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Neuroscience and its Applications

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Letter to Editor

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Abstract

Neuroscience is a subject matter which is multidisciplinary and not only combines anatomy, physiology, molecular biology, cytology, developmental biology, computer science but also mathematical modeling. The scope and subject matter of neuroscience and its applications has extended over the time to include various approaches to study nervous system at different scales. Many methods and measures used by neuroscientists has been extended from molecular and cellular studies of individual neurons to imaging of sensory, motor, and cognitive tasks in the brain.

Keywords: Neuroscience; Biology; Sensory; Imaging

Abbreviation: STIMO: Stimulation Movement Overground.

Stem Cells Repair Parkinson's Damaged Cells

Developed brain is notoriously bad at repairing itself after damage caused by trauma or strokes or from degenerative illness such as Parkinson's.

In new research at University of Wisconsin – Madison, scientists demonstrated concept of stem cell treatment model of Parkinson's disease. Stem cells which are adaptable for better natural repair. They discovered that neurons that are derivative from stem cells can integrate well into the correct regions of the brain also it can able to link with native neurons and restore motor functions. The key is identity. By meticulously trailing the transplanted stem cells, researchers discovered that cell's identity is "dopamine" producing cells in the case of Parkinson's; defined the connections they made and how they performed.

To renew the circuits in the Parkinson's disease model, scientists began by coaxing human embryonic stem cells to differentiate into "dopamine" producing neurons – the cells that die in Parkinson's. Scientists transplanted these new modified neurons into brains of mice, the brain region most affected by Parkinson's degeneration. Several months later after new neurons integrated into the brain, results showed that it had improved motor skills. The nerve cells also established connections with regulatory regions of the brain that fed in the new neurons and prevented from being overstimulated [1,2].

Microbes Hold the Key for Treating Neurological Disorders

Researchers at Baylor College of Medicine propose a new remedy for treating neurological disorders. Microbes in the gut may contribute to specific symptoms associated

with complex neurological disorders. Their discoveries also suggest that microbe inspired therapies may one day help to treat them.

Scientist and Researchers discovered that mouse models for neurodevelopmental disorders; hyperactivity is controlled by the host's genetics, whereas social behavior deficits are mediated by the gut microbes. One of the most significant discovery from their research for treating neurological disorder is that from a therapeutic perspective, they found that treatment with a specific microbe that helps the production of compounds that is associated to biopterin family in the gut or treating with metabolically active biopterin molecule shows improved social behavior but not in motor activity for treating neurological disorders. The work contributed by the scientists offers diverse way of thinking about neurological disorders in which human and microbial genes relate with each other and contribute to the condition. Their discovery also proposes that effective treatments would likely require to be directed to the brain and the gut to fully address all symptoms. Additionally, they also open possibility to other complex conditions for instance viral infection, diabetes, cancer, or other neurological disorders that may have microbiome component [3,4].

Treating Paralysis Through Neurotechnology

In this research of treating Paralysis patients, scientists used a new technique called STIMO (STImulation Movement Overground) which creates a new therapeutic framework structure to improve recovery from spinal cord injury. All patients who had paralysis participated in this learning process recovered; voluntary control of leg muscles that was paralyzed for many years. "Using this method most of the patients effected by paralysis were able to walk using body weight support within one week." was confirmed by neurosurgeon.

This research accomplishes an unprecedented level of precision in electrically stimulating spinal cords for treating paralysis through neurotechnology. In this technique they implanted an arrangement of electrodes over the spinal cord which allows to aim individual muscle groups in the legs. Using this method of treating paralysis through neurotechnology, selected specific configurations of electrodes activate specific regions of spinal cord and mimics the signals that brain would deliver to produce walking [5,6].

Treating Alzheimer's with Low Dosage of Aspirin

A low dose aspirin treatment may signify a new avenue for reducing Alzheimer's disease. This new study identifies a new role for one of the most widely used medications in the world.

