synchronization between areas of the visual and parietal cortex, and between areas of the parietal and motor cortex. Despite the long distance between synchronized areas, the synchronization occurs with zero phase lag (Roelfsema et al., 1997), which has also been demonstrated in (1) hippocampal-prefrontal synchrony during working memory (Harris and Gordon, 2015), (2) synchronization between visual area V4 and lateral prefrontal cortex during visual memory (Liebe et al., 2012), (3) synchronization of activity and silence in neocortical neurons during slow-wave oscillations (Volgushev et al., 2006), and (4) synchronization in lateral geniculate nucleus of both hemispheres during visual processing (Neuenschwander and Singer, 1996).

The zero lag synchronization between distant areas is remarkable, considering that synaptic transmission and axon conduction will cause time delay. The wireless communication, which acts instantaneously, may solve the problem of time delay. However, the wireless stimuli, either extracellular fields or EM waves, are weak. They cannot excite a neuron from the resting membrane potential. Hence, although the electric fields and EM waves may reach the entire brain, they will affect only the neurons that are already oscillating near the threshold. Furthermore, both wireless stimuli may modulate neuronal excitability by "shaking off" the microtubule antenna from the AIS membrane. Therefore, the effects of wireless stimuli also depend on the binding between the microtubule antenna with the membrane. The highly sensitive hippocampal CA1 and CA3 neurons to the external fields (Francis et al., 2003; Reato et al., 2010) indicates that their microtubule antennas are loosely bound with the membrane.

Two Brains, One Percept

The case of Tatiana and Krista Hogan could be a very rare example of ephaptic coupling **between two brains**. The twin girls were born in 2006, joined at the skull and brain - a medical condition called "conjoined craniopagus twins". Miraculously, the twins are still alive today. Other than the joined heads, they are healthy and happy. The twins have individual brains, each consisting of all structures that a normal brain has. What makes the twins unique is that they often have the same conscious perception.

When a girl was watching television, another girl, while her vision was angled away from the television, could still perceive and laugh at the images before her sister's eyes. The tickle of one girl's foot was immediately perceived by another girl. Their doctor, Douglas Cochrane of British Columbia Children's Hospital, interpreted this as the existence of a "thalamic bridge" linking the thalamus of one girl to the thalamus of her sister, on the basis of brain images. However, according to a New York Times article, "Brain imaging is inscrutable enough that numerous neuroscientists, after seeing only one image of hundreds, were reluctant to confirm the specific neuroanatomy that Cochrane described".

The conscious perception arises from the large scale neural synchrony (Lee, 2016). In the case of Tatiana and Krista Hogan, the extracellular electric fields resulting from ionic processes may propagate from one brain to the other, facilitating synchronization between two brains. With the ephaptic coupling and radiative EM coupling, the two girls may perceive the same thing without any wired connection between their thalami.

Discussion

The instantaneous action of wireless communication offers an easy solution to zero-lag long range synchronization. It also provides an economical method to link distant brain areas. In the case of Tatiana and Krista Hogan, the two joined brains could be linked by wireless communication. This paper has described how the brain may transfer information without going through synaptic transmission. The proposed mechanism is based on experimental findings, such as the unique microtubule structure at AIS and the effect of Tau proteins on excitability. However, future experiments are still necessary for direct proof.

Wireless communication in the brain can be divided into two categories: the ephaptic coupling via non-radiative extracellular electric fields and the EM coupling via EM waves radiated from accelerated ions in the ion channels. The ephaptic coupling has been well documented. The EM coupling involves transmitting and receiving components. As described above, the receiving component is located at AIS where microtubules may serve as the receiving antennas for the transformation of the absorbed EM energy into neuronal excitability. Experiments have shown that the radio waves emitted from mobile phones can affect excitability and synchronized EEG activity (Valentini et al., 2007; Roggeveen et al., 2015a; Roggeveen et al., 2015b). The radiation power from mobile phones is too weak to induce thermal effects. Its specific effects on excitability and EEG activity supports the notion that the brain is capable of converting the received EM waves into excitability, thereby modulating synchronization. However, direct proof for the brain-emitted EM waves is still lacking.

The EM frequency used by the brain is estimated to be on the order of 10 MHz, which falls in the range of radio waves. Broadcasting stations are regularly transmitting radio waves at this frequency. Such environmental noises may exceed the signals from the brain. In that case, a well-shielded Faraday cage might be necessary. The power generated by the brain is predicted to be around 0.01 W. Although the power outside the brain will be attenuated by brain tissues and the skull, it could still be detectable.

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