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**Review Article** 

## New Auditory Signal Pathway to Receptor

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### Received date: May 15, 2024; Accepted date: May 24, 2024; Published date: May 31, 2024

Citation: Jan Myjkowski, (2024), New Auditory Signal Pathway to Receptor, *J, Clinical Case Reports and Studies*, 5(4); DOI:10.31579/2690-8808/198

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## Abstract

The paper points out the shortcomings and doubts of "Bekesy's travelling wave" theory of hearing. Announce in 1928, it is a typical mechanical theory of hearing [1]. It has been supplemented many times later on, but its foundation in terms of the role of the basilar membrane and resonance, as well as hydrodynamics of cochlear fluids is constant. The theory does not properly explain all the processes that make up the reception, processin and transmission of auditory information

Key Words: hydrodynamics; phenomenon; mechanical theory

## Introduction

The paper points out the shortcomings and doubts of "Bekesy's travelling wave" theory of hearing. Announced in 1928, it is a typical mechanical theory of hearing [1]. It has been supplemented many times later on, but its foundation in terms of the role of the basilar membrane and resonance, as well as hydrodynamics of cochlear fluids is constant. The theory does not properly explain all the processes that make up the reception, processing and transmission of auditory information. This paper is an impetus for a new analysis of hearing mechanisms consistent with the current state of knowledge at the submolecular level in agreement with physics and quantum chemistry [2]. There are already some signs of a new vision of hearing, but a broader discussion is lacking. Belief in Bekesy's dogma is still too strong. The paper presents evidence of existence of a simple and rapid auditory signal pathway to the receptor through the cochlear bone housing [3].

## Problems with Bekesy's travelling wave theory:

1. Cochlear implant surgery for partial deafness causes immobilization of the basilar membrane, but does not ruin the hearing that was there before. This indicates a different signal pathway to the receptor, without the mediation of the basilar membrane and cochlear fluids.

2. Traveling wave damping – cochlear fluids on both sides of the basilar membrane have properties that dampen vibrations of the basilar membrane. There is a deficiency of accurate analysis or calculations.

3. The diameter of the cochlear ducts decreases from the base to the top. According to the theory, the width and thickness of the basilar membrane increases on its way to the cap. The size of the cochlear canals at the base is 1.7 mm, and the width of the basilar membrane separating these canals is 0.25 mm? At the top of the cochlea - according to the theory - the width of the basilar membrane is 0.75 mm. [4].

4. Natural vibrations of the basilar membrane were calculated for vibrations in the air, without taking into account cochlear fluid attenuation and without taking into account the mass of the organ of Corti on the basilar membrane. In humans, natural vibrations of the basilar membrane are 16 Hz - 20 kHz. Mammals (mouse, cat, bat) can hear up to 100- 200 kHz. Their natural vibrations cannot reach 200 kHz. The results of studies on natural vibrations of human tissues range between 5 and 100 Hz. [5]

5. The resonance of the longitudinal wave with the transverse wave creates a problem of information transmission. Wave resonance occurs when the frequency and direction are consistent and the damping is lower than the energy of the forcing wave. With a threshold wave and a slightly larger wave, resonance will not arise, because the damping exceeds the energy of the forcing wave.

6. The resonance of a 14.5 m wave at 100 Hz on a 35 mm long basilar membrane may be only residual. The maximum of the amplitude of this wave transmitting energy for the forced wave is 7.25 m. We hear well. There must be another transmission mechanism to ensure hearing.

7. The OHC is not in contact with the basilar membrane. How it pulls the basilar membrane, during the amplification of quiet sounds. At the lower pole of the OHC there is a network of synapses of afferent and efferent innervation. It seems unlikely that with each contraction of the OHC pulling up the basilar membrane, these delicate 50-nm structures that perform such an important function would be subjected to destructive force.

8. Quiet tones amplified by 40 dB are still heard as quiet. Tones that are not picked up, by this method, cannot be amplified. There is intracellular, molecular amplification, for amplifying the energy of signal received but too negligible to reach the brain. [6]

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9. A mechanical method can be used to amplify completely different, strange waves on the basilar membrane, already transmitting different information. Each wave period may contain new information.

