

The Auditory cell: A Hearing Organ Receptor

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Abstract

In published studies, much attention is paid to the path that auditory information is transferred to the receptor by means of sound wave resonance in the cochlear fluid with natural vibrations of the basilar membrane. Much focus is put on the traveling wave on the basilar membrane and to liquid flows in the inner ear that supposedly trigger the tip-links mechanism. Sound signal amplification is explained by contractions of external auditory cells and pulling up of the basilar membrane. The mechanism of how sound wave energy works on the hearing receptor is not discussed.

In the study, particular emphasis is put on the change in the pathway taken by the signal on its way to the receptor and the processes that are taking place in the auditory cell alone, including the receptor, with respect to receiving, processing, and transferring auditory information. Attention was drawn to the manner in which the signal is amplified on its way to the receptor. All the transformations occur on a molecular level and a submolecular level.

Keywords:

Introduction

Bekeşy's travelling wave theory holds that sound wave resonance in the cochlear fluid with natural vibrations of the basilar membrane is the mechanism that transfers information to the receptor. It provides a description of the cochlear fluid flow and the tip-links mechanism that supposedly transfer and encode auditory information. Soft tone amplification is purely mechanical [1,2].

It should be noted that:

- a) natural vibrations of the basilar membrane of mammals that can hear sounds up to 100 kHz do not create any resonance with high-frequency sound waves,
- b) a sound wave is a longitudinal wave, a travelling wave is a transverse wave, the resonance of these waves is unlikely, whereas precise transfer of information is impossible.
- c) basilar membrane vibrations in the cochlear fluid are dampened. If the dampening of the reflected wave is greater than the energy of the incident wave, no resonance occurs. This situation pertains to audible threshold and peri-threshold tones. This points to a different pathway
- d) the tones that are audible are tenth of a millisecond long, whereas 1 or 2 periods of a wave does not cause resonance of the wave and the traveling wave [3,4],
- e) the basilar membrane ballasted with the organ of Corti and connective tissue on the lower surface, is vibrating in the cochlear fluid that substantially dampen the vibrations [5].
- f) the speed of sound wave propagation in the cochlear fluid is 1450 m/s and is constant, whereas the speed of the travelling wave is variable ranging from 50 to 2.9 m/s (Bekeşy). The difference in the speed varies from 29 times to

500 times in the vicinity of the cupula. With the wave speeds that different, information transfer is impossible.

- g) the transfer of the quantized energy of a sound wave, harmonics, phase shifts, and the quantitative via the travelling wave, via the cochlear fluid and the tip-links mechanism is impossible. The energy that codes the auditory information is transmitted by the sound wave without a shift in the mass of the environment. In the same way the information is transmitted also via the bone labyrinth of the cochlea to the receptor [6],
- h) the rocking motion of the stapes at a high frequency disrupt the transfer of information on the cochlear fluid. When one half of the footplate is generating progressive motion, the other half is generating a regressive wave at the same time.
- i) the tympanic membrane fails to reflect 99.9% of sound wave energy. It absorbs and transmits about 80% of the sound wave energy to the middle ear, part of the energy is transferred onto the bone labyrinth of the middle ear. Part of the sound wave energy from the ossicles of the middle ear is transmitted onto the bony labyrinth of the cochlea.
- j) in stapedotomy surgery, there is no high-frequency sound transmission.
- k) in middle ear, soft tones are amplified by 33 dB whereas in the inner ear, by 40-50 dB, and continue to be heard as soft tones.
- l) decreasing sound wave energy on its way to the receptor precludes hearing of threshold sounds.
- l) the latency in ECoG testing of soft tones at 1.5 ms precludes signal propagation via the travelling wave, the cochlear fluid, and the mechanical amplification.

Mechanical amplification of soft tones by means of an OHC contraction is time-consuming. The received tone has to trigger depolarisation of OHC so

that an OHC contraction can take place. Each depolarization of a cell causes an OHC contraction – including very loud tones. There is a problem regarding amplification of high-frequency sounds. An auditory cell cannot shrink down to 100 kHz. Depolarization of a cell is determined by the work of ion channels of the cell wall [7].

Pulling by the basilar membrane increases amplitude of the unknown wave that runs across the basilar membrane at that time. The information carried by both waves simultaneously gets mixed up. In the case of multi-tones, soft tones that are amplified reach the centre with a delay, separately from the transmitted information of loud tones. They distort subsequent information transmitted at that time that require no amplification.

