

Visual Snow Syndrome and Its Relationship to Tinnitus

Matthew Renze

International Conference on the Management
of the Tinnitus and Hyperacusis Patient

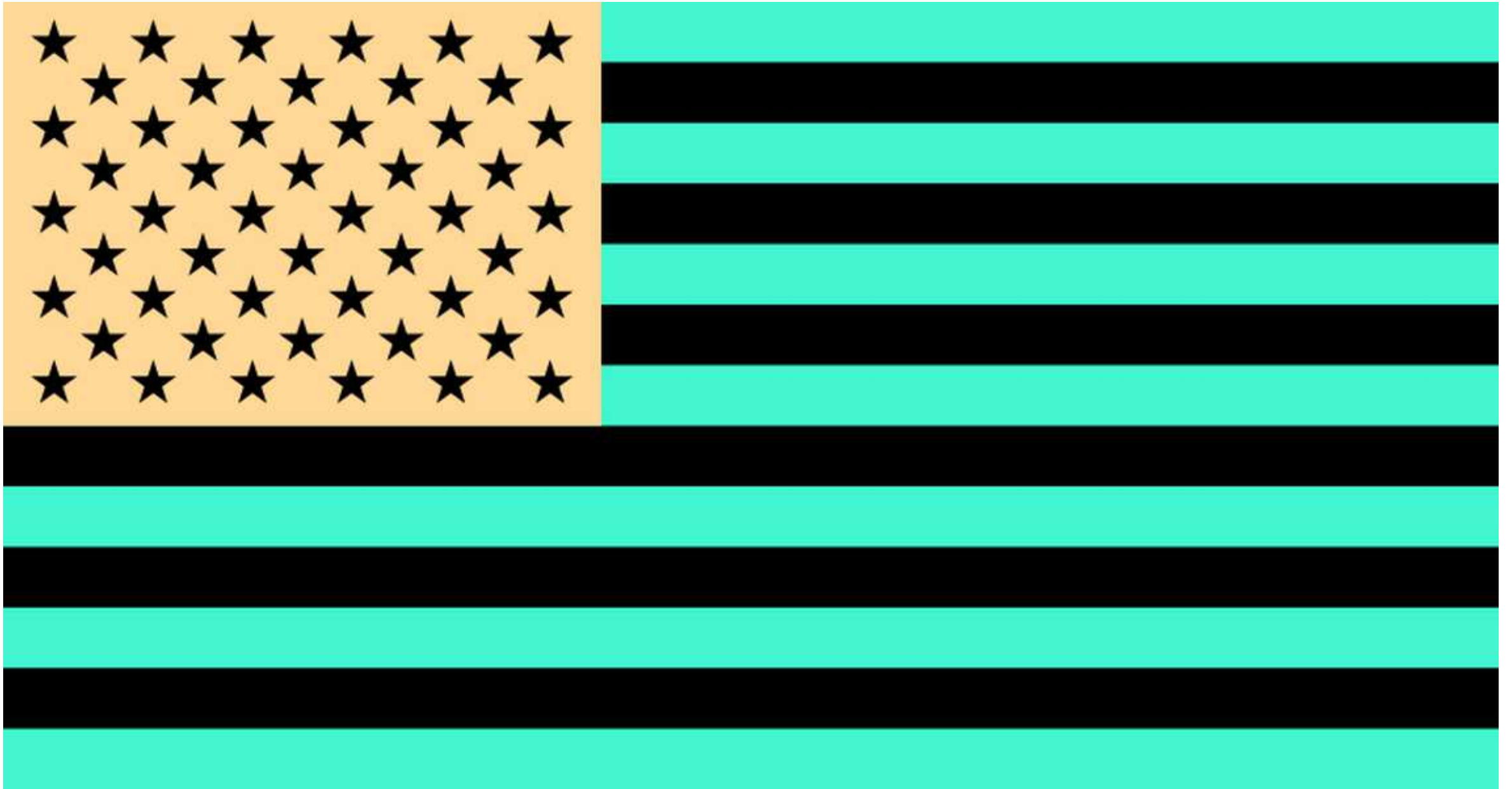
February 7, 2014











Other Visual Symptoms

Vibration in text

Trailing images

Bright-light issues

Night-vision issues

Halos at night



Other Auditory Symptoms

Loud-noise issues

Conversation issues

Environmental-noise issues

Ear noise with volume changes



Other Tactile Symptoms

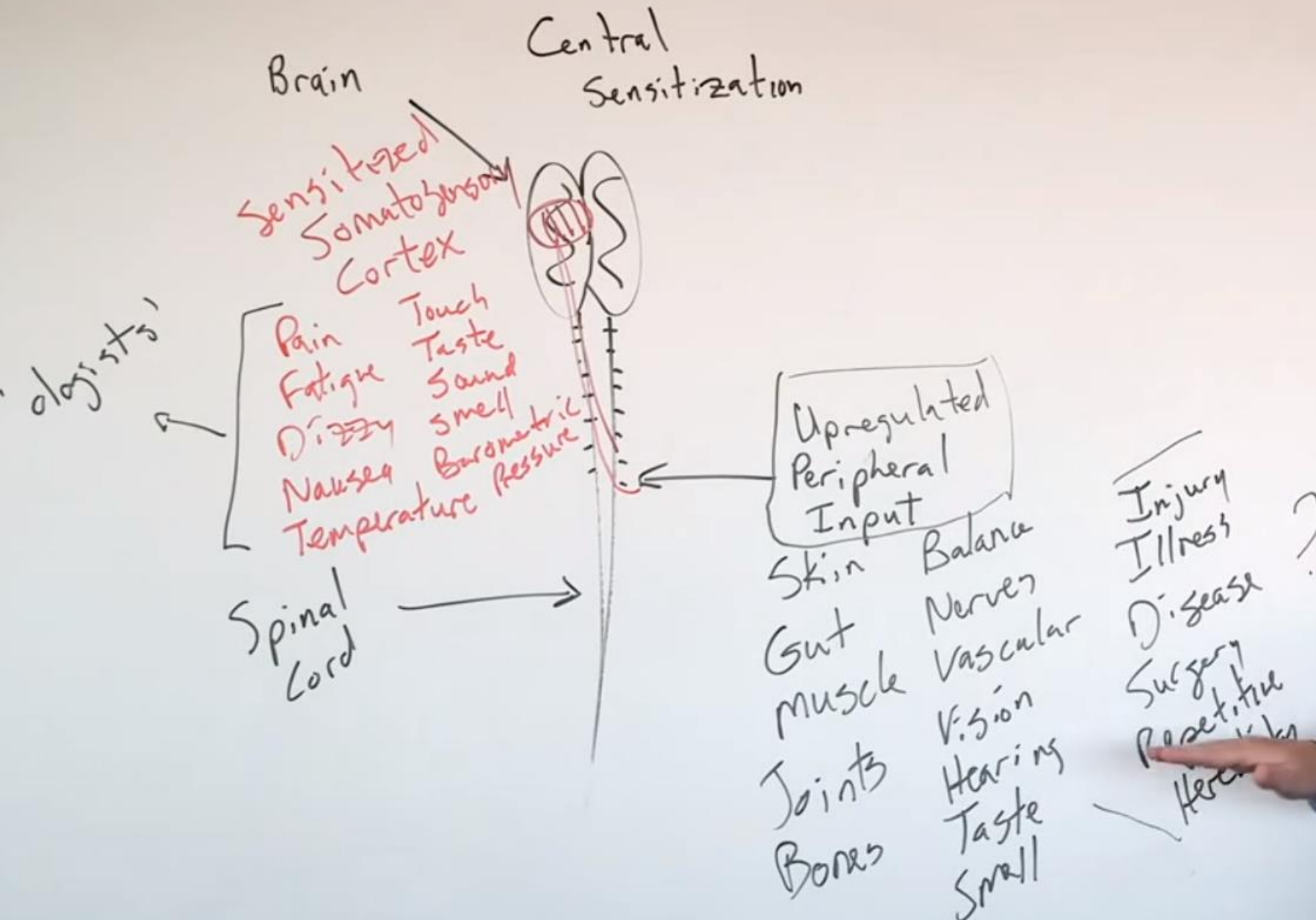
Pulsating buzzing

Fine tremors











‘Visual snow’ – a disorder distinct from persistent migraine aura

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Visual Snow

“continuous tiny dots in the entire visual field similar to noise of an analog television”



