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Outer Hair Cells can Amplify the Fluid Traveling Wave by Changing Organ-of-Corti area in the Short-Wave Region

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Abstract. There are two kinds of cochlear amplification: "non-propagating amplification" that is widespread but doesn't couple to the traveling wave, and "traveling-wave amplification" that enables wide-band outer hair cell (OHC) motility to produce sharply tuned amplification. Traveling-wave amplification has been thought to be produced by a "OHCs-act-on-the-basilar-membrane" mechanism. Here it is hypothesized that in the traveling-wave short-wave region, OHC motility causes oscillatory longitudinal cortilymph flow in the organ-of-Corti (OoC) tunnels, producing OoC area changes that drive scala media (SM) fluid pressure and motion, and by the forces on SM, OHC motility amplifies the traveling wave.

INTRODUCTION

Cochlear amplification has been thought to be produced by outer hair cell (OHC) somatic motility acting on the basilar membrane (BM) at the right phase to add energy cycle-by-cycle to the traveling wave. However, this "OHCs-act-on-the-BM" hypothesis has several obstacles: OHCs do not directly contact the BM, OHCs have no anchor for their push/pull on the BM, and how OHC contractions are properly timed to add energy to the traveling wave is unknown. Adding to the conundrum, the motions of many organ-of-Corti (OoC) structures, e.g., the reticular lamina (RL), are amplified at frequencies that are far from frequencies amplified on the BM. These issues are addressed here by dividing cochlear amplification into two types, and by a new hypothesis for traveling-wave amplification.

Traveling-wave properties and two types of amplification. Although the traveling wave is often talked about as if it were mainly on the BM, the energy of the traveling wave is carried by motion of the fluids in scala tympani, scala media and scala vestibuli (ST, SM and SV) [1]. BM motion is produced by the pressure difference (SV minus SM) across the cochlear partition. Traveling-wave pressure has been measured in the scalae [2-4] but fluid motions have been measured only by the motions of the structures abutting the scalae and not deeper in the scalae. Starting at the cochlear base, the traveling wave propagates apically with a speed that decreases as it approaches the best-frequency (BF) region; near the stapes the wavelength is long, but near the BF the wavelength is much shorter. These are denoted the long-wave and short-wave regions [5].

The long- and short-wave regions approximately demarcate the locations of the two types of cochlear amplification: "traveling-wave amplification" and "non-propagating amplification" (Fig. 1). Traveling-wave amplification is manifest by increases in BM motion amplitude and traveling-wave pressure [2-5]. A crucial aspect of traveling-wave amplification is that increases (and decreases, e.g., from two-tone suppression (2TS)) are carried

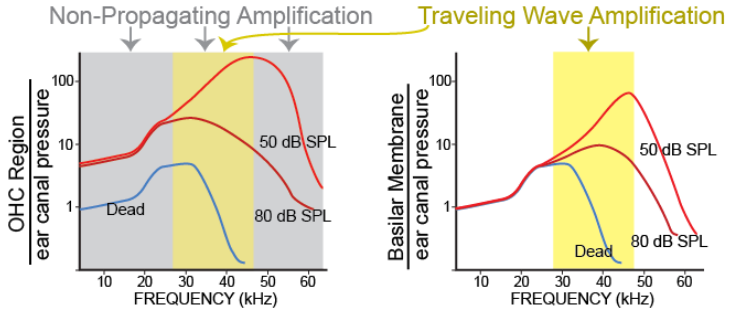

FIGURE 1. Frequency regions that show traveling-wave amplification (yellow) and non-propagating amplification (gray) on outer-hair-cell (OHC) region motion (left) and basilar-membrane (BM) motion (right). Lines are cartoon versions of data from the gerbil base [6]. Both amplifications are present in the center of the left panel.

