

## Deciphering Tinnitus from the Shoulders of Giants: A Kuhnian Shift may be Required

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### Abstract

The mechanism contributing to the causation of tinnitus continues to evade us. It is unlikely that our current thinking is progressing in the right direction. The literature on the subject is mounting but with no real insights. Perhaps we are all barking up the wrong tree!

The objective of this paper is to introduce, if possible, a paradigm shift that may produce a different trend in thinking and hopefully change our direction and lines of research.

This is attempted by employing the basic technique of logical thinking aided by modern computer logic and also incorporating neuroscience, artificial intelligence, psychology and philosophy. It is admitted that this hypothesis is subject to confirmational empiricism.

**Keywords:** Subjective tinnitus; Tinnitus mechanisms

### Introduction

Idiopathic tinnitus is a conscious “phantom” noise, with the descriptive interpretation provided by the sufferer and hence investigators have been misled in ascribing tinnitus to the ear. As a result, a significant effort has been expended over the years in explaining the exact site of the anatomical pathology in the ears [1,2]. Understandably such attempts have been futile.

There is little doubt now that tinnitus is a conscious central percept [3-6] and is not perceived during sleep or anesthesia. Some literature [7-12] suggests a peripheral initiator (hearing loss) to explain the plasticity incentive for the central states. And in the presence of normal hearing, an ultra-high frequency loss [13] and a “hidden hearing loss” [14] is offered to explain the central changes. Ironically the latter possibilities and the absence of any loss whatsoever can only suggest that the tinnitus may originate centrally.

Tinnitus has several similarities with pain and the neuroses and possibly addiction [15-20]. These are essentially heuristic behaviors for a three-dimensional world which had survival value for our ancestors and will be referred to as “tinnitus similars” in this argument.

These “tinnitus similars”, like tinnitus, occupy the attention by way of (negative) automatic thoughts. Such “tinnitus similars” have:

1. Reiterative qualities
2. no immediate impending threats
3. similar aggravating factors
4. may be associated with each other [35]
5. Imaging identifies similar regions [21-26]
6. similar treatments may help
7. also occur in lower species.

Such “tinnitus similars” may have the same pathological mental state.

### Known Characteristics of Tinnitus

The qualia (subjective qualitative experience) of the tinnitus are private and privileged only to the sufferer. As this is not publicly observable, it does not occupy a physical space for it to be accessible to our current methods of detection. The clinical inferences have behavioral and mainly distress manifestation overtones. Although attempted, the exact qualia are difficult for others to appreciate, and this explains the existence of the various tinnitus matching tests that inhabit the audiology template with no consistent inferences. Even musicians often have difficulty locking their tinnitus into these tests [27].

The point is made at this stage that tinnitus, like thoughts, feelings etc occupies the consciousness domain, being private and individual.

This paper aims to address the symptom of the phantom sound manifestation only. The distress manifestation of tinnitus are fairly well explained by invoking limbic connections [28,29].

### Hearing and Tinnitus may have Separate Pathways

At this stage of our knowledge, it is admitted that this is subject to confirmational empiricism. The following pointers however may suffice to suggest the plausibility that the hearing and tinnitus pathways may be separate:

1. Tinnitus can occur in the presence of normal hearing, indicating that separate pathways are highly probable. A single path will necessarily create problems, allowing only hearing or only tinnitus. Also, if hearing and tinnitus had a common path, absence of one would necessarily summon the other into action. This is not supported

by the empirical evidence. The Heller and Bergman and other studies lend support to this co-occurrence [30-32].

2. Only some patients with hearing loss develop tinnitus [3,33]. If all individuals with hearing loss developed tinnitus, only a single path may allow such. But as not all individuals with hearing loss develop tinnitus, separate paths are more likely.

3. Even the documented reorganization of the central auditory components with hearing loss [7-12] does not explain [5] the non occurrence of tinnitus in the many other hearing loss patients (with the such expected reorganization), and hence further supporting separate paths.

4. Absence of both hearing as well as tinnitus is plausible with separate paths. Absence of hearing allows the tinnitus path uninterrupted access to conscious 'attention' but only if other more pressing attention occupiers are not in competition, in which case tinnitus also loses. Similarly in the absence of a hearing loss, the occurrence of tinnitus may be explained by the presence of a "tinnitus similar" facilitator [34,35].

5. Somatosensory tinnitus occurs in the absence of a hearing loss, further contributing to the plausibility of dual paths.