Although, exact cause of Alzheimer's disease progression is unknown, it is assumed that impaired clearance of toxic amyloid beta especially from the hippocampus is a leading mechanism. Activating the cellular machinery is responsible for removing waste from the brain has consequently emerged as a promising strategy for slowing the disease. Based on studies illustrating a relation between aspirin reduced risk and prevalence of Alzheimer's disease. Scientists demonstrated that common over the counter medication decreases amyloid plaque pathology by simulating lysosomes – one of the components of cells that aids to clear cellular debris. The study also adds to aspirin's established uses for pain relief and for the treatment of cardiovascular diseases [7,8].

Treatment that Eliminates Parkinson Disease and Generates New Neurons

Scientists at University of California San Diego School of Medicine studied a protein called PTB which is well known for binding RNA and influencing which genes are turned "on" or "off" in a cell. To comprehend the role of a protein like PTB, scientists often manipulate cells to reduce the amount of that protein.

Scientists in the lab made a discovery by applying it what could one day be a new therapeutic approach for Parkinson's diseases and other neurodegenerative diseases (Figures 1-9). A single treatment was sufficient to inhibit PTB in mice converted native astrocytes, star shaped support cells of the brain into neurons that produce the neurotransmitter dopamine. As a result, Parkinson's diseases symptoms disappeared [9,10].

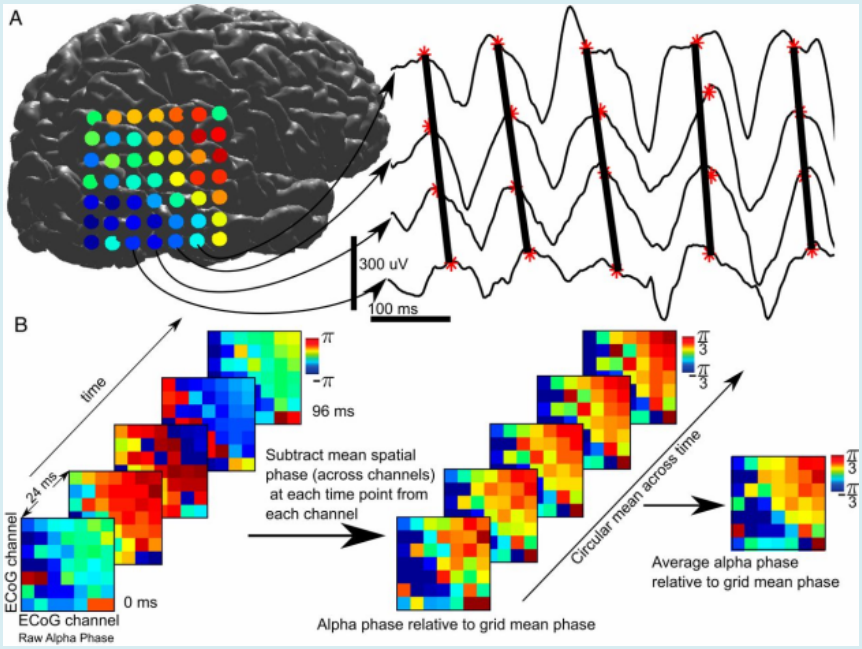

Figure 1: Figure Illustrates Analysis Stream for Ecog Traveling Waves. (A) Alpha Propagates as A Traveling Wave in the Raw Broadband Data. (B) Analysis Stream for Visualizing Traveling Waves in Ecog. Started with Raw Alpha Phase of the Grid over Time. Then, for Each Time Point, Circular Distance (Distance on the Unit Circle) between Each Contact and the Grid's Mean Phase (Across all Contacts) at that Time Point. Lastly, Circular Mean of this Difference to Get Each Contact's Average Phase Advance or Delay [11].