Visual Snow Syndrome

Palinopsia

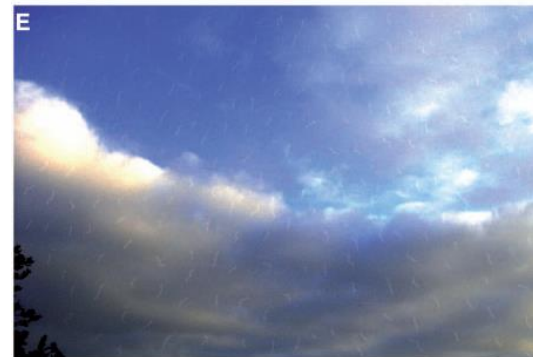
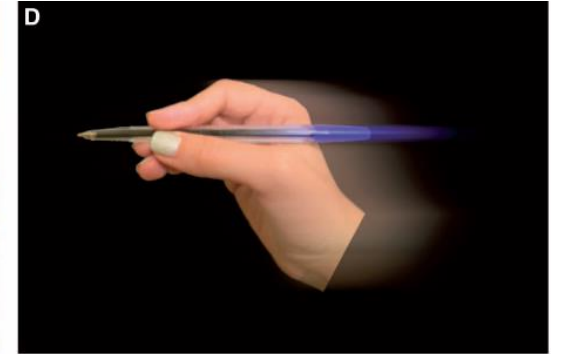
Floaters

Blue-field entoptic phenomena

Photophobia

Nyctalopia

Tinnitus

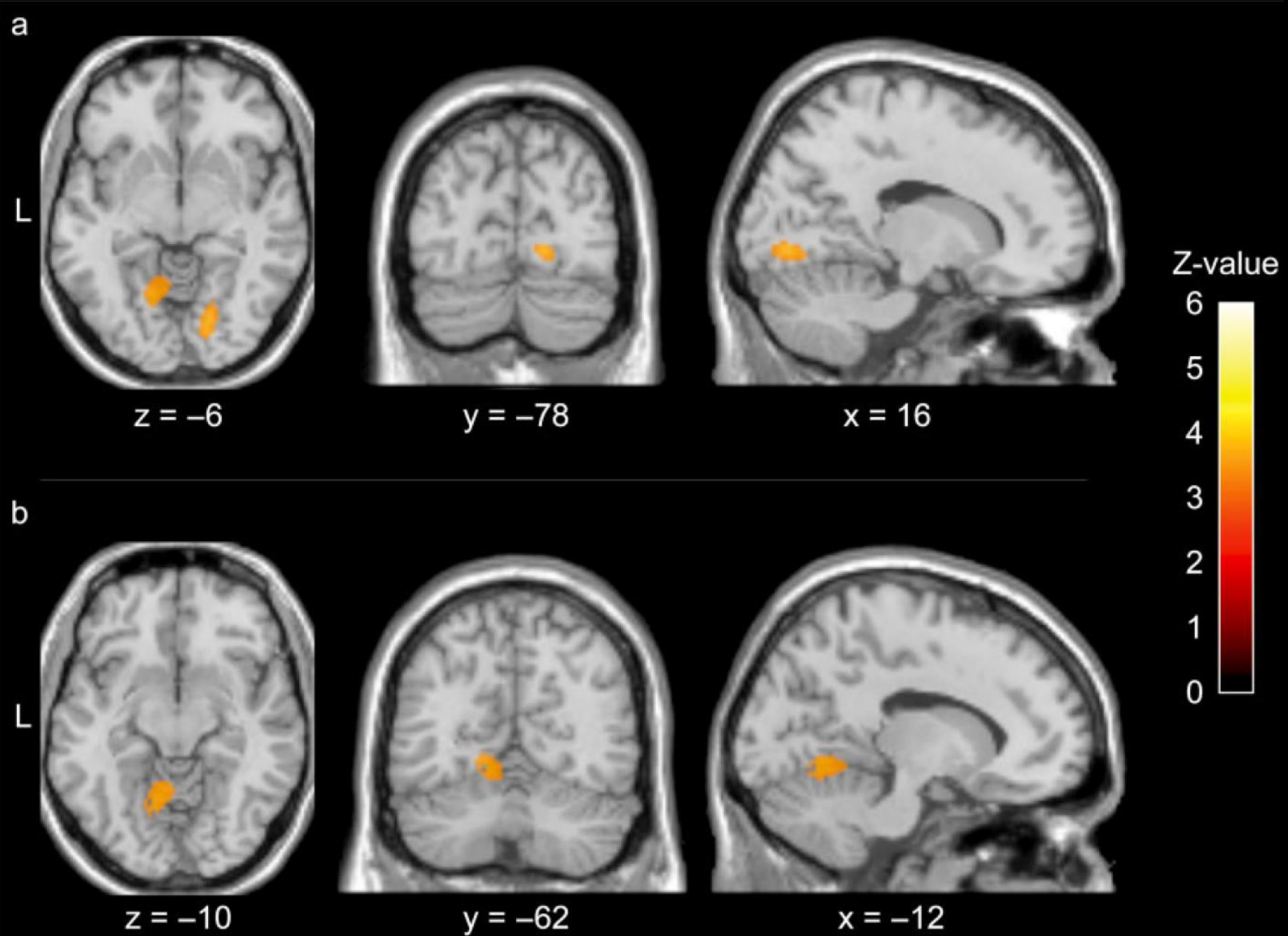


2014 Wolff Award Paper

The Relation Between Migraine, Typical Migraine Aura and “Visual Snow”

Christoph J. Schankin, MD; Farooq H. Maniyar, MD; Till Sprenger, MD; Denise E. Chou, MD;
Michael Eller, MD; Peter J. Goadsby, MD, PhD

Objective.—To assess the relationship between the phenotype of the “visual snow” syndrome, comorbid migraine, and typical migraine aura on a clinical basis and using functional brain imaging.



What does this have to do with tinnitus?

Associated Visual Snow Symptoms

Symptom	Study 1	Study 2
Palinopsia (Afterimages)	86%	84%
Floaters	81%	58%
Blue-field Entoptic Phenomena	79%	76%
Photophobia	74%	72%
Nyctalopia	68%	63%
Spontaneous Photopsia	63%	53%
Tinnitus	62%	64%

Neurologic Ophthalmology and Otology (RK Shin and DR Gold, Section Editors)

Visual Snow: a Potential Cortical Hyperexcitability Syndrome

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Clinical Study

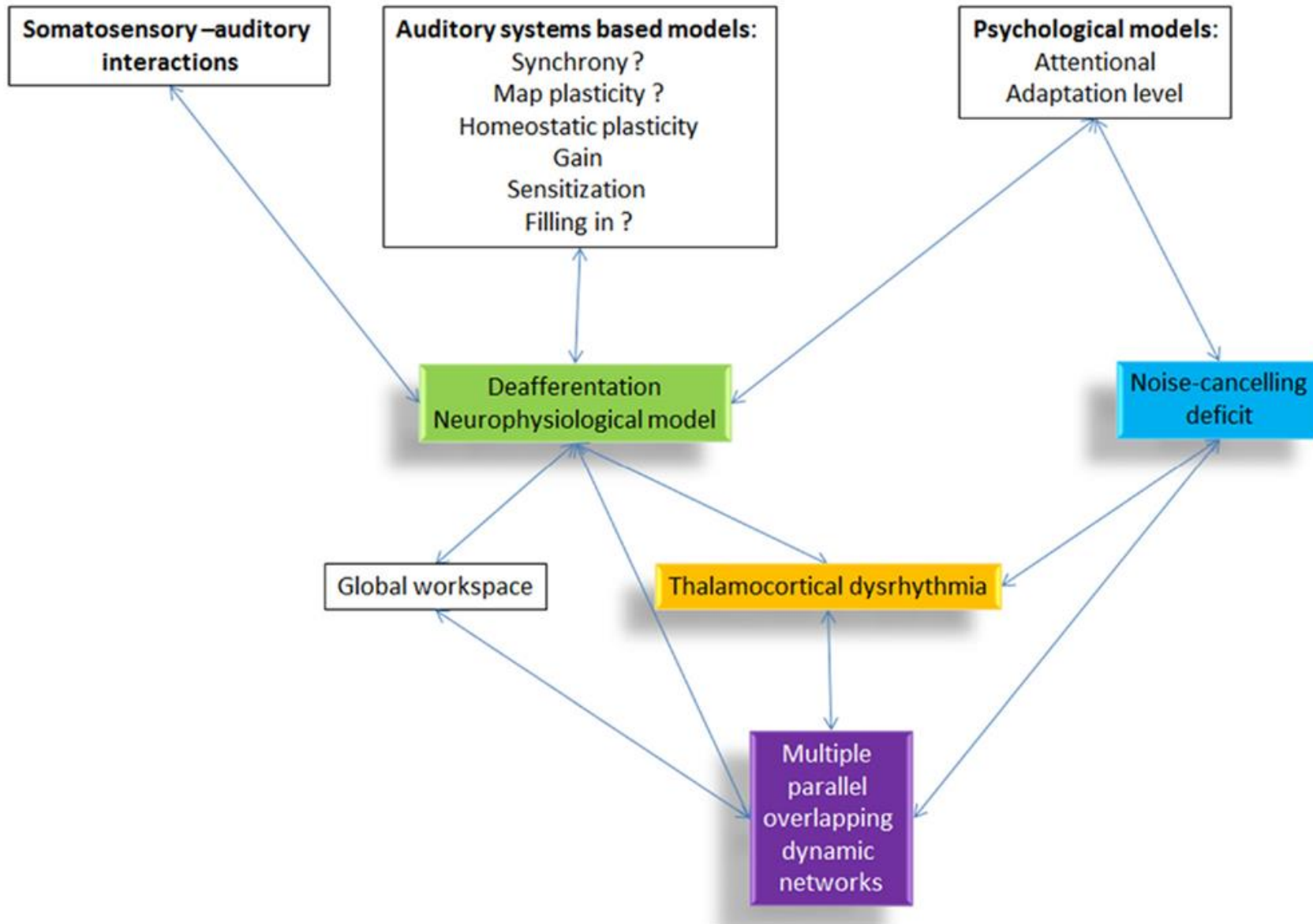
Visual snow: A thalamocortical dysrhythmia of the visual pathway?

