Modelling the Generation of Tinnitus in a Silent Environment

Richard Gault[1], T.M. McGinnity[1,2] Sonya Coleman[1]
[1]Intelligent Systems Research Centre, Ulster University, UK
[2]Nottingham Trent University, UK
Email: gault-r2@email.ulster.ac.uk, tm.mcginnity@ulster.ac.uk,
sa.coleman@ulster.ac.uk

***Abstract*—Tinnitus is the phantom perception of a sound heard in or around the head in the absence of an identifiable source affecting 10-15% worldwide. The majority of tinnitus sufferers have some form of hearing loss. The multiple pathologies that generate and sustain tinnitus in a diverse tinnitus population make it challenging to establish a homogeneous cohort for experimental studies. People with no hearing loss or previous experience of tinnitus also begin to perceive phantom sounds when situated in a sound proof room for five minutes or less. This is consistently observed across multiple studies. Studies that induced tinnitus through acoustic deprivation in healthy subjects provide a more controlled environment to observe tinnitus. Although experimental work shows what is happening it does not explain how the tinnitus related activity is generated. Computational modelling of tinnitus following hearing loss has shown that underlying mechanisms, such as adaptive gain, can generate hyperactivity in the regions of hearing loss. These models do not account for the generation of tinnitus in people with no hearing loss. In this work we model the development of tinnitus related activity in cases of no hearing loss and induced acoustic deprivation. The tinnitus related activity disappears once the model is returned to normal ambient noise.**

I. Introduction

Tinnitus is the conscious experience of a sound, typically characterised as a ringing or buzzing noise that originates from an unidentifiable source affecting 10-15% worldwide with 1-2% having a diminished quality of life [1]. The underlying mechanisms that generate and sustain the tinnitus sensation remain obscure. To date there is no objective measure or cure for tinnitus. Consequently interventions focus on managing the distress of sufferers. Treatment methods include sound therapy [2], cognitive behavioural therapy [3] and mixtures of counselling and noise stimulation [4].

Hearing loss is commonly a comorbidity with tinnitus [5]. In cases where the perceived tinnitus has a tonal quality, the dominant pitch of the tinnitus has been found to lie in the region of hearing loss [6]. Even with no apparent hearing loss it is possible to have damage within the auditory pathway. This can be observed using threshold equalising noise, which highlights 'dead regions' within the cochlea, and auditory brainstem response to stimulus, which can be used to assess the function of the auditory pathway. Tinnitus sufferers have shown signs of hidden hearing loss in studies using threshold equalising noise [7] and auditory brainstem response paradigms [8] where subject's audiogram results were within normal hearing limits. Hearing aids are now used to not only provide amplification for those with hearing loss but as a treatment method for tinnitus. Schaette and McAlpine [8] found using auditory brainstem response data that the auditory system of subjects with tinnitus amplifies the reduced signal that is found in regions of hearing loss. Hearing aids may therefore provide amplification of the sound in regions of hearing loss so that the auditory system does not have to amplify the sound itself. Additionally the hearing aids will provide amplification of other frequencies allowing the listener to attend more easily to other sounds rather than focus on their phantom sound. However, hearing aids do not provide relief for every person. The diversity of the tinnitus population and the subjective nature of the problem make it challenging to identify similarities across tinnitus subjects when conducting experiments. Rather than study subjects with pre-existing tinnitus whose tinnitus may have changed over a period of time it is possible to induce tinnitus temporarily in healthy subjects with no previous experience of tinnitus. Heller and Bergman [9] found that approximately 94% of people with normal hearing will develop tinnitus when placed in a sound proof room for five minutes. More recent studies have shown similar findings with between 64-83% of people developing tinnitus like sounds [10], [11], [12]. Schaette et al. [13] demonstrated that it is possible to induce reversible tinnitus by having those with no hearing loss wear an ear plug in one ear for seven days to simulate hearing loss. Once the ear plug was removed the phantom sound disappeared in all cases where tinnitus developed.

