Homeostatic plasticity and spike-time-dependent plasticity in computational modeling of tinnitus generation and its management by sound therapy

Hirofumi Nagashino, Yohsuke Kinouchi, Ali A. Danesh, and Abhijit S. Pandya

***Abstract*—**Tinnitus is considered as an auditory perception in one ear, both ears or in the head without any external source. A very effective method of tinnitus management is referred to as sound therapy. Computational and dynamical models with plasticity using a neural oscillator or neuronal networks have been proposed by our team in order to investigate mechanisms of tinnitus generation and the clinical effects of sound therapy. In the present paper, two models are proposed, a neuronal network model with homeostatic plasticity (HP) and another model with both HP and spike-time-dependent plasticity (STDP). The results are compared in reference to their effects on inhibition of oscillations as a model of tinnitus management. The outcome data show that the model with both HP and STDP is more robust than the model with STDP only or HP only in the sense that oscillation can be inhibited in a larger range of the intensity of external constant input.

***Keywords*—** tinnitus, neuronal network model, sound therapy, spike-time-dependent plasticity, homeostatic plasticity, oscillation, inhibition

I. Introduction

TINNITUS is considered as an auditory perception in one ear, both ears or in the head without any external source [1]. Tinnitus is not a real sound; it is an actual brain electrical activity. This annoying auditory phenomenon is generated by many factors such as noise exposure and/or chemical and medicinal exposure. Other contributing factors include aging, metabolic and endocrine disorders, neurologic atypicalities, and cardiovascular disturbances.

For many years, tinnitus has been considered as a difficult-to-manage clinical condition. Throughout the history many scientists and clinicians have attempted to find ways to help those who suffer from this condition. Tinnitus generation is still a mystery. There are many proposed theories that have attempted to explain its generation. Some of these theories relate the percept of tinnitus to hyperactivity of the auditory cortex and some attribute the changes in inhibitory and excitatory neurotransmitters of the auditory system. The most favored tinnitus generation theory describes tinnitus as a product of brain reorganization as a consequence of hearing loss [2]. Based on the tonotopic organization maps of the auditory cortex, it has been shown that those cortical areas that represent the corresponding frequency region of hearing loss are "invaded" by adjacent frequencies. This reorganization and neuroplasticity has been credited in generating tinnitus.

Additionally, the mechanisms of tinnitus generation have been described based on neurophysiological models [3], [4]. The role of neural plasticity to explain the neural correlates of tinnitus also has been reported [5]-[11]. Auditory electrophysiological recordings have addressed the thalamic plasticity via top down modulation [12]. A scientific literature review showed that cochlear damage decreases auditory nerve activity and this change leads to plastic adjustments, a shift in the balance of excitation and inhibition, and increase of spontaneous firings in the central auditory system [8], [9]. Neuroimaging studies such as magnetic resonance imaging (MRI) have shown structural brain changes in individuals with tinnitus [13].

Computational modeling has been applied for better understanding of tinnitus [14]-[17]. There are many areas in the brain that contribute to tinnitus generation; however, it has been shown that the thalamo-cortical network is important for its generation [13], [18]. A neural network model of thalamo-cortical correlates with plasticity toward understanding of the tinnitus has been reported [14]. A tinnitus model based on the neurophysiological model of Jastreboff [3], combined with the adaptive resonance theory of cognitive sensory processing [19] has been proposed for identification of neural correlates of tinnitus [20]. Using models of corticothalamic feedback dynamics, the effect of auditory selective attention on the tinnitus decompensation has also been investigated [20], [21].

A variety of therapeutic approaches for tinnitus have been used for the management of tinnitus [22]-[24]. These include use of medications, supplemental vitamins and

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micronutrients, psychotherapy and biofeedback, electrical stimulation, transcranial magnetic stimulation, and more importantly and least invasively sound therapy or acoustic therapy. Tinnitus has many types and subcategories depending on what caused it. Attempts have been made to categorize tinnitus based on its characteristics which in turn can facilitate the selection of management methods [25].

The process of sound therapy is one of the most effective methods. The tinnitus patients who have gone under sound therapy protocol report diminished annoyance from tinnitus [26]. Potentially, patients may perceive a reduction in tinnitus loudness following acoustical stimulation through sound therapy. This cessation of tinnitus following the use of sound therapy has been termed "residual inhibition". Sound therapy employs a variety of stimuli such as music, white noise, narrow band noise and environmental sounds to facilitate the habituation process to tinnitus. The mechanisms of tinnitus management by sound therapy; however, have not been thoroughly clarified. Some attribute the success with sound therapy to brain plasticity [27] while others consider it a habituation process [28].

