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Perpetual Motion in the Ear

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Abstract

The paper analyzes the mechanism of amplification of quiet sounds by contraction of the outer hair cell – described in Bekesy's traveling wave theory. Attention was drawn to the lack of amplification adjustment. Failure to take into account the variable energy demand depending on the frequency of the amplified sound. The energy required to amplify higher frequencies increases proportionally to the square of the increase in frequency. The electrochemical energy of chlorine and anions in the hair cell membrane does not meet this condition. Prestin, without drawing external energy, cannot produce new energy to generate vibrations of the basilar membrane together with the organ of Corti and the cochlear fluids. Perpetual motion does not exist in Nature. It exists in Bekesy's traveling wave theory.

Keywords: Molecular Medicine, Molecular Mechanisms of Hearing, Hair Cell Physiology, Outer Hair Cell (Ohc), Prestin Protein

Introduction

The Mechanisms According to Bekesy's Theory

A machine that operates independently, performing continuous work without using external energy – it is a machine called a "perpetual motion machine". This situation occurs in the ear according to the traveling wave theory. It should be clarified that this is not Bekesy's idea, but that of his followers, invented in the 1970s and 1980s. It involves mechanical amplification of quiet tones by contracting the OHC and pulling on the basilar membrane to amplify its vibrations by 40-50 dB [1,2]. Nature couldn't have invented this Vibrations of the basilar membrane are wave motion. The energy required to create vibrations is directly proportional to the vibrating mass and to the amplitude of the deflections of the vibrating element and proportional to the square of the vibration frequency. Closely connected to the basilar membrane is the massive organ of Corti with hair cells that vibrate together with the hairs in accordance with the frequency of the sound wave. The vibrations take place in a fluid that has damping properties. The cochlear fluid mass vibrates in unison with the basilar membrane. The amplification applies to all frequencies – low intensities [3].

The energy required for vibration increases dramatically as the frequency increases. For each frequency a different amount of external energy is needed. To amplify a 1000 Hz, 20 dB sound (0.1 nm amplitude) by any amount requires 100 times more energy than to amplify a 100 Hz, 20 dB sound. Any OHC depolarization that is maximal (according to theory) causes OHC contraction. There is no information as to whether each OHC contraction pulls up the basilar membrane, regardless of sound intensity. It is unlikely that the entire OHC could depolarize and contract to 100 kHz simultaneously. Theoretically, local depolarization could exist. An important role is played by cell wall ion channels, which have a limited channel cycle time [4]. The refractory period and channel inactivation limits the frequency of voltage-dependent ion channel opening. Testing the frequency of OHC contractions by stimulating it with an electric current is a misconception. Ion channels that determine the frequency of depolarization are turned off.

The fundamental question concerns the source of energy to perform the continuous work that causes the wave motion of the basilar membrane - (according to the traveling wave theory). On the outer surface of the OHC wall is a globular protein, prestin, which has a sensor that responds to changes in the concentration of chlorine and anions on both sides of the hair cell [5]. The sensor is the termination of the prestin protein chain. Changes in the electrochemical potential of chlorine are thought to generate conformational changes in prestin, which are thought to

shorten the OHC by 4%-5% in length (OHC length 20-100 µm = 20,000-100,000 nm). 4% of this length = 200 - 1000 nm). If a cell of 100 µm shortens by 4%, it shortens by 4000 nm! If one end of the OHC moves 2000 nm, the wave amplification by the basilar membrane pull-up exceeds 100 dB? The long hair cell, compressed by the globular prestin molecules that surround it, - according to the theory - becomes shorter? Prestin, encoded by the SLC26A5 gene on chromosome 7, is a transporter of chloride and carbonate anions. The change in the electrochemical potential of chlorine acts on the prestin voltage sensors and causes the initiation of conformational changes that are not encoded by the information contained in the sound wave.

Pulling on the basilar membrane does not contain information encoded in the sound wave traveling to the receptor [6]. The claim that prestin is a molecular engine that ensures OHC shortening and lengthening is not credible. Prestin does not derive energy from ATP, like other molecular engines: dynein, kinesin, myosins. Prestin draws energy for its conformational changes from the difference in electrochemical potential of the hair cell membrane, created mainly by the difference in the levels of K+, Na+, Ca++ and Cl- ions on both sides of the hair cell membrane. The equilibrium potential for chlorine is similar to the equilibrium potential for potassium. During each cell depolarization, electrochemical energy of the hair cell wall generated by potassium, sodium, calcium, and chloride ions causes the movement of chloride ions from the cell to the outside through the chloride channels, despite the high level of chlorine outside the cell (cell interior – 10 mmol/l, cell exterior – 140 mmol/l) [7].