6. If ototoxic drugs and excessive noise damage the hearing pathway and contribute to a hearing loss then it is unlikely that tinnitus will travel the same functionally damaged path to produce the sensation of sound. Yet tinnitus thrives in the presence of a hearing loss. Hence another (undamaged) path for tinnitus is more likely.

In summary, tinnitus is independent of a hearing loss, thus throwing serious doubt on the available theories [34] which attempt to explain tinnitus in the presence of a hearing loss, attributing central changes to sensory deprivation [7-12,36,37]. It is possible that the various documentations (excitatory and inhibitory, neural plasticity, neuromodulation, neuroprotection etc.) provided may be co-related occurrences (compensatory) rather than causations. It appears that ultimately the explanations may lie at the final conscious 'attention' level, access being only available via separate paths.

In some individuals, genetic synaptic pruning by the relevant caspases results in destruction of the tinnitus path and this contributes to the unavailability of the tinnitus path in the presence of a hearing loss in later life. It is also possible that the hearing path may be similarly destroyed early, thus contributing to a congenital genetic hearing loss. These latter individuals may still complain of tinnitus.

With the available theories, it is assumed that a hearing loss is present. Then various mechanisms are recruited with the final inference being "constitutes a reasonable candidate mechanism leading to the sensation of tinnitus".

But exactly how this occurs is not explained. It is also not explained why these changes could not be attributable to the tinnitus itself. Some studies refute such mechanisms [5,6].

It is proposed here that tinnitus does not start de novo, but is the clear "winner" in the presence of a hearing loss, at the conscious 'attention' level in the attention game.

In cases where tinnitus occurs in the absence of a hearing loss (from 250 Hz to 8 KHz), the credibility of the audiology of such studies is questioned and the loss of ultra high frequency losses (10-20 KHz) is recruited to explain the theories. But it must be remembered that even

if one case of tinnitus occurs without any hearing loss [38,39], then these theories become untenable.

An innate tinnitus, possibly evolutionary (see below), incorporating all the hearing frequencies is proposed here. Competitively therefore, the "dead" frequencies will gain prominence in the symptomatic tinnitus. This appears to be the empirical audiological fact [40,41]. This also explains why tinnitus is best masked by incorporating the "dead" frequencies.

The two paths are in very close proximity that may not be discernible to our current methods of detection. As both paths have the same outcome, which is the perception of sound, this proximity is most likely. Also evolution tends to engineer a close fit between functionality and economy of structure and this arrangement certainly satisfies our argument.

The difficulty in hearing experienced in the presence of tinnitus is indicative of the proximity of these paths and the tendency to be mutually interfering (ephaptic transmission) at subcortical levels. This may also explain the masking of tinnitus by noise.

The masking noise tends to crowd out the tinnitus path, causing the generation of tinnitus to break down and this contributes to residual inhibition, before tinnitus can find its way back, on cessation of the masking noise.

Sensation along the hearing path is initiated by sound at the ear and finally perceived consciously by the brain. Tinnitus (and "tinnitus similar") may initiate at a "reiterative (nagging) center" (the site with the "halting problem", see below) in the subcortex and finally be perceived consciously, attention allowing, by the brain.

Electromagnetic provocation (electroconvulsive therapy, vagal stimulation, transcranial magnetic stimulation) [42], can be expected to interfere with the "reiterative center" and cause disruption of its activity, atleast momentarily and hence contribute to the transient therapeutic efficacy in these conditions (tinnitus et similar).

The brain is a massively parallel processor (see below) and in this regard, the concept of a "reiterative center" should not relate its function to a single distinct part but to several localizable parts to include emotional, cognitive, attentional and memory components. The current functional MRIs bear testimony to this fact.

These established Hebbian connections are further reinforced with the advance of symptomatic tinnitus.

## Conscious and Subconscious Components

Tinnitus is a mental state [43] which triggers further mental states involving the limbic and autonomic nervous systems.

Initially the limbic system turn-on is initiated by the cortex (reached possibly via the ventral parts of the thalamus) and then relegated to the subcortex (possibly the medial and dorsal parts of thalamus). Once such links are established, it appears that cortical control is lost and difficult to regain as is evidenced by the significant failures of the counselling therapies.