The studies mentioned above can only observe the external characteristics of tinnitus. As shown in [8] computational models of the tinnitus can be used to understand the underlying mechanisms that generate the observable behaviour. In [8] the auditory brainstem response to sound was measured. In subjects with tinnitus there was a reduction in the amplitude of the activity in the initial stage of the auditory pathway however later stages displayed normal levels of activity compared to healthy controls. The authors explained the apparent amplification in the signal by an increase in the auditory gain. In essence the brain amplified the spontaneous activity of the auditory pathway. Consequently it was possible to develop increased activity above spontaneous levels; this is referred to as hyperactivity. Other tinnitus models also focus on the plastic

changes that occur in the auditory system following hearing loss (see [14] for review). These models do not explain the occurrence of tinnitus with no hearing loss and in particular why phantom sounds are perceived when a person with normal hearing is placed in a sound proof room.

This paper presents preliminary work in the development of a phenomenological model of tinnitus that emulates basic tinnitus behaviour once someone with no hearing loss is placed in silence. Furthermore the tinnitus like behaviour disappears once normal environmental sounds are restored as observed in practice [13], [11], [12], [10].

The remainder of this paper is as follows: Section II outlines an overview of related research. In Section III we provide an outline of the experimental paradigm that will test our proposed model along with the parameters used. Section IV presents the results of the simulations. Sections V provides a summary of the conclusions along with a guide to the future development of this work.

II. Background and Related Research

A. Tinnitus related behaviour

In 1953 Heller and Bergman [9] famously conducted a study wherein subjects were placed in a sound proof chamber that had an ambient noise of between 15-18dB. In total 94% subjects with normal hearing and no tinnitus experienced phantom sounds after minutes of being inside the chamber. In the same study, 73% of subjects from an additional group, who did have hearing loss, developed tinnitus within five minutes of acoustic deprivation. In this study it is unclear the precise wording of the instructions given to the participants. It is possible that subjects would expect to hear a sound given that they were asked to document any change of sound they experienced whilst in the sound proof room therefore increasing the likelihood of them perceiving a sound.

Further studies were inspired to investigate the possibility of introducing the anticipation or persuasion of an existing sound. Del Bo et al. [12] revisited the Heller and Bergman study and found that 83% of subjects with normal hearing experienced phantom sounds when placed in an sound proof room. Furthermore they introduced a false loudspeaker into the room and found that the number of subjects that reported hearing a phantom sound rose to 92% illustrating the influence of expectation. Tucker et al. [10] investigated the gender differences of those who develop tinnitus in silence. Although they found no significant difference between genders, 64% of subjects developed tinnitus after situated in a sound proof room for a number of minutes. The exact time needed for a phantom sound to develop is unknown. It would be challenging to introduce a timing element in to the studies mentioned without introducing anticipation. Knobel and Sanchez [11] investigated the influence of top down mechanisms generated from auditory attention. The paradigm consisted of three components where subjects were placed in a sound proof booth for five minutes and in every case subjects were asked to comment on any changes in the sound and light level. In one session the subject was asked to pay particular attention to any changes in the light that may or may not occur. In another they were asked to focus on any changes to the sound that may or may not occur and finally subjects were asked to complete a cognitive task; the classical logic puzzle towers of Hanoi. The highest prevalence of visual and auditory hallucinations occured during the visual and auditory tasks respectively. In every session it was the phantom sounds that were most common over visual perception, up to 68%. However when instructed to complete the cognitive task this value fell to approximately 20%. This showed the influence auditory attention can have on the emergence of tinnitus in a quiet environment. The cognitive task provided a distraction for subjects meaning they did not attend to low sound levels in the room. Treatment and intervention measures for tinnitus often work on developing distraction techniques to assist sufferers [15].

Schaette et al. [13] investigated acoustic deprivation via a different approach by having subjects with normal hearing and no previous experience of tinnitus wear an ear plug in one ear for a week attenuating sound by between 10 to 30dB. They found that 78% of subjects developed tinnitus. Once the ear plug was removed the phantom auditory perceptions went away. In this way it shows that the reduced stimulation received in the auditory system through hearing loss, or a hearing imbalance between ears, is substantial enough to induce an amplification in the auditory gain generating hyperactivity in regions of acoustic deprivation. This concept was reinforced with a computational model showing hyperactivity induced by adaptive gain.

