

OHC motility as a means to concentrate delivery of cochlear stimulus energy

The Outer Hair Cells (**OHC**) and the Organ of Corti act to increase the energy that affects zones of Basilar Membrane (**BM**) detecting the various frequency components of a stimulus.

Two mechanisms are usually thought to contribute to this:

1. *'Negative damping' or 'positive feedback' : BM resonance is enhanced by active OHC force, in phase with velocity so as to increase frequency selectivity & sensitivity by accurately opposing viscous damping. This may not be very important (see right panels).*
2. *'Energy Feed', whereby active forces feed energy into the travelling wave as it moves along the BM towards the detectors that are most sensitive to its frequency components.*

This poster considers a third way in which OHC motility may enhance energy delivery:

3. *'Active Stiffening' : reducing energy loss from the travelling wave, by stiffening the BM for frequencies below the local resonance. These frequency components are then more efficiently transmitted to where they are sensed, more apically. This could be achieved by negative feedback, in which OHCs act to stabilise the BM up to a limiting frequency.*



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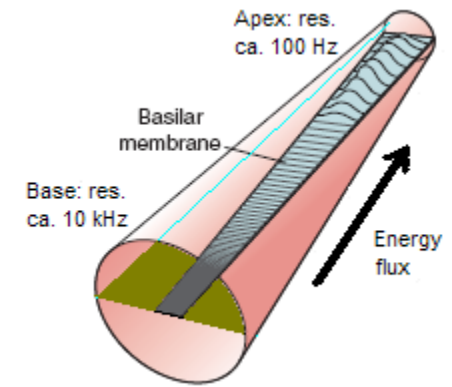
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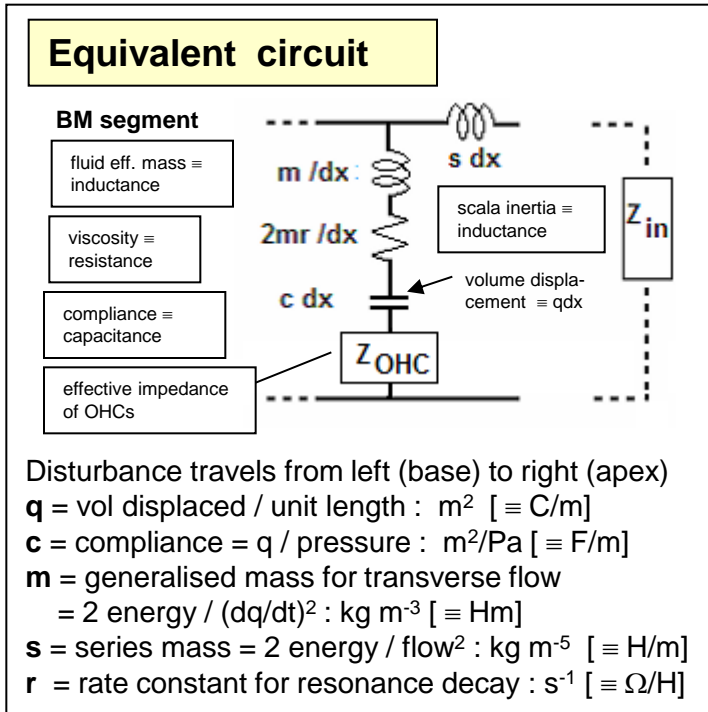
Travelling waves on a resonant transmission line:

The issues addressed here and the simulations employed only treat the BM as a simple 1-dimensional structure: a resonant transmission line. Of course the Organ of Corti is more complex in ways that are doubtless important, but these need not be introduced here.

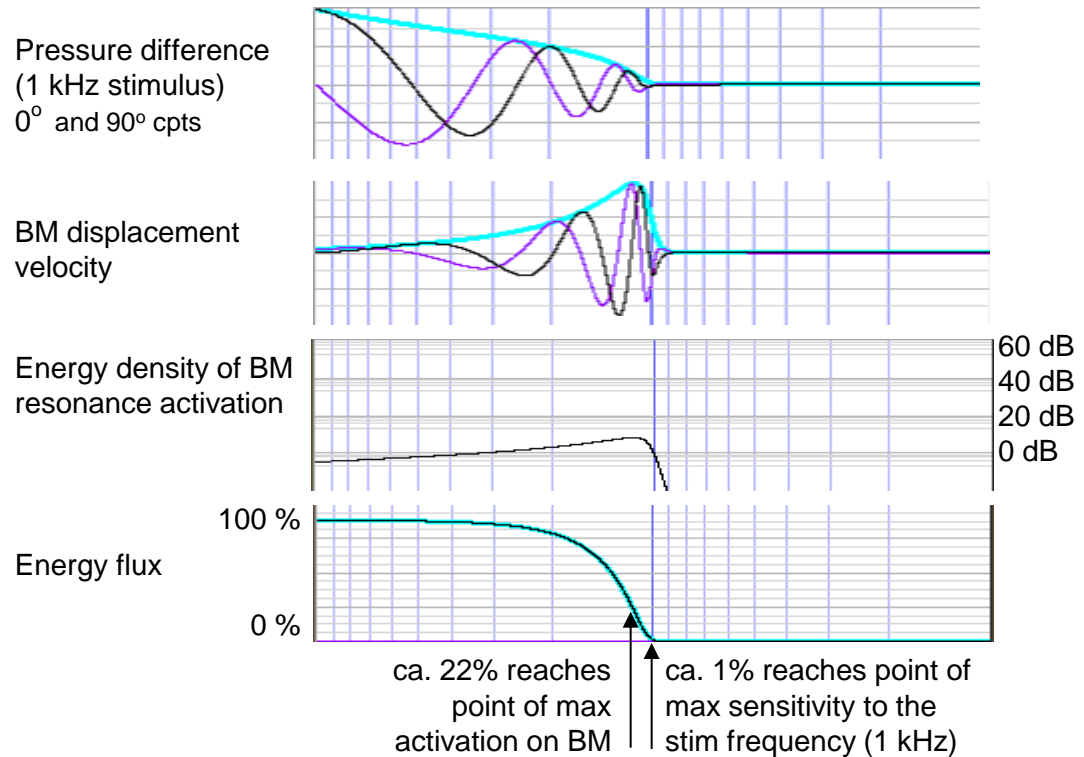
Simulation treats a continuous transmission line (resonance $f=10\text{ kHz}$ to 100 Hz) as below. The time constant for passive amplitude decay is generally set uniformly at 2 ms ($r=0.5\text{ ms}^{-1}$).



