\chapter{Description of Human Ear Anatomy}

\label{sec:human-ear-anatomy-model}

\phantomsection

\section{ Calculation of Basilar Membrane Velocity }

\label{sec:bm-calculation-model}

Our work implements the \citealt{furst2015} model of the mammalian ear, which is composed of an outer ear, a middle ear, and an inner ear. The outer

ear includes the pinna, the ear canal, and the ear drum. The middle ear is an air-filled cavity

behind the ear drum, which includes three small ear bones called the ``ossicles.’’ The inner ear includes

a snail-shaped structure called the cochlea (see schematic drawing in Figure \figref{fig:cochlear-model}). The sound is

directed by the outer ear through the ear canal to the eardrum. When sound strikes the ear

drum, the movement is transferred through the three bones of the middle ear to a flexible tissue

called the oval window, finally reaching the upper fluid-filled ducts of the cochlea (see \cref{fig:cochlear-model}). The upper cochlear ducts are called the ``scala vestibuli,’’ and the bottom duct is referred to as

the ``scala tympani.’’ The space between the top and bottom ducts is called the ``scala media.’’

\begin{figure}[ht]

\setlength{\subfigcapmargin}{.1in}

\centering

\includegraphics[width=3in]{figs/ear-anatomy}

\caption{Drawing of human ear anatomy from Ref. \cite{HumanEarAnatomy}.}

\label{fig:human-ear-detail-anatomy}

\end{figure}

The task of the middle ear is to match the impedance of the sound pressure in the air with that of the fluid. Movement of the fluid inside the upper cochlear duct results in a pressure difference

between the upper and lower ducts. This pressure difference in turn causes the basilar

membrane (the membrane that separates the scala tympani and scala media) to move.

\begin{figure}[ht]

\setlength{\subfigcapmargin}{.1in}

\centering

\mbox{\subfigure[cochlea cross section]{\includegraphics[width=3in]{figs/Cochlea-crosssection}

\label{fig:cochlea-cross-section}}

\subfigure[cochlea diagram ]{\includegraphics[width=3in]{figs/cochlea\_drawing\_fixed}

\label{fig:cochlea-diagram}}

}

%}

\caption{Structure of cochlea.}

\label{fig:cochlea-diagrams}

\end{figure}

\begin{figure}[ht]

\setlength{\subfigcapmargin}{.1in}

\centering

\includegraphics[width=3in]{figs/cochlea\_model\_2\_new}

\caption{Cochlear model of applied pressures.}

\label{fig:cochlear-model}

\end{figure}

Two types of auditory receptor cells inhabit the scala media: the inner hair cells (IHCs) and outer hair cells (OHCs). The

defining feature of these cells is a hair bundle on the top of each cell. The hair bundle comprises

dozens to hundreds of stereocilia, which are cylindrical actin-filled rods. The stereocilia are

immersed in endolymph, which is a fluid rich in potassium and characterized by an endocochlear

potential of +80 mV. The stereocilia move with the displacement of the BM, and their

deflection mechanically opens gated ion channels that allow any small, positively charged ion

(primarily potassium and calcium) to enter the cell. The influx of positive ions from the

endolymph into the scala media depolarizes the cell, resulting in a receptor potential. The roles

played by the OHCs and IHCs in the functioning of the cochlea are very different. While the OHCs act

as local amplifiers, the IHCs innervate the auditory nerve. The OHCs lay on the BM, and their upper part is embedded in a gel-like membrane called the tectorial membrane

(TM). An increase in the OHC receptor potential decreases its length \cite{Brownell1985}, which in

turn enhances the BM movement. The hair bundles of the IHC move freely in the scala media.

The change in their receptor potential then opens voltage-gated calcium channels that release

neurotransmitters at the basal end of the cell, which in turn trigger action potentials in the attached

nerve.

Modeling the human ear requires a detailed model of the cochlea and the middle and outer

ears \cite{cohen2004int,Saboddd2013682,Zwislocki1950,zweig1976cochlear,Borsboom1980485,Furst1982}.