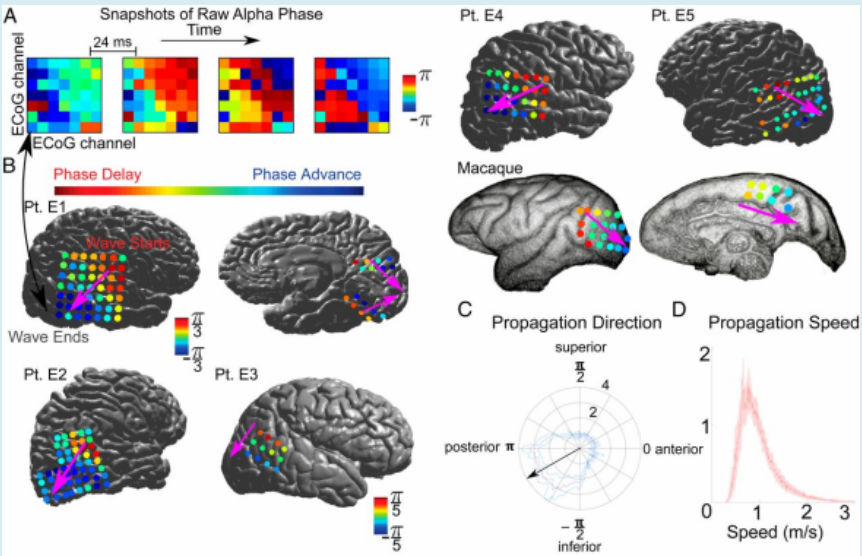

Figure 2: Figure illustrates Alpha propagates from anterosuperior to posteroinferior cortex. (A) Alpha-phase snapshots from Pt. L1 demonstrate propagation from the grid's top-right (anterosuperior) to bottom-left corner (posteroinferior). (B) Average circular distance of each contact's alpha phase from the spatial mean phase during eye closure. In all patients, alpha propagates toward posteroinferior areas. Overlaid arrow is the direction of the grid's average phase gradient. Color runs from ±π3 in Pts. E1, E2, and E5 and macaque; and from ±π5 in Pts. E3 and E4. (C) Average probability distribution of traveling wave directions across time such that the bottom left contact is the most posteroinferior (±SEM across patients). (D) Average probability distribution of traveling wave speeds (±SEM across patients) [11].