During repolarization, chloride ions move into the hair cell. The variable electrochemical potential of chlorine on the cell membrane is responsible for the movement of chloride ions in both directions across the hair cell membrane. The equilibrium potential for chlorine is ~ 90 mv. It was assumed (invented) that the variable electrochemical potential of chlorine is responsible for the contractions of the OHC, which pull up the basilar membrane – as a mechanism for amplifying quiet tones. The electrochemical potential of chlorine is the driving force behind changes in the level of chloride ions on both sides of the cell membrane. It stabilizes the electrochemical potential of the cell membrane. It does not have the ability to transmit the encoded quantized sound wave energy necessary for amplification gradation. It does not have enough energy to generate high-frequency vibrations of the basilar membrane and the organ of Corti. The force to produce high-frequency vibrations is expressed in Newtons - kg x m/s2. According to AI:
20 dB ----amplitude-0.1 nm----100 Hz----2.76 x 10^{-9} N
20 dB ---amplitude 0.1 nm----1000 Hz ----2.76 x 10^{-7} N
60 dB ---amplitude 10 nm ----1000 Hz ----2.76 x 10^{-5} N
60 dB--- amplitude 10 nm ---10 000 Hz ---2.76 x 10^{-3} N
When the wave amplitude increases 100 times (by 40 dB), the energy necessary for wave propagation increases 100 times.

If we add an increase in frequency from 100 Hz to 10,000 Hz, the wave energy required for wave propagation increases 1,000,000 times. Electrochemical energy cannot increase like that. Work performed according to theory, without external energy – it's perpetual motion! Another problem is what wave is amplified by pulling on the basilar membrane. A silent sound wave that generates an electrochemical potential is a wave received by the receptor and the signal is transmitted to the auditory cell, where it is analyzed. If the wave energy is too low to reach the receptor, the wave is amplified at the molecular level in the hair cell. Mechanical amplification cannot apply to the foreign wave on the basilar membrane (which, according to Bekesy's theory, runs at this time as a traveling wave). The intensity of this wave is unknown and the message contained in this wave may have nothing to do with the received, amplified wave. The wave on the basilar membrane is delayed relative to the amplified wave by tenths of a millisecond.

There is no explanation of how to amplify quiet polytones with numerous aliquots, length of sound, and phase shifts when the polytone contains quiet and loud sounds of different frequencies. Are quiet ones separated from loud ones? Amplified and separately transmitted to the IHC and then to the brain? The next problem is the varying energy requirements for amplifying quiet tones at different frequencies. Conformational changes in prestin do not provide external energy - different for all frequencies. Mechanical amplification of quiet tones in the inner ear is impossible. There is intracellular amplification. Contractions of the hair cell pulling up the basilar membrane loaded with the organ of Corti produce vibrations of different frequencies. This vibrating conglomerate has mass, speed of movement and acceleration, and inertia [8]. An increase in frequency by a certain amount causes an increase in inertia proportional to the square of that amount. The generation of an increase in basilar membrane vibration (according to Bekesy's theory) requires a large increase in energy. The energy associated with depolarization is only used to initiate conformational changes in prestin; it does not have the energy to pull up the basilar membrane.

Signal Amplification According to the Submolecular Theory of Hearing

in all senses there is intracellular, regulated, molecular amplification. Most chemical reactions and energy transfer between small molecules take place in 10-14 s. These are reactions at the atomic and electronic level. "Difficult" reactions take place 1000 times slower, but it is still 10-11 s. Intracellular enhancement is a whole complex of factors such as: phosphorylation and dephosphorylation of ion channels responsible for the conductivity of cell membranes, ATP concentration, cAMP and cGMP levels, cell pH, osmotic pressure, presence of ligands, and the work of Ca++ATPase pumps. These membrane-bound pumps play an important role in maintaining fluctuating calcium levels within the cell. Intracellular enhancement is also related to the activity of calcium-binding proteins, where calmodulin plays an important role by influencing the production and breakdown of cAMP and cGMP.

It activates protein kinases and phosphatases, regulates the functioning of the calcium pump. It affects the contraction of muscle and non-muscle cells by activating the cAMP-independent myosin light chain kinase. Calmodulin also affects the transmitter's exocytosis. Binding 4 calcium atoms to calmodulin increases its effect 1000 times. The process of enzyme production or the rate of their breakdown is regulated. Calcium is a second messenger of information in the cell, acting faster than the other second messengers: cAMP, cGMP, DAG, IP3, which are produced in

connection with an increase in calcium levels or activated by G protein. The stage of production of second messengers is one of several mechanisms of intracellular amplification. One enzyme molecule can produce several hundred-second messengers. Received tones of which energy is too low to reach the brain are amplified. Intracellular signal amplification is one of the main pillars of the "Submolecular Theory of Hearing" [9].

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