Previously we proposed computational and dynamical models employing a neural oscillator [15], [30], [31] or a neuronal network [32]-[35] in order to replicate tinnitus and its management by sound therapy. We have demonstrated that those models conceptually imitate tinnitus perception and exhibit tinnitus inhibition with sound. This inhibition is provided by applying a variety of input with constant amplitude, sinusoidal waveform or noise that represent the role of acoustic stimuli which are used for treatment of tinnitus. By employing these models we could inhibit the oscillations (i.e., tinnitus). This was accomplished by incorporating neural plasticity through parameters in a way that their values can be modified. By hypothesizing that the oscillation and the equilibrium in the model correspond to perception and inhibition of tinnitus, respectively, we reported that these phenomena could explain the fact that the habituated human auditory system temporarily halts perception of tinnitus following sound therapy. However, a model that has larger range of input intensity for inhibition of oscillation is preferable. In order to explore it, we propose a model with different plasticity in the present paper.

For plasticity of our previous models we employed Hebbian hypothesis [15], [30]-[33], or spike-timing-dependent plasticity (STDP) [34], [35] in one of the couplings between the components.

Hebbian hypothesis [36] has been adopted in a number of neural network models for many years. As a newer and biologically plausible hypothesis for synaptic plasticity in the nervous system, "spike-timing-dependent plasticity (STDP)", has been proposed [37]. It does not replace the idea of Hebbian hypothesis; however, it describes Hebbian synaptic plasticity more specifically. This hypothesis has been adopted in a number of computational models of neuronal networks [38].

As another hypothesis for the plasticity in the nervous system, homeostatic plasticity (HP) was proposed [39]. The HP is applied to nervous systems that require stability of the activities and its role has been widely investigated [40]. The role of HP in hearing loss-induced tinnitus has been investigated [41]. A computational model with HP for tinnitus with hearing loss has been proposed [17], [42]. That model, however, is not a dynamical system. Further modeling of a dynamical system for tinnitus with HP [43]-[45] is required.

In the present paper, we propose a dynamical model with HP. The current model has the same structure as the previous one [35]. It is composed of the model neurons described by simplified Hodgkin-Huxley equations [46]-[48]] as we employed in the previous studies [32]-[35]. The plasticity is given to inhibitory coupling [49] between neurons, which is based on the neurophysiological consideration [18].

We show the results of analysis of a neuronal network model with HP only and another model with both HP and STDP. We demonstrate the results of computer simulation of this model. The results show that the present model is more robust than the model with STDP only, which was reported in [35] and the model with HP only in the sense that oscillation can be inhibited in a larger range of the intensity of external constant input.

II. A Neuronal Network Model

The neuronal network model that we analyze in this paper is shown in Fig. 1. In the model the firing sequences in the nervous system are simulated. The present model only replicates the inhibition of tinnitus by external sound stimulation. Modeling the habituation would need much larger network configuration. The present model is a conceptually simplified system of a tinnitus generation network. However, we believe that the neural mechanism proposed here could form components of models involving large-scale neural correlates for providing a neurophysiological framework [2].

The model is composed of two excitatory neurons and one inhibitory neuron as shown in Fig. 1. This mechanism includes a positive feedback loop of the excitatory neurons E_1 and E_2 mutually coupled, and a negative feedback loop with the excitatory neuron E_1 and the inhibitory neuron I that are also mutually coupled. The negative feedback loop controls the firing rate. The mechanism can be bistable with a sustained firing state and a non-firing state.

The coupling strength between neurons is denoted by C_{ij} ($i, j \in \{1, 2, 3\}$). The neuron E_1 receives external stimuli S that is afferent signal due to the acoustic stimuli that are employed in sound therapy.

We express the dynamics of the model by a simplified version of Hodgkin-Huxley equations (HH) [46]-[48]. We employed it instead of HH to reduce the computational complexity and the related simulation time by reducing the number of state variables for each neuron from four to two.

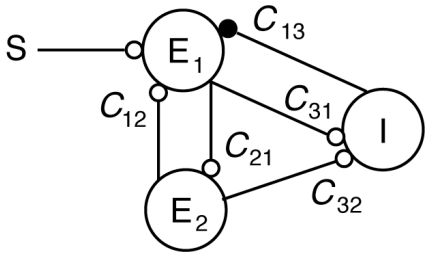


Fig. 1 A neuronal network model.