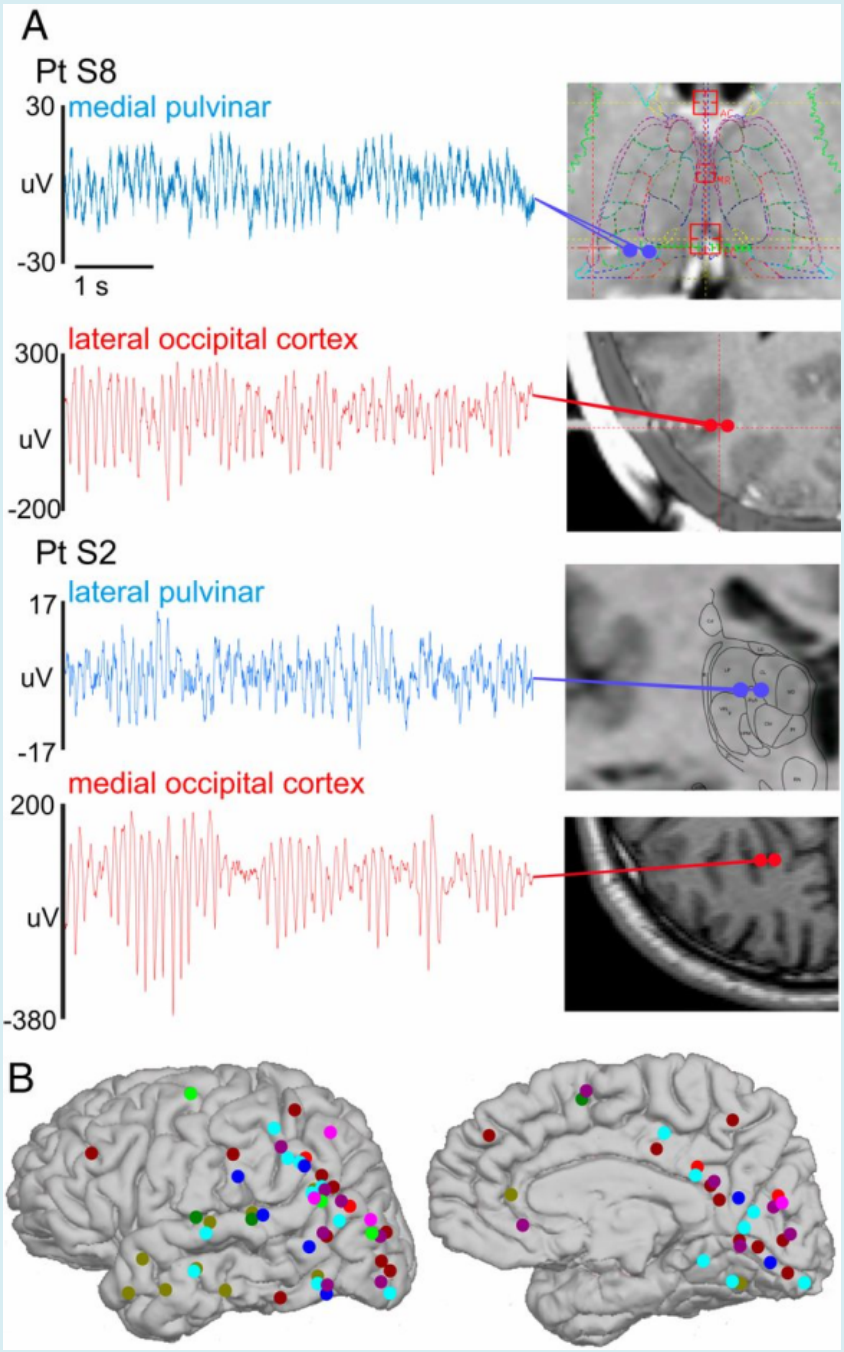

Figure 3: Figure illustrates Robust alpha rhythms can be recorded in human pulvinar and cortex. (A) Representative 6-s LFPg traces of simultaneous thalamic and cortical alpha activity. Prominent, largely continuous alpha rhythms can be recorded in various locations within the pulvinar as well as posterior cortex. (B) Cortical implant locations in all SEEG patients displayed on Pt. S3's brain. Each color signifies a different patient [11].