A common approach is to model the inner ear as a one-dimensional structure

with the cochlea regarded as an uncoiled structure with two fluid-filled compartments

with rigid walls that are separated by an elastic partition called the basilar membrane (BM). The cochlear

partition, whose mechanical properties are describable in terms of point-wise mass density,

stiffness, and damping, is regarded as a flexible boundary between the scala tympani and the scala

vestibuli. Thus, at every point along the cochlear duct, the pressure difference $P(x, t)$ across

the partition drives the partition’s velocity. By applying fundamental physical principles such

as the conservation of mass and the dynamics of deformable bodies, the differential equation

for P is obtained as \cite{furst2015}

\begin{equation}

\label{eq:pressure-relate-to-speed}

\frac{\partial^2 P(x,t)}{\partial x^2} = \frac{2\rho \beta}{A} \frac{\partial^2 \xi\_{BM}(x,t)}{\partial t^2},

\end{equation}

where $\xi\_{BM}$ is the BM displacement, $A$ is the cross-sectional area of the scala tympani and

scala vestibuli, $\beta$ is the BM width, and $\rho$ is the density of the fluid in both the scala vestibuli

and the scala tympani. The pressure on the BM ($P\_{bm}$) is a result of both the difference in fluid

pressure and the pressure caused by the OHCs ($P\_{ohc}$). The pressures of the

BM, TM, and OHC are related as given in \cite{Barzelay2011} \cref{fig:cochlear-model}, which can be interpreted as

\begin{equation}

\label{eq:pressure-equation}

\left .

\begin{array}{lll}

P\_{BM}(x,t) & = & P(x,t) + P\_{OHC}(x,t) \\

0 & = & P\_{OHC}(x,t) + P\_{tm}(x,t)

\end{array} \right\}.

\end{equation}

The mechanical properties of both BM and TM are simulated as second-order oscillators, which yields

\begin{equation}

\label{eq:ocillates-properties}

\left .

\begin{array}{lll}

P\_{bm} & = & M\_{bm}(x)\cdot \frac{\partial^2 \xi\_{bm}(x,t)}{\partial t^2} + R\_{bm}(x)\cdot \frac{\partial \xi\_{bm}(x,t)}{\partial t} + K\_{bm}(x)\cdot \xi\_{xm}(x,t) \\

P\_{tm} & = & M\_{tm}(x)\cdot \frac{\partial^2 \xi\_{tm}(x,t)}{\partial t^2} + R\_{tm}(x)\cdot \frac{\partial \xi\_{tm}(x,t)}{\partial t} + K\_{tm}(x)\cdot \xi\_{tm}(x,t)

\end{array} \right\},

\end{equation}

where $K\_{bm}$, $K\_{tm}$, $R\_{bm}$, $R\_{tm}$, $M\_{bm}$, and $M\_{tm}$ are the effective stiffness, damping, and mass per unit

area of BM and TM, respectively (see \cref{tab:Lambda-parameters}). The TM displacement is defined as $\xi\_{tm}$.

Since the OHCs lie between the two membranes, their displacement is

\begin{equation}

\xi\_{ohc} = \xi\_{tm} - \xi\_{bm}.

\end{equation}

Each OHC is modeled by two sections: the apical and basal parts. The apical part is directed

toward the endolymph of the gap between the TM and the reticular lamina (RL), while the

basolateral part is embedded in the perilymph next to the supporting cells, which are aligned

along the BM. Motion of the stereocilia of the OHCs caused by the relative displacement of the BM

and the TM affects the conductance of the apical part of the OHC, which in turn causes a

flow of potassium and calcium ions to the endolymph. Thus, a voltage drop develops across

the basal part of the OHC membrane \cite{Dallos2003}.

