

# THE EFFECT OF AGING ON COCHLEAR AMPLIFIER: A SIMULATION APPROACH USING A PHYSIOLOGICALLY-BASED ELECTRO-MECHANICAL MODEL OF THE COCHLEA

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## 1. INTRODUCTION

Age-related hearing loss is one of the top three most common chronic health conditions affecting individuals aged 65 years and older (Frisina, 2010). Models have been exploited as powerful analytical tools to sharpen our understanding of various mechanisms and systems. Many of the existing models for human peripheral auditory system use computational elements such as filters, etc. to represent each stage of the system regardless of tiny biological details (Irino and Patterson, 2001); therefore, they cannot be used for differentiating the underlying physiological roots of various cochlear hearing impairments.

Here the electrical, acoustical, and mechanical elements of the cochlea are explicitly integrated into a transmission-line model. The aim is to choose these elements to have a physiological interpretation of the human cochlea insofar as is known<sup>a</sup>. As a result, the model enables fundamental simulation of specific cochlear lesions such as metabolic presbycusis.

The prevailing hypothesis that high-frequency hearing loss in older adults may be due to metabolic presbycusis whereby there is a reduction in the endocochlear potential (EP) as a result of age-related cellular degeneration in the lateral wall of the cochlea (Schmiedt, 1996; Dubno *et al.*, 2008; Lang *et al.*, 2010). Besides of aging, similar degenerations are observed in patients with diabetes type 2 millitus (Fukushima *et al.*, 2006). This physiologically-based model is used to quantitatively validate this hypothesis.

## 2. MODELING METHOD

A sound pressure field in the air is transmitted via the outer and middle ear to the inner ear. It causes the stapes to vibrate resulting in a travelling wave along the organ of Corti propagating from base towards apex (Bekesy, 1960). The organ of Corti rests on the basilar membrane (BM); the displacements on BM trigger an active electro-mechanical element - outer hair cell (OHC) – which boosts the passive vibrations. The amplified vibration is converted to neural signals by the inner hair cells (IHCs).

<sup>a</sup> In spite of the new measurement techniques, this is still a challenge remembering that human cochlea is covered by bones and is rather inaccessible for real-time measurements; therefore, animal data plays an important role for creating reasonable assumptions of physiological details.

In order to study this complex mechano-electro-acoustical system the cochlear duct is assumed to be ‘N’ discrete partitions extending from the stapes on the left to the apex on the right. These partitions are structurally coupled to each other via longitudinal components; forming a transmission line as shown in fig 1. Each cochlear partition consists of passive loads (BM, RL and TM) together with an active component (OHC). Each passive load (BM, RL and TM) is modeled by a classical second-order ‘mass-spring-damper’.

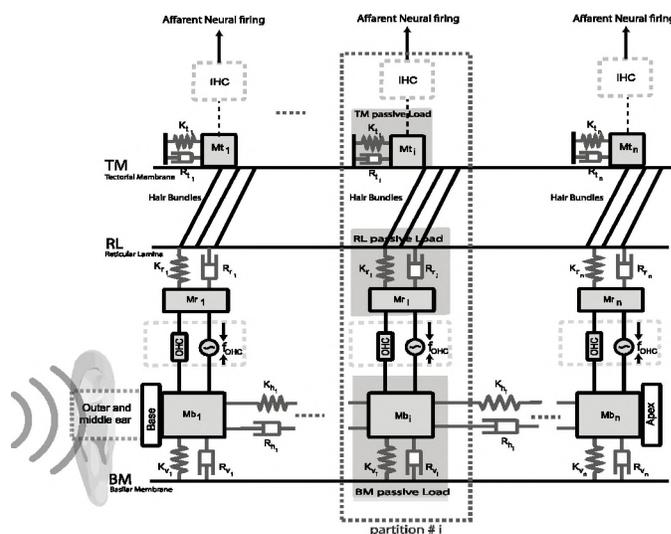


Figure 1: The transmission line consisting of ‘N’ vertical partitions.

### 2.1 Active processes: Cochlear amplifier

The OHC stretches against RL on one side and BM on the other side (Kim and Mountain, 2011) pulling them together. This molecular force generator -acting between BM and RL- is known as somatic motility. Somatic motor is triggered by the receptor current produced by the MET mechanism. Stria vascularis, in turn, supplies the necessary charge for MET by secreting K<sup>+</sup> into the cochlear endolymph of scala tympani. This 3-stage system is depicted in fig 2; it is then solved for the parameters.