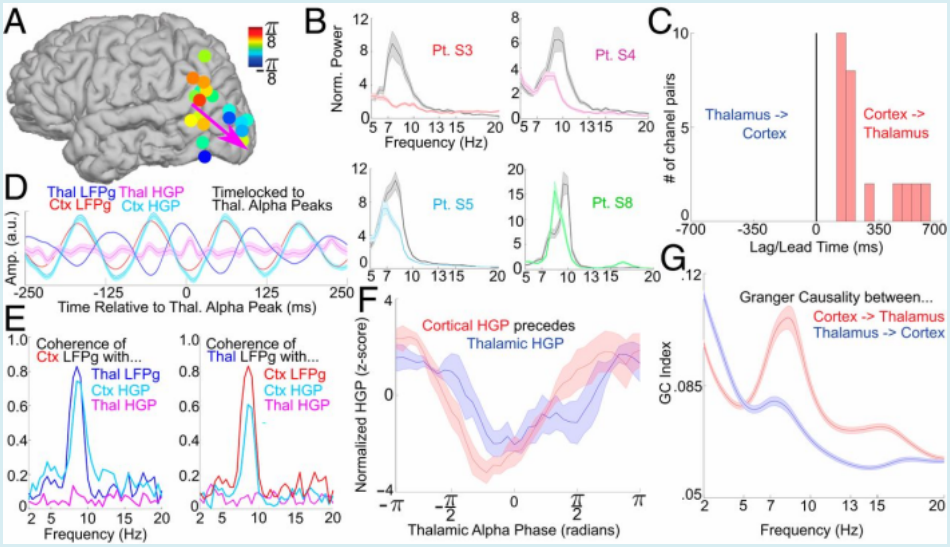

Figure 4: Figure illustrates Cortical alpha leads thalamic (pulvinar) alpha. (A) Average alpha-phase lag/leads in bipolar contacts (n = 5). Note that anterosuperior channels lead inferoposterior ones, in accord with our ECoG recordings. (B) Power spectra of the thalamic (color) and cortical (gray) channel with the greatest alpha power. (C) The difference in start times between all cortical and thalamic alpha bursts (start time in thalamus to start time in cortex) in the 28 channel pairs with a significant thalamic or cortical lead ($P < 0.05$, Bonferroni corrected, binomial test). Alpha bursts start (on average) in cortex for all 28 channel pairs. (D) Cortical and thalamic LFPg and HGP from representative channels locked to peaks in thalamic alpha LFPg—cortical, but not thalamic, HGP is phasic with thalamic LFPg. (E) Coherence spectra of thalamic and cortical LFPg with thalamic and cortical HGP and LFPg from the same channel pair in D; the coherence of thalamic LFPg with cortical HGP (but not thalamic HGP) suggests that the cortex may drive thalamic alpha activity. (F) Normalized thalamic and cortical HGP at different thalamic alpha phases averaged across channels; note that cortical HGP slightly leads thalamic HGP. (G) GC spectra averaged over all thalamocortical contact pairs; corticothalamic causality shows a strong alpha peak. Amp., amplitude; a.u., arbitrary units; ctx, cortical; norm., normalized; thal, thalamic [11].

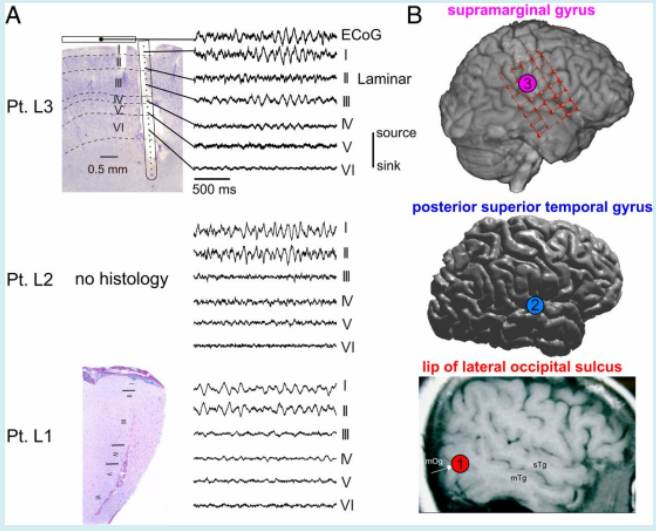

Figure 5: Figure illustrates Laminar recordings of cortical alpha. (A) Nissl stains of the explanted tissue surrounding the laminar probe in Pts. L1 and L3, in addition to representative laminar CSD traces from each layer in each subject. Note that despite being made in distinct cortical locations, alpha oscillations were always strongest in layer I/II. Furthermore, in L3, the trace of a simultaneously recorded overlying ECoG contact is near identical to the underlying laminar layer I. (B) Locations of each laminar probe in all patients [11].