A. Formulation of the model without plasticity

We describe the basic dynamics of the model as

$$\frac{dv_1}{dt} = \frac{G(v_1,\, m_1,\, n_1,\, h_1) + C_{12}z_2 - C_{13}z_3 + D + S}{C_m}, \tag{1}$$

$$\frac{dh_1}{dt} = \alpha_h(v_1)(1-h_1) + \beta_h(v_1)h_1, \tag{2}$$

$$\frac{dv_2}{dt} = \frac{G(v_2,\, m_2,\, n_2,\, h_2) + C_{21}z_1}{C_m}, \tag{3}$$

$$\frac{dh_2}{dt} = \alpha_h(v_2)(1-h_2) + \beta_h(v_2)h_2, \tag{4}$$

$$\frac{dv_3}{dt} = \frac{G(v_3,\, m_3,\, n_3,\, h_3) + C_{31}z_1 + C_{32}z_2}{C_m}, \tag{5}$$

and

$$\frac{dh_3}{dt} = \alpha_h(v_3)(1-h_3) + \beta_h(v_3)h_3. \tag{6}$$

where v is the membrane potential, m, n and h are the variables associated with activation of sodium ion channel, inactivation of sodium ion channel and activation of potassium ion channel in the neuron E_1, E_2 or I. The functions $G(v,m,n,h)$, m and n are expressed as

$$G(v,m,n,h) = \bar{g}_{Na}m^3h(V_{Na}-v) + \bar{g}_K n^4(V_K - v) + \bar{g}_l(V_l - v) \tag{7}$$

$$m = \alpha_m(v)/\{\alpha_m(v) + \beta_m(v)\} \tag{8}$$

and

$$n = 0.8(1-h) \tag{9}$$

respectively. In the original HH model [41] m and n are expressed by differential equations. In the simplified version that we employ in the present study, m is expressed by the function of the membrane potential v, as Eq. (8), and n is expressed by the function of the variable h, as Eq. (9), since the change of m and n rapidly converges compared with v and h. The functions $\alpha_m(v)$ and $\beta_m(v)$ in Eq. (8) are expressed respectively as

$$\alpha_m(v) = 0.1(25-v)\Big/\left\{e^{(25-v)/10} - 1\right\} \tag{10}$$

and

$$\beta_m(v) = 4\,e^{-v/18} \tag{11}$$

Functions $\alpha_h(v)$ and $\beta_h(v)$ in Eq. (2), (4), (6) are expressed respectively as

$$\alpha_h(v) = 0.07\,e^{-v/20} \tag{12}$$

and

$$\beta_h(v) = 1\Big/\left\{e^{(30-v)/10} + 1\right\}. \tag{13}$$

The parameters of the neuron model were fixed as C_m=1[μF/cm^2], $\bar{g}_{Na} = 120[\mathrm{mS/cm^2}]$, $\bar{g}_K = 36[\mathrm{mS/cm^2}]$, $\bar{g}_l = 0.3[\mathrm{mS/cm^2}]$, V_{Na}=115[mV], $V_K = -12$ [mV], V_l=10.6 [mV], based on the values in the original HH model [41].

The output of the neuron j to its postsynaptic neurons is denoted by z_j and expressed as function of the membrane potential v_j as

$$z_j = \begin{cases} 1 & (v_j \geq 6) \\ 0 & (v_j < 6) \end{cases}. \tag{14}$$

Moreover, a bias term D is introduced in the equation of the membrane potential v_1 of the neuron E_1, Eq. (1) in order to enable the neurons to elicit sustained firings keeping z_j at 0 when the neurons are not firing.

B. Introduction of plasticity

All the couplings in the model could have plasticity. Based on the physiological consideration in [xx] and for simplicity of the modeling, we assume in the current model that only single coupling of inhibition between neurons has plasticity. In the present model both HP and STDP are introduced.

C. Formulation of HP

We incorporate HP in the present model as a dynamical process. We assume that the plastic coupling coefficient C_{13} changes depending on the activity of the neuron E_1. The dynamics of C_{13} is modeled in such a way that the higher the activity of E_1 is, the larger C_{13} grows. When E_1 does not fire, C_{13} converges to C_S. The change of the synaptic coefficient due to HP is expressed as

$$\frac{dC_{13}}{dt} = \frac{-C_{13} + C_S + pz_1}{\tau}, \tag{15}$$

where C_S is the stationary value of C_{13} when E_1 does not fire, p is a parameter that gives the quantity of the modification of C_{13}, and τ is the time constant of C_{13}.