At any moment 'attention' occupies the focus of consciousness, although a state of "fringe consciousness" which is a latent awareness of the background, to which we can shift attention when needed, lurks. This absolute attention workspace may be occupied by one of several options to include sense data, thoughts, tinnitus etc. and the competition for this workspace is like radio channels competing for a

narrow frequency band with a “winner take all” equilibrium [44]. It may be at this level that tinnitus loses to hearing, in hearing individuals, and in the absence of hearing, tinnitus tends to thrive. The reiterative propensity of the “tinnitus et similar” allows them an added mathematical probability advantage of ‘winning’ in the attention game. The ‘attention’ workspace is also biased towards emotion laden stimuli [45] which further advantages tinnitus which has a definite emotional salience.

Counselling aids the patient to take control of this space and oust the negative intruders by introducing positive thoughts (“voluntary” top-down attention) and as this space is limited, this can work. Sound therapy also works (“reflexive” bottom-up attention) by attempting to occupy this space. The concept of space is employed here to facilitate our human comprehension, although another (nonmetaphysical) dimension may be involved.

As tinnitus is absent during sleep and anesthesia, the subjective experience of tinnitus lies in the realm of phenomenal consciousness. Consciousness thus provides us the key to our inquiry.

Tinnitus, like pain, is a broad term and encompasses several subjective qualities in different patients. It is also possible that anxiety and depression also have different subjective qualities. Further excavation in this regard is required.

Functional MRIs tend to localize areas of activation in tinnitus [46]. Exactly how this translates into tinnitus at the conscious level is not explained. The currently identified shortcomings of these imaging techniques include reverse inferences [5] and pre-emptive blood flow (in anticipation). To date these studies have provided inconsistent and contradictory results and “a vague picture of the neuronal correlates of tinnitus” [47].

Because consciousness is essentially subjective, it is an entity, objective science will never be able to explain according to McGinn [48] who argues that there must be a physical truth about consciousness, but it is conceptually impossible for humans to grasp.

## What we can Learn from Computers

Neurons only fire when their inputs reach a certain threshold. The available theories ignore [35,36] this fundamental concept. Hence, as neurons have threshold firing only, tinnitus is most likely to be a “halting problem” as mentioned below.

Computers use the von Neumann architecture, employing memory to contain both data and a program for operating on the data. The human brain possibly also uses memory to store and manipulate emotions, ideas, sensations, etc. and employs parallel connectome processing at the subcortex level. The tinnitus sound (and “tinnitus similar”) is also available at the subcortical level to draw upon, depending upon the Hebbian connections established for access to the conscious ‘attention’ level.

The senses employ a form of multi-layer nets in perception. Such nets are also good at pattern recognition. Each layer finds patterns in the layer below it and it is this ability to create the internal representations from the external world that is finally perceived [49]. In tinnitus such a layer of neurons may be subject to a “halting problem”.

Incidentally artificial neural nets which employ multi-layer nets also utilize the technique of back propagation of errors. Backpropagation approximates the non-linear relationship between the input and the

output by adjusting the weighting values internally. Such a technique may also be employed in the hindbrain for the phenomena of ‘homeostasis’ (regulation of body temperature, respiration, etc.) where a fixed output is desired irrespective of the variability of the inputs.

Computer scientists are able to build such machines that mimic human abilities and still not understand the mechanism of those abilities.

As such, the mechanism of the “halting problem” of the Turing machine (a mathematical concept) remains an enigma.

It is important to be wary of computational models [50,51]. Such models are usually capable of handling only the known input variables, parameters, constraints, usually with limited interactive processing and interdependent manipulation. Also unlike in biological systems, this processing is essentially bottom-up, without cortical control and eventually is subject to automatic generalization. In other words, computational models can sometimes err seriously.

## Does Evolution Contribute?

It is proposed that tinnitus may be an evolutionary incident, initiating in earlier times as “siren” hearing, to warn the organism to be on guard constantly for predators. The “siren” sound creates an atmosphere of present-centeredness which may have adaptive value for the organism by forcing the recruitment of a broad network of task-related neural resources. The triggered limbic and autonomic events may be such responses. Habituation will not occur as this noise is centrally induced. Prior to the long period of evolution of the basic tasks required of an auditory system, to include acoustic feature discrimination, sound source localization, frequency analysis, and auditory scene analysis, this “siren” hearing may have had survival value. As tinnitus is lost during sleep, such organisms had to ensure safe quarters during sleep. Such a natural “siren” may be comparable in computer terms to the “halting problem” of the Turing machine. As evolution proceeded, and possibly to reduce energy consumption, “alarm” (normal) hearing evolved with cortical representation. “Siren” hearing was relegated to the subcortex with access to conscious attention. In our argument this provides an abductive (inference to the best explanation) advantage. Due to the eons of time involved, this of course cannot be subjected to falsifiability. This concept may also bear an evolutionary similarity to saccadic vision [52] (employed for tracking moving prey or predators by our ancestors). This inference also helps to support the concept of separate paths for hearing and tinnitus, having evolved at different times for different needs.