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apically by the traveling wave so that amplification (or 2TS) produced in each OoC cross-section *accumulates* as the traveling wave moves forward (e.g., [7, 8]). In contrast, non-propagating amplification is present everywhere that the traveling wave amplitude is enough to substantially deflect OHC stereocilia and cause OHC contractions/elongations, but it is local and not coupled to the traveling wave. This conclusion is supported by 2TS of non-propagating amplification having an effect only locally [9, 10]. Also, solutions that reduce OHC motility and remove non-propagating amplification far basal of a tone's BF have no effect on stimulus-frequency otoacoustic emissions, showing that the motion far basal of BF from non-propagating amplification doesn't couple to backward traveling waves [11, 12]. In the Theory section, we consider what makes OHC motility produce traveling-wave amplification in some regions and non-propagating amplification in others.

THEORY

Short-wave-region longitudinal cortilymph flow. Although OHCs contract/elongate wherever there is substantial traveling-wave motion, we hypothesize that OHC length changes produce oscillatory fluid flow into and longitudinally along the OoC tunnels only (or predominately) in the short-wave region. There are no measurements of cortilymph flow in intact cochleae, but in excised preparations, electrical stimulation of OHCs produced oscillatory cortilymph flow along the tunnel of Corti [13]. Model simulations indicate that, in the short-wave-region, oscillatory cortilymph flow along the tunnels has a half-decay length that is more than the wavelength of the traveling wave [13-15]. In the short-wave region, contractions of the OHCs at one phase of the traveling wave are hypothesized to squeeze the OoC and force some cortilymph out of the region being squeezed and into the OoC tunnels of adjacent sections. In the same cochlea, one-half wavelength along the cochlea, OHCs would be expanding and sucking fluid out of the tunnels. The net effect of this is to produce cortilymph flow longitudinally along the tunnels from contracting regions to expanding regions (Fig. 2C), which would result in an oscillatory fluid flow along the tunnels. To provide an idea of the dimensions involved in this oscillatory flow, in the gerbil 20 kHz region near BF, half of the traveling-wave wavelength is ~100μm [16] which is approximately the distance between centers of the two tunnels in a transverse section. In contrast, far basal of BF, in the long-wave region, there would be relatively little cortilymph flow along the tunnels because the traveling-wave wavelength is much longer (it can be an order of magnitude more than in the short-wave region [16]), so there would be both a much larger mass of fluid to be moved and more damping from the long distance along the tunnels (Fig. 2A). The result is that OHC contractions can be expected to produce longitudinal cortilymph flow mainly in the short-wave region.