D. Formulation of spike-time-dependent plasticity (STDP)

Secondly we incorporate spike-time-dependent plasticity (STDP) in the present model. We assume that the inhibitory coupling, the coupling strength from the neuron I to the neuron E_1, C_{13}, also has STDP. The key idea of this hypothesis on inhibitory synapses is that when the postsynaptic neuron fires before the presynaptic neuron, the synaptic strength becomes stronger (long term depression), and when the presynaptic neuron fires before the postsynaptic neuron fires, the synaptic strength becomes weaker (long term potentiation). Hence, C_{13} decreases when E_1 fires after I fires, and increases when I fires after E_1 fires. The time difference between firings of neuron I and neuron E_1, t_{31}, is defined as

$$t_{31} = t_3 - t_1 \tag{16}$$

where t_1 and t_3 are the latest firing times of E_1 and I, respectively as shown in Fig. 2. The value of coupling strength with plasticity C_{13} at time $t+\Delta t$, $C_{13}(t+\Delta t)$, is given by addition of the value at time t, $C_{13}(t)$, and the change of C_{13}, ΔC_{13},

$$C_{13}(t+\Delta t) = C_{13}(t) + \Delta C_{13}\ , \tag{17}$$

where Δt is the time step of calculation, and ΔC_{12} is given as

$$\Delta C_{13} = -\frac{dC_{13MAX}}{T_1} t_{31} + dC_{13MAX} \tag{18}$$

when $0 < t_{31} < T_1$,

$$\Delta C_{13} = -\frac{dC_{13MIN}}{T_2} t_{31} - dC_{13MIN} \tag{19}$$

when $-T_2 < t_{31} \le 0$, and

$$\Delta C_{13} = 0, \tag{20}$$

when $t_{31} \le -T_2$ or $t_{31} \ge T_1$.

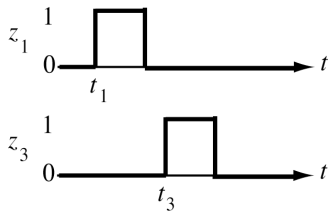


Fig. 2 Definition of firing time.

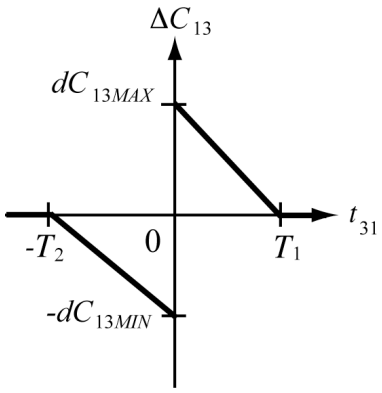


Fig. 3 Modeling of STDP in inhibitory coupling strength C_{13}.

III. Results

We demonstrate the results of computer simulation of the model. Throughout the simulation the parameter values $D = 18$ [μA/cm^2] , $C_{21} = 10$, $C_{31} = 10$, $C_{32} = 20$ were employed.

A. Analysis of the model without input or plasticity

Without stimulation or plasticity, the model has two stable solutions, an oscillatory state by sustained firings and a non-firing state. They are bistable for a parameter region.

First, we performed the simulation changing the value of the coupling coefficient C_{12} by one in the range $0 < C_{12} \le 30$ with the value C_{13}=10. The non-firing state exists for any value of C_{12} in the range. On the other hand, the oscillatory state exists when $C_{12} \ge 23$. That is, the two solutions coexist when $C_{12} \ge 23$. It corresponds to the clinical fact that a number of patients of tinnitus claim that they do not always hear sound when there is no external sound. The larger C_{12} brings the larger basin of the oscillatory solution in the state space of the model in the region.

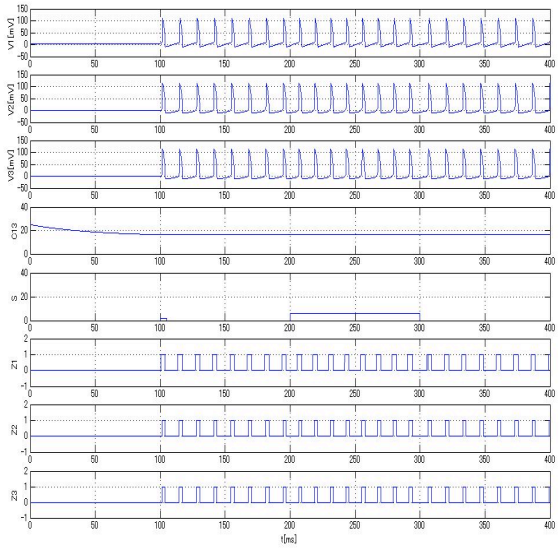
Secondly, we performed the simulation changing the value of the coupling coefficient C_{13} by one in the range $0 < C_{13} \le 30$ with the value C_{12}=25. The non-firing state exists for any value of C_{12} in the range. On the other hand, the oscillatory state exists when $0 \le C_{13} \le 22$ and $27 \le C_{13} \le 30$. Also in this case the two solutions coexist when $0 \le C_{13} \le 22$ and $27 \le C_{13} \le 30$.