In the Heller and Bergman study [29], 94% of 80 normal individuals experienced tinnitus in quiet surroundings. Considering such a high proportion which is also available in other similar studies [30,31] including one with a placebo suggestion [31], the possibility that tinnitus may lie in our evolved cognitive architecture cannot be ruled out.

The animal models support that tinnitus exists in retrohuman species, at least as far back as rodents [53]. If the occurrence of neuroses in animals is extrapolated as a “tinnitus similar” to tinnitus itself, this further supports our argument.

Further, as per our argument, tinnitus similar being an adaptive evolutionary behaviour, the oft-noted association of such with the “fight or flight” autonomic response is easily explained.

Thus it appears that animals can experience tinnitus but the human characteristics of language and narrative; the tendency to attribute causes to events in the world; and perhaps the ability to experience emotions like awe make tinnitus a concern for some individuals.

### **Some Considerations of the Current Literature on Tinnitus**

In the elucidation of tinnitus, it may benefit to visualize a three stage model:

1. Stimulus
2. Mechanism
3. Effect (tinnitus percept)

In general, if the first two are known we can predict the effect (percept). If the last two are known we can retrodict the stimulus (cause). If the first and last are known we can provide the mechanism (explanation).

However, when only one is known we need a hypothesis, which when confirmed by experimentation leads to a theory.

In the tinnitus literature, no such visualization process is in evidence to date. Only the effect (tinnitus in this case) is known. Fragmented mechanisms to explain the experimentally obtained facts are provided. Hence statements like, "Thus, correlations between brain activity and/or connectivity and tinnitus can tentatively be turned into causal relationships" [54] are necessarily incomplete.

Further the authors by their own admission appear to "tentatively" confound correlations with causes. There are basic problems:

1. Correlations are patterns, occurrences, or changes that vary in relation to each other. Is this variation proven here?
2. Although inferences from correlations assert a predictable relationship between variables, they do not account for it; thus they are less powerful than causal inferences.
3. Can the brain activity and/or connectivity signify two or other more different, even opposite things i.e may be even attempts to nullify the tinnitus?
4. Is tinnitus responsible for the brain activity and/or connectivity rather than the other way around?
5. Is there a basis for thinking that the relationship is anything other than a mere coincidence?
6. Could tinnitus itself have multiple causes? Are we looking for one cause only?

Further, the fact that a particular mechanism leads from the first to the last (tinnitus) provides no confirmation that it is the only mechanism that could produce that outcome. It also does not confirm that such is the mechanism that actually produced that result in reality.

Nevertheless the plausible relevance/irrelevance of some literature to this hypothesis is mentioned:

### **The Neurophysiological model of tinnitus [55]**

The Jastreboff Neurophysiological model does not provide a mechanism for the generation of the acoustic component of tinnitus and attempts a dissociation approach (tinnitus retraining therapy) at alleviating the symptoms and distress caused by tinnitus. This is not at

odds with the "voluntary" top-down attention (education) and the "reflexive" bottom-up attention (sound therapy) to crowd out the 'attention space', as indicated in this hypothesis.

### **Cognitive model of tinnitus [56]**

As mentioned above, this hypothesis only attempts to deal with the acoustic component of tinnitus. However, if an evolutionary basis for tinnitus is accepted, the cognitive component of tinnitus is essentially the remnant of the type 1 error (false positive) response which is etched into our constitution, and which was the more reliable interpretation necessary for the survival of our ancestors when a predator clue emerged. Imagine an ancestor interpreting an unfamiliar sound as nonthreatening (false negative or type 2 response). Not many such interpreters would survive and reproduce. Having got out of (the perceived) harm's way pronto, the ancestor is now subject to (negative) thinking to involve the identity of the supposed predator (fear) and methods of deceit/escape etc. Persistence of this thinking fosters anxiety and depression. A hypervigilance state thus established may reduce the cognitive capacity needed to perform tasks that require voluntary, conscious, effortful, and strategic control.

Extinction of this basic response is the aim of Cognitive Behavioural Therapy (CBT).