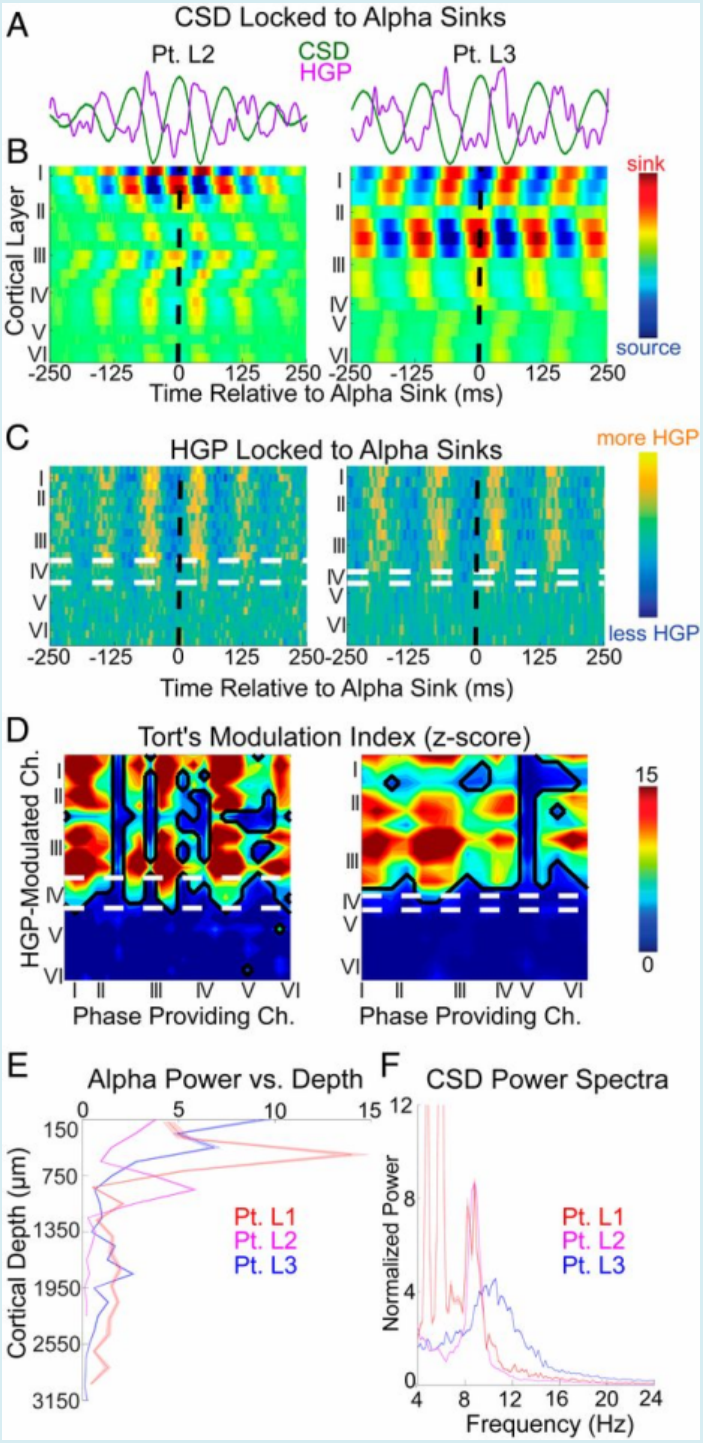

Figure 6: Figure illustrates Alpha CSD and HGP are maximal in supragranular cortex. (A) Average CSD and HGP waveforms of a single channel on the same time axes as B (±SEM across alpha sinks). (B and C) CSD (B) and HGP (C) averaged on current sinks in channels 3 and 6 in Pts. L2 and L3, respectively; white and black dashed lines indicate layer IV boundaries and the time of the alpha sink, respectively. (D) Z score of the MI between alpha phase and HGP across all channels (Ch.). (E) Average alpha power throughout the cortical depth (±SEM across epochs). (F) Power spectra of the channel with greatest alpha power in each subject (±SEM across epochs) [11].