10. Waves with a duration of tenths of a millisecond are received [7]. The forcing wave has only 1 or 2 wave periods, full resonance is not possible. Hearing is made possible by a different signal pathway to the receptor.

11. The difference in wave speed in the cochlear fluid and the traveling wave averages 29 times - (1450: 50 m/s). How is the information encoded with such transmission compression? The traveling wave on the basilar membrane from the oval window to the cap -35 mm - "runs" approx. 1 ms. During this time, the sound wave in the cochlear fluid travels a 1450 mm distance with information.

12. A pigeon hears sounds of 5 Hz - the length of wave in the fluid is 290 m. Can resonance occur on a basilar membrane with a length of 5 mm? The center of the forcing wave is at 145 m wave? [8]. Resonance is impossible. Pigeon can hear very well. The signal reaches the receptor by a different route, not through the basilar membrane and cochlear fluids.

13. Owl can hear 0.001 nm waves at the input. In the cochlea, the wave amplitude fades several hundred times. A sound wave approx. 100 times smaller than the diameter of a hydrogen atom will not induce a travelling wave on the basilar membrane. Owl can hear perfectly. It has very good directional hearing [8]. This indicates a different signal pathway to the receptor.

14. We hear 10 dB and 10 kHz - according to the theory and calculations in accordance with Bekesy's theory, in the duct, at the beginning of the atrium, wave is 0.0000000747 nm - how do we hear it? When the wave energy in the cochlea is fading? There is a long way to go to the receptor.

15. We listen to very quiet music at 10 dB, we have very good hearing. Sound wave after amplification in the middle ear is transmitted to the cochlear fluids and induces a wave traveling on the basilar membrane with an amplitude of 0.00026 nanometers (for a frequency of approximately 10 kHz). This is a magnitude 154 times smaller than the diameter of a hydrogen atom, and there is no way for this wave of such amplitude to induce fluid flow, tilting the hairs of hair cells, which are more than 100,000 times thicker than the amplitude of this wave. If there is no receptor stimulation, then there is no mechanical amplification! We hear it. That's because the signal to the receptor takes a different route, through the bone housing of the cochlea [9].

16. The depolarization and contraction of OHC depends on the operation of ion channels, of which work cycle is 2-4 ms. Theoretically, the whole cell can contract after depolarization up to 250/s. [10]. But OHCs contract to 200 kHz ! Maybe the depolarization and contraction of the auditory cell does not involve the whole cell at the same time? Depolarization can involve parts of the membrane.

17. How do the basilar membrane and cochlear fluids encode polytonal information with aliquots, phase shifts, accent, and length of sound?

18. Is there only one mechano-dependent potassium channel on 1 hair of the hair cell? One cadherin strand supports one channel? It should be checked.

19. How do cadherin strands regulate the gating of mechano-dependent potassium ion channels? They regulate the opening and closing of channels according to the frequency and energy of the sound wave, according to the coded information?

20 How is information encoded by the tilting of hairs of hair cells and the tension of cadherin strands pulling the mechanism that gates the openness of mechano-dependent channels? They pull the "mechanism", how do they close the channel?

21. Is OHC just an amplifier for IHC? Does it use its own afferent innervation, receive and transmit information to the brain?

22. There are vibrating elements in the middle and inner ear that have mass. If there is motion, acceleration and mass - then there is inertia. Bekesy's theory does not take this into account. There is no calculation of inertia in the middle and inner ear. Inertia in the ear:  $(2\pi x \text{ frequency})^2 x$  amplitude x mass g/mm/s<sup>2</sup>. "Inertia is directly proportional to amplitude and proportional to the square of frequency. The results of the calculations indicate that a different mechanism is responsible for the transmission of high frequencies, where the sound wave, which has no mass, conveys information via a different path [11]. If in the labyrinth the inertia of the fluid is the basis for maintaining balance, in the organ of hearing inertia plays an important role in hearing high frequencies when the supplied mechanical energy of the sound wave is lower than the inertia of the vibrating elements having mass. Therefore, high frequencies cannot be conducted through the basilar membrane and cochlear fluids. The sound wave, having no mass, goes to the receptor by a different route. The calculations are convincing here. Weight of the vibrating elements of the tympanic cavity = 70 mg. Weight of the basilar membrane with organ of Corti + fluids - unknown.

