The effect of cochlear damage on auditory cortical responses:

Plasticity – adaptive and mal-adaptive

Auditory pathways from cochlea to cortex are organized for frequency

![Diagram showing frequency organization from cochlea to cortex](image)
Adult-onset hearing loss and cortical plasticity

Basal cochlear lesions produce high-frequency hearing losses

Normal cochlea regions

Damaged cochlea regions
Basal cochlear lesions produce high-frequency hearing losses

What cortical plasticity looks like
What cortical plasticity looks like

Is it plasticity or is it “residue”? 
Cochlear nerve fibre tuning reflects basilar membrane tuning at a point.

Hence damage to a point on the basilar membrane abolishes the tuning curve for the neuron getting input from that point.

Cortical tuning curves reflect convergent input from many originally-cochlear inputs.
“Residue” can cause frequency plasticity but with elevated thresholds.

A1 map thresholds are consistent with plasticity, not residue.
How much and what sort of a loss causes adult cortical plasticity?

Rajan & Irvine 1998:

Frequencies with losses of 10 dB or more (7 / 8 cats) or 20 dB or more (1 / 8 cats) were not represented in the CF map.

Instead, A1 regions which would normally have represented those frequencies were occupied by an expanded CF representation of frequencies with smaller losses.

What frequencies get “taken over”? 

Seki & Eggermont 2002
Plasticity needs a region of large (total?) loss

Rajan & Irvine 1998:

It must be noted that...

frequencies with small 10 dB or
20 dB losses bordered higher
frequencies with even larger
CAP losses, extending basally to
frequencies with no responses.

Thus the impetus for the A1
map changes may come not
simply from the presence of
losses > 10-20 dB, but from the
presence of a total loss of input
at even higher frequencies.

Small cochlear lesions……

… do not cause cortical
plasticity
Conductive HL does not cause plasticity

EE prevents plasticity in the case of mild HL, consistent with cortical changes

Is it cortical or sub-cortical?

Map plasticity emerges fully at cortex

Cochlear lesioning

Plasticity
(Kanke, Brown & Irvine, 02)

Patchy plasticity
(Irvine, Rajan & Smith, 03)

No plasticity
(Rajan & Irvine, 98)

Salicylate o/d

A 1kHz Compound Action Potential

B Inferior Colliculus Evoked Potential

C Auditory Cortex Evoked Potential

Qu et al. Hear Res 2000
Summary: When does Adult cortical plasticity occur?

1. When the loss is sensori-neural
2. When there is a sloping hearing loss leading to a cochlear dead region
3. Over time
4. In a fixed acoustic environment

Neonatal hearing loss and cortical plasticity
Neonatal plasticity is more extensive - but messier…

Neonatal plasticity only allows sensitive plasticity over the same range as in adults
Neonatal plasticity generally shows good precision & timing of responses

Summary: Features of neonatally-induced deafness for cortical plasticity

1. Much more extensive than in adults
2. Consists of an immediate region of good sensitivity and a more extensive region of increasingly poorer sensitivity
3. Other response properties relatively normal
Does plasticity occur in adult humans with Adult-onset hearing loss - and what are the perceptual consequences?

Cortical plasticity of edge frequencies
In our tactile systems, more brain “space” is devoted to processing information from regions for which we have greater tactile acuity.

Better frequency discrimination at the “edge” of steeply-sloping hearing loss

Thai-Van et al., Brain, 2002
As predicted, the improved frequency discrimination at the “edge” of steeply-sloping hearing loss only occurs when there is also a cochlear dead region.

Enhanced DLFs near fe, consistent with cortical reorganization, occurred for subjects whose audiograms had both steep & shallow slopes, and regardless of hearing aid use.

Enhanced DLFs usually occur near fe for subjects with:
(a) a high-frequency DR in both ears;
(b) a high-frequency DR in one ear and a hearing loss in the other ear;
(c) a low-frequency DR.

Kluk & Moore, Hearing Res 2006

Improved edge frequency discrimination may help speech extraction

Vickers et al 2007 JASA

[DR] Subjects with fe at 0.75 kHz, obtained 42% correct with speech low pass filtered at 0.75 kHz & 47% correct when speech was low pass filtered at 1.27 kHz (1.7fe).

In contrast, subjects w/o DR scored only 30% correct with speech low pass filtered at 0.75 kHz & 34% correct when cut-off frequency = 1.27 kHz.
Does use of a hearing aid alter the better frequency discrimination at the edge?

– a new phase of plasticity?

Is mal-adaptive plasticity responsible for tinnitus?
Cortical map plasticity may underlie tinnitus in some subjects

Mullinickel et al 2002
PNAS

Euclidean distance between trajectory of standard tones and
(a) location of tinnitus frequency in tinnitus subjects
or
(b) corresponding comparison frequency in control subjects

Is dominant tinnitus pitch related to hearing loss in a manner related to cortical map reorganization?

When audiometric hearing loss is present, the frequencies reported by patients to correspond to their tinnitus are in the frequency region of threshold shift in the audiogram (Noreña et al. 2002; Roberts et al. 2008; see Fig. 1). The dominant pitch most commonly reported for NIHL-induced tinnitus matches that of a 3-kHz tone (Penner 1980).

Eggermont & Roberts Cell Tissue Res 2015

Sound frequencies judged to resemble tinnitus (Likeness Rating) and the center frequency of band pass masks giving optimal forward suppression of tinnitus (residual inhibition, RI Depth) mark the region of audiometric threshold shift (from Roberts et al. 2008). A likeness rating of 40 denotes a sound beginning to resemble tinnitus. Sound thresholds (broken lines) are considered normal when ≤20 dB HL. WN RI depth after a white noise masker.
Tinnitus may not be related to edge frequency / cortical reorganization

Is dominant tinnitus pitch at the audiometric edge of hearing loss or well within HL region?

NO: Roberts et al. 2008; Pan et al. 2009: Could not confirm prediction of over-representation of edge frequencies in tonotopic maps after noise trauma, implying that tinnitus pitch would match the edge frequencies.

YES: Moore & Vinay 2010: Was this failure related to octave errors in pitch matching? Following training to avoid these errors, mean pitch matches were close to the edge frequency, with r = 0.94.

NO (again!): Schecklmann et al. 2012: confirmed relationship between tinnitus pitch & max. HL but not to the edge frequency.

Increased cortical spontaneous activity and tinnitus?

Seki and Eggermont (2003)
Sub-cortical sites may also cause tinnitus

Increased cortical spontaneous activity may drive sub-cortical spontaneous activity changes

Table 1
Time-line of significant changes in spontaneous firing rate after noise trauma.

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↑ significant increase; = no significant change. Seki and Eggermont (2003)
Evidence for multiple sites of tinnitus generation –

A range of other inputs modulate tinnitus

Approx. 2/3 of people with tinnitus can alter the loudness and pitch of their tinnitus via somatic manoeuvres – e.g., jaw clenching, tensing neck muscles etc

and

tinnitus can arise from somatosensory insults

Roberts et al J Neurosci 2010

Shore et al Nat Neurol Rev 2016