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Opinion

An Integrative Tinnitus Model Based on Sensory Precision

William Sedley,^{1,*} Karl J. Friston,² Phillip E. Gander,³
Sukhbinder Kumar,² and Timothy D. Griffiths^{1,2,3}

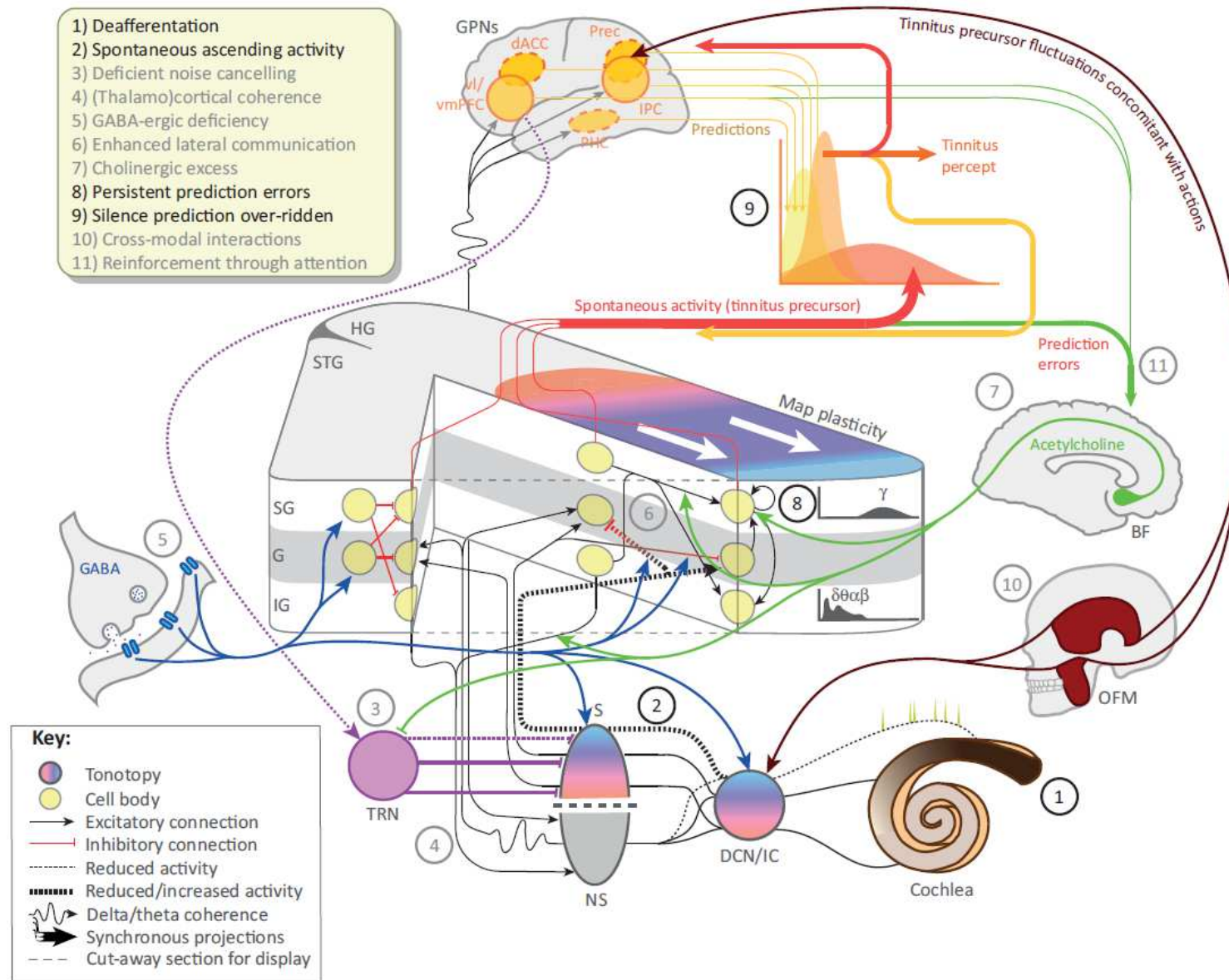
Tinnitus is a common disorder that often complicates hearing loss. Its mechanisms are incompletely understood. Current theories proposing pathophysiology from the ear to the cortex cannot individually – or collectively – explain the range of experimental evidence available. We propose a new framework, based on predictive coding, in which spontaneous activity in the subcortical auditory pathway constitutes a ‘tinnitus precursor’ which is normally ignored as imprecise evidence against the prevailing percept of ‘silence’. Extant models feature as contributory mechanisms acting to increase either the intensity of the precursor or its precision. If precision (i.e., postsynaptic gain) rises sufficiently then tinnitus is perceived. Perpetuation arises through focused attention, which further increases the precision of the precursor, and resetting of the default prediction to expect tinnitus.

Trends

Existing tinnitus models, including mutually exclusive mechanisms, invoke causes from the ear to high-level cortical brain networks.

The generic framework of predictive coding explains perception as the integration of sensory information and prior predictions, each weighted by its precision.

In our model, previously proposed neural correlates of ‘tinnitus’ largely relate to hearing damage, rather than to tinnitus *per se*, and reflect an increase in the precision of sponta-





Visual Snow: a Potential Cortical Hyperexcitability Syndrome

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This article is part of the Topical Collection on Neurologic Ophthalmology and Otology

Keywords Visual snow · Cortical hyperexcitability · Migraine visual aura · Persistent visual phenomena of migraine

Opinion statement

The purpose of this review is to provide an overview of visual snow (VS) and provide information regarding current treatment options for VS. Visual snow (VS) is a rare disorder manifesting with a persistent visual phenomenon of seeing numerous tiny snow-like dots throughout the visual field, and it can cause debilitating visual and psychological consequences. It is emerging as a disorder separate from, but associated with, migraine visual aura, and neuronal cortical hyperexcitability is being considered as a theoretical mechanism for the persistent-positive visual symptoms. There are few studies that have investigated the treatment of VS, but as our understanding of this entity begins to change, we expect that new treatment approaches and treatment trials will emerge in the next decade. Currently, our approach is to consider pharmacologic treatment for all patients with VS who report decreased quality of life as a result of VS. Resolution of the disorder is difficult to accomplish with treatment, but in our experience, even when symptom intensity is simply reduced, many patients find that there is an improvement in their quality of life that is beneficial. Our preferred treatment options include: (1) oral lamotrigine with a slow increase from 25 mg daily to a maintenance dose of 200–300 mg daily in divided doses as tolerated, and this is typically achieved by advancing the dose in increments of 25–50 mg weekly following the first 2 weeks of therapy; (2) oral acetazolamide with an initial dose of 250 mg daily followed by a slow increase over 1–2 weeks to a total of 1000 mg daily in divided doses, and higher doses can be tolerated by some without increasing the risk-benefit ratio; or (3) oral verapamil long-acting at 120–240 mg daily, and if side effects limit the dose the can be initiated, then lower doses with short-acting verapamil two or three times daily

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Clinical Study

Visual snow: A thalamocortical dysrhythmia of the visual pathway?

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Visual snow

ABSTRACT

In this paper we review the visual snow (VS) characteristics of a case cohort of 32 patients. History of symptoms and associated co-morbidities, ophthalmic examination, previous investigations and the results of intuitive colourimetry were collected and reviewed. VS symptoms follow a stereotypical description and are strongly associated with palinopsia, migraine and tinnitus, but also tremor. The condition is a chronic one and often results in misdiagnosis with psychiatric disorders or malingering. Colour filters, particularly in the yellow-blue colour spectrum, subjectively reduced symptoms of VS. There is neurobiological evidence for the syndrome of VS that links it with other disorders of visual and sensory processing such as migraine and tinnitus. Colour filters in the blue-yellow spectrum may alter the koniocellular pathway processing, which has a regulatory effect on background electroretinographic rhythms, and may add weight to the hypothesis that VS is a thalamocortical dysrhythmia of the visual pathway.