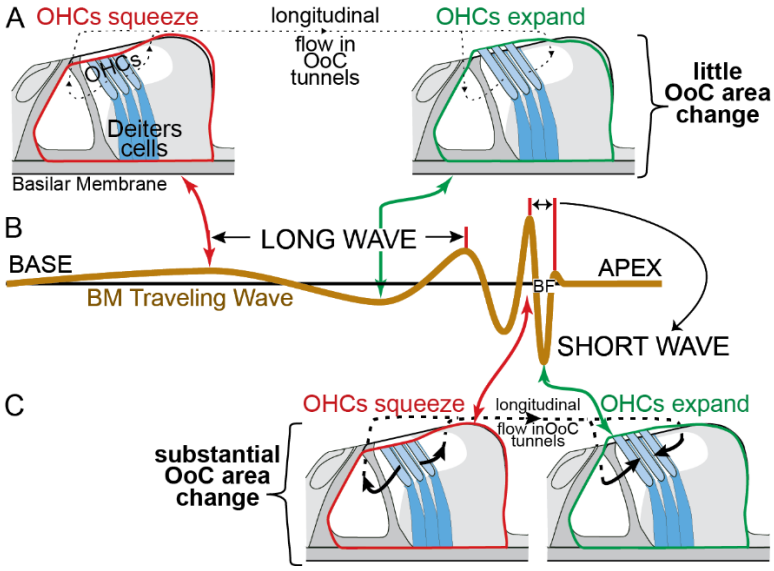

FIGURE 2. Contracting and expanding OHCs cause oscillatory longitudinal flow of cortilymph in the short-wave region but very little in the long-wave region. **A**. Organ of Corti (OoC) transverse sections at points of the traveling wave with OHC contractions (left) and expansions (right). **B**. A diagrammatic snapshot of BM displacement from a tone, showing the best frequency (BF) and example long-wave and short-wave wavelengths. **C**. OoC transverse sections as in A, but in the short-wave region where there is a short distance from OHCs squeezing to OHCs expanding and substantial flow along the tunnels between these OHC regions (shown by black arrows and dashed lines). OoC area changes are shown by red outlines (left) and green outlines (right). These indicate little net OoC area change in A and substantial area change in C. In the long-wave region of A, there is tissue movement that keeps the OoC area little changed, but in the short-wave region of C, there is an oscillatory OoC area change because fluid flows longitudinally in and out of the section. The cross-sections show a time instant, but in reality the flows are cyclic at the tone frequency. The cross sections are cartoon approximations in which motions at the OHC bottoms are not shown.

The role of Deiters cells. The Deiters cells (DC) connect OHCs to the BM, and the DC phalangeal process (PhP) connects the bottom of an OHC to the RL at a location slightly more lateral than the OHC and several OHCs more

apical [17, 18]. The Deiters-cell connection of the OHC and the BM is the basis of the OHCs-act-on-the-BM hypothesis (e.g., Fig. 3 in [19]). In feed-forward cochlear models, the PhP has been hypothesized to apply OHC forces at locations basal and apical to the OHC (e.g., [20, 21]). A problem for these hypotheses is that at frequencies far below CF, the OHC-DC junction (OHC-DCj) has transverse motion that is many times larger than BM motion (e.g., [6]). This implies that the DC doesn't couple OHC motion effectively to the BM, i.e., that DCs are relatively elastic, at least at low frequencies. An alternate hypothesis for the role of Deiters cells and their PhP is that the OHCs, PhPs and the RL act like a bellows that enables OHC contractions/expansions to change the area of the enclosed fluid space and help to force cortilymph around the OHCs into the tunnels (Fig. 3).

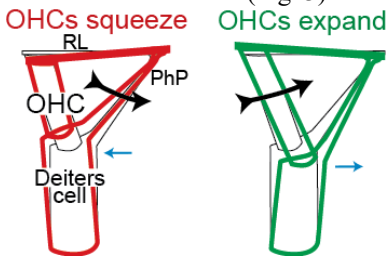

Figure 3. Cartoons of an OHC and attached Deiters cell (positions at rest are shown by thin background lines) contracting (left, red thick outline) or expanding (right, green thick outline) and forcing fluid out of, or into, the space enclosed by the OHC, the Deiters-cell phalangeal process (PhP), and the reticular lamina (RL) and into, or out of, the OoC tunnels. Thin, blue arrows indicate that OHC squeezing/contracting produce longitudinal movement of the Deiters cells.

Effects of OoC area changes. An important consequence of cortilymph flowing out of a cochlear cross section and into the OoC tunnels of adjacent OoC cross sections, is that this changes the cross-sectional area of the OoC: where OHCs contract the OoC area is smaller than at rest, and where OHCs expand, the OoC area is larger than at rest (Fig. 2C). The change in OoC area is primarily along the SM edge of the OoC, not the ST edge, because the RL is much less stiff than the BM and moves more. This OoC area change creates pressures and movements in the SM+SV fluids. In the long-wave region the distance between regions with OHCs contracting and elongating is far, which makes it difficult to force cortilymph along the tunnels, so the squeezing/expansion of the OHC region produces compensatory movement locally, e.g., fluid and tissue movement toward the Hensen cells, so that the net OoC area change is very little in the long-wave region (Fig. 2A). Opposite-phase movements of the RL and Hensen cell edges have been documented [9, 22, 23]. More work is needed to determine the exact shape changes in intact, normal cochleae.