B. The auditory system in changing sound levels

The ear continually deals with dynamically changing environmental noise. Auditory neurons do not have the capacity to deal with the 120dB range of sound intensities that can be received by the ear. When placed in a sound proof chamber the ambient noise drops steeply from approximately 60dB to 15-18dB. To account for such changes in the sound intensity, the auditory system aims to stabilise the mean activity of the auditory pathway so that sound intensities appear relative to the volume of the ambient noise [16]. This phenomena has been shown in various stages of the auditory system including the auditory midbrain [17] and auditory nerve [18]. Wen et al. [18] found that adapting the activity of auditory nerve fibres around the mean sound intensity and the mean firing rate can model biological behaviour as observed in animal studies. The significance of this adaptation is thought to be the key in neural encoding of sound levels. Reducing the range of intensities the auditory system is tuned to deal with at a particular time allows for higher resolution and better discrimination between the sound levels of the environment around us that is dynamically changing. Although this concept has been used to model empirical data [18], [19], [20] the underlying mechanisms that facilitate this stabilisation of volume are unknown.

III. Methodology

In this section we define the auditory nerve and learning rules for the dynamic range adaptation of the auditory periph-

TABLE I
TERMS FOR AUDITORY NERVE FIRING RATE

Term	Meaning	Value
f_{sp}	Spontaneous firing rate	50Hz
I_{th}	Sound intensity threshold	0dB
f_{max}	Maximum firing rate	250Hz
P_{sp}	Probability of spontaneous activity	$\int_{-\inf}^{I_{th}} p_I(x)dx$

ery that will comprise our proposed model.

A. Modelling the auditory nerve

The auditory nerve fibre is modelled as a function of the sound intensity at the nerve fibres characteristic frequency [21]. A Gaussian probability density function $p_I(I)$ for a sound intensity I is given by

$$p_I(I) = \frac{\exp\left(\frac{(I-\mu)^2}{2\sigma^2}\right)}{\sqrt{2\pi\sigma^2}} \tag{1}$$

where the mean $\mu = 40$dB and the standard deviation $\sigma = 15$dB.

The firing rate of the auditory nerve fibre is assumed to be independent of the characteristic frequency. The firing rate of the auditory nerve fibre for a given sound intensity I is defined as

$$f(I) = \begin{cases} f_{sp} & \text{for } I < I_{th} \\ f_{sp} + (f_{max} - f_{sp}) \frac{\int_{I_{th}}^{I} p_I(x)dx}{1-P_{sp}} & \text{otherwise.} \end{cases} \tag{2}$$

Table I outlines the terms used in this calculation along with the parameter values used in simulations.

B. Dynamic range adaptation

Figure 1 is a schematic illustrating how dynamic range adaptation modules modulate the sound intensity and firing rate of the auditory nerve fibre. Sound intensity, I, enters the model and is adjusted relative to the previous sounds heard. The sound intensity is then adjusted towards the mean intensity denoted μ. The rate at which the intensity stabilises to the mean rate is controlled by η_1. Similarly the firing rate is calculated with the current sound intensity input, the effective intensity I_e, and adjusted to tend towards the mean firing rate $\bar{f}$. Finally the signal is passed through a positive half-rectifier to ensure the firing rate f_{out} is not negative.

a) Sound intensity adaptation: The sound intensity I that stimulates the auditory nerve at time t is adapted relative to the previous intensities that have stimulated the ear. The sound intensity relative to the previous sound intensity is given by

$$I_{rel}(t) = I(t) - I(t-1) I_e(t-1) \tag{3}$$

where the effective sound intensity, I_e, is given by

$$I_e(t) = \eta_1 (\mu - I_{rel}(t)) + I_{rel}(t). \tag{4}$$

Here $\eta_1 = 0.25$ is the learning or adaptation rate and μ is the mean sound intensity as before. The effective intensity, I_e, is the input to the firing rate adaptation module.