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1. Introduction

Visual snow (VS) refers to the persistent visual experience of flickering fine achromatic dots or static in the whole visual field of both eyes likened to “static analogue television noise” [1]. A recent series of publications highlight the very similar subjective stereotypical descriptions between patients of this frequently distressing phenomenon [2,3]. The symptom frequently occurs with other visual symptoms such as photopsia, nyctalopia, palinopsia (the persistence of previously viewed stimuli) and entoptic phenomena, as well as other disorders of sensory perception such as migraine with or without aura, tinnitus and tremor [1,4].

VS can be associated with stress, depression and previous illicit drug use, though no clear causative agent has been identified [3]. This results in patients with these symptoms often being misdiagnosed as having migraine with aura, hallucinogen persisting perception disorder (HPPD) or malingering. As a consequence treatments are often inappropriate, ineffective or absent.

Recently Schankin et al. suggested that VS is a unique clinical syndrome, distinct from migraine with aura, and recommended set diagnostic criteria to help identify VS patients [3]. The overlapping symptomatology and therefore potentially pathophysiology between migraines and VS patients cannot be dismissed. Indeed, migraine, tinnitus, photopsia and palinopsia all appear to relate to

an increase in sensitivity of sensory perception [1–3]. However as distinct from the presumed cortical spreading depression theory in migraine, VS patients are thought to have differences in regional metabolism resulting in modulation of neuronal sensitivity and excitability [3].

VS patients frequently report persistence of symptoms at all times including when the eyes are closed and with few patients reporting relief of intensity of snow symptoms in bright light [1–3]. We noticed however that some patients report relief of symptoms from tinted lenses. Intuitive colourimetry, the assessment of optimum tint, has been used in the past to alleviate symptoms of perceptual disorders and visual stress reported by patients with dyslexia, migraine or photosensitive epilepsy [5,6]. When offered to VS patients we identified a pattern of symptom relief from these coloured filters, particularly those in the yellow-blue spectrum.

In this paper we present a review of our patients with VS symptoms to whom we offered the option of undergoing intuitive colourimetry, review the previous hypotheses of VS and propose a new hypothesis – that VS is an imbalance of koniocellular and magnocellular pathway function creating a thalamocortical dysrhythmia that results in a disorder of visual processing.

2. Subjects and methods

Data was collected from 32 VS patients presenting to tertiary referral neuro-ophthalmology services in Sydney, NSW between 2012 and 2014. Patients underwent a standardised series of

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0967-5868/© 2015 Elsevier Ltd. All rights reserved.

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Opinion

An Integrative Tinnitus Model Based on Sensory Precision

William Sedley,^{1,*} Karl J. Friston,² Phillip E. Gander,³
Sukhbinder Kumar,² and Timothy D. Griffiths^{1,2,3}

Tinnitus is a common disorder that often complicates hearing loss. Its mechanisms are incompletely understood. Current theories proposing pathophysiology from the ear to the cortex cannot individually – or collectively – explain the range of experimental evidence available. We propose a new framework, based on predictive coding, in which spontaneous activity in the subcortical auditory pathway constitutes a ‘tinnitus precursor’ which is normally ignored as imprecise evidence against the prevailing percept of ‘silence’. Extant models feature as contributory mechanisms acting to increase either the intensity of the precursor or its precision. If precision (i.e., postsynaptic gain) rises sufficiently then tinnitus is perceived. Perpetuation arises through focused attention, which further increases the precision of the precursor, and resetting of the default prediction to expect tinnitus.

Why Understanding Tinnitus Matters

Fourteen percent of adults experience chronic tinnitus [1], while over 50% of normal-hearing adults experience subtle ongoing tinnitus within a silent environment [2,3]. Hearing loss is the biggest risk factor, followed by increasing age [1]. No widely applicable treatment reliably suppresses or eliminates tinnitus; in part, this is due to incomplete understanding of underlying pathophysiology. Improved understanding might also help clinicians to explain the condition to patients, and offer a unique window into sensory processing – without the confounding effects of an external stimulus. Furthermore, tinnitus may share commonalities with other aversive sensory conditions such as chronic pain [4,5].

The Symptomatology and Pathophysiology of Tinnitus

Tinnitus is the experience of persistent sound, in one or both ears or inside the head, in the absence of an external source [6]. In ‘objective’ tinnitus there is a measurable internal sound source such as turbulent blood flow, while the majority of tinnitus cases are ‘subjective’, where no such source exists. Tinnitus is perceived as fairly quiet, often masked by sufficient levels of environmental sounds, but a minority of cases are reported as extremely loud, and some are exacerbated by environmental sound [7]. Sounds are usually simple, with common forms resembling pure tones (‘ringing’), Gaussian noise (‘hissing’), or buzzing. More complex sounds are reported, and a minority of cases comprise music, for which we have recently proposed a related but distinct brain model to the tinnitus model described here [8]. Most people experience transient tinnitus at times, either spontaneously or following loud or prolonged noise exposure. Once tinnitus has been present for weeks to months, unless a reversible cause of hearing impairment is present, it typically becomes permanent. While it does not usually resolve spontaneously, the natural history tends to be of **habituation** (see Glossary) over time. However, a minority of patients report increasingly severe symptoms [7].

Trends

Existing tinnitus models, including mutually exclusive mechanisms, invoke causes from the ear to high-level cortical brain networks.

The generic framework of predictive coding explains perception as the integration of sensory information and prior predictions, each weighted by its precision.

In our model, previously proposed neural correlates of ‘tinnitus’ largely relate to hearing damage, rather than to tinnitus *per se*, and reflect an increase in the precision of spontaneous activity in the auditory pathway, which acts as a tinnitus precursor.

Perception of tinnitus emerges if the precision of the precursor rises sufficiently to override the default (but hypothesis) percept of ‘silence’.

Tinnitus becomes chronic when perceptual inference mechanisms learn to expect ‘tinnitus’, engaging connections between auditory and parahippocampal cortex.