What are the consequences of OoC area changes? Could OoC area changes produce traveling-wave amplification? First, we consider a phase of OHC contractions idealized for producing cochlear amplification by the OHCs-act-on-the-BM method, i.e., OHC contractions, presumed to pull upward (toward SV) on the BM, timed to be when BM velocity is upward (Fig. 4A-E). This phase is the target phase for cochlear amplification considered by Lee et al. [24] and Dong and Olson [25]. As a first approximation, the OoC area parallels OHC length (Fig. 4D). The resulting RL movement exerts forces on the SM fluid, which could add energy to the traveling wave, i.e., produce traveling-wave amplification. For instance, if the OoC area change produces a pressure in SM that parallels the OoC area, this would produce a SM pressure increment that is in phase with the SM pressure (Fig. 4C, E). This pressure, and the momentum induced in SM fluid by the RL motion, might produce traveling-wave amplification, but determining this needs analysis with a detailed model. If it does, then the OoC-area-pump and OHCs-act-on-BM might both produce traveling-wave amplification at the same phase of OoC motions.

An alternate to setting the phase to be idealized for OHCs-act-on-the BM amplification, is to use the measurements of Cho and Puria [6] for OoC motion at ½ octave below BF (Fig. 4, right). These measurements had phases that were delayed by ~¼ period from the idealized phases (Fig. 4, right is delayed from Fig. 4 middle). With the phases of Figure 4, right, OHC contractions do not produce traveling-wave amplification by the basic OHCs-act-on-the-BM method; while OHCs are contracting BM velocity is first positive then negative (compare brackets in Fig. 4A and H) so the net effect is zero. The phases shown in Figure 4, right, are perhaps an oversimplification because the measurements of Cho and Puria [6] are more complex and frequency dependent, but overall these measurements are not favorable for OHCs-act-on-BM cochlear amplification. Nonetheless, the Cho and Puria data showed that there was more than 40 dB traveling-wave amplification. The hypothesis proposed here is that this amplification came from the OoC-area-change applying forces to the SM fluid.

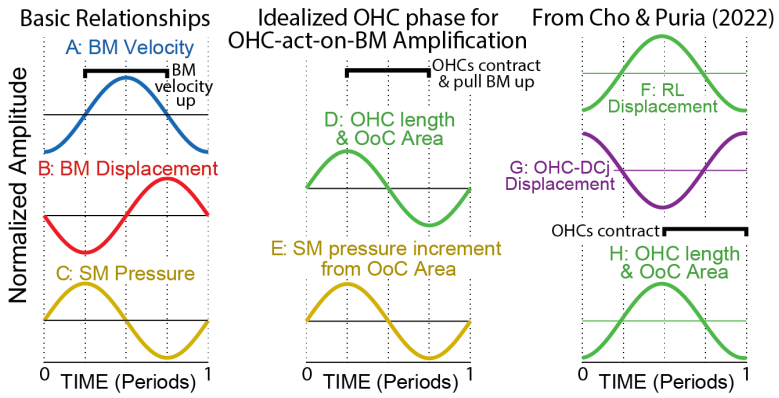

Figure 4. One-cycle amplitude vs. time plots for cochlear responses to a tone. Left column: Traveling wave relationships. Middle column: Idealized OHC phase chosen for amplifying by the OHCs-act-on-BM hypothesis, i.e., phase set so OHCs pull the BM up when the BM is moving upward – phases denoted by black brackets at the column tops. Right column: phases from measurements of Cho and Puria [6], at ½ octave below BF, for (F) the RL over the third row of OHCs, and (G) the OHC-Deiters-cell junction (OHC-DCj). Note that the OHC contraction time (bracket in H) is half while the BM is moving up and half while the BM is moving down (compare to bracket in A), so the effects cancel.

How does the OoC-area-change produce traveling-wave amplification? The analysis in Figure 4 was based on considering traveling-wave amplification as solely due to forces exerted by the OoC-area change *within a given cochlear cross section*. Such forces are shown in Figure 5 by the purple double-tipped arrows. For this kind of motion to amplify the fluid traveling wave, the forces must be at the correct phase (or close to the correct phase). However, another way that the OoC area change may add energy to the traveling wave is by the *longitudinal motion* of the corrugations in the RL surface produced by the OoC-area-changes (Fig. 5, blue arrows pointing apically). This OoC-area-change-wave would push or drag, the SM fluid apically as the traveling wave moves apically, and this is hypothesized to add energy to the traveling wave.