An OHC model is described by an equivalent electrical circuit in \cref{fig:cochlear-circuit} \cite{cohen2004int,Mountain1995}. The

apical part is described by its variable conductance ($G\_a \approx Î± â‹…\xi\_{ohc}$) and its constant capacitance

($C\_a$), while the basal part is described by its constant conductance and capacitance $G\_b$ and $C\_b$,

respectively. The electrical potential of the endolymph is $V\_{sm}=80$~mV, and the perilymph

resting potential is $\psi\_0 = âˆ’70 $~mV. Solving the equivalent electrical circuit by using Kirchhoff’s laws

yields the differential equation for $psi\_{ohc}$ (i.e., the membrane voltage of the OHCs):

\begin{equation}

\label{eq:membrane-voltage}

\frac{d \psi\_{OHC}}{dt} +\omega\_{OHC} (\psi - \psi\_0) = \eta \xi\_{OHC},

\end{equation}

where $\omega\_{ohc} \approx G\_b / C\_b =1000$~Hz, which represents the cutoff frequency of the OHC’s membrane

and $\eta =\alpha â‹…V\_{sm} / (C\_b + C\_a)=\text{const.}$ (see \cref{tab:Lambda-parameters}).

\begin{figure}[ht]

\setlength{\subfigcapmargin}{.1in}

\centering

\includegraphics[width=3in]{figs/CochlearCircuit}

\caption{Equivalent electrical circuit of outer hair cell}

\label{fig:cochlear-circuit}

\end{figure}

The length $\Delta l\_{ohc}$ of OHCs changes due to the electrical potential developed on the OHC membrane and is usually described as a sigmoid function \cite{Zheng,DallosHe2000,NeelyLyu}:

\begin{equation}

\label{eq:delta-lohc}

\Delta l\_{ohc} = \alpha\_s \frac{e^{-2 \alpha\_l \psi} - 1}{e^{-2 \alpha\_l \psi} + 1} = \alpha\_s tanh(-\alpha\_l \psi),

\end{equation}

where $\alpha\_l$ and $\alpha\_s$ are constants (see \cref{tab:Lambda-parameters}).

The pressure $P\_{ohc}$ developed by each OHC is obtained from the spring properties of an OHC.

We define $\gamma\_{ohc}(x)$ to be the OHC effective index; it represents the effective distribution of

OHCs along the cochlear partition. Therefore, the OHC pressure is

\begin{equation}

\label{eq:ohc-pressure}

P\_{ohc}(x,t) = \gamma\_{ohc}(x) \cdot K\_{ohc}(x) \cdot [\xi\_{ohc}(x,t) - \Delta l\_{ohc}(x,t)],

\end{equation}

where $K\_{ohc}$ is the OHC stiffness (\cref{tab:Lambda-parameters}). A cochlea with no active OHC is obtained by

$\gamma\_{ohc}(x) = 0$, whereas $0.5 \le \gamma\_{ohc}(x) \le 0.6$ yields an optimal cochlea that best fits the physiological data \cite{Barzelay2011}.

The ear model described by \crefrange{eq:pressure-equation}{eq:ohc-pressure} is solved by applying the initial boundary conditions, which are

\begin{equation}

\label{eq:pressure-initial-conditions}

\left .

\begin{array}{lll}

\left . \frac{\partial P}{\partial x} \right |\_{x=0} & = & 2\rho C \frac{\partial^2 \xi\_{ow}(t)}{\partial t^2} \\

P(L\_{co},t) & = & 0

\end{array} \right\},

\end{equation}

where $L\_{co}=3.5 $~cm is the cochlear length, $\xi\_{ow}$ is the oval window displacement, and $C\_{ow}$ is the

coupling factor of the oval window to the perilymph. To obtain $\xi\_{ow}$, the middle ear

model was applied \cite{Talmadge1998}, as expressed by

\begin{equation}

\label{eq:ow-equations}

\frac{d^2 \xi\_{ow}(t)}{dt^2} + \gamma\_{OW} \frac{d \xi\_{ow}(t)}{dt} + \omega\_{OW}^2 \xi\_{ow}(t) = \inv{\sigma\_{OW}} \left[P(0,t) + \Gamma\_{me} P\_{in}(t) \right],