### **Three neural networks model (eg attention, distress, memory) [54]**

This paper points to the limitation of functional imaging studies in identifying specific neuronal correlates of tinnitus (attention, distress, memory). Brain stimulation is offered as able to identify the neuronal correlates of the various clinical aspects of tinnitus. It should be obvious that the evolutionary hypothesis suggested above is not at odds with this view. However it must be remembered that the tinnitus response is a "joint effort" by the whole patient (mind and body) to react to a perceived threat and is best investigated holistically.

### **Extra/lemniscal model of tinnitus [57]**

Apart from imparting a conceptual viability to dual pathways, the extra/lemniscal (somatosensory) characteristic is not of relevance to this hypothesis for several reasons:

- The separate tinnitus path concept in this hypothesis is not exclusory of any particular type of tinnitus.
- The paths for hearing and tinnitus are entirely separate except ephaptically in this hypothesis.
- Being an evolutionary phenomenon, there is no age restrictions to the path.

Moller et al. introduced the concept of the non-classical pathways in 1992, as occurring only in children and only in some forms of tinnitus and autism. To date neither a overarching (commonality) reason or cause to account for such connectivity, and only in some of these individuals, has not been provided. Considering that it occurs only in "some cases", it is more likely than not, that such paths may only be an incidental finding and not specifically destined.

### **'Hidden hearing loss' contribution to tinnitus [14]**

In the quest for a hearing loss to lend credence to the 'auditory deprivation for central plasticity' hypothesis, unreliable audiometry elsewhere, very high frequency loss and 'hidden hearing loss' have all

been summoned. It is profitable to note that in the King-Kopetzky Syndrome (KKS), a normal audiogram is also obtained. However in these patients who actually complain of a hearing disability, no imploration for a 'hidden hearing loss' is made. The term Auditory Processing Disorder (APD) is also used here, which ascribes a mechanistic cause but no evidence to support this term is provided. If brain stem imaging studies in this disorder (not available yet) also reveal the same findings as that in tinnitus then it may be possible that the such findings could be related to the auditory processing mechanism and not necessarily directly to tinnitus. If the findings are not similar, then the 'hidden hearing loss' gets closer to biting the dust. Incidentally in KKS, it is proposed that a combination of psychological, social, and biological factors lead to the experience of 'hearing difficulties' and the 'impairment' may not be auditory. It may be purely psychological or psychologically 'amplified' [58]. Psychological reverberations also abound in tinnitus.

Also the Schaette et al. paper attempts to translate hearing loss findings from the mice peripheral system to the human brain stem. In the paper this is further conflated with tinnitus.

Ultimately whilst Schaette et al. attempt to provide a hearing loss cause for tinnitus, this hypothesis maintains that a hearing loss only helps to uncover tinnitus at the consciousness level. Another way of looking at this would be that if tinnitus is considered genetic then hearing loss may only provide an epigenetic footing.

Schaette et al., posit a "hidden hearing loss" in tinnitus.

In the Heller and Bergman study, 94% of 80 normal individuals experienced tinnitus in quiet surroundings. Such a high proportion is also available in other similar studies including one with a placebo suggestion.

Extrapolating the Schaette et al., postulate, 94% of individuals in the Heller and Bergman study may have a "hidden hearing loss". Such an eventuality is clearly untrue and absurd.

However, both the above assertions could be true, if it is held that an intermittent and spontaneous hearing loss can occur, but only under the Heller and Bergman study conditions. Again, clearly this is another absurdity.

Taken together these studies only serve to indicate that a hearing loss is not required for tinnitus.

### What predictions can be made by this hypothesis?

1. Being an evolutionary phenomenon, tinnitus may only succumb to psychotherapy in some cases
2. The most effective therapy is Cognitive Behavior Therapy. So efforts at better methods of such delivery need to be addressed.
3. Drug therapy must aim at cognition-altering or attention- altering medication without affecting reason /consciousness.

### Conclusion

A credible mechanism for tinnitus must conclusively explain how tinnitus occurs in the absence of a hearing loss. It must also explain why tinnitus only occurs in some but not all cases of hearing loss.

It is proposed here that hearing and tinnitus occupy separate proximate paths competing for conscious 'attention'. The identification of the exact mechanism of this "winner takes all

attention" at the conscious level is essential for further progress. Thus the suggested "Kuhnian shift" demands a search at the consciousness level.

In other words the most important question about tinnitus may be the one we don't yet know how to ask.

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