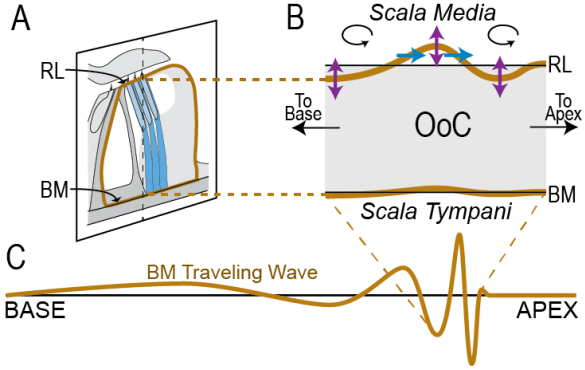

FIGURE 5. Organ-of-Corti (OoC) area changes acting on Scala Media (SM) fluid around the peak of the traveling wave. **A**. An oblique view of an OoC cross-section showing the locations of the reticular-lamina (RL) and basilar membrane (BM) points diagramed in B. **B**. A *longitudinal* section through the OoC (at the dashed line in A) showing motions at one point in time in the RL abutting SM fluid, and in the BM abutting scala tympani fluid. The purple double-tipped arrows at top show motion directions for OoC expansion (up) and contraction (down) that act on SM fluid within a given OoC cross section. The blue horizontal arrows indicate the direction of the forward travel of RL corrugations and the direction of the net forces they may exert on SM fluid. The motions shown in B represent the net effect of RL motion (which includes the whole OoC surface that abuts SM) on SM fluid in the short-wave region where there are changes in OoC area. In the long-wave region where there are minimal OoC area changes, the motion of the OoC top, lateral of the OHCs (near the Hensen cells), would be approximately opposite in phase to the motion over the OHCs, so the net effect on SM fluids would be small. **C**. A cartoon traveling wave with dashed lines showing the region considered in B.

Human traveling-wave amplification. The anatomy and motion of the cochlear partition is different in human temporal bones than in smaller laboratory animals. The cochlear partition is much wider in humans than that in lab animals and has a non-body "bridge" between the bone of the spiral lamina and the medial edge of the BM [26]. In response to tones, the point of greatest motion in the human temporal bone is at the bridge-BM boundary, not near the center of the BM as in lab animals [26]. An important consequence of this is that when the BM moves up, in human

temporal bones the OoC rotates clockwise, whereas in lab animals it rotates counter-clockwise [26]. With many OHCs-act-on-BM theories, the OoC rotation in humans would lead to cochlear amplification at all frequencies, but a wide range of data indicate that the pattern of cochlear amplification is similar in humans and lab animals (e.g., [27, 28]). This seeming anomaly would be removed if traveling-wave amplification came from an OoC-area-change drive (as in Figs. 2, 5) and not from OHCs-act-on-the-BM traveling-wave amplification.

Another way humans are somewhat different from lab animals is in frequency selectivity, which is much sharper in humans when measured in frequency bandwidth, but not when measured in the spread of tuning along the BM [29, 30]. However, in addition to the human cochlea being much longer than cochleae of lab animals, humans have much longer otoacoustic emission delays [30]. These two things together indicate that the wavelength of the traveling wave near corresponding BFs is similar in humans and lab animals. If, as is proposed here, the wavelength controls traveling-wave amplification, then the similarity of the sharpness of tuning in humans and lab animals, when expressed in distance along the BM, may be due to their similar traveling-wave wavelengths near BF.