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Cortical Hyperexcitability

Thalamocortical Dysrhythmia

Predictive Coding





Symptom Aggravators

Stress

Lack of sleep

Cold/flu

Alcohol

Caffeine

Excessive computer use

Too much sodium

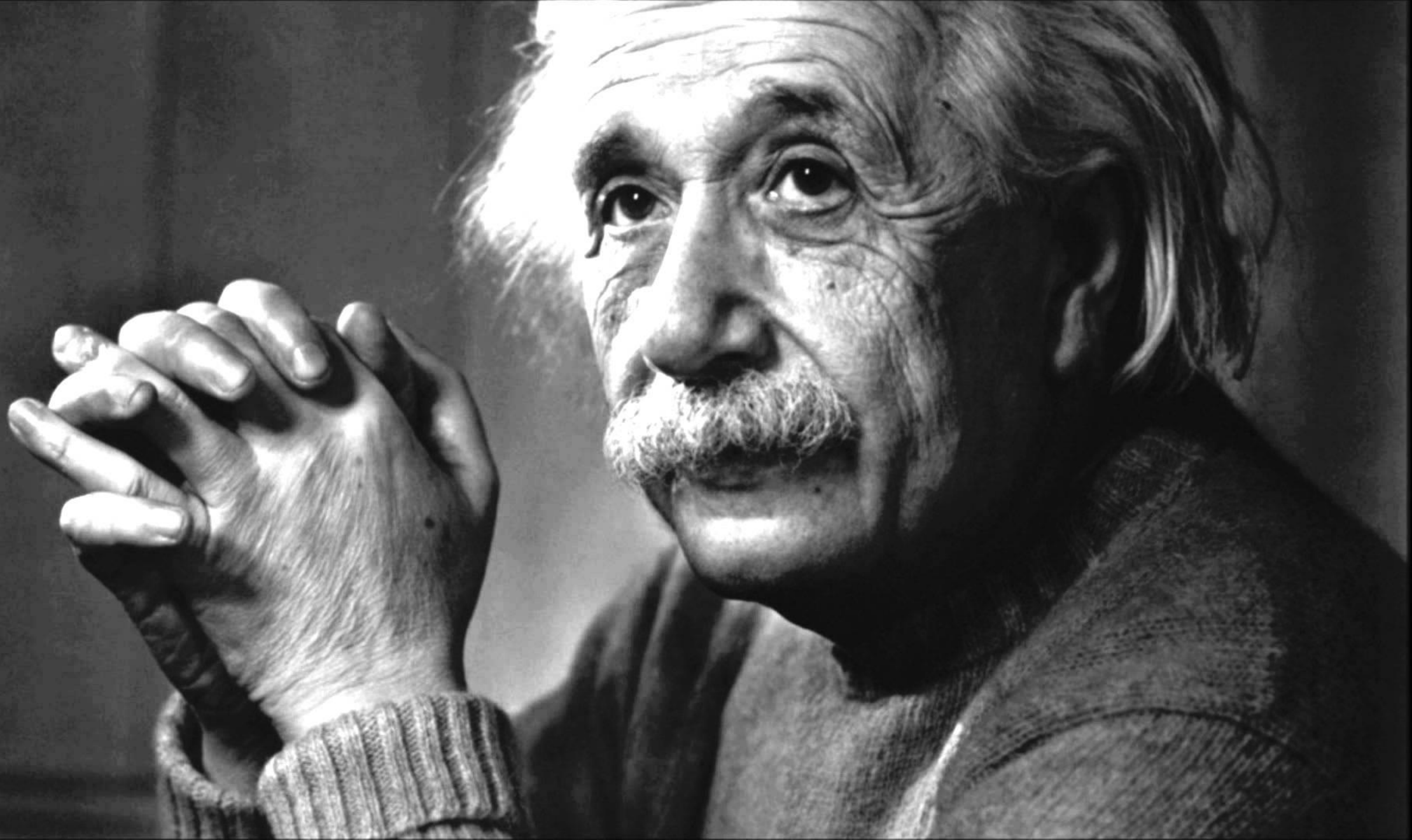
Too much sugar



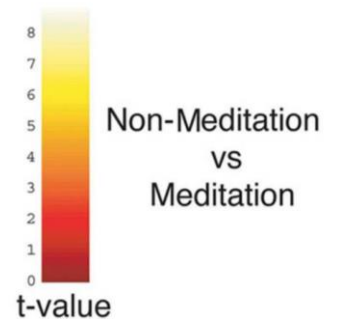
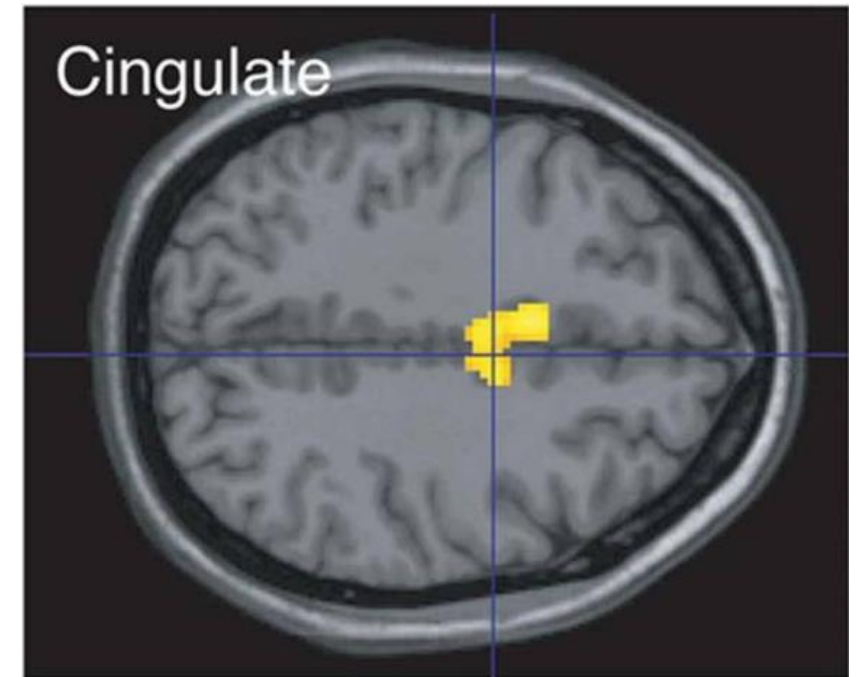
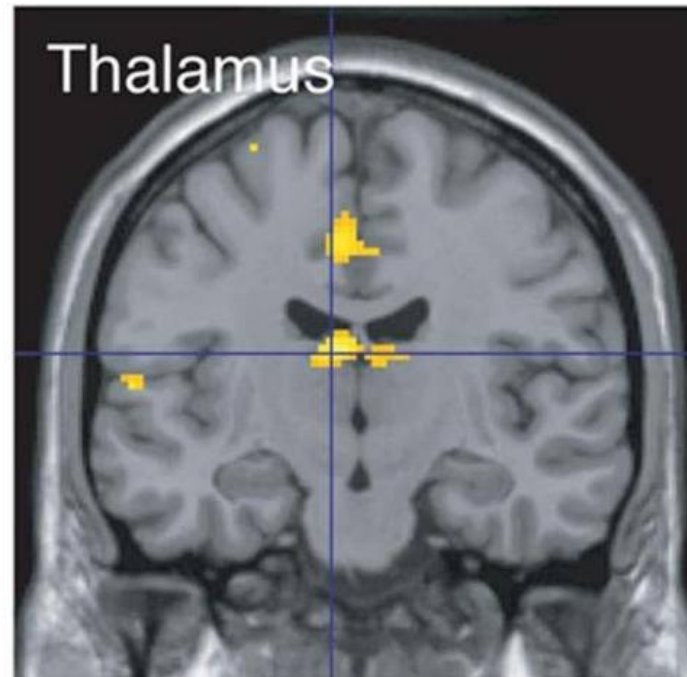
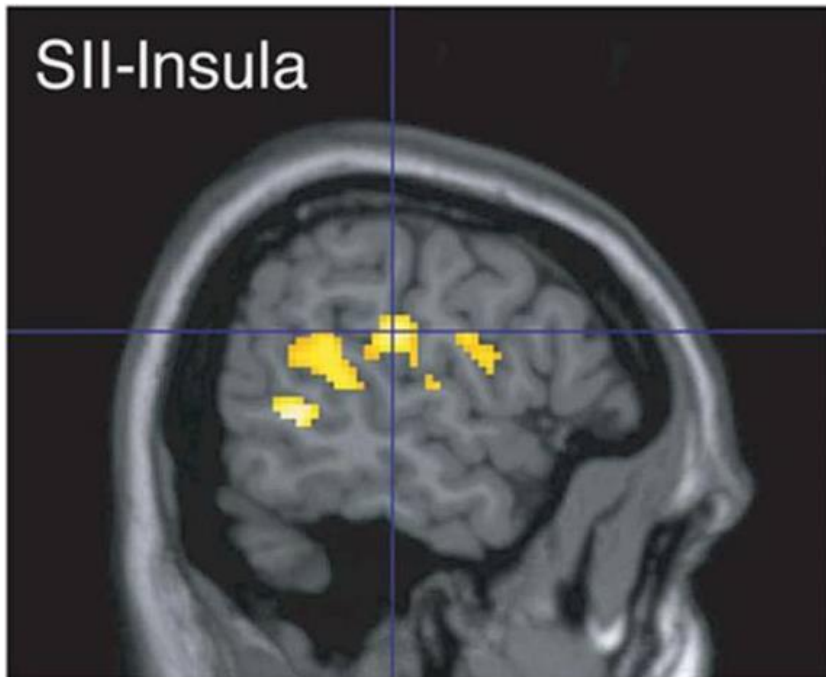


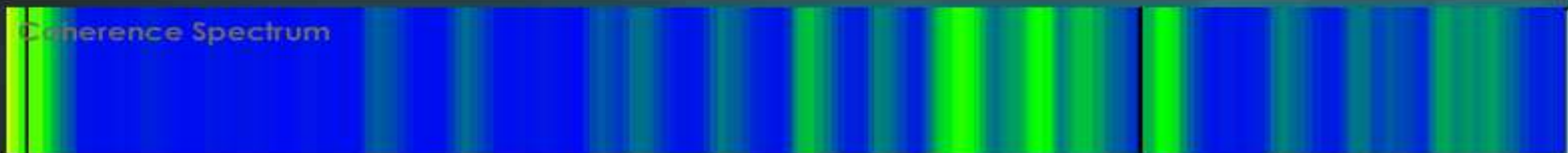
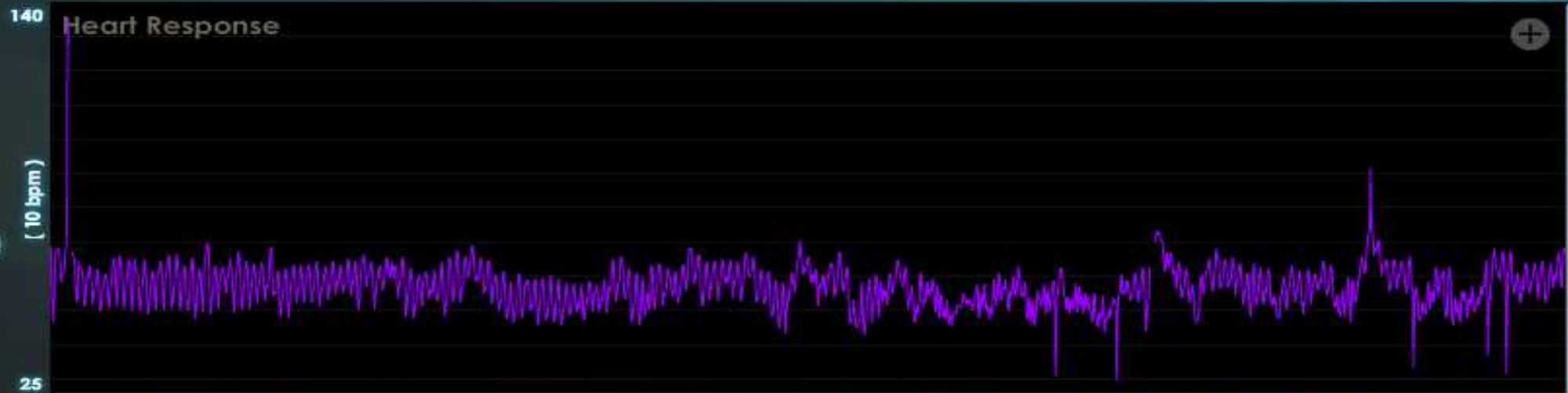