\end{equation}

where $\sigma\_{OW}$, $\gamma\_{OW}$, and $\omega\_{OW}$ are the oval window’s areal density, resistance, and resonance frequency \citealt[(see Section~2)]{furst2015}. The mechanical gain of the ossicles is denoted by $\Gamma\_{me}$. The initial conditions are

\begin{eqnarray}

\label{eq:ow-initial-conditions}

\left .

\begin{array}{lll}

\xi\_{bm}(x,0) & = & \left . \frac{\partial \xi\_{bm}(x,t)}{\partial t} \right|\_{t=0}= 0, \\

\xi\_{tm}(x,0) & = & \left . \frac{\partial \xi\_{tm}(x,t)}{\partial t} \right|\_{t=0}= 0, \\

\xi\_{ow}(0) & = & \left . \frac{\partial \xi\_{ow}(t)}{\partial t} \right|\_{t=0}= 0,\\

\psi\_{OHC}(x,0) = \psi\_0.

\end{array} \right\}

\end{eqnarray}

\section{Model of Inner Hair Cell Auditory Nerve Synapse}

\label{sec:anr-calculation-model}

The BM motion is transformed into neural spikes of the auditory nerve by the

IHCs. The deflection of the hair-cell stereocilia opens mechanically gated ion channels

that allow small, positively charged ions (primarily potassium and calcium) to enter the

cell \cite{White}. Unlike many other electrically active cells, the hair cell itself does not fire an action

potential. Instead, the influx of positive ions from the endolymph in the scala media depolarizes

the cell, resulting in a receptor potential. This receptor potential opens voltage-gated

calcium channels, allowing calcium ions to enter the cell and trigger the release of neurotransmitters

at the basal end of the cell. The neurotransmitters diffuse across the narrow space between the

hair cell and a nerve terminal, where they then bind to receptors and thus trigger action

potentials in the nerve. In this way, the mechanical sound signal is converted into an electrical

nerve signal. The \ac{ihc} chronically leak $Ca^{+2}$, which causes a tonic release of neurotransmitter

to the synapses. This tonic release is thought to explain how hair cells

respond so quickly to mechanical stimuli. The quickness of the hair cell response may also be

due to that fact that it can increase the amount of neurotransmitter release in response to a

change of as little as $100\ \mu$V in membrane potential.

Many models have been developed to explain the IHC’s transduction abilities \cite{SumnerC}. Some

models focus on possible mechanisms for adaptation \cite{Zilany}.

One commonly simplified modeling approach to explain the IHC’s role in the auditory system

posits a nonlinear system that combines AC and DC responses followed by a random generator

that creates spike trains \cite{Zilany}. The model presented in this chapter is consistent with

these principles.

The BM displacement stimulates the IHC cilia to move, and its velocity $\dxi\_{ihc}$ corresponding to the

BM velocity $\dxi\_{bm}$ is given by the nonlinear function

\begin{equation}

\label{eq:dxi-ihc}

\begin{array}{lll}

\dxi\_{ihc} & = & \alpha\_1 tanh(\alpha\_2 \dxi\_{bm}) \\

& \approx & \alpha\_1 [\alpha\_2 \dxi\_{bm} - \frac{(\alpha\_2 \dxi\_{bm})^3}{3} + \frac{2(\alpha\_2 \dxi\_{bm})^5}{15} s] \\

& \approxeq & \dxi\_{bm}.

\end{array}

\end{equation}

Since the BM displacement in this model is nonlinear, as described by the mechanical model

above, we ignore the nonlinear terms in \cref{eq:dxi-ihc} and assume that $\alpha\_1 â‹…\alpha\_2 =1$ to approximate $\dxi\_{bm}$.

The electromechanical receptors located in the IHC membrane increase the

electrical potential ($\psi\_{ihc}$ of the IHC membrane. A common approach to model the IHC’s

role in the auditory system is based on a nonlinear system that combines AC and DC responses.