DISCUSSION

Active processes within the cochlea increase the motion responses of the structures within the cochlea. This increase is called "cochlear amplification." When the only cochlear structure that could be measured was the BM and cochlear amplification occurred only at BF and slightly basal, the term "cochlear amplification." was unambiguous. However, optical coherence tomography (OCT) allowed motion of structures within the OoC to be measured and revealed more extensive motion amplification than was seen on the BM. This has produced confusion about the relationship of the various amplifications. Here we propose that two kinds of cochlear amplification should be distinguished because of their different properties and consequences. Two kinds of cochlear nonlinearities were distinguished by Fallah et al. [10] that may correspond to the division made here, but identifying these as amplification, not just nonlinearity, seems important for understanding their origins. Our division has allowed us to form a series of hypotheses that indicate how both kinds of cochlear amplification are produced and how they differ.

The most important hypotheses put forth here are: (1) in the short-wave region OHC motility causes oscillatory longitudinal flow of cortilymph along the OoC tunnels, but in the long-wave region there is little cortilymph flow, (2) the longitudinal cortilymph flow causes oscillatory changes in OoC area, and (3) the changes in OoC area drive traveling wave cochlear amplification by applying forces to SM fluid. Perhaps, the action of an OoC-area-pump strictly in the transverse plane (which requires good matching of OoC-area forces and SM pressure/motion) may not fully explain the data. Perhaps instead of, or in addition to, an "OoC-area pump" we should think of an OoC-area *longitudinal wave* as adding energy to the traveling wave (Fig. 5, blue arrows). The OoC-area longitudinal wave appears to have the advantage of not needing an exact match between the phase of the OoC-area change and the local SM pressure/motion. Overall, exactly how the OoC-area-change adds energy to the traveling wave is not yet clear.

The phase relationships between basilar-membrane motion and OHC contractions appears to be different between the mouse 9 kHz region [24] and the gerbil 45 kHz region [6]. Although methodological differences cannot be ruled out (e.g., the measurements were not actually from the structures the data were assigned to, and/or the data were contaminated by longitudinal motion), these differences across species provide a reason for thinking that traveling-wave amplification may differ across species and/or frequencies.

The idea that OHC contractions reduce OoC area and force fluid into the OoC tunnels, and this produces cochlear amplification, was proposed many years ago by Mountain and coworkers [13, 31, 32] as part of their "fluid pump" model. In their model, longitudinal fluid flow in the tunnel was one of two interacting traveling waves, and traveling-wave amplification was produced by the interaction transferring energy from one wave to the other. When this model was proposed, the only known cochlear amplification was BM-motion amplification; motion in the long-wave region was thought to be passive and was not considered in detail. The present hypothesis differs from that of Mountain and coworkers [13,31-32] in how traveling-wave amplification is produced. Here the main drive of traveling wave amplification is hypothesized to be the change in OoC area adding energy to the traveling-wave from forces on the scala media fluid. However, an interaction between two traveling waves as envisioned by [13,31-32] is not ruled out by our traveling-wave-amplification-from-OoC-area-change hypothesis. Cochlear traveling-wave amplification produced by RL and TM forces acting on SM fluid has recently been proposed by Altoe et al [33] from their analysis of the motions of the TM and RL.

For the data of Cho and Puria [6] shown in Figure 4F-G, our analysis indicates that traveling wave amplification was *not* produced by OHCs-act-on-BM cochlear amplification with the motion at the bottom of the OHC coupled directly to the BM by the Deiters-cell body. It is possible that OHCs-act-on-BM cochlear amplification might be

produced by a feed-forward mechanism [e.g., see 20, 21]. However, the models demonstrating that traveling-wave amplification can be produced by feed-forward mechanisms have derived their OHC motility phase from BM motion or traveling-wave pressure using a formula, and not from the deflection of OHC stereocilia produced in a realistic way from RL and TM motions. Whether these feed-forward models work when using actual measured phases of OoC motions has yet to be demonstrated.

The hypotheses put forth here need testing. The most straightforward test is to measure motions along the SM-facing side of the OoC to determine whether there are OoC area changes with the hypothesized timing. It may also be feasible to measure the predicted motion within the tunnel of Corti, as was done in [13] using tunnel-crossing fibers visualized as in [34]. The degree to which energy is added to the traveling wave by the OoC area change producing forces acting in a transverse direction on SM fluid, and/or by an apically moving RL-deformation wave, need to be tested by models that include these motions along with SM pressure and 3-D motions within the SM fluids.