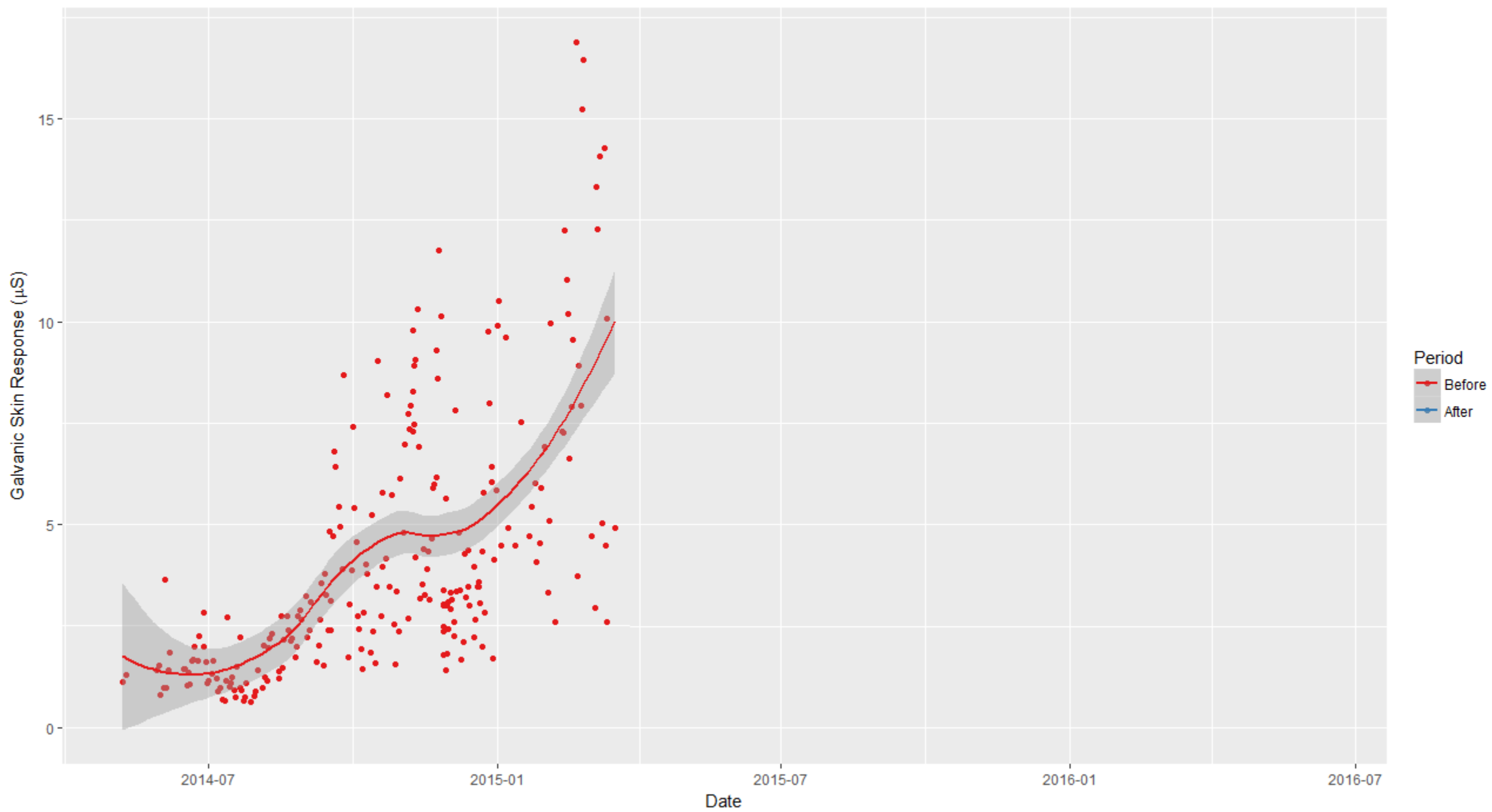


Meditation reduces pain-related neural activity in the anterior cingulate cortex, insula, secondary somatosensory cortex, and thalamus