The DC level represents the firing responses without any synchrony to the input stimuli

and the AC level represents the synchronized firing response (typical at low frequencies). The

DC component includes a high-pass filter followed by a moving-average filter of 2 ms duration.

The AC component consists of a low-pass filter. To account for physiological

observations that demonstrate a reduction in synchronization as the frequency of the stimulus

increases, a low-pass filter with a cutoff frequency of 300~Hz was chosen with an attenuation of 1800~Hz.

In practice, $\psi\_{ihc}$ is obtained by

\begin{equation} \label{eq:psi-ihc}

\begin{array}{lll}

\psi\_{ihc}(x,t) & = & e^{\gamma\_{ihc}(x)} \Big\lbrack \eta\_{AC} \dxi\_{ihc}(x,t) \star h\_{ihc}(t) \\

& + & \eta\_{DC} \int\_{t-\delta}^{t} \big\{\dxi\_{ihc}(x,\tau) [1-h\_{ihc}(t)]\big\}^2 d\tau \Big\rbrack, \\

\end{array}

\end{equation}

where $x$ represents the location of the IHC along the cochlear partition, $h\_{ihc}(t)$ is the impulse

response of the low-pass filter that represents the IHC response, and $\eta\_{AC}$, $\eta\_{DC}$, and $\Delta$ are

constants \cref{tab:Lambda-parameters}. The parameter $\gamma\_{ihc}(x)$ represents the IHC efficiency index, which was

defined as a function of $x$, to allow variability in IHC efficiency along the cochlear partition.

For normal cochlea, we chose $\gamma\_{ihc}(x)=8$, which was found to match the experimental data. The

efficiency of the IHC is reduced with decreasing $\gamma\_{ihc}(x)$.

This IHC receptor potential opens voltage-gated calcium channels, allowing

calcium ions to enter the cell and trigger the release of neurotransmitters at the basal end of the cell.

The neurotransmitters diffuse across the narrow space between the hair cell and a nerve terminal where they

then bind to receptors and thus trigger action potentials in the nerve.

The neural activity in the auditory system is irregular since a specific neuron might respond

with a single spike or several spikes to a given stimuli. The origin of the stochastic

activity of neurons is poorly understood. This activity results in both intrinsic noise sources

that generate stochastic behavior on the level of the neuronal dynamics and extrinsic sources

that arise from network effects and synaptic transmission. Another source of noise that

is specific to neurons arises from the finite number of ion channels in a neuronal membrane patch \cite{furst2015}.

A number of different ways have emerged to describe the stochastic properties

of neural activity. One possible approach relates to the train of spikes as a stochastic point

process. For example, studies suggested that the spontaneous activity of the cochlear

nucleus can be described as a homogeneous Poisson process. Further investigations of the auditory system describe the neural

cochlear model for hearing loss

response as a non-homogeneous Poisson point process (NHPP) whose instantaneous rate

depends on the input stimuli \cite{furst2015,GrayPR,RiekeF}.

In the present chapter, we refer to the neural activity as \ac{nhpp}, and thus only the instantaneous

rate (IR) should be extracted. To derive the IR, we use the Weber--Fechner law,

which describes the relationship between the magnitude of a physical stimulus and the

intensity or strength that people feel. This type of relationship can be described by the differential equation

\begin{equation}

\label{eq:weber-fechner}

\mathit{d}P = K \frac{\mathit{d}S}{S},

\end{equation}

\subsection{ Auditory nerve response }

\label{sec:anr-response-theory}

where dP is the differential change in perception, $dS$ is the differential increase in the stimulus,

and $S$ is the stimulus at the given instant. Integrating the above equation gives $P =k â‹… lnS + C$. Let

us define $\lambda\_{AN}(x, t)$ as the IR obtained by the auditory fiber attached to location $x$ along the

cochlear partition, and let us further assume that it relates to the perception of the physical parameter.

Conversely, $\psi\_{ihc}(x, t)$ is the IHC electrical potential and corresponds to the stimulus.