B. Analysis of the model with input and HP only

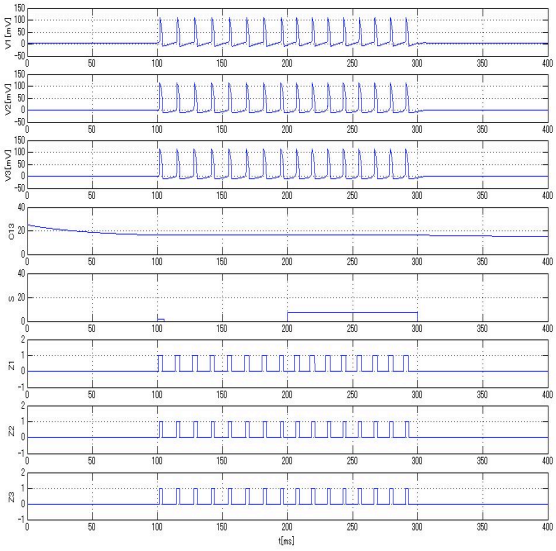
The inhibition of oscillation by constant input with amplitude I as stimulus S to neuron E_1 was examined with plasticity. The parameter values $C_S = 15$ and $\tau = 50$ [ms] were employed for plasticity. The time scale of the change of the synaptic strength is much smaller than the clinical process. It was arranged so that the simulation is completed in a reasonable time. The initial value of the coupling strength C_{13} is denoted by C_0. Simulations were performed where the parameter C_0 = 25, in which only non-firing solution exists stably. The amplitude I of the input was changed by 1 μA/cm^2 in the range of $0 < I \le 15$ [μA/cm^2]. Fig. 4 and Fig. 5 show the examples of simulation results. In the figures, the rows illustrate the membrane potentials v_1, v_2, v_3, the coupling strength C_{13}, input S, output of the neurons z_1, z_2 and z_3, and time difference between firings of neuron I and neuron E_1, t_{31}, respectively from the top.

At first the neurons do not fire since the model is in the parameter region where only non-firing solution exists stably. The coupling strength C_{13} decreases towards its stationary value $C_S = 15$. The model enters the parameter region where both non-firing and firing solutions. However, the non-firing state is sustained since the state of the model system is in the basin of the non-firing solution in the state space of variables v_j and h_j. For a short period of time from $t = 100$ [ms], appropriate input S constant with time whose amplitude is appropriate is applied. Then the neurons start firing since the state variables move to the basin of the firing solution. From $t = 200$ to 300 [ms], input S constant with time whose amplitude is I is applied to the neuron E_1 for 100ms. The neurons continue to fire for the period. After the input is removed at $t = 300$[ms], the behavior of the model depends upon the amplitude of the input which is applied from $t = 200$ to 300 [ms].

As shown in Fig. 4, when p=5, the input with I=7 [μA/cm^2] for 100ms makes the network stop the oscillation after the input is removed, while the input with I=6 [μA/cm^2] fails to stop the oscillation. For p=5, the amplitude I=7 or 8 [μA/cm^2] was required for inhibition of oscillation. When p=10, the input with I=9 [μA/cm^2] for 100ms makes the network stop the oscillation after the input is removed, while the input with I=10 [μA/cm^2] fails to stop the oscillation, which is shown in Fig. 5. For p=10, the input with I=7, 8 or 9 [μA/cm^2] was required for inhibition of oscillation. When p=1, the input with I=6, 7 or 8 [μA/cm^2], and when p=20, the input with I=9 or 10 [μA/cm^2], respectively, for 100ms was required to make the network stop the oscillation after the input is removed. These results are summarized in Table 1.