The tip-links mechanism does not serve its role in the information transfer close to the threshold of hearing. The sound wave amplitude, which is 8 picometres on entry and fades on its way through the basilar membrane and the cochlear fluid by about 100 times [8], cannot lean or bend auditory hair cells. The amplitude of this wave is about a million times smaller than the diameter of the auditory hair cells. The movements of the hair cells supposedly code and transmit information to the molecular system of the hearing receptor by tightening cadherin filaments. How do cadherin filaments transfer the energy of overtones, phase shifts, and the quantitative to sound-sensitive molecules? That is not possible. They do not ensure energy quantization.

The above reservations pertaining to Bekesy's travelling wave theory allow one to put forward a new hypothesis about the pathway a signal takes to the receptor as well as the molecular mechanism for receiving and processing auditory information. Coded with quantized sound wave energy, auditory information is transmitted from the auricle, the middle ear and the lateral wall encircling the oval window to the bone labyrinth of the cochlea, further straight to the hearing receptor. The transfer takes place on a molecular level and an electron level. Further analysis and processing of the information takes place in an auditory cell. Low frequency sounds that are easily transmitted through all environments propagate also via the cochlear fluid with the reservation that the information is transferred by a sound wave without moving the mass of the environment [9].

Auditory cell

The sound wave reception mechanism with a transformation and transmission of mechanical energy to the electrical energy of the receptor further to the energy of chemical bonds of intracellular information messengers takes place on a submolecular level and an electron level. The stimulus adequate for the hearing organ is sound wave energy transmitted by vibrating particles of the environment to the receptor. All the atoms in the matter of the environment that transfers sound waves and molecules and atoms that comprise the hearing receptor are in constant motion. It is progressive motion – oscillatory and rotatory motion or a mixture of the above. Motion is linked to kinetic energy of molecules. Kinetic energy aside, molecules of the receptor have also potential energy composed of chemical bonds and electrostatic attraction and electromagnetic forces. The sum of these energies makes up the inner energy of a molecule's body. The total inner energy is determined also by temperature and the mass of the molecule. Supplying external energy - in the case of a hearing receptor – that of a sound wave, to the molecule of the (hearing) receptor causes an increase in the internal energy of the sound-sensitive molecule that receives the signal.

The vibrating particles of a sound wave have a positive, negative, or neutral electric charge. Neutral particles transfer potential energy determined by the speed of vibrations and frequencies and electron energy by means of contact of the electron clouds of molecules. Each atom has electrons that form an electron cloud around the nucleus of the atom. The size of that cloud is determined by the number of orbits that electrons are positioned on. An electron can change its orbit; however, to jump to an orbit closer to the nucleus, it has to receive additional energy. Changing 1 orbit from 2 to 1 requires 3.4 eV. These shifts are quantized, meaning that there is either a jump or no jump – with no other possibility in-between. If an atom in a molecule of an acceptor receives a quantum of energy from another atom or a particle (sound wave), then the electron of the acceptor jumps to an orbit that is closer to the nucleus – its internal energy rises – incrementally –

quantized. The so-called excited state of an atom that, unlike the basic state, is impermanent, lasts about 10^{-8} seconds and immediately strives to return to the basic state by emitting 1 photon of energy – when the issue lies in 1 atom moving by a single orbit. If in the combination a molecule of the receptor + sound wave energy there are countless jumps like that, or jumps by 2 orbits or more, then there are 10^{20} possibilities for transmitting various kinds of quantized energy. This yields unlimited number and diversity of transmitted bits of information. The duration of simple shifts in energy between molecules is 10^{-14} s. More complex transformations last 1,000 times longer but still, that is 10^{-11} s. The information-coding energy is transferred from the sound-sensitive molecules that receive the information reaches to the molecules tasked with mechanically-gated potassium ion channels. The transferred energy acts on the activation gates and inactivation gates of the potassium ion channel that determine the flow of potassium from endolymph to the auditory cell. The energy of a sound wave causes changes in the rotation of atoms, shifts in the angles of bonds, oscillation, consequently leading to conformational changes in molecules, while the formed conformers by changing their shape and size, by fulfilling their role involving closing and opening of an ion channel. This role is regulated by the information encoded in the sound wave with the precision of electron energy.

The mechanically-gated potassium ion channel has a selectivity filter that ensures flow of potassium ions exclusively. With the potassium ion channel fully open in 1 ms as many as 6,000 potassium ions flow from endolymph into the auditory cell.