b) Firing rate adaptation: The second stage of the dynamic range adaptation uses the mean firing rate of the auditory nerve fibres to stabilise the firing rate of the auditory nerve over time. The mean firing rate of the auditory nerve is calculated by

$$\bar{f} = P_{sp} f_{sp} + \frac{1}{2}(1 - P_{sp})(f_{max} + f_{sp}). \tag{5}$$

Using the values outlined in Table I gives a mean firing rate is 145Hz which is achieved with a sound intensity of $\mu = 40$dB. To calculate the firing rate after adaptation, the firing rate, $f(I_e)$, is first calculated according to equation (2). The firing rate is then updated using the rule

$$f(I) = \eta_2 \left(\bar{f} - f(I_e)\right) + f(I_e) \tag{6}$$

where the adaptation rate is controlled by $\eta_2 = 0.25$. To ensure that the firing rate is non-negative a positive half-rectifier is applied to the signal such that

$$f_{out} = [f]_+ = \max(0, f). \tag{7}$$

C. Accounting for attention

The top down mechanisms that control auditory sensitivity are poorly understood but are thought to be responsible for controlling auditory attention. In this work we facilitate the influence of top down mechanisms as modelling attention is beyond the scope of this paper. We introduce additional scalars A_1 and A_2 in a multiplicative sense with η_1 and η_2 respectively transforming equations (4) and (6) into

$$I_e^{(A)} = A_1 \eta_1 (\mu - I_{rel}(t)) + I_{rel}(t) \tag{8}$$

and

$$f^{(A)}(I) = A_2 \eta_2 \left(\bar{f} - f(I_e)\right) + f(I_e). \tag{9}$$

In this approach we set A_1 and A_2 to be 0 when attention is away from the information coming into the auditory nerve and 1 otherwise. These parameters are included so that in future work the attention can be modelled in a more sophisticated way.

D. Simulations

The proposed model was implemented using MATLAB 2013a (Mathworks Inc, Natick, MA). The same stimulus was used in every paradigm. A train of sound intensities is fed into the model simulating seven minutes of sound in time steps of 1 second. The first minute consists of continuous stimulation at 55dB equivalent to the volume of general speech. This is followed by 3 minutes of quiet or silence (15dB) simulating a person inside a sound proof room. The final three minutes consist of continuous sound at 55dB representing a person coming out of acoustic deprivation and back to normal environmental noise of people speaking. Four paradigms were used to test the behaviour of the proposed model:

1) Dynamic range adaptation with $A_1 = A_2 = 1$,
2) Adaptive firing rate with suppressed sound intensity adaptation i.e $A_1 = 0, A_2 = 1$,
3) Adaptive sound intensity with suppressed adaptive firing rate i.e $A_1 = 1, A_2 = 0$,

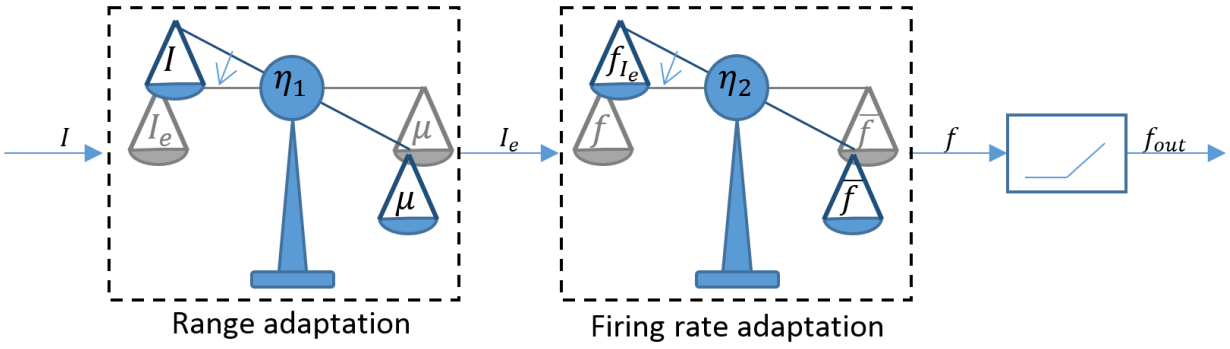


Fig. 1. The two adaptation modules provide stabilisation of the sound intensity and firing rate about their respective steady states. The final module represents a positive half-rectifier.

4) Suppression of both forms of adaptation setting $A_1 = A_2 = 0$ to simulate no attention to the information contained in the auditory nerve.

