Neural coding of ITD with bilateral cochlear implants: Effects of auditory experience

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ABSTRACT
Human bilateral cochlear implant users do poorly on tasks involving interaural time differences (ITD), a cue which provides important benefits to the normal hearing, especially in challenging acoustic environments. Yet the precision of neural ITD coding in acutely-deafened, bilaterally-implanted cats is essentially normal (Smith and Delgutte, 2007). One explanation for this discrepancy is that neural plasticity induced by the extended periods of binaural deprivation typically experienced by cochlear implant users degrades neural ITD sensitivity. To test this hypothesis, we recorded from single units in inferior colliculus (IC) of two groups of bilaterally-implanted, anesthetized cats: acutely-deafened cats, which had normal binaural hearing until experimentation, and congenitally deaf white cats, which received no auditory inputs until the experiment. Rate responses of only half as many neurons showed significant ITD sensitivity to low-rate pulse trains in congenitally deaf cats compared to acutely deafened cats. For neurons that were ITD sensitive, ITD tuning was broader and best ITDs were more variable in congenitally deaf cats. A signal detection model constrained by the observed physiology supports the idea that the degraded neural ITD coding resulting from deprivation of binaural experience contributes to poor ITD discrimination by human implantees.

INTRODUCTION
Increasing numbers of patients are receiving cochlear implants (CI) bilaterally with the goal of restoring the benefits of binaural hearing, including accurate sound localization and improved speech intelligibility in noise. Such benefits are indeed observed, but differ from those experienced by normal-hearing listeners in that they result primarily from acoustic head shadow effects: sound localization relies primarily on interaural level differences (ILDs) (van Hoesel, 2004), and improvements in speech reception in noise are largely consistent with attending to the ear with the best signal to noise ratio (Litovsky et al., 2006). Unlike normal-hearing listeners, bilateral CI users receive little benefit from interaural time difference (ITD) cues. In particular, they experience little “binaural squelch”, which requires neural processing of ITD, and is important for understanding speech when multiple competing sources are widely distributed in space, minimizing head shadow benefits (Zurek, 1992).

Sound localization and speech reception abilities of bilateral CI users are consistent with performance on basic psychophysical tasks. ILD discrimination is exquisitely fine (~0.2 dB) using direct stimulation, and is comparable to normal hearing (1-2 dB) when listening through clinical processors (van Hoesel and Tyler, 2003; Laback et al., 2004; Grantham et al., 2008). On the other hand, ITD discrimination is typically poorer than normal. For the best performers, just
noticeable differences (JNDs) in ITD are on the order of 50 μs for low-rate pulse trains, comparable to JNDs in normal hearing listeners presented with similar stimuli (Laback et al., 2007). However, ITD JNDs with bilateral CIs are highly variable across subjects, reaching several hundreds of msec in some listeners, and degrade rapidly for pulse rates above 300 pulses per second (pps) (Lawson et al., 1998; van Hoesel and Tyler, 2003; Laback et al., 2007; van Hoesel, 2007).

In contrast to the typically poor ITD discrimination exhibited by human CI listeners, neural ITD coding in the inferior colliculus (IC) is essentially as precise in acutely deafened, bilaterally-implanted cats as in normal-hearing cats (Smith and Delgutte, 2007). Specifically, the two groups of animals have similar distributions of neural best ITD and sharpness of ITD tuning.

An important difference between the human psychophysics and animal physiology is duration of deafness. The acutely-deafened cats studied by Smith and Delgutte (2007) had normal binaural hearing until experimentation, whereas human cochlear implant wearers typically experience long periods of auditory deprivation before receiving their first implant and, in many cases, an additional period of binaural deprivation before the second implantation. Such extended periods of deprivation may degrade neural ITD sensitivity by inducing changes in brainstem neural circuits involved in ITD processing.

We tested this hypothesis by making single-unit recordings in the IC of two groups of bilaterally-implanted, anesthetized cats: acutely-deafened cats (normal binaural hearing before experiment), and congenitally deaf white cats (no auditory experience). We found about half as many ITD-sensitive neurons in the congenitally deaf animals as compared to the acutely-deafened animals and differences in ITD tuning among the neurons that were ITD-sensitive. We developed a computational model to assess the effect of these physiological differences on psychophysical ITD discrimination.