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COMMENTS & QUESTIONS

[*Post-Talk Q&A*]

Marcel van der Heijden: Excellent talk. You did [not] mention our study, 2018 Hotspot paper, where we actually do present some evidence for longitudinal motion within the OoC, but contrary to your scheme, it is large [in the] low [frequency] tail, so the longitudinal motion that we actually measure in the OHC-DC region is dominant in the low [frequency] tail, in the long-wave region rather than in the short-wave, CF, region. I don't know why that it, but it's a pretty strong experimental fact. How do you account for that?

Author: There is no incompatibility between that and what I am saying. First of all, I would point out that the experiments I used for the second example, Cho and Puria (2022), is something [are experiments] where they were able to orient their optical system very much perpendicular to the OHCs so they were mostly measuring transverse motion and not longitudinal motion. There certainly [could] be longitudinal motion [in that experiment]. I expect that there is a lot [but it is perpendicular to the measuring angle].

Marcel van der Heijden: There is a lot of it [longitudinal motion]. So even if you tilt a bit and you don't have perfect control, it [longitudinal motion] can be completely dominant, especially at low frequencies. That's what we observed. So don't count on it [your optical system] being perpendicular. That's not important, because it's [longitudinal motion] so big it will dominate easily, even if you don't want it.

Author: Well, I think there is that [longitudinal motion at frequencies way below CF], and that it's not producing cochlear amplification, it's producing … it's part of what is seen in non-propagating amplification. It's not producing an area change. I suppose that it could, in the short-wave region, produce an area change, but if everything is moving together, the area in those regions is not changing. If, in the short-wave region, some part is moving this way and some part is moving that way, there could be an area change due to that [motion of solid structures]. So, it [the finding that the Deiters cells show transverse motion] is completely compatible [with my proposed theory]. That would be saying that, in addition to the fluid structures that I've talked about, the cellular structures also have some degree of flow that's like that [the hypothesized oscillatory longitudinal flow of fluid in the tunnels].

Marcel van der Heijden: So you are basically arguing that there needs to be a differential, ... a longitudinal gradient in longitudinal motion. That's what you need for your model. You need a gradient, not uniform longitudinal motion.

Author: Well, a gradient with, [coupled to] the wavelength, and our data would certainly be consistent with it [the structural longitudinal motion] changing with the wavelength of the traveling wave. It's the wavelength of the traveling wave that's, in fact, the controlling factor.

Author [added post meeting]: Our hypothesis that the OHC, Deiter-Cell-PhP, and the RL, form a bellows that acts to force fluid out of the peri-OHC space (see Fig. 3, which has been updated from the figure shown in the preliminary manuscript), may also explain why the Deiter-Cell body is driven in the longitudinal direction. As the OHCs expand or contract, they push or pull the top of the Deiters-cell body in a longitudinal direction (e.g., as shown in the movie of Motallebzadeh et al., 2018). Thus, the longitudinal motion of the Deiters-cell body, and the surrounding

OoC tissue, may be a *biproduct* of the OHC-induced bellows-like motion that produces the oscillatory flow of cortilymph in the OoC tunnels.

Karolina Charaziak: Hi John, very nice presentation. I have one question regarding the motion of the lateral compartment in the long wave region. Based on your cartoon, it looks like the lateral compartment moves together, or in upper ……[not understood]

Author: If this is the lateral compartment up here [pointing to side]. [then indecipherable Charaziak and Guinan both talking]

Karolina Charaziak: My question is, in general, do you think the deformation can be coupled to fluid and can be the source of backward pressure waves for otoacoustic emissions generated in the long wave region from non-propagating amplification?

Author: I'm lost. I didn't get the question properly.

Karolina Charaziak: I was wondering if you think the change in the lateral compartment … in opposition to the OHC contractions in the long-wave region, could be the source of otoacoustic emissions that come from the long wave region, as an artifact of non-propagating amplification?