IV. Results

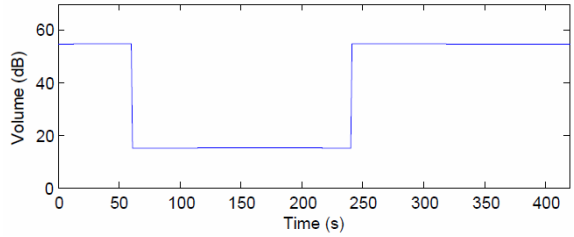
The following sections outline the results of the four paradigms investigated This section concludes with a brief discussion of the findings.

a) Paradigm 1: In this simulation we set the attention parameters $A_1 = A_2 = 1$. Figure 2b shows the effective firing rate over the simulation period. As the model is subjected to the same volume for a sustained period of time (between 60 seconds and 240 seconds and 241 seconds to the end) the effective sound intensity returns to its steady state. Additionally the firing rate adaptation causes a subsequent modulation towards the mean firing rate (Figure 2c). The result means that during the period of silence, despite the sound intensity being 15dB the firing rate is above the spontaneous rate; hyperactivity indicative of tinnitus. The perceived loudness is proportional to the firing rate of the auditory nerve fibres firing rate [22]. Therefore the increase in auditory nerve activity gives rise to an increase in the loudness perceived.

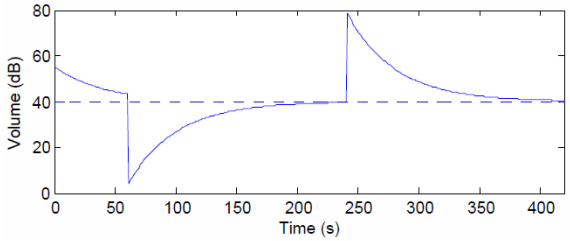
b) Paradigm 2: In this simulation we set the attention parameters $A_1 = 0$ and $A_2 = 1$. Figure 3b shows that the effective firing rate does not change while the parameter A_1 suppresses the adaptation. However the firing rate adaptation causes hyperactivity during the period of silence (Figure 3c).

c) Paradigm 3: With attention parameters $A_1 = 1$ and $A_2 = 0$ we simulated suppression of the firing rate adaptation. Figure 4b shows that the effective sound intensity modulates over time tending towards the mean sound intensity while the parameter A_2 suppresses the firing rate adaptation. Despite the suppression the already increased sound intensity causes hyperactivity during the period of silence (Figure 4c).

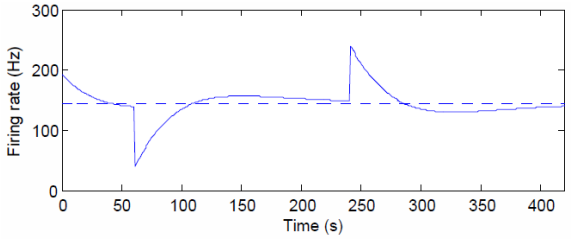
d) Paradigm 4: Figure 5 shows the how the effective sound intensity and firing rate do not change from their natural levels when simulating a lack of attention towards the characteristic frequency. Consequently no hyperactivity is developed (Figure 5c).



(a) Sound intensity of input in decibels (dB).

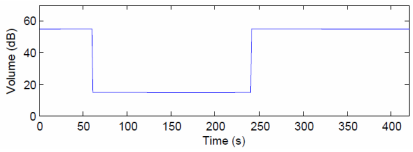


(b) The effective sound intensity of the input after the initial stage of adaptation. The mean sound intensity is marked with a dotted line.

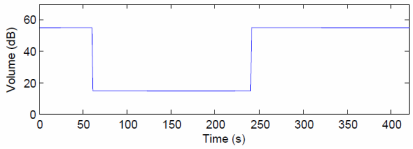


(c) The firing rate of the auditory nerve after both sound intensity and firing rate adaptation. The mean firing rate is marked with a dotted line.

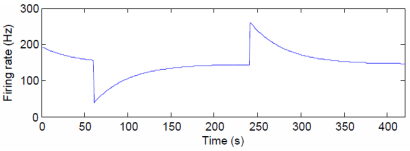
Fig. 2. Hyperactivity in the auditory nerve during the period of reduced sound intensity between 61 and 120 seconds.