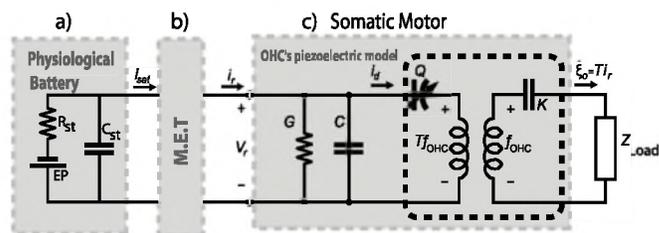


Figure 2: *a*) stria vascularis is modeled by its body resistance and capacitance ( $R_{st}$  and  $C_{st}$  respectively) together with an ideal voltage source (EP). Stria vascularis functions as a 'battery' providing the whole cochlear amplifier with the necessary electrical charge *b*) The MET mechanism which plays the role of a motor driver for the somatic motor. *c*) Circuit model of the OHC somatic motor according to piezoelectric model of Liu and Neely (2009).

## 2.2 Metabolic presbycusis

As a result of age-related cellular degeneration in the lateral wall of the cochlea, stria vascularis is not able to maintain the maximum EP (89mV) anymore. As the EP decreases, the MET produces less receptor current which, according to the 3-stage circuit of fig 2, eventually leads to a decline in the force/displacement generated by the somatic motor.

## 3. RESULTS

Figure 3 illustrates how the cochlear amplification is affected by a decrease in EP. The results are illustrated at 30% and 70% of cochlear length from the base to compare how the amplification declines while the EP decreases from 89mV to its half value (44.5mV).

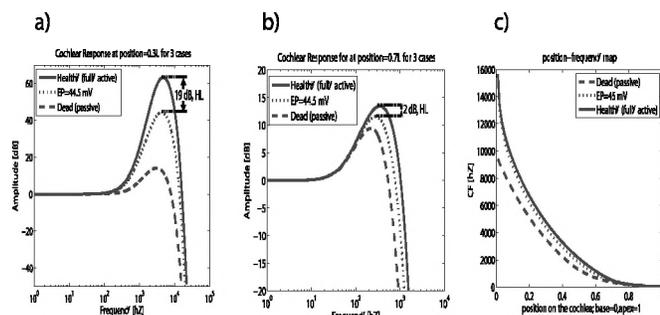


Figure 3: The effect of EP degeneration on magnitude and sensitivity of cochlear amplification (a, b) and position-frequency (c). Note that these are simulated at a low intensity corresponding to a 120 nm displacement of RL, according to small signal analysis. *a*) magnitudes at location 30% of total cochlear length from the base. *b*) at 70% of total length from the base *c*) illustrates how the CFs are shifted backwards as a result of EP degeneration.

When the EP is decreased to its half value, there is a 19-dB loss at the peak; CF is also shifted downwards from 4700 Hz to 4212 Hz. Once the active processes are totally dead (passive cochlea) the curve is flattened, the amplification is significantly decreased to only 14.12 dB and the CF is further shifted backwards to 3026 Hz. A similar decline is seen in fig 3.b however there is only a 2-dB loss at the peak of the curve and the CF is shifted backwards from 340 Hz to 271 Hz as a result of a 50% decrease in EP.

## 4. DISCUSSION

Comparing fig 3.a with fig 3.b indicates that the decrease in EP affects cochlear amplifications in higher frequencies more than it does in lower frequencies. This is consistent with Dubno *et al.* (2008) where they argue that the reason why presbycusis is associated with high frequencies is that cochlear amplifications is greater in higher frequencies than in lower frequencies therefore the lack of amplification is

significantly felt in higher frequencies. Figure 3 also reveals another aspect of cochlear amplification: The cochlear position-frequency map changes when the active processes are deteriorated. The CFs of the curves tend to move backwards in a presbycusis cochlea. This is consistent with Robles and Ruggero (2001) where they indicate that in a passive cochlea ( $EP=0$ ) the CFs are shifted backwards.

Clinical tests can be performed to further improve the parameters and predictions of the model. Moreover, this biologically-based model is developed with the aim of being connected to models of nervous pathways and more central parts of the auditory system in a signal-cognitive manner as suggested by Stenfelt and Ronnberg (2009).

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