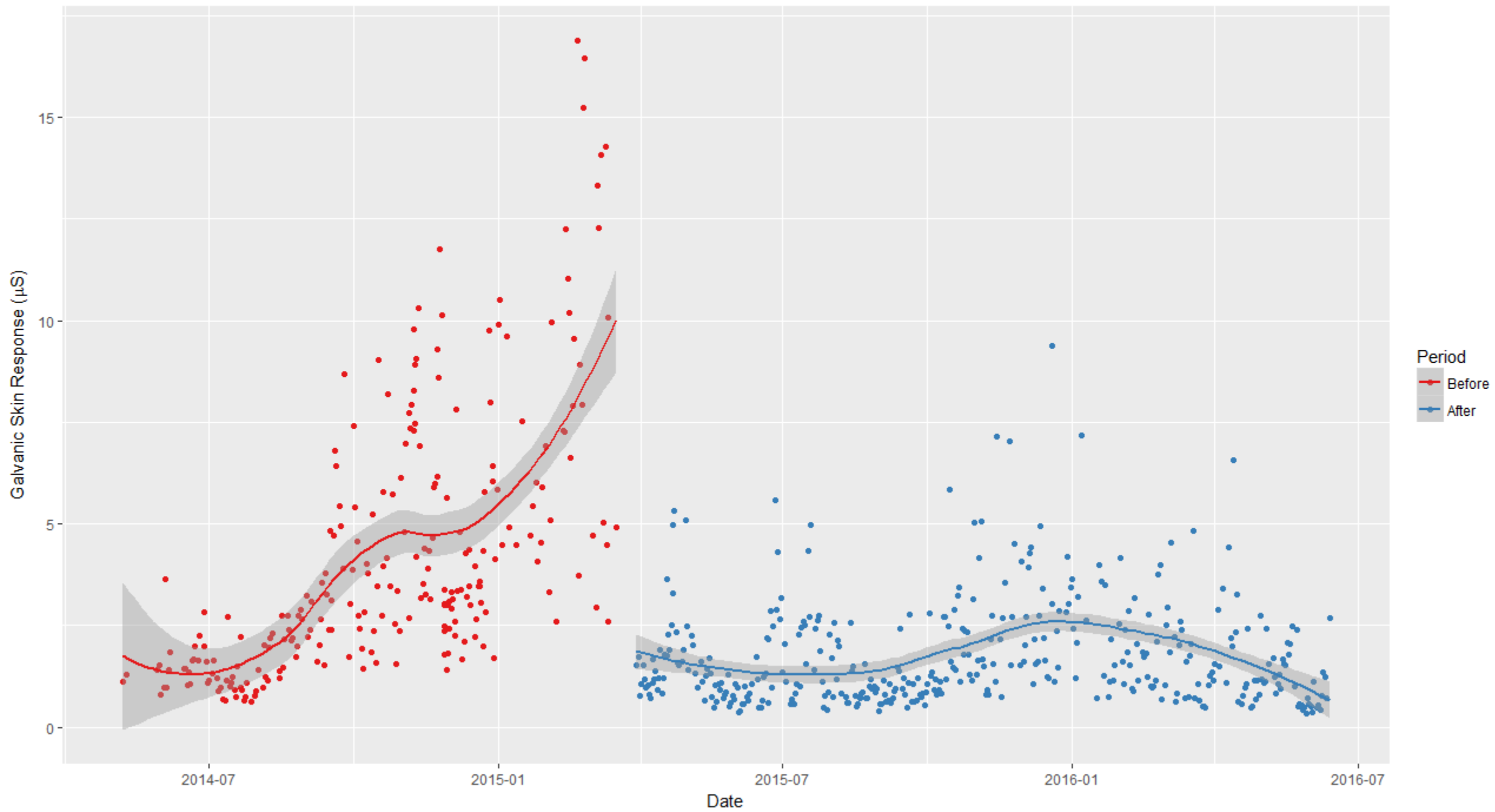




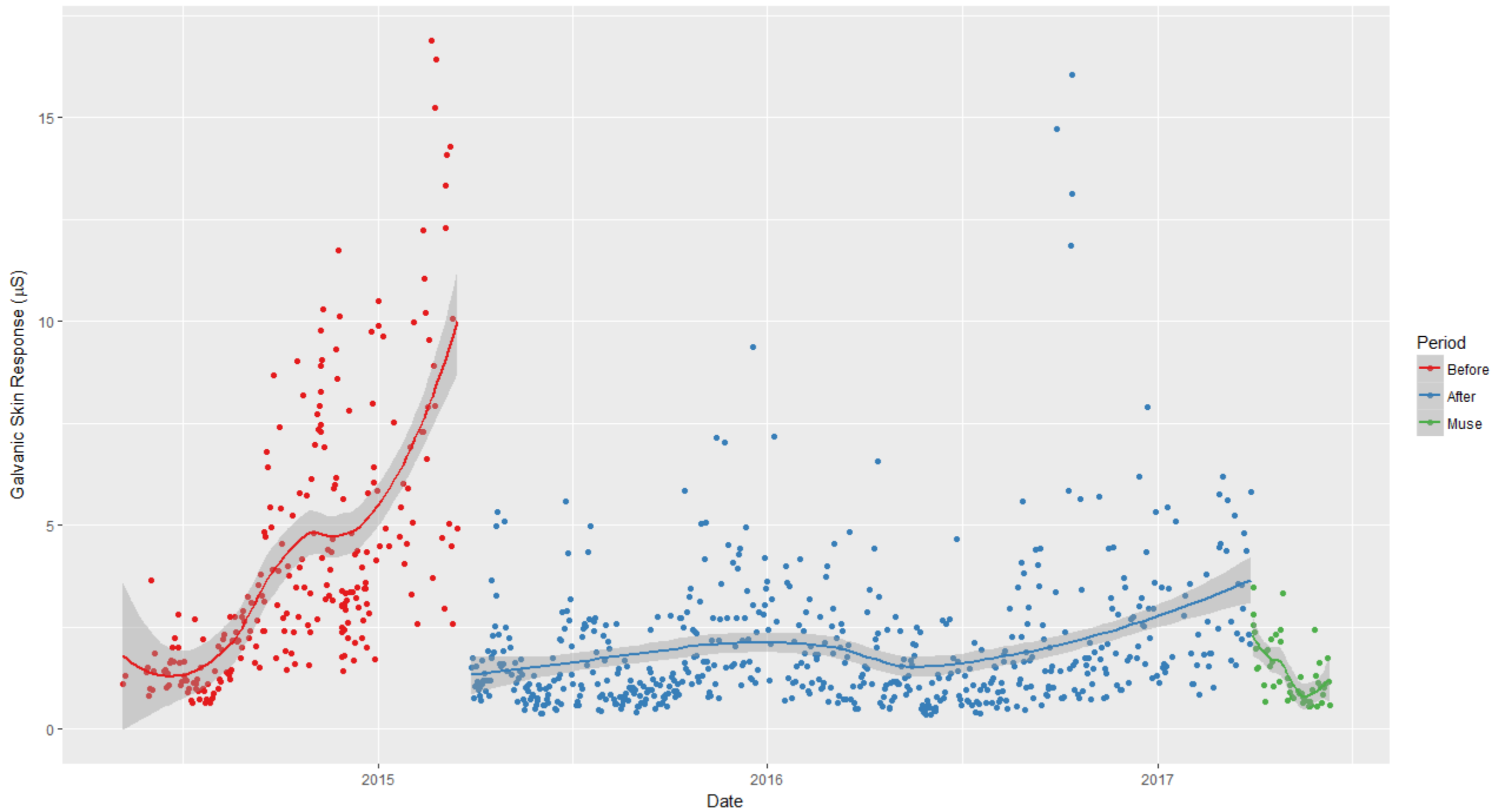
Average GSR Before and After Meditation Retreat



Average GSR Before and After Meditation Retreat



Average GSR Before and After Meditation Retreat





Current Research

Visual Snow – a disorder distinct from persistent migraine aura

<http://brain.oxfordjournals.org/content/137/5/1419.long>

The Relationship Between Migraine, Typical Migraine Aura and Visual Snow

<http://brain.oxfordjournals.org/content/137/5/1419.long>

Current Research

Visual Snow: a Potential Cortical Hyperexcitability Syndrome

<https://www.ncbi.nlm.nih.gov/pubmed/28349350>

Visual snow: A thalamocortical dysrhythmia of the visual pathway?

[http://www.jocn-journal.com/article/S0967-5868\(15\)00653-0/fulltext](http://www.jocn-journal.com/article/S0967-5868(15)00653-0/fulltext)

An Integrative Tinnitus Model Based on Sensory Precision

<https://doi.org/10.1016/j.tins.2016.10.004>

Future Research

Third study completed

Fourth study in the works

Research is crowd funded



EYE ON VISION
FOUNDATION

www.eyeonvision.org

Help Cure Visual Snow



5.8K SHARES

f SHARE

🐦 TWEET

📍 ORLANDO, FL

🏛️ CHARITY

\$77,594 of \$150k

Raised by 747 people in 23 months

Donate Now

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Eye ON Vision Foundation Corp