(a) Input Stimulus.

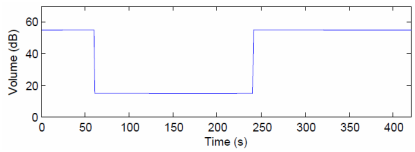


(b) Effective sound intensity (without sound intensity adaptation).

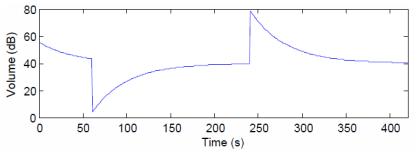


(c) Auditory nerve firing rate (with firing rate adaptation)

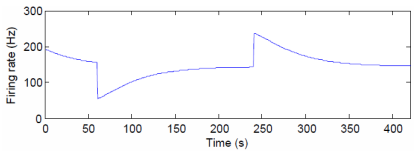
Fig. 3. Increased auditory nerve activity during silence even with no increase in effective sound intensity.



(a) Input Stimulus.

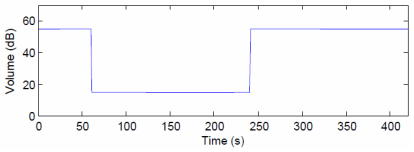


(b) Effective sound intensity (with sound intensity adaptation).

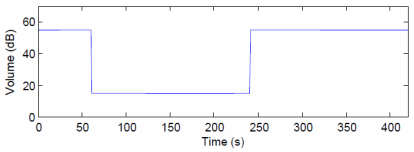


(c) Auditory nerve firing rate (without firing rate adaptation)

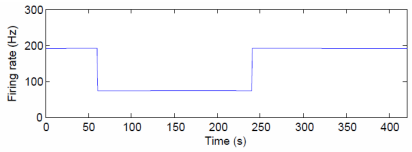
Fig. 4. Increased auditory nerve activity during silence despite no firing rate adaptation.



(a) Input Stimulus.



(b) Effective sound intensity (without sound intensity adaptation)



(c) Auditory nerve firing rate (without firing rate adaptation)

Fig. 5. No increased activity as no attention directed towards auditory nerve suppresses adaptation.

A. Discussion

a) Hyperactivity generated in silence: In three of the simulations, seen in Figures 2c, 3c and 4c, hyperactivity developed within minutes of acoustic deprivation onset. The resultant firing rate activity corresponds to that of approximately 40dB of sound despite the true levels being 15dB. Therefore this activity would generate a more noticeable effect and a phantom sound would be perceived. The only paradigm to display no hyperactivity occurred when simulating a lack of attention. This is to be expected from empirical results [11]. The main difference between the first paradigm, where full dynamic range adaptation was allowed to modulate both the sound intensity and firing rate, and paradigms two and three, where only one modulation affected the outcome, was how the firing rate returned to its stable state. The resultant dual adaptation produces a hyper-excitation of the auditory nerve (roughly 120 seconds into stimulation) as would be expected in a biological system (Figure 2c). Without this dual adaptation there is no hyper-excitation as shown in Figures 3c and 4c. Thus the the dual adaptation is necessary to mimic biological behaviour.

b) Tinnitus related activity disappears when normal sound levels are restored: When normal ambient noise of 55dB is reinstated on the fourth minute an effective increase of 40dB in the sound intensity causes a steep jump in the auditory nerve firing rate (Figure 2c). It should be noted that the firing rate in response to 55dB when coming out of silence is significantly more than the firing rate before the silence. Consequently the ambient noise would be perceived as louder than the equivalent volume at the beginning of the simulation. The firing rate then begins to reduce to an average level in response to the sustained input of 55dB. Over time as the effective volume drops by only 15dB it is reasonable to assume that there would not be a noticeable change in the environmental noise and consequently normally activity is resumed at this point.