Therefore, by applying the Weber--Fechner law, we obtained the relationship

$\lambda\_{AN}(x, t) = ln(\psi\_{ihc}(x, t)) + C$. However, the AN’s IR should satisfy

$0 < \lambda\_{spont} \le \lambda\_{AN}(x, t) \le \lambda\_{sat}$,

where $\lambda\_{spont}$ and $\lambda\_{sat}$ are the spontaneous and saturation rates of the

AN, respectively. Therefore, $\lambda\_{AN}(x, t)$ is given by

\begin{equation}

\label{eq:an-response}

\lambda\_{AN}(x,t) = min\{\lambda\_{sat},\lambda\_{spont}+max\{0,A\_{ihc}(x)\*ln(u(\psi\_{ihc}(x,t)))\}\}.

\end{equation}

where

$A\_{ihc}$ is constant (see \cref{tab:Lambda-parameters}).

In general, the AN response is divided between three types of fibers according to their

spontaneous rates: a high spontaneous rate (HSR) that usually codes low-level stimuli, a

medium spontaneous rate (\ac{msr}), and a low spontaneous rate (\ac{lsr}) that generally codes high

level stimuli. To include all types of ANs, we substitute into Eq. (13) the

relevant constants $[\lambda^{(H)}\_{spont}$, $A\_H; \lambda^{(M)}\_{spont}$, $A\_M; \lambda^{(L)}\_{spont}$, and $A\_L]$

for the \ac{hsr}, \ac{msr}, and \ac{lsr} that yield the

instantaneous rates $[\lambda^{(H)}\_{AN}(x,t)$, $ \lambda^{(M)}\_{AN}(x,t)$, and $\lambda^{(L)}\_{AN}(x,t)]$, respectively.

The different types of ANs are distributed uniformly along the cochlear partition, with frequency of 61\%, 23\%, and 16\% for high, medium, and low rates, respectively.

\section{Hearing Threshold Based on Auditory Nerve}

\label{sec:jnd-calculation-model}

The hearing threshold, which is defined as the lowest threshold of acoustic pressure sensation, is

usually determined by quantitative psychoacoustical experiments in which the human ability

to detect the smallest difference in the physical property of a stimulus is obtained. This difference

is referred to as a ``just-noticeable difference’’ (JND). In such experiments, a subject must

distinguish between two similar time-dependent stimuli $s(t, \alpha)$ and $s(t, \alpha + \Delta \alpha)$, where $\alpha$ is a

given physical property. The $JND(\alpha)$ is the minimum $\Delta \alpha$ a person can perceive. The

parameter Î± represents any physical property of the stimulus that can be measured, such as

frequency or level in monaural stimulus.

Comparing the behavioral JND and the neural activity is possible if one assumes that the neural

system estimates the measured parameters. \citealt{Siebert} obtained such a comparison by comparing the

JND of a single tone’s frequency and level to the neural activity of the AN. Siebert’s findings were based on the assumption that the AN response

behaves as a NHPP, and the brain acts as an unbiased optimal estimator of the physical

parameters. Thus, the JND equals the standard deviation of the estimated parameter and

can be derived by lower bounds such as the Cram\’er-Rao lower bound. \citealt{Heinz}

generalized Sibert’s results to a larger range of frequencies and levels.

In a psychoacoustical JND experiment, the JND is obtained when $d'=1$, which

is expressed by \citealt[Section~4]{furst2015}

\begin{equation}

\label{eq:evaluated-dtag}

d' = \frac{E[\hat{\alpha} \given \stalpha] - E[\hat{\alpha} \given (\stalpha + \Delta \alpha)]}{std(\hat{\alpha} \given \stalpha)} = \frac{\Delta \alpha}{std(\hat{\alpha} \given \stalpha)},

\end{equation}

where $E[\hat{\alpha} \given \stalpha] = \stalpha$, with $\stalpha$ being the true value of $\alpha$ and $\hat{\alpha}$ being the estimated value of $\alpha$. Therefore,

$d' = 1$ yields the relation $\Delta \alpha = std(\hat{\alpha} \given \stalpha)$, which implies

\begin{equation} \label{eq:jnd-std}

JND(\stalpha) = std(\halpha \given \stalpha).