23. Damping at resonance reduces the amplitude of the forced wave. What is the energy flow from the forcing wave to the forced wave depending on the amount of damping - there are no calculations... How does this affect the encoding and reception of information? There are no analyses.

24. According to the theory, the traveling wave grows from the oval window towards the cap - moving away from the source of vibrations. For long waves, resonance in the first half of the basilar membrane is impossible, and this wave on the basilar membrane grows from the oval window? The problem arises in case of polytones with multiple aliquots - each frequency has its own maximum wave? How does fluid flow and tilting of the hairs of hair cells occur? Is there a different fluid flow for each wave at the same time?

25. How does a 50 Hz wave with a length of 29 m resonate on a 1.0 mm long basilar membrane in a hummingbird when the length of the basilar membrane is 29,000 times smaller than the length of the forcing wave? Hummingbirds can hear well.

26. How are quiet polytone sounds conducted, requiring time-consuming amplification? They are separated from the loud sounds, amplified and separately conveyed to the brain after the time required for amplification? It's difficult to accept.

27. If a 10 kHz wave resonates on the 4th mm of the basilar membrane, at 0.2 ms, then 6-10 kHz resonates on 0-4 mm of the basilar membrane (Ph.D. dissertation, Kamieniecki, Polit. Warsaw, 2018). Can there be 0.00066 mm of basilar membrane per 1 frequency, responsible for frequency resolution? This is unlikely to happen.

28. Stapedotomy improves low and mid frequencies [12]. The action of piston is used, without the action of the incus-stapes joint and the oscillating movements of the stapes, which are important in the transmission of high frequencies. In oscillating movements, half of the stapes plate produces a forward wave and the other half of the plate simultaneously produces a backward wave. This leads to disturbances in fluid flow, destructive interference and disruption of information transmission. Information is transmitted to the bone housing of the cochlea via a sound wave and then directly to the receptor.

29. OHC contraction amplifies quiet sounds rather than amplifying loud sounds by pulling on the basilar membrane? What is the mechanism responsible for the regulation of basilar membrane pull by OHCs?

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30. The basilar membrane, is a fibrous tissue, comes from the germ layer of connective tissue, and has no afferent or efferent innervation. It has no tension adjustment. All-important organs have such innervation.

31. Bekesy wrongly assumed that the sound wave travels on both sides of the basilar membrane and the resulting pressure differences create a traveling wave due to the resonance of the sound wave with the basilar membrane. Physiologically, the sound wave runs in the atrial duct. The sound wave is separated from the basilar membrane by Reissner's membrane, the endolymph fluid, tectorial membrane, the fluid of the hypopharyngeal region and the mass of the organ of Corti with hearing receptors, lying on the basilar membrane. The sound wave passes through the receptor (according to Bekesy), without transmitting any information to reach the basilar membrane, induce a traveling wave that starts a new path of information transmission to the receptor. Nature could not accept such a solution.

32. To simplify the calculations, Bekesy proposed to "straighten the cochlea," creating a straight cochlear duct tapering in the middle. This changes the laws of physics - there is no wave reflection, reflection attenuation, absorption attenuation, interference attenuation. Adopting such a calculation methodology with the removal of Reissner's membrane distorts the results. For 10 dB and 10 kHz, the vibration amplitude of the stapes plate is 0.000011757 nm, the wave amplitude at the base of the stapes is 0.0000008747 nm, the amplitude of the basilar membrane is 0.00026 nm (W. Gambin - according to Bekesy's calculation methodology). It's not possible for energy of a 10 dB wave with an amplitude of 0.05 nm at the input to decrease 570,000 times in the initial section of the atrial duct. The paradox is that we hear a tone of 10 dB. How is this possible? There is only one logical answer. The sound wave travels to the receptor by a different route bypassing the cochlear fluids and basilar membrane, with no loss of energy. The path leads from the tympanic membrane, the ossicles of the tympanic cavity from the stapes plate to the bone housing of the cochlea, and with a speed of 4,000 m/s the signal heads to the receptor without much energy loss[10].