**METHODS**

**Physiology**

Experiments were performed on 11 barbiturate-anesthetized cats, divided into two groups. Four cats were acutely deafened with ototoxic drugs (Xu et al., 1993) one week before implantation and experimentation. Seven were congenitally deaf white cats, in which the organ of Corti degenerates before the onset of hearing (West and Harrison, 1973; Heid et al., 1998). All cats were bilaterally implanted with 8-contact intracochlear electrode arrays (Cochlear Corp). Single-unit recordings were made from the IC using methods described previously (Smith and Delgutte, 2007), except that the recordings were made with 16-channel NeuroNexus probes.

Stimuli were low-rate (10-80 pps) trains of biphasic current pulses (anodic/cathodic, 50-μs/phase) delivered using a wide bipolar configuration. ITD was varied either statically or dynamically. Static-ITD pulse trains were 300 ms in duration, with a 300-ms silent interval between presentations. The ITD of each pulse was constant within a train, but varied across presentations. Dynamic-ITD stimuli were 1-s duration, continuous pulse trains with a pulse rate of 40 pps. Every pulse was presented at a different ITD to create a “binaural pulse beat” stimulus. For both static- and dynamic-ITD stimuli, ITD was typically varied from -2000 μs (ipsilateral-leading) to +2000 μs (contralateral-leading) in 200-μs steps.

Neural responses were measured as a function of ITD at stimulus levels between 1-6 dB re. single-pulse threshold. Static ITD stimuli were presented at pulse rates between 10-80 pps.
ITD sensitivity of single-unit rate responses was quantified using an analysis of variance-based signal-to-noise ratio (SNR) metric:

\[ ITD \text{ SNR} = \frac{\text{Variance in firing rate due to variation in ITD}}{\text{Total variance of firing rate}} \]  

(Eq. 1)

\( ITD \text{ SNR} \) is the fraction of the variance in neural firing rates accounted for by the variation in stimulus ITD, as opposed to random, across trial variability. It ranges between 0 and 1, indicating no ITD sensitivity and perfectly reliable ITD coding, respectively. An F-test was used to determine if the dependence of firing rate on ITD was statistically significant (p<0.025).

Summary data (Fig. 2-3) are shown for the stimulus condition (level and pulse rate) that maximizes the ITD SNR.

**ITD discrimination model**

A computational model (Hancock and Delgutte, 2004) previously used for predicting normal-hearing ITD discrimination based on physiological properties of IC neurons was extended to the bilateral CI case. Figure 5A shows a diagram of this signal detection model. The model comprises a two-dimensional grid of model neurons, each of which has a Gaussian-shaped rate-ITD curve (e.g. Fig. 3A). The standard deviation of the Gaussian (sharpness of ITD tuning) varies systematically along one axis of the grid (corresponding to the tonotopic axis in the normal-hearing case), with a lognormal distribution fit to the physiological data. The ITD of maximum slope (see Results) has a normal distribution along the other axis of the grid.

The model simulates a two-alternative forced choice ITD discrimination experiment by comparing model neural firing rates in response to a reference ITD and a test ITD. Model rates are first summed along the columns of the grid (i.e. across different sharpness of tuning). For each column \( i \), the summed rates \( r_i \) are used to compute a standard separation \( D_i \) (analogous to \( d' \)):

\[
D_i = \frac{r_i ITD_{test} - r_i ITD_{ref}}{\sqrt{\frac{1}{2} \left( \sigma_i^2 ITD_{test} + \sigma_i^2 ITD_{ref} \right)}}
\]  

(Eq. 2)

The firing rate variance \( \sigma^2 \) is assumed to be proportional to the mean firing rate (Hancock and Delgutte, 2004). The individual standard separations are combined optimally (Green and Swets, 1988), assuming statistically independent firing rates across columns, to get the overall standard separation \( D \):

\[
D^2 = \varepsilon \sum_i D_i^2
\]  

(Eq. 3)

Percent correct discrimination is computed from \( D \) using the inverse normal distribution. The test ITD is adjusted to find the value yielding 75% correct discrimination. The difference between this test ITD and the reference ITD is taken as the model just noticeable difference (JND) in ITD. The model has only one free parameter, the detection efficiency \( \varepsilon \), which is an overall scale factor on the JNDs predicted by the model. For all simulations, \( \varepsilon \) was held constant at the value which produces accurate predictions of normal-hearing ITD discrimination performance for broadband noise (Hancock and Delgutte, 2004).
RESULTS