\end{equation}

When the estimate is based on neural activity that behaves as a NHPP, there are two possible

ways to analyze the performance: The first way is referred to as ``rate coding’’ (RA), which

means that the performance is analyzed on the basis of the number of spikes. The second way

is referred as ``all information coding’’ (AI), indicating that, in addition to the number of spikes

in the interval, the timing of the discharge spikes is also considered.

Let us define $N(0,T)$ as the random variable that represents the number of spikes in the time

interval $[0, T]$ . For RA coding, the probability density function (pdf) of getting $n$ spikes in

the time interval $T$ is obtained by

\begin{equation}

\label{eq:ra-pdf}

P\_{RA}( N(0,T) = n ) = \inv{n!} \left[\int\_0^T \lambda (t,\alpha) \mathit{d}t\right]^{n} \exp \left\{-\int\_0^T \lambda (t,\alpha) \mathit{d}t \right\},

\end{equation}

where $\lambda(t,\alpha)$ is the instantaneous rate of the nerve fiber that depends on both the time $t$ and

the physical parameter $\alpha$. Given the RA pdf (\Cref{eq:ra-pdf}), the resulting Cram\’er-Rao lower bound

(CRLB) is obtained by \cite{ISO226}

\begin{equation} \label{eq:ra-crlb}

CRLB\_{RA}(\stalpha) = \left\{\frac{T}{\bar{\lambda}(\stalpha)}\left[\left.\frac{\partial\bar{\lambda}(\stalpha)}{\partial\alpha} \right|\_{\alpha=\stalpha}\right]^2\right\}^{-\inv{2}},

\end{equation}

where

$\bar{\lambda}(\stalpha) = \inv{T}\int\_0^T \lambda(t,\alpha) dt$ is average rate for the interval $[0,T]$.

For AI coding, the pdf for getting $n$ successive neural spikes at a

set of time instances is $t\_1,t\_2, \dots, t\_n$, where $0 \le t\_1<t\_2< \cdots < t\_n \le T$ is obtained by

\begin{equation}

P\_{AI}( N(0,T) = n,t\_1, \dots, t\_n ) = \inv{n!} \Pi\_{k=1}^n \lambda(t\_k,\alpha) \exp \left\{-\int\_0^T \lambda (t,\alpha) \mathit{d}t \right\},

\end{equation}

and the CRLB is

\begin{equation} \label{eq:ai-crlb}

CRLB\_{AI}(\stalpha) = \left\{\int\_0^T\inv{\lambda(t,\stalpha)}\left[\left.\frac{\partial\bar{\lambda}(\stalpha)}{\partial\alpha} \right|\_{\alpha=\stalpha}\right]^2\right\}^{-\inv{2}}.

\end{equation}

For the unbiased system, the rule is

\begin{equation} \label{eq:crlb-est}

std(\halpha \given \stalpha ) \geq CRLB\_{RA}(\stalpha) \geq CRLB\_{AI}(\stalpha).

\end{equation}

In an optimal unbiased system, the standard deviation of the estimator can achieve the lower

bounds. Since $JND(\stalpha)=std(\hat{\alpha} \given \stalpha)$ (\Cref{eq:jnd-std}), $JND(\stalpha)$ can be estimated by calculating

$CRLB\_{RA}(\stalpha)$ or $CRLB\_{AI}(\stalpha)$. Comparing the estimated thresholds to experimental results can

resolve the question of whether the brain estimates the auditory thresholds according to RA or

AI coding.