Orlando, FL

Tax ID: 205394501



Created July 15, 2014

✉️ Jen Ambrose

<https://www.gofundme.com/visual-snow>



Topic
Better Living

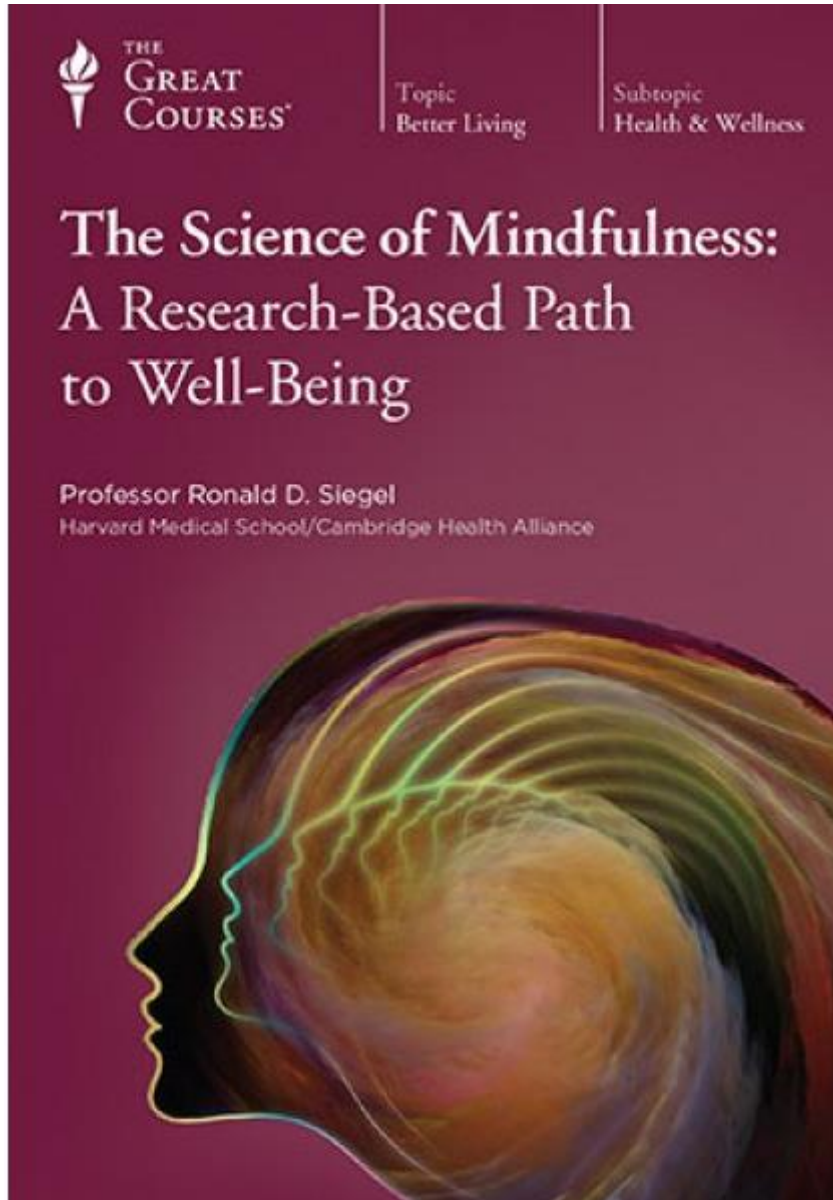
Subtopic
Health & Wellness

The Science of Mindfulness: A Research-Based Path to Well-Being

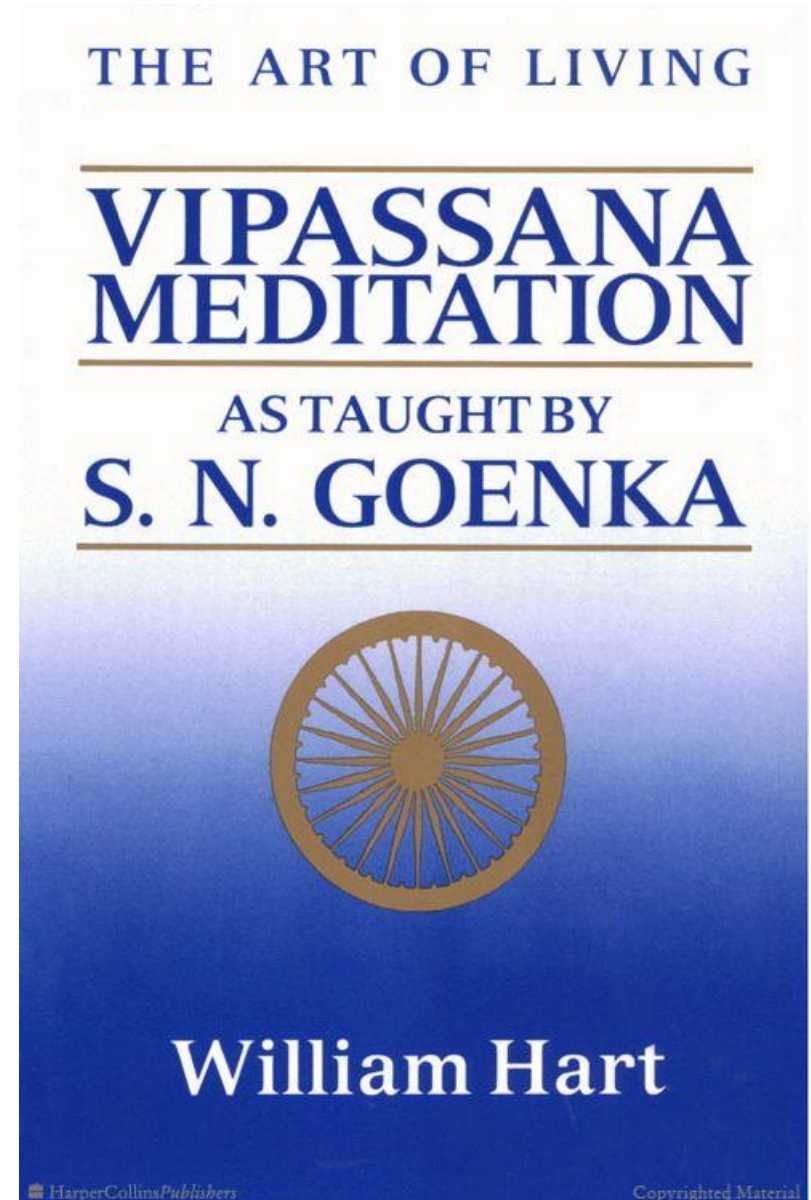
Professor Ronald D. Siegel
Harvard Medical School/Cambridge Health Alliance



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Conclusion

- 1.
- 2.
- 3.

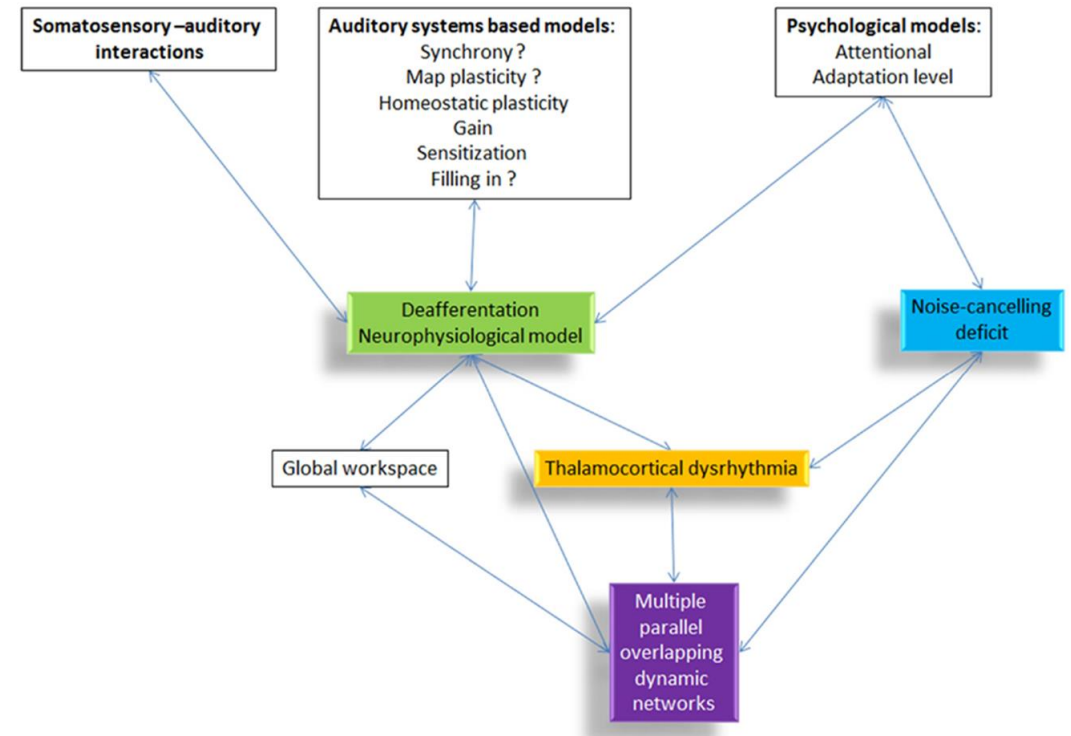
Conclusion

1. Tinnitus is associated with VSS
- 2.
- 3.

Symptom	Study 1	Study 2
<u>Palinopsia</u> (Afterimages)	86%	84%
Floaters	81%	58%
Blue-field Entoptic Phenomena	79%	76%
Photophobia	74%	72%
<u>Nyctalopia</u>	68%	63%
Spontaneous <u>Photopsia</u>	63%	53%
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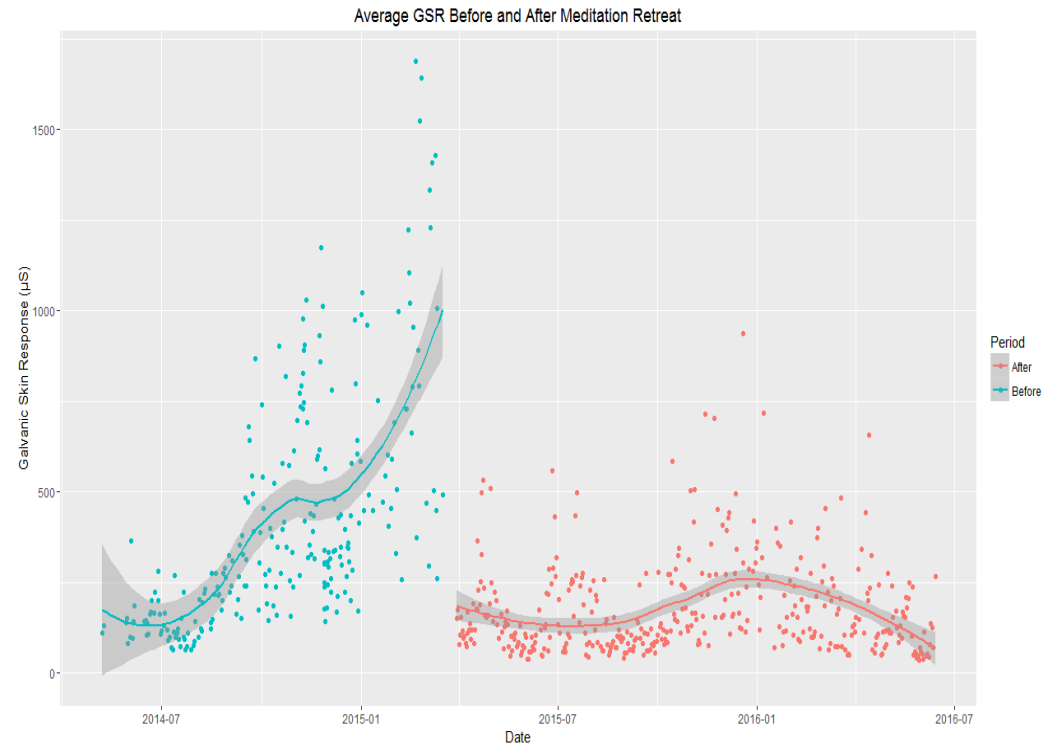
Conclusion

1. Tinnitus is associated with VSS
2. VSS provides insight into tinnitus
- 3.



Conclusion

1. Tinnitus is associated with VSS
2. VSS provides insight into tinnitus
3. Symptoms can be managed



It is possible to be in
physical, mental or emotional pain,
but to not be suffering from it.

Suffering is how we respond to pain.

Contact Info

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Thank You! :)