Physiology

The results demonstrate that neural ITD coding is degraded in congenitally deaf cats compared to acutely-deafened cats. Figure 1 illustrates the typical qualitative differences in response properties of IC neurons across the two groups of animals. Responses to a 10-pps pulse train varied in ITD are shown for one IC neuron from an acutely-deafened cat (top row) and one from a congenitally deaf cat (bottom row). In the acutely-deafened cat, the spikes are precisely locked to each stimulus pulse (Fig. 1A), and the firing rate is strongly modulated by ITD (Fig. 1B), as indicated by the large, highly-significant value of the ITD SNR metric (0.69, p<0.001). The ITD tuning is peak-shaped with a best ITD of 200μs and shows a preference for contralateral-leading stimuli. This neuron has no spontaneous activity, as is the case for most IC neurons in the acutely-deafened cat.

In contrast, in the congenitally deaf cat, spikes have long latencies and poor timing with respect to the stimulus pulses (Fig. 1C). The firing rate is not obviously modulated by ITD, consistent with the small, statistically insignificant value of ITD SNR (0.15, p=0.95). The spontaneous rate for this example is 4.1 spikes/s, close to the median spontaneous rate of 3.0 spikes/s for the congenitally deaf group as a whole.

Figure 2A shows the distributions of ITD SNR for the two groups of animals. In congenitally deaf cats, only 45% (55/114) of IC neurons have statistically significant SNRs (p<0.025), compared with 81% (55/68) in acutely-deafened cats. The median ITD SNR is significantly lower in the congenitally deaf group (0.19 vs. 0.37, p<0.005, rank-sum test). Among the units
for which the ITD SNR is significant, however, the median ITD SNRs are similar (0.44 congenitally deaf vs. 0.47 acutely-deafened). Thus, about half as many IC neurons are ITD-sensitive in congenitally deaf cats compared to acutely-deafened cats, but where ITD sensitivity exists, it is similar in magnitude between the groups.

We next examined whether the ITD-sensitive neurons show differences in shapes of ITD tuning curves between the two groups of implanted cats. We classified the shape of ITD tuning using the four template functions (Fig. 2B) of Smith and Delgutte (2007): peak (positive-going Gaussian), trough (negative-going Gaussian), biphasic (difference of two Gaussians), or sigmoidal (cumulative Gaussian). Each neuron was assigned the shape whose template best fit the rate-ITD curve in the least-squares sense. Figure 2B compares the incidence of each tuning shape between the two groups. There were slightly more peak and biphasic shapes in the acutely-deafened group and more troughs and sigmoids in the congenitally deaf group, but the difference was not statistically significant ($p=0.47, \chi^2$-test).

Fig. 2: Summary statistics on ITD tuning for acutely-deafened and congenitally deaf cats. A: Distributions of ITD SNR. B: Incidence of rate-ITD curve shapes for neurons with statistically significant ITD SNRs.

ITD tuning is examined in more detail using the metrics illustrated in Fig. 3A: halfwidth, best ITD, and ITD of maximum slope (ITDMS). The analysis was limited to peak-shaped rate-ITD curves and the peak portion of biphasic curves, and the metrics were computed from the best fits.

In acutely-deafened cats, the distribution of best ITD (Fig. 3B) has a clear contralateral bias (mean = 114 μs) and a comparatively narrow distribution (standard deviation = 406 μs). In congenitally deaf cats, the distribution is centered closer to the midline (mean = 55 μs) and is much broader (s.d. = 843 μs, $p<0.001$, Levene’s test).

Figure 3C shows the distributions of halfwidth. In acutely-deafened cats, the distribution is relatively narrow (mean = 685 μs), whereas in congenitally deaf cats, it is broad with a large mean (1078 μs). The difference in means is significant ($p<0.05$, rank-sum test).