Author: I don't think so for two reasons. One is that this is the lateral compartment [pointing to part of Fig. 2A] and it is moving in the [opposite direction to the OHCs, as you stated], and as long as the [OoC] area is the same, it [the movement of the lateral compartment and the OHC region] won't produce a net [oscillatory] flow …. a net displacement of scala media fluid.

Also, in the Goodman et al. (2020 and MoH 2022) experiments, where a tone was put in and the ototoxic drugs [being forced through the cochlea, apex to base] pass the CF region [and flow into the region >2 octaves more basal], that tone would be producing motion [of the RL and lateral compartment] in the long wave region, [but] there is [Goodman et al. observed] no change in the otoacoustic emission [the SFOAE] even though its [the drug] is wiping out the OHC motility – but it's [the drug flowing through the far-basal region] not changing the otoacoustic emission [the SFOAE]. So the motion is not coupled to the traveling wave. More evidence on this will be presented by Goodman (who is sitting next to you) in a few days.

Karolina Charaziak: Great, and I will present opposing evidence. [Laughter]

Author [added post meeting]: In her MoH talk Karolina presented evidence of SFOAE-like residuals at the probe-tone frequency when there was a suppressor tone 2.1 octaves higher in frequency than the probe tone. In contrast, the relevant Goodman et al.'s evidence [see the 2022 MoH proceedings] is for SFOAEs produced by a single probe tone and did not require any other tone to demonstrate the SFOAE (pharmacological reduction of OHC motility was used). The Goodman et al., data imply that the residual measured by Charaziak with a suppressor tone 2.1 octaves higher than the probe tone, is not there when the suppressor tone is not there, i.e., the SFOAE-like residual was produced by nonlinearity from the suppressor tone and is not a component of the SFOAE produced by the probe tone alone.

[*Online Forum*]

Riccardo Marrocchio: This is an interesting paper in which a new theory regarding traveling wave amplification is proposed. I think it would be helpful, to better understand the main ideas of the new theory, to add some explanatory figures. Also, it may be interesting to add a section regarding proposed experiments to test these hypotheses.

Author: The final manuscript version has figures. A brief statement of proposed experiments has also been added. A longer description of possible experiments is in a longer version of this work submitted to Hearing Research.

Jonathan H Siegel: I find the speculation hard to evaluate. Hensen cells have little cytoskeleton, so why wouldn't they be expected to deform to minimize local organ of Corti area changes?

Also, in light of the paper by Charaziak (MOI_08), do you still stand by the statement "Also, solutions that block OHC motility and remove non-propagating amplification far basal of a tone's BF have no effect on otoacoustic emissions, showing that the motion increases from non-propagating amplification don't couple to backward traveling waves [11]?" Regarding the reference to the Goodman et al paper (ref 11), it provided no evidence for the absence of sources of SFOAEs originating far basal to the probe's peak since this study demonstrated the SFOAE using suppressor tones close to the frequency of the probe tone. The effect of a suppressor tone is generally localized to its own place, not very far basal.

Author: We do expect the Hensen cells to deform and minimize local organ of Corti area changes in the long wave region, where we hypothesize that there is minimal fluid forced into the OoC tunnels. However, in the short-wave region, we expect behavior like that found by Kavarataki and Mountain (2007) in which OHC contractions force fluid into tunnels and there are OoC area changes.

For the questions regarding the Charaziak paper and the Goodman et al paper, see my reply to her question presented right after the talk. Also see the Goodman et al paper from this meeting, which was able to measure SFOAEs without using any suppressor tone (instead it used pharmacological blocking of OHC mobility throughout the cochlear length). Goodman et al's results showed that the SFOAE produced by a single tone has little or no component that arises more than two octaves basal of CF. The large SFOAE-like residuals measured with far-basal, so-called "suppressor," tones are produced by the "suppressor" producing a local discontinuity that reflects the probe-frequency energy. These SFOAE-like residuals are not present when just the probe tone alone is there, i.e., they are not showing a component of the SFOAE produced by a single probe tone.

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