(a)

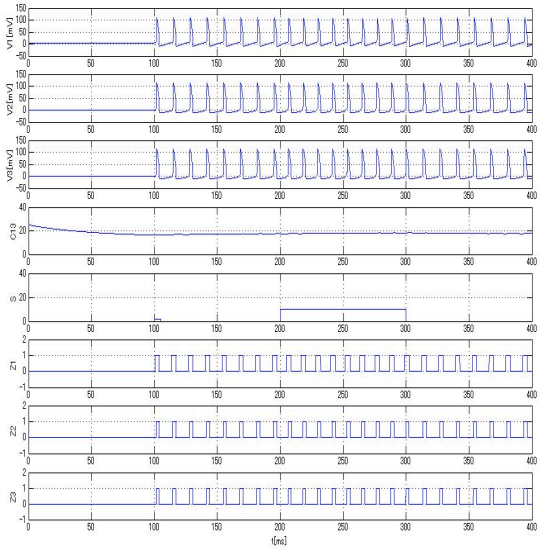


(b)

Fig. 4 Simulation results in the model with homeostatic plasticity only, C_0=25, p=5, (a) an unsuccessful result, I=6 [μA/cm^2], (b) a successful result, I=7 [μA/cm^2].

Table 1. Inhibition of oscillation in the model with homeostatic plasticity only. O: Inhibition is accomplished. X: Inhibition is not accomplished.

p	I[μA/cm^2]							
	4	5	6	7	8	9	10	11
1	X	X	O	O	O	X	X	X
5	X	X	X	O	O	X	X	X
10	X	X	X	O	O	O	X	X
20	X	X	X	X	X	O	O	X



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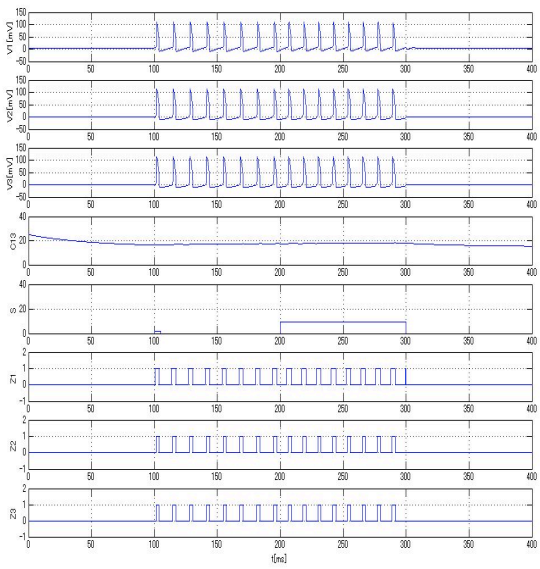
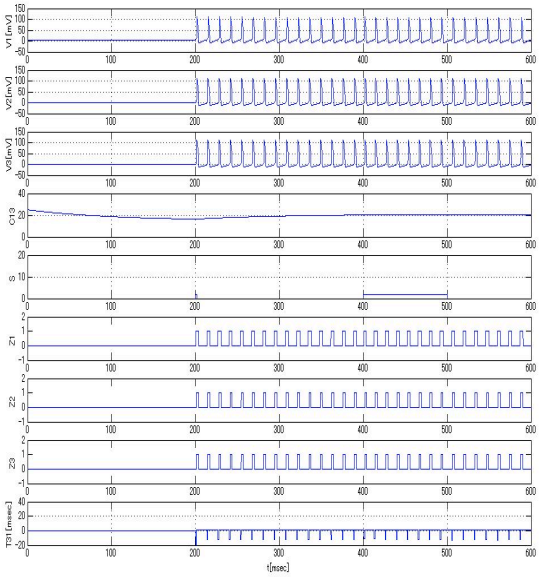


(b)

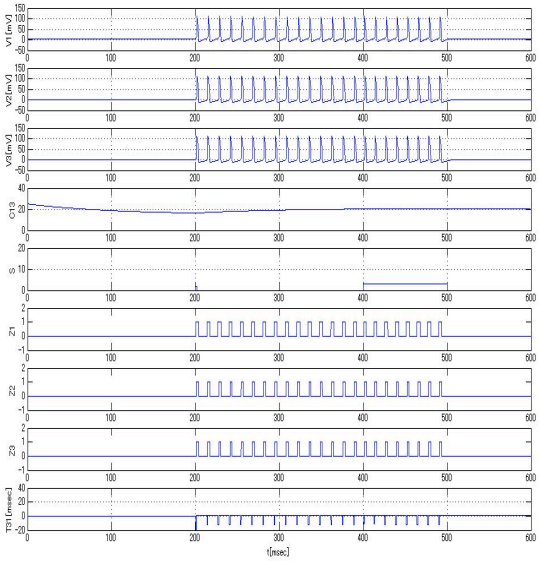
Fig. 5 Simulation results in the model with homeostatic plasticity only, C_0=25, p=10, (a) an unsuccessful result, I=10 [μA/cm^2], (b) a successful result, I=9 [μA/cm^2].