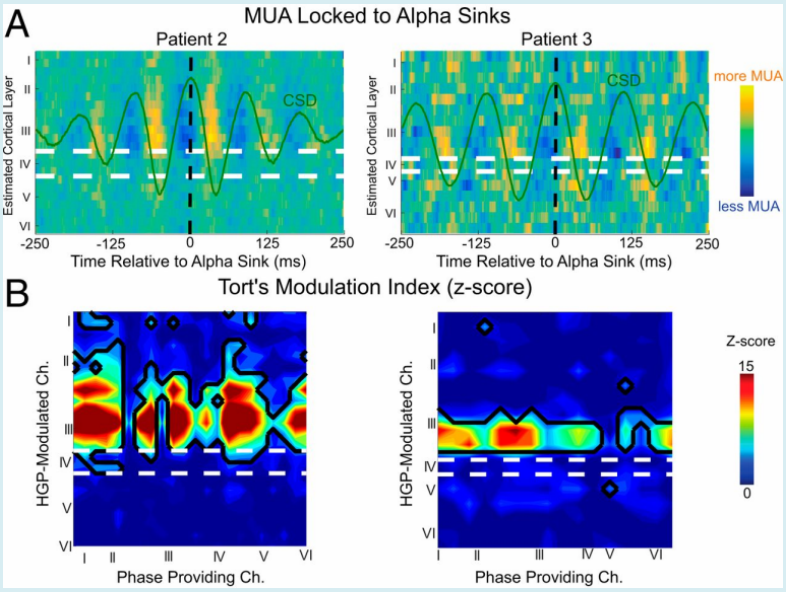

Figure 7: Figure illustrates MUA is modulated in layer III. (A) MUA averaged on current sinks on the channels with the greatest alpha power (channels 3 and 6, respectively), which is most clearly modulated within lower layer III. (B) Tort's MI between alpha CSD phase and MUA amplitude over all laminar contact pairs—note that firing is correlated with alpha phase in both superficial and deep cortex. Ch., channel [11].

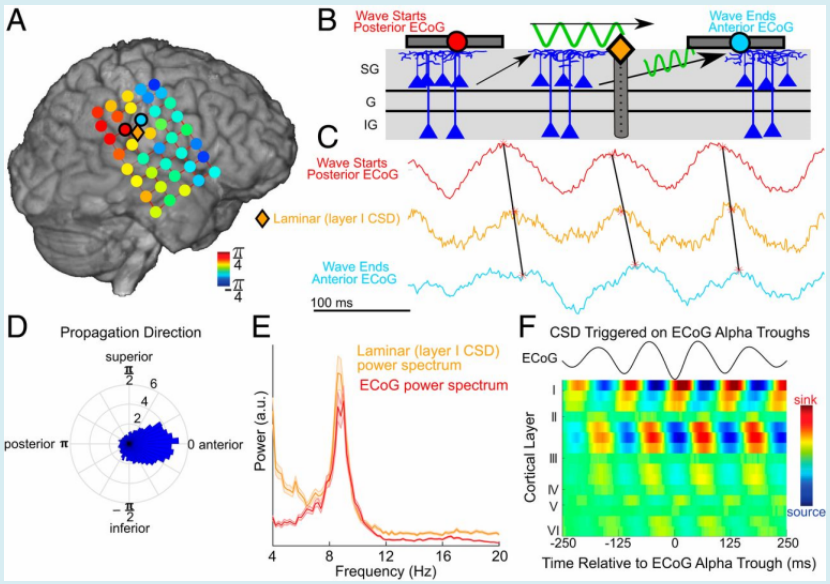

Figure 8: Figure illustrates Simultaneous ECoG–laminar recordings reveal traveling alpha waves which propagate through supragranular cortex. (A) Average circular distance of each ECoG (circles) and layer I laminar (diamond) contact's alpha phase from the spatial mean phase throughout the ECoG grid. Note that the laminar's alpha phase is intermediate to neighboring ECoG contacts, suggesting that ECoG and the laminar probe recording the same traveling wave at different scales. (B) Representative drawing of a traveling alpha wave (as measured with ECoG) propagating through superficial layers (as measured by a laminar probe). (C) Example traces from ECoG contacts posterior (red) and anterior (blue) to the laminar probe. Alpha phase in the laminar is intermediary to the ECoG contacts. (D) Distribution of traveling wave directions; mu waves propagate from posterior (higher-order) toward anterior (lower-order) cortex. (E) Power spectra from simultaneous laminar and ECoG recordings; they share a near-identical alpha peak. (F) Laminar CSD averaged on troughs in the nearest ECoG contact. Note that alpha activity is superficial [11].