**What happens to the energy fed in at the base?
[Passive cochlea, stimulus 1 kHz]**



base: res=10kHz res=1kHz apex: res=100Hz



Countering energy loss from the travelling wave

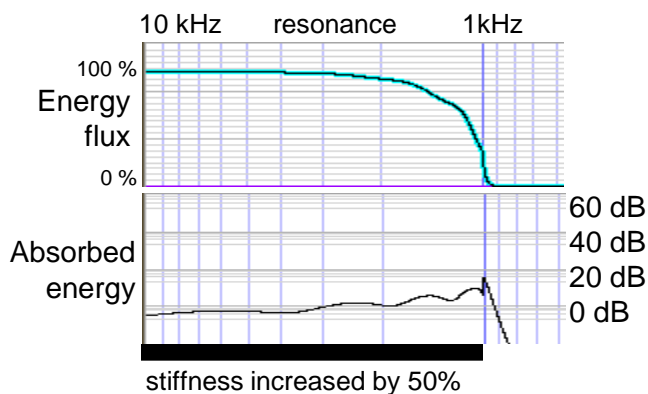
Energy is progressively absorbed from a passive travelling wave before the resonant point is reached. The most sensitive part of the cochlea may be exposed to less than 1% of stimulus energy.

Early after stimulus onset (less than the time constant for viscous energy dissipation), lost energy goes into building up BM displacements in the basal cochlea. Once a steady state is reached, the lost energy dissipates by viscosity. If OHCs act to stiffen the BM between stapes and the resonant point, energy loss can be reduced.

Frequency-independent stiffening

If the basal cochlea were rendered wholly stiff to all disturbance, energy delivery could in principle increase 100-fold (20 dB). Simulation (below) shows that a 15 dB increase could be achieved with just a 50% increase in stiffness.

Though substantial, this increase is much less than the OHCs are believed to account for. The mechanism would also be highly non-linear.

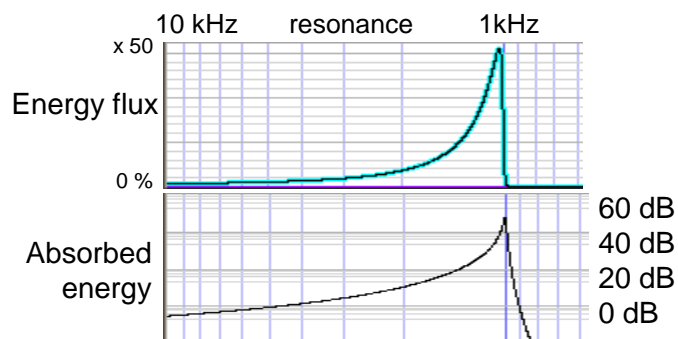


Note that the abrupt change of stiffness here causes standing waves by reflection.

Stiffening by OHC negative feedback

If the OHCs **oppose** BM movement with force proportional to displacement (a linear homeostatic mechanism), this effectively stiffens the local BM up to a limiting frequency. The high frequency roll-off has 2 desirable consequences:

1. stiffening is greatest for low frequency components for which the energy needs to be transmitted further apically
2. the phase lag results in energy feed, amplifying the energy sent forward in the travelling wave



Negative feedback (stiffness at low freq x 4.1 everywhere), with uniform 3dB roll-off at 100 Hz (1st order filter, time constant 1.5 ms). Stimulus: 1 kHz. Viscosity x2 to reduce sharpness of the activation peak. There is a ca.50-fold (17 dB) increase in energy flux and ca. 40dB increase of peak sensitivity compared with the passive model.