C. Analysis of the model with input, HP and STDP

In order to examine the effect of additional STDP on the oscillation in the model, temporarily constant input with amplitude I as stimulus S was supplied to neuron E_1 in the model with plasticity of the inhibitory coupling strength C_{13}. The parameter values $dC_{13MAX} = 0.001$, $dC_{12MIN} = 0.001$, $T_1 = 15\,[\mathrm{ms}]$, $T_2 = 5\,[\mathrm{ms}]$, and $\Delta t = 0.01\,[\mathrm{ms}]$ were employed for STDP. For HP C_S=15, p=10 and $\tau = 50\,[\mathrm{ms}]$ were employed. The time scale of the change of the synaptic strength is much smaller than the clinical process.

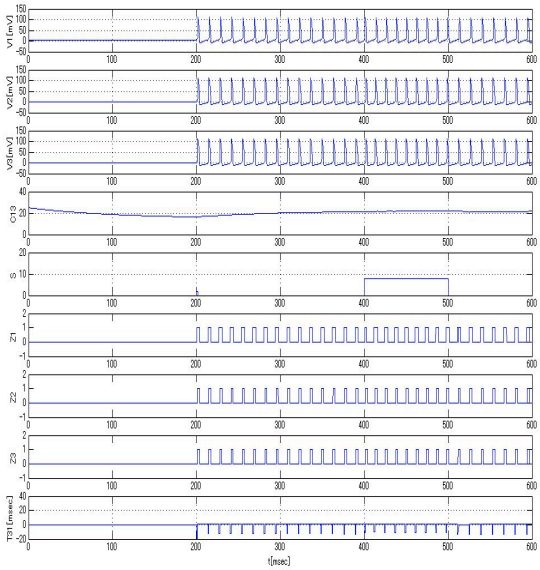


(a)



(b)

Fig. 6 Simulation results in the model with both homeostatic plasticity and spike-time-dependent plasticity, $C_0 = 25$, p=1, (a) an unsuccessful result, I=2 [μA/cm^2], (b) a successful result, I=3 [μA/cm^2].



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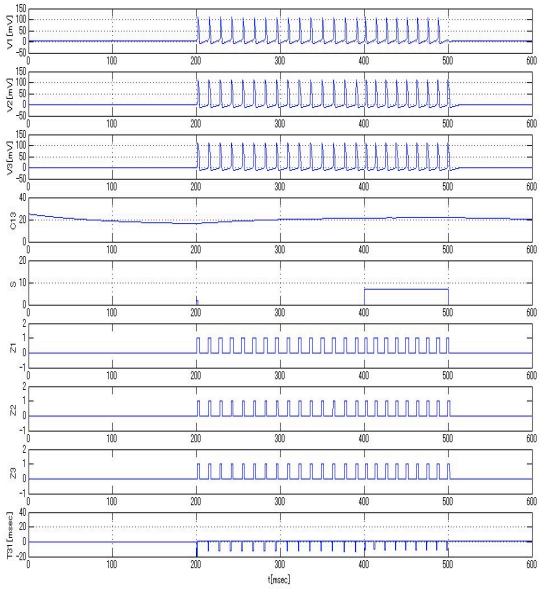

(b)

Fig. 7 Simulation results in the model with both homeostatic plasticity and spike-time-dependent plasticity, $C_0 = 25$, p=10, (a) an unsuccessful result, I=8 [μA/cm^2], (b) a successful result, I=7 [μA/cm^2].

It was arranged so that the simulation is completed in a reasonable time. Simulations were performed with the initial value of the coupling strength C_{13}, C_0=25, at which no oscillation occurs without external input. The amplitude I of the input was changed by 1 μA/cm^2 in the range of $0 < I \leq 15$ [μA/cm^2].