Finally, Fig. 3D shows the distributions of the ITD of maximum slope, where the firing rate changes most steeply with ITD. In the acutely-deafened group, 71% of the maximum slopes fall within the range of naturally-occurring ITDs for the cat ($\pm 350 \mu s$). For the congenitally deaf group, the distribution is broader with a stronger ipsilateral bias, consistent with the larger halfwidths and more variable best ITDs in that group, and only 35% of the slopes lie within the
In summary, about half as many IC neurons are ITD-sensitive in congenitally deaf cats as in acutely-deafened cats. Neurons that are ITD-sensitive show similar degrees of sensitivity between the two groups, and similar distributions of rate-ITD curve shapes. There are quantitative differences between the two groups in the characteristics of ITD tuning. In congenitally deaf cats, the tuning tends to be less sharp and best ITDs are distributed more widely.

Model of psychophysical ITD discrimination

An important question is the extent to which the changes in ITD coding due to congenital deafness affect ITD perception. The answer depends on the assumptions one makes about the central processing of the ITD information available in the responses of the population of IC neurons. Our approach was to use a signal detection model to assess the impact of congenital deafness on ITD discrimination. For this purpose, we adapted to the deaf case a model that was previously shown to account for ITD discrimination in normal-hearing listeners (Hancock and Delgutte, 2004).

The normal-hearing model is a grid of neurons (e.g. Fig. 5A), each of which is characterized by a rate-ITD curve whose shape is determined by two parameters: best ITD and best frequency (BF). An essential physiological constraint on the model is that in the IC, best ITD is inversely
correlated with BF, such that the product of the two is nearly constant along the tonotopic axis (McAlpine et al., 2001; Hancock and Delgutte, 2004; Joris et al., 2006). Equivalently, best ITD is positively correlated with halfwidth of ITD tuning (i.e. broadly tuned neurons tend to have large best ITDs) because the main lobe of the rate-ITD curve widens as BF decreases (Yin et al., 1986). This correlation between best ITD and halfwidth tends to align the rising slopes of the rate-ITD curves near 0 ITD, and misalign them at larger ITDs. In the model, summing firing rates across BF (or, equivalently, halfwidth) takes advantage of this alignment of the rising slopes to produce fine ITD acuity on the midline, while the misalignment elsewhere decreases acuity more laterally, consistent with psychophysical data (Mossop and Culling, 1998).

It is \textit{a priori} unclear whether this important relationship between best ITD and halfwidth holds in the case of bilateral CI. In normal-hearing animals, the dependence of halfwidth on BF primarily reflects the effects of cochlear filtering (Yin et al., 1986), while small interaural mismatches in the BF of the inputs to the binaural coincidence detector neurons likely contribute to the dependence of best ITD on BF (Joris et al., 2006). With cochlear implants, however, these peripheral mechanisms are bypassed.

Nevertheless, Fig. 4A shows that a positive correlation between best ITD and halfwidth does exist in the IC of acutely-deafened, bilaterally-implanted cats. For this purpose, we combined our sample (n=31) of peak-shaped and biphasic rate-ITD curves with a larger sample (n=77) from Smith and Delgutte (2007). The two variables are significantly correlated ($r^2=0.41$, $p<0.001$). Mean best ITD and mean halfwidth were computed after dividing the data into quartiles based on halfwidth (Fig. 4A, black symbols). Figure 4B plots the corresponding average rate-ITD curves.

![Fig. 4: Best ITD is correlated with halfwidth in acutely-deafened cats. A: Scatter plot of best ITD vs. halfwidth for peak/biphasic shaped rate-ITD curves. Symbols show mean values in quartiles based on halfwidth. B: Average rate-ITD curves corresponding to symbols in A. C: ITD of maximum slope is uncorrelated with halfwidth.](image-url)
for each quartile, illustrating the alignment of slopes at the midline despite the wide variation in halfwidths. As a result, the ITD of maximum slope is uncorrelated with halfwidth, as shown in Fig. 4C ($r^2=0.0013$, $p=0.70$).

Based on these results, we constructed a population model for the bilateral CI case in which halfwidth and ITD$_{MS}$ vary along the two independent axes of the grid of model neurons (Fig. 5A), because these parameters are uncorrelated in the physiological data (Fig. 4C). In every other respect, the bilateral CI model operates in identical fashion to the normal-hearing model (see Methods). Importantly, the sole free parameter of the model, the detection efficiency $\varepsilon$ (Eq. 3), was fixed at the value that produced accurate predictions of normal-hearing ITD discrimination for broadband noise (Hancock and Delgutte, 2004).