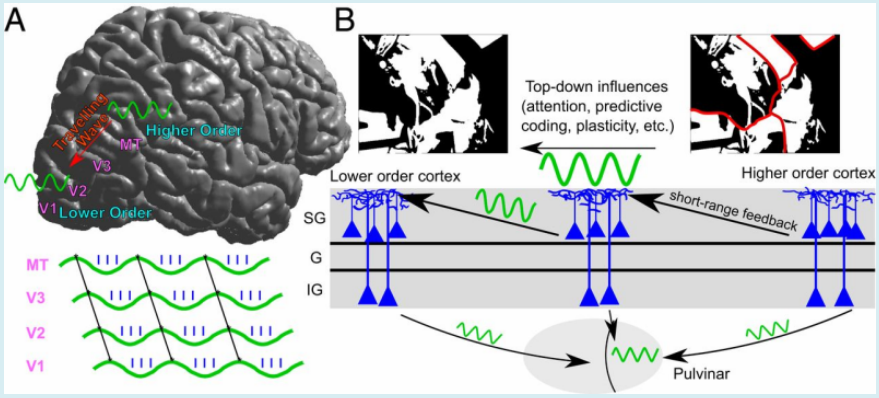

Figure 9: Tentative model for how alpha's physiology could mediate feedback. (A) Alpha propagates as a traveling wave from higher-order (middle temporal, visual area 3) toward lower-order visual areas 1/2 cortical areas. (B) Alpha is strongest within supragranular cortex and may carry top-down information via short-range feedback connections to constrain lower-level processing; for instance, alpha may play a role in resolving ambiguous visual imagery, such as the picture of a woman and a horse's snout shown above. Cortical alpha in layer VI might influence alpha activity within the pulvinar [11].

References

1. Xiong M, Tao Y, Gao Q, Feng B, Yan W, et al. (2021) Human Stem Cell-Derived Neurons Repair Circuits and Restore Neural Function. Cell Stem Cell 28(1): 112-126.

2. Hamilton E (2020) Stem cells can repair Parkinson's-damaged circuits in mouse brains. University of Wisconsin-Madison.

3. Buffington SA, Dooling SW, Sgritta M, Noecker C, Murillo OD, et al. (2021) Dissecting the contribution of host genetics and the microbiome in complex behaviors. Cell 184(7): 1740-1756.

4. Baylor College of Medicine (2021) Microbes may hold the key for treating neurological disorders.

5. Wagner FB, Mignardot JB, Le Goff-Mignardot CG, Demesmaeker R, Komi S, et al. (2018) Targeted neurotechnology restores walking in humans with spinal cord injury. Nature 563 (7729): 65-71.

6. Sanctuary H, Barraud E (2018) Ecole Polytechnique Fédérale de Lausanne Breakthrough in treating paralysis: Targeted neurotechnology restores walking in humans with spinal cord injury.

7. Chandra S, Jana M, Pahan K (2018) Aspirin induces Lysosomal biogenesis and attenuates Amyloid plaque pathology in a mouse model of Alzheimer's disease via PPARα. The Journal of Neuroscience 38(30): 6682-6699.

8. Society for Neuroscience (2018) Treating Alzheimer's with aspirin: Low-dose aspirin regimen could reduce Alzheimer's disease pathology.

9. Qian H, Kang X, Hu J, Zhang D, Liang Z, et al. (2020) Reversing a model of Parkinson's disease with in situ converted nigral neurons. Nature 582(7813): 550-556.

10. Buschman H (2020) One-time treatment generates new neurons, eliminates Parkinson's disease in mice: Inhibiting a single gene converts many cell types directly into dopamine-producing neurons. University of California-San Diego.

11. Halgren M, Ulbert I, Bastuji H, Fabó D, Erőss L, et al. (2019) The generation and propagation of the human Alpha rhythm. Proceedings of the National Academy of Sciences of the United States of America 116(47): 23772-23782.