Fig. 6 and Fig. 7 show examples of simulation results. In the figures, the rows illustrate the membrane potentials v_1, v_2, v_3, the coupling strength C_{13}, input S, output of the neurons z_1, z_2 and z_3, and time difference between firings of neuron I

Table 2. Inhibition of oscillation in the model with both homeostatic plasticity and spike-time-dependent plasticity. O: Inhibition is accomplished. X: Inhibition is not accomplished.

p	I [μA/cm^2]							
	2	3	4	5	6	7	8	9
1	X	O	O	O	X	X	X	X
5	X	O	O	O	X	X	X	X
10	X	X	O	O	O	O	X	X
20	X	X	X	X	O	O	O	X

and neuron E_1, t_{31}, respectively from the top. At first from time t=0[ms] to t=200[ms] input S=0. The coupling strength C_{13} decreases according to HP so that the firing of neuron E_1 is easier to occur. It decays to the value in which oscillatory solution also exists. At t=200[ms] a trigger input with very short duration and with intensity I_t is given to E_1. The constant input I was applied to E_1 for 100[ms] from t=400[ms] to 500[ms]. The neurons fire with higher rate for this period. Consequently it gives an effect of plasticity that is different from the one given while no input is applied. From t=500[ms] to t=600[ms] input is not applied. As shown in Fig. 4 and Fig. 5, when I_t=1.3[μA/cm^2], the input with I=4 [μA/cm^2] for 100ms makes the network stop the oscillation after the input is removed, while the input with I=3 [μA/cm^2] fails to stop the oscillation. For the inhibition of oscillation the input amplitude of a suitable range is required.

As shown in Fig. 6, when p=5, the input with I=3 [μA/cm^2] for 100ms makes the network stop the oscillation after the input is removed, while the input with I=4 [μA/cm^2] fails to stop the oscillation. For p=5, the amplitude I=3, 4 or 5 [μA/cm^2] was required for inhibition of oscillation. When p=10, the input with I=7 [μA/cm^2] for 100ms makes the network stop the oscillation after the input is removed, while the input with I=8 [μA/cm^2] fails to stop the oscillation, which is shown in Fig. 5. For p=10, the input with I=4, 5, 6 or 7 [μA/cm^2] was required for inhibition of oscillation. When p=1, the input with I=3, 4 or 5 [μA/cm^2], and when p=20, the input with I=6, 7 or 8 [μA/cm^2], respectively, for 100ms was required to make the network stop the oscillation after the input is removed. Table 2 summarizes these results.

For I_t=1.3, the amplitude I=5, 6 or 7 [μA/cm^2] was required for inhibition of oscillation. Table 1 demonstrates the inhibition is accomplished or not with different values of I_t and I. With larger I_t, smaller range of I was appropriate for inhibition.

The plastic coupling coefficient C_{13} increases slightly during the stimulation.

D. Discussion

In summary, it was observed that the model succeeds in demonstrating the effect of the introduction of the external stimulus S. This leads to termination of firing of the neurons.

Tables 1 and 2 show that the range of input intensity in which the oscillation is inhibited in the model with STDP and HP is wider than the range of input intensity in the model with HP only for some values of the parameter p.

Comparing the results in the model with STDP and HP and those in the model with STDP only described in [xx], we can see that the range of input intensity in which the oscillation is inhibited in the model with both STDP and HP is wider than the range of input intensity in the model with STDP only.

It can be stated that that the model with both STDP and HP is more robust than the model with STDP only or HP only in the sense that oscillation can be inhibited by a larger range of the intensity of external input.

In the models with STDP only, HP only, and both STDP and HP, the plastic coupling coefficient does not change to the value in which the firing solution does not exist during the stimulation. The oscillation stops due to the change of the state of the model as well as the change of the coupling coefficient by the input. Hence, further investigation of simulation or different modeling is required in order to reproduce the inhibition of oscillation by synaptic plasticity only.

IV. Conclusion

The results of computer simulation of a computational and dynamical neuronal network model with HP only and the one with both HP and STDP for tinnitus generation and its management by sound therapy were described in this paper. The structure of the models is the same as that of the model with STDP only that was previously proposed.

It has been shown through computer simulations that the model with both STDP and HP is more robust than the model with STDP only or HP only in the sense that oscillation can be inhibited by a larger range of the intensity of external input that can be hypothesized as activation by sound stimulus in sound therapy.

In the present model, the inhibition of the oscillation was realized by both the change of the plastic coupling strength and the change of the state of the model by supplying the input. More investigation for improvement of the model is required in order to demonstrate that only the synaptic plasticity brings the inhibition of oscillation and the model is more robust in the input amplitude for inhibition of oscillation.

Our future work will expand this model so that it can more effectively explain underlying physiology of tinnitus, and explore better stimulation for its inhibition through sound therapy techniques.

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