Fig. 5B compares psychophysical data from two bilateral CI subjects with model predictions. The gray line shows mean ITD JND as a function of reference ITD in response to 50-pps pulse trains for two post-lingually deafened adults who were among the best performers (Hancock and Noel, 2008). JNDS are small on the midline (90 $\mu$s), but increase as the reference ITD increases, similar to the trend exhibited by normal-hearing listeners (Mossop and Culling, 1998). An “acutely-deafened” model was created using the constraints imposed by the physiological data obtained from this group of animals. Specifically, all model neurons were ITD-sensitive, and the values of halfwidth and ITD$_{MS}$ were independently distributed according to the data of Fig. 4C.

**Fig. 5:** Physiologically-based model of psychophysical ITD discrimination with bilateral CI. A: Grid of model neurons. Each model neuron has a Gaussian-shaped rate-ITD curve. Halfwidth varies in one dimension, ITD of maximum slope (ITD$_{MS}$) in the other. Rates are summed across halfwidths before computing D-values, and then the Ds are combined optimally across ITD$_{MS}$. B: ITD JND vs. reference ITD for bilateral implant subjects (gray line) and various model configurations.
The prediction of the acutely-deafened model is shown by the solid black circles in Fig. 5B. The JND is about 50 $\mu$s on the midline and increases with reference ITD. Although the trend in the model prediction roughly parallels the psychophysical data, the simulation clearly illustrates the discrepancy that motivated this study: neural ITD coding in acutely-deafened cats predicts better ITD discrimination than is actually observed in bilateral CI users, even very good performers.

We hypothesize that the abnormal neural ITD coding observed in congenitally deaf cats may better predict the psychophysics because human implantees experience long periods of binaural deprivation before receiving a second implant. We explored this hypothesis by constructing a “congenitally deaf” model incorporating the observed neural abnormalities. Specifically, the decreased incidence of ITD-sensitive IC neurons was modeled by making half of the model neurons fire at a constant rate as a function of ITD. Furthermore, the alteration of neural ITD tuning properties was simulated by increasing the widths of the distributions of halfwidth and ITDMS as shown in the data of Fig. 3B,D. Imposing the congenital deafness constraints increases the predicted JNDs by nearly an order of magnitude relative to the acutely-deafened model, while preserving the general trend (Fig. 5B, triangles).

The ITD discrimination performance predicted by the congenitally deaf model is about a factor of three worse than the psychophysical data shown. Possibly, neural ITD coding is less degraded in these good-performing subjects than in the extreme case of congenital deafness. Alternatively, the central processor may learn to ignore the uninformative (i.e. ITD-insensitive) neurons within the IC neuron population in order to optimize performance. This condition was simulated by making all neurons sensitive to ITD in the congenitally deaf model (Fig. 5B, squares). In this configuration, the model produces a close match to the psychophysical data on the midline, but a more shallow increase in JND off the midline.

**DISCUSSION**

ITD discrimination by human bilateral CI users is generally poor compared to normal hearing listeners, and is highly variable across subjects. In contrast, neural ITD coding by single units in the IC of acutely-deafened, bilaterally-implanted cats is essentially normal. A confounding factor in comparing the human psychophysical data to the animal neurophysiology is the extent of previous binaural hearing experience. While the cats were deafened within a week of experimentation, humans often experience months or years of deafness and/or monolateral stimulation before receiving a second cochlear implant. Here, we have begun to address this issue by comparing neural ITD coding in acutely-deafened and congenitally deaf cats, which contrast maximally in binaural experience.

The main finding is that there are only about half as many ITD-sensitive neurons in the IC of congenitally deaf cats as in acutely-deafened cats. In addition, congenital deafness affects ITD tuning among the neurons that do exhibit ITD sensitivity: rate-ITD curves featuring a peak tend to be broader and have a wider range of best ITDs in the congenitally deaf animals. However, the degree of sensitivity (as quantified using the ITD SNR metric) is comparable between the groups of deaf cats for ITD-sensitive neurons; this metric depends on the range of firing rates and the response variability as well as on the shape of the ITD tuning curve. Overall, our results suggest that deprivation of auditory experience comprising the neonatal period has a major impact on the function of the neural circuits processing ITD. The deficits observed in the IC appear to be inherited by the auditory cortex, which exhibits less sensitivity to ITD and weaker preference for contralateral stimulation in congenitally deaf white cats compared to acutely-
deafened cats (Kral et al., 2009).