V. Conclusion and Future Work

Tinnitus has consistently appeared in people with normal hearing once subjected to acoustic deprivation [9], [10], [12], [11], [13]. To date the generation of tinnitus behaviour has not been modelled for cases of acoustic deprivation and normal hearing. Our results show that dynamic range adaptation used to stabilise the mean sound intensity and mean firing rate over time can develop hyperactivity in the auditory system in periods of acoustic deprivation. Additionally this behaviour goes away once normal environmental levels are restored. Our work also shows the requirement for the dual adaptation of relative sound intensity and firing rate adaptation to mimic the steady state recovery of a biological system.

This paper deals with the adaptation of a healthy auditory system in response to acoustic deprivation. This does not account for the generation of tinnitus following hearing damage as modelled in other works (for example, see [14] for a review). Future work is aimed at extending this model to include the generation of tinnitus in both acoustic deprivation and no hearing loss as well as hearing loss and normal environmental noise levels. This work has contributed a new temporal element to previous auditory nerve models that have been used to model tinnitus [21]. According to this model, and in agreement of experimental work [9], the generation of tinnitus behaviour occurs on a time scale of minutes once acoustic deprivation has been induced. The precise timing of tinnitus onset following acoustic deprivation is not known and

would be challenging to investigate experimentally without introducing expectation.

The time scale of the adaptation is determined by the two rate parameters η_1 and η_2 relative to the time step used in simulations. The exact values of these parameters are unknown for two reasons. Firstly, as mentioned above the precise timing of the onset of tinnitus is challenging to determine from experimental findings without introducing an expectation or attentional bias. Secondly, it would not be possible to uncouple the individual volume and firing rate adaptation in experimental work. Consequently, there are infinitely many possible solutions for η_1 and η_2. For convenience, the learning rates are kept equal as there is no evidence to suggest that one adaptation occurs faster than the other. Parameter estimation was used to determine the values of η_1 and η_2 so that the stabilisation of the mean firing rate occurred on average around three minutes (in line with reported studies [11], [9]).

Our model includes the facilities to investigate the effect of attention on generating tinnitus behaviour. When simulating the attention away from the characteristic frequency associated with the auditory nerve, that is $A_1 = A_2 = 0$, there was no development of abnormal activity in the auditory system. However our model does not capture the complexity of attention at this time; a more complex and non-binary model of attention would be a particularly interesting advancement. The consequence of having an accurate model of attention could have real implications for the suppression of tinnitus activity in sufferers.

This model focusses on the early stages of the auditory periphery and in particular the auditory nerve as it is the main signal carrier of the sound input to the rest of the auditory system. Future work will extend this model to incorporate additional components of the auditory system, such as the inferior colliculus within the auditory midbrain [8], [19]. Additionally, the tonotopic ordering of frequencies in the auditory system could easily be implemented to include an estimate of the pitch of the perceived tinnitus similar to previous work [14].

In conclusion this model replicates hyperactivity of the auditory system after minutes of acoustic deprivation for normal hearing as observed experimentally. The basic tinnitus behaviour emulated in this modelled is suppressed once normal environmental conditions are restored due to dynamic range adaptation. The basic mechanisms of attention, either attend or not attend, are incorporated into this model and illustrate that it may be possible to stop the onset of adaptation by top down mechanisms as postulated by previous studies using a cognitive task [11].

Acknowledgement

This work was completed under a PhD studentship supported by the Department of Education and Learning (DEL), Northern Ireland (UK). The authors would like to thank Roland Schaette for his help while implementing the auditory nerve model used in this work.