Mechanism

Active BM stiffening in this model enhances sensitivity by 3 mechanisms:

(i) Concentration of energy delivery to the region most sensitive to each frequency component

(ii) Energy feed into the travelling wave, amplifying the stimulus energy

(iii) Negative damping at the resonance point, allowing activation to build up for longer times

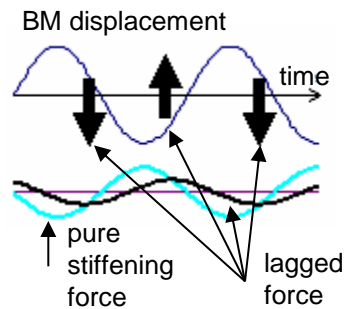
Comparable increases may arise from each source, depending on model parameters. To achieve enhancement of the order of 40 dB as suggested experimentally for the contribution of OHCs, it is probably not sufficient just to make the basal Organ of Corti more rigid in response to a detected stimulus (left panel above, (i) alone).

Conclusion

Most cochlear models assume OHCs enhance rather than oppose BM displacement at low frequencies. Surprisingly, the issue seems somewhat unclear experimentally. Mammano & Ashmore ('93) showed that extracellular current causing apical depolarisation of OHCs (as occurs with upward displacement of the BM) led to downward movement of the Reticular Lamina (RL) and a smaller upward movement of BM. This might be taken to support conventional models, but there is some uncertainty about whether the basal end of the OHCs may have been significantly hyperpolarised in these experiments. Undoubtedly the RL and BM are squeezed together with OHC depolarisation, expanding the tissue radially. This could have unexplored consequences, including possible downward pressure on the attached BM and a reduced mass of fluid moving perpendicular to the BM.

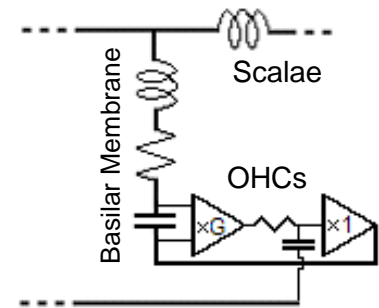
The effect of lag

Mechanisms (ii), (iii) arise naturally if OHCs attempt to oppose detected movements of the BM, since there are intrinsic delays and phase lags. A lagged stiffening force has a component that is in phase with velocity, feeding energy into the travelling wave and opposing viscous drag.



Equivalent Circuit

The simplest equivalent circuit is shown below. At low frequencies this reduces the effective BM capacitance (analogous to compliance) by a factor $1+G$, equivalent to introducing a series capacitance C_{BM}/G . The effect of the OHC filter is to increase the series capacitance at higher frequencies and introduce a negative series resistance.



ABSTRACT (British Society of Audiology, Cambridge, Sept 2006) :

OHC motility as a means to concentrate delivery of cochlear stimulus energy

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Motility of Outer Hair Cells (OHC) is thought actively to cancel viscous energy dissipation, thereby achieving high frequency selectivity at the basilar membrane (BM). But OHC motility could also serve other functions, e.g. viscosity modulation, reduction of after-effects of transients, concentration of energy flux to optimal sites, or induction of flow in subtektorial space. Experiments with a physical model (Gardner-Medwin, 2006) have suggested that passive damping of resonance with a locally compliant BM zone (in an otherwise relatively stiff separation between cochlear ducts) would not be as limiting as was suggested by Gold's (1948) analysis of damping for a fluid-immersed vibrating string.

The issue addressed here is the possible role of OHC motility in cutting down energy absorption from a travelling wave as it progresses from the oval window towards the region of BM with maximum sensitivity to its frequency components. Simulation of a purely passive travelling wave at 1 kHz suggests that with plausible parameters only 20-30% of the initial energy flux may reach the place on the BM where displacement is maximal, and that less than 1% may reach the point (further towards the apex) where BM resonance matches the stimulus frequency, and where the BM would be intrinsically most sensitive to delivery of the stimulus energy. The rest of the energy has been absorbed on the way. This means that by simply stiffening the BM on the basal side of a region of activation, sensitivity may be increased by amounts of the order of 20dB. A moderate stiffening gives proportionately a much larger enhancement of the energy reaching the resonant zone.

OHC motility could stiffen the BM in different ways that might contribute to concentration of energy flux. One is potentially linear: by detecting and opposing any movements of the BM up to a cutoff frequency that is somewhat below the local resonant frequency. This would be analogous to a stretch reflex in muscle, with movements actively opposed cycle-by-cycle. The second is non-linear: alteration of the frequency-independent stiffness of the tissue, analogous to stiffening of a muscle that is steadily activated or in rigor. Interestingly, high frequency roll-off of the first (negative feedback, homeostatic) mechanism would yield negative damping at frequencies near the local resonant frequency, giving scope for simultaneous enhancement by three aspects of the same mechanism.

Gardner-Medwin A.R. 2006. Viscous damping of acoustic resonance with a restricted zone of wall compliance Proc Physiol Soc 3 (UCL) DC2, 210P

Gold T. 1948. Hearing. II. The Physical Basis of the Action of the Cochlea. Proc R Soc Lond B Biol Sci 135, 492-8