To apply the above-mentioned method for determining the auditory threshold, we

consider the responses of all 30\,000 AN fibers that innervate each ear. Because the AN

fibers are statistically independent \cite{Heinz2010}, the $d$ theorem can be applied, which yields

\begin{equation}

\label{eq:d-summary}

(d')^2 = \sum\_{m=1}^M (d'\_m)^2,

\end{equation}

where M is the number of nerve 1 fibers and $d'\_m$ is the $d$ (\Cref{eq:evaluated-dtag}) that was derived for the $m$th fiber. Moreover,

\begin{equation}

\label{eq:std-summary}

std(\halpha \given \stalpha ) = \inv{\sum\_{m=1}^M[std\_m(\halpha \given \stalpha )]^{-2}},

\end{equation}

where $std\_m(\halpha \given \stalpha )$ is the standard deviation of the estimator obtained by the $m$th fiber. Since

the threshold is obtained when $\mathit{d}'=1$, it implies that, in an optimal system,

\begin{equation}

JND(\stalpha ) = \inv{\sqrt{\sum\_{m=1}^M[CRLB\_m( \stalpha )]^{-2}}},

\end{equation}

where $CRLB\_m(\stalpha)$ is the CRLB of the $m$th fiber.

Let us define the number of fibers attached to each location along the cochlear partition as

$M (x)$.

Thus, $\sigma\_{x \in [0,L\_{co}]} M(x)=30\,000$, where $L\_{co}$ is the cochlear length. For every location, three

IRs were derived: $\lambda^{(H)}\_{AN}(x,t)$, $ \lambda^{(M)}\_{AN}(x,t)$, $\lambda^{(L)}\_{AN}(x,t)$ \cref{eq:an-response}, which correspond to the HSR, MSR, and LSR fibers, respectively. They are distributed uniformly along the cochlear partition with the

corresponding weights $w\_L$, $w\_M$, and $w\_H$ (see \cref{tab:Lambda-parameters}). Therefore,

\begin{equation}

\label{eq:fisher-sum}

JND(\stalpha) = \inv{\sqrt{F\_H + F\_M + F\_L}}

\end{equation}

such that

\begin{eqnarray}

\left .

\begin{array}{lll}

F\_H = & \sum\limits\_{x \in [0,L\_{co}]} & \sum\limits\_{m=1}^{\omega\_H \cdot M(x)}\left\{ CRLB\_m^{(H)}(\stalpha) \right\}^{-2}, \\

F\_M = & \sum\limits\_{x \in [0,L\_{co}]} & \sum\limits\_{m=1}^{\omega\_M \cdot M(x)}\left\{ CRLB\_m^{(M)}(\stalpha) \right\}^{-2}, \\

F\_L = & \sum\limits\_{x \in [0,L\_{co}]} & \sum\limits\_{m=1}^{\omega\_L \cdot M(x)}\left\{ CRLB\_m^{(L)}(\stalpha) \right\}^{-2}.

\end{array}

\right \}

\end{eqnarray}

Replacing CRLB in \cref{eq:fisher-sum} with the corresponding $CRLB\_{RA}(\stalpha)$ or $CRLB\_{AI}(\stalpha)$, $JND(\stalpha)$ is

estimated by either RA or AI coding.

To calculate both $CRLB\_{RA}(\stalpha)$ and $CRLB\_{AI}(\stalpha)$, the derivative of the instantaneous

rate should be derived. We used the following approximation:

\begin{equation}

\label{eq:crlb-approx}

\partial \lambda(t,\alpha) / \partial \alpha |\_{\alpha=\stalpha} \approx \frac{\lambda(t,\stalpha + \Delta \alpha) - \lambda(t,\stalpha)}{\Delta \alpha}.

\end{equation}

Therefore, in deriving $JND(\stalpha)$ for any stimulus $s(t, \stalpha)$, the IRs for both stimuli $s(t, \stalpha)$ and

$s(t, \stalpha + \Delta \alpha)$ should be calculated. Two types of thresholds are presented for tones in a quiet environment

and in the presence of noise. The quiet threshold is derived by substituting $\stalpha = 0$, which yields

$\lambda(t,\stalpha)=\lambda\_{spont}$. For the thresholds in the presence of noise, $s(t, \stalpha)$ is equal to the noise, and

$s(t, \stalpha + \Delta \alpha)$ is equal to the noise + tone with a level of $\Delta \alpha$.