By what mechanisms might congenital deafness affect neural ITD coding? In congenitally deaf cats and mice, there are abnormalities in the endbulbs of Held, the specialized synapses between auditory nerve fibers and the spherical bushy cells (SBCs) of the cochlear nucleus (Ryugo et al., 1997; Ryugo et al., 1998; Lee et al., 2003). Disruption of this critical synapse in a normally precisely-timed pathway is likely to impair the encoding of ITD by the medial superior olive (MSO), which receives excitatory inputs from SBCs bilaterally (Ryugo et al., 1997).

Similarly, cochlear lesions result in abnormalities in the calyces of Held, the giant synapses formed in the medial nucleus of the trapezoid body (MNTB) by cochlear nucleus globular bushy cells (Jean-Baptiste and Morest, 1975). The MNTB in turn makes an inhibitory projection to MSO (Kuwabara and Zook, 1992) and this projection is thought to play a role in shaping ITD tuning (Brand et al., 2002). The normal development of these inhibitory synapses depends on auditory experience (Kapfer et al., 2002), but can be partially restored in congenitally deaf animals by chronic electric stimulation of the cochlea (Tirko et al., 2009).

We extended a physiologically-based model of psychophysical ITD discrimination for normal hearing to assess the potential perceptual consequences of the abnormal neural ITD coding resulting from congenital deafness. The modeling results broadly support our hypothesis that deprivation of binaural experience accounts for less than optimal ITD discrimination in bilateral CI users. As expected, when the model is constrained using the physiological data from acutely-deafened cats, it predicts better ITD discrimination than observed psychophysically in two good-performing subjects (Hancock and Noel 2008). On the other hand, constraining the model using data from the congenitally deaf cat resulted in poorer predicted performance than observed psychophysically. There are several possibilities for reconciling model predictions with the psychophysical data. The central processor might be able to improve performance by discarding uninformative neurons lacking ITD sensitivity (e.g. Fig. 5B, squares). Alternatively, the degradations in ITD tuning of IC neurons in post-lingually deaf subjects might be less severe than in the congenitally deaf case, leading to model predictions intermediate between those for the acutely-deafened case and the congenitally deaf case, and therefore more in line with the psychophysical data. Model predictions span nearly an order of magnitude depending on the degree of alteration in ITD coding assumed. It is possible that variations across subjects in both the number of ITD-sensitive neurons and the sharpness of tuning of the sensitive neurons underlie the large variability in ITD acuity observed psychophysically.

Interestingly, the data used to constrain the model may provide insights into the development of ITD coding in normal hearing. In normal-hearing cats, guinea pigs, and gerbils, best ITD and the halfwidth of ITD tuning curves are positively correlated (McAlpine et al., 2001; Brand et al., 2002; Hancock and Delgutte, 2004). Consequently, neural firing rates are typically most sensitive to changes in ITD near the midline, where perceptual ITD acuity is finest. Cochlear filtering and traveling wave delays are likely to contribute to this trend (Joris et al., 2006), in addition to neural mechanisms including propagation delays, synaptic filtering, and inhibition. Fig. 4 shows that the trend persists in bilaterally-implanted cats even though cochlear mechanics are bypassed. The following scenario might account for this observation. Early in development, there might be a broad range of sharpness of ITD tuning and an unfocused distribution of best ITDs, similar to the congenitally deaf data in Fig. 3B,C. Auditory experience might provide selective pressure to create a network maximally sensitive to changes in ITD about the midline by strengthening inputs that favor correlation between the halfwidth and best ITD, and pruning
those that don’t, regardless of whether the properties of each particular input strengthened or pruned are shaped primarily by cochlear filtering or by alternative central mechanisms.

The results described here raise additional questions: Does the period of auditory deprivation have to encompass the neonatal period to produce the abnormalities in ITD coding observed in the IC of congenitally deaf cats, or do some of these abnormalities also occur in the case of adult-onset deafness? Can chronic bilateral stimulation through cochlear implants reverse some of these abnormalities and improve neural ITD coding in deaf animals? If so, are there certain stimulation paradigms or training regimens particularly effective for this purpose? Answers to such questions will shed further light on the development and plasticity of neural ITD coding, and hopefully suggest methods for improving the ability of bilateral CI listeners to use ITD information.

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