33. In studies on human hair cells, hair deflections ranged from 50 nm for basal OHCs to 150 nm for apical OHCs. One should compare the amplitude of threshold wave of 0.01 nm at the input, decreasing hundreds of times along the way, with the possibility of deflecting OHC hairs 10,000 times thicker than the wave at the input. Moreover, the deflection of a hair with a thickness of 100 nm via the cadherin thread is supposed to open ion channel with a diameter of 1 nm at the frequency of up to 200 thousand/s. (in a bat). These movements must encode auditory information (amplitude, frequency, harmonic components, phase shifts, accent, length of sound and melody). The channel opening takes milliseconds or fractions of a millisecond. There must be channel closing after each opening. Cadherin thread is not capable of closing channels. It was thought up (J. Hudspeth) that myosin interacting with actin is responsible for channel closing. None of the myosins are able to work at this rate. A more probable hypothesis is that the sound wave energy acts directly on the mechanism responsible for the gating of mechanodependent ion channels [13], by acting on the sound-sensitive proteins (sound-sensitive molecules) of the ion channel, influencing the conformational changes of the protein regulating the activation and inactivation gates responsible for opening and closing ion channels.

34. Billions of creatures in the world do not have the basilar membrane and cochlear fluids in their hearing organs, but they receive information contained in sound waves up to a frequency of 300 kHz. So there is a direct sensitivity and ability to receive information from wave through the receptor.

35. Acoustic otoemission, according to Kemp, involves contraction of the OHC and pulling on the basilar membrane, which generates sound waves found in the external auditory canal. The hair cell contracts after depolarization, leading to the formation of an action potential. If there are

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OHC contractions, the potential in the auditory nerve must be tested. The outer hair cells have no contact with the basilar membrane, they are located between the Deiters cells, so there is only sliding and friction, without an effective pull-up of the basilar membrane on which the OHCs rest along with massive organ of Corti. The entire conglomerate is embedded in fluid - so the dampening effect of inner ear fluid must be taken into account. Moreover, it is not possible for the basilar membrane to vibrate on its own - it vibrates together with the organ of Corti, fluid spaces, nerves, vessels, hairs of hair cells, cadherin connections (tip-links) and cochlear fluids! Do such vibrations produce sound of a specific frequency? Acoustic emission is common in nature. It is formed in brick walls, oil tanks, bridges, even in a bag of rice and in knee where there is no OHC. The acoustic otoemission mechanism described by Kemp is questionable. There may be other reasons for this phenomenon.

# Physiological possibility of traveling wave formation on the basilar membrane. (Author).

The stapes plate generates waves in the atrial fluid. The wave, traveling in a straight line, encounters the wall of the cochlear duct, which is concave in two planes. Reflections from the concave surface cause the concentration of the reflected wave, which reaches the basilar membrane through the flaccid Reissner's membrane. Multiple reflections result in the summation of wave energy in the basilar membrane. A transverse wave is formed on the basilar membrane, which grows from the oval window toward the cap. High-frequency waves are reflected first. The absorbed energy of long waves, reflected later, is accumulated in a further and further part of the basilar membrane, causing a wave on the basilar membrane. But can such a traveling wave be created when the amplitude of the wave in the atrial canal is even 100 times smaller than the diameter of a hydrogen atom? A signal of this amplitude reaches the receptor - by a different route.

## **Conclusion:**

The collected information, analysis and evidence let us put forward the thesis that there is a simple and fast route of the auditory signal from the middle ear through the bone housing of the cochlea directly to the receptor. This path is the most reasonable and documented for high-frequency tones. There is already plenty of evidence. Further simple research is proposed to confirm this thesis. Acoustic otoemission also requires new analysis. Acoustic otoemission is a common phenomenon and is used in medical research, but there is no logical explanation of the mechanism behind the phenomenon.

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DOI:10.31579/2690-8808/198

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