References

[1] L. Sanchez, "The epidemiology of tinnitus," *Audiological Medicine*, vol. 2, no. 1, pp. 8–17, 2004.
[2] J. Hobson, E. Chisholm, and A. El Refaie, "Sound therapy (masking) in the management of tinnitus in adults," *The Cochrane Library*, 2010.
[3] P. Martinez-Devesa, R. Perera, M. Theodoulou, and A. Waddell, "Cognitive behavioural therapy for tinnitus," *Cochrane Database Syst Rev*, vol. 9, 2010.
[4] P. J. Jastreboff and M. M. Jastreboff, "Tinnitus retraining therapy (trt) as a method for treatment of tinnitus and hyperacusis patients," *JOURNAL-AMERICAN ACADEMY OF AUDIOLOGY*, vol. 11, no. 3, pp. 162–177, 2000.
[5] L. Del Bo and U. Ambrosetti, "Hearing aids for the treatment of tinnitus," *Progress in brain research*, vol. 166, pp. 341–345, 2007.
[6] M. Sereda, M. Edmondson-Jones, and D. A. Hall, "Relationship between tinnitus pitch and edge of hearing loss in individuals with a narrow tinnitus bandwidth," *International journal of audiology*, vol. 54, no. 4, pp. 249–256, 2015.
[7] N. Weisz, T. Hartmann, K. Dohrmann, W. Schlee, and A. Norena, "High-frequency tinnitus without hearing loss does not mean absence of deafferentation," *Hearing research*, vol. 222, no. 1, pp. 108–114, 2006.
[8] R. Schaette and D. McAlpine, "Tinnitus with a normal audiogram: physiological evidence for hidden hearing loss and computational model," *The Journal of Neuroscience*, vol. 31, no. 38, pp. 13 452–13 457, 2011.
[9] M. F. Heller and M. Bergman, "Tinnitus aurium in normally hearing persons," *Ann Otol Rhinol Laryngol*, vol. 62, no. 1, pp. 73–83, 1953.
[10] D. A. Tucker, S. L. Phillips, R. A. Ruth, W. A. Clayton, E. Royster, and A. D. Todd, "The effect of silence on tinnitus perception," *Otolaryngology–Head and Neck Surgery*, vol. 132, no. 1, pp. 20–24, 2005.
[11] K. A. B. Knobel and T. G. Sanchez, "Influence of silence and attention on tinnitus perception," *Otolaryngology–Head and Neck Surgery*, vol. 138, no. 1, pp. 18–22, 2008.
[12] L. Del Bo, S. Forti, U. Ambrosetti, C. Serena, D. Mauro, G. Ugazio, B. Langguth, and A. Mancuso, "Tinnitus aurium in persons with normal hearing: 55 years later," *Otolaryngology–Head and Neck Surgery*, vol. 139, no. 3, pp. 391–394, 2008.
[13] R. Schaette, C. Turtle, and K. J. Munro, "Reversible induction of phantom auditory sensations through simulated unilateral hearing loss," *PLoS One*, vol. 7, no. 6, p. e35238, 2012.
[14] R. Schaette and R. Kempter, "Computational models of neurophysiological correlates of tinnitus," *Frontiers in systems neuroscience*, vol. 6, 2012.
[15] F. Gerhards and D. Brehmer, "[distraction and relaxation training in acute tinnitus: effects of a complement to otorhinolaryngological treatment]." *HNO*, vol. 58, no. 5, pp. 488–496, 2010.
[16] B. Wark, B. N. Lundstrom, and A. Fairhall, "Sensory adaptation," *Current opinion in neurobiology*, vol. 17, no. 4, pp. 423–429, 2007.
[17] I. Dean, N. S. Harper, and D. McAlpine, "Neural population coding of sound level adapts to stimulus statistics," *Nature neuroscience*, vol. 8, no. 12, pp. 1684–1689, 2005.
[18] B. Wen, G. I. Wang, I. Dean, and B. Delgutte, "Dynamic range adaptation to sound level statistics in the auditory nerve," *The Journal of Neuroscience*, vol. 29, no. 44, pp. 13 797–13 808, 2009.
[19] I. Dean, B. L. Robinson, N. S. Harper, and D. McAlpine, "Rapid neural adaptation to sound level statistics," *The Journal of Neuroscience*, vol. 28, no. 25, pp. 6430–6438, 2008.
[20] M. S. Zilany and L. H. Carney, "Power-law dynamics in an auditory-nerve model can account for neural adaptation to sound-level statistics," *The Journal of Neuroscience*, vol. 30, no. 31, pp. 10 380–10 390, 2010.
[21] R. Schaette and R. Kempter, "Development of tinnitus-related neuronal hyperactivity through homeostatic plasticity after hearing loss: a computational model," *European Journal of Neuroscience*, vol. 23, no. 11, pp. 3124–3138, 2006.
[22] P. U. Diehl and R. Schaette, "abnormal auditory gain in hyperacusis: investigation with a computational model," *Frontiers in neurology*, vol. 6, 2015.