The inflow of positively charged K^+ ions into the cell triggers depolarization of that cell. If that change in the membrane potential increases over the level ca. 10 mV, potential-dependent Ca^{++} ion channels are activated. Local depolarisation increases, subsequent depolarisation-dependent Ca^{++} channels and Na^+ channels on the lateral surface of auditory cells open up. The interior of the cell has a negative potential - 80 mV, owing to a high number of proteins with a negative charge and the ongoing work of sodium-potassium pumps that discharge 3 sodium ions outside the cell in exchange for 2 potassium ions imported into the cell. Outside the cell, there is a high level of Na^+ , Ca^{++} , and Cl^- , which combined with the high electric potential creates a high electrochemical potential for sodium and calcium ions. The wave of Ca^{++} ions flowing into the cell causes calcium ions to be released from mitochondria, endoplasmic reticulum and the nucleus of the cell. The level of calcium outside the cell is 10,000 times higher than the basic calcium level in the cell that can increase even up to 100 times. Following excitation, the membrane potential changes to receptor potential. Inside the cell, calcium bonds with calcium-dependent proteins, thus changing their properties. The most important one is calmodulin.

The information is divided into constitutive actions related to the ordinary role of a cell and regulated actions related to producing, transporting and discharging the transmitter to the synapse. After the calcium level is increased and the information is transferred, the calcium level in the auditory cell drops instantaneously. Calcium pumps and ion exchangers discharge calcium ions outside the cell, and some calcium is imported back to the mitochondria, endoplasmic reticulum and the nucleus of the cell. The lower the calcium level in the cell, the more the cell is susceptible to receiving a new signal. Intracellular transmitters (messengers), varying calcium levels and the action of intracellular proteins are responsible for intracellular amplification [10].

An important factor in the forming of the membrane potential is the different permeability of that membrane for different ions. The cell membrane at rest is most permeable to potassium ions; for this reason, the resting potential of the cell membrane is similar to the resting potential of potassium. The natural drive toward an equilibrium state regarding concentration and potential on both sides of the membrane, in line with the second law of thermodynamics, eliminating membrane potential as a result. This is counteracted by the presence of ion pumps and ion transmitters, the active mechanism that creates a difference in the concentration and potential on both sides of the membrane. They form a chemical and electric gradient. The cell is negative on the inside with the potential of ca. -80 mV compared to the environment.

It is assumed that as a reference point, the potential of the environment of a cell is zero.

When it comes to auditory cells, the situation is exceptional since the side and bottom walls of these cells are in contact with perilymph or interstitial fluid with a low concentration of potassium ions and a high concentration of sodium ions (K^+ 7-8 mEq/l, Na^+ -140 mEq/l), whereas auditory hair cells and the top part of the auditory cell are surrounded by endolymph with a high potassium concentration and a low sodium concentration (K^+ -150 mEq/l, Na^+ -15 mEq/l). This is exceptionally important for the reception of auditory information.

The performance of ion channels can be affected by a number of factors such as phosphorylation and dephosphorylation of channel proteins, ATP concentration inside and outside the cell, levels of cAMP, cGMP, the cell pH, mechanical energy (sound wave), osmotic pressure, oxidation-reduction potential, or the presence of ligands.

The mechanism that is highly significant for the hearing process is located in stereocilia, which are proteins – molecules sensitive to a given frequency and sound wave intensity, genetically determined, responsive to an adequate stimulus.

Sound wave energy causes an inflow of potassium ions into the cell but at the same time, they are exported outside the cell via potassium channels, the so-called leak channels, in the side wall of the cell. Certain equilibrium in the K^+ levels is maintained. This equilibrium is not affected by an overly low inflow of K^+ ions via the mechanically-gated potassium ion channels of the auditory hair cells. Exceeding a certain threshold causes a domino effect in the auditory cell alone. The higher the density of potassium ion channels on the hair cells and in the top part of the auditory cell, the lower the excitation threshold of an auditory cell. It is unlikely that there is only one potassium channel for every 2 auditory hair cells (according to the travelling wave theory) [9].

Positively charged potassium ions that flow into the cell from endolymph cause depolarization. Cell depolarization is a factor that triggers conformational changes of proteins responsible for the conductivity of voltage-dependent calcium and sodium ion channel in the side and bottom part of the cell.

Sodium channels react to excitation much quicker than potassium channels; hence, the cell membrane becomes permeable to sodium ions first. The electro-chemical potential is significantly higher for sodium ions than to potassium ions. The driving force for ions is greater and affects the speed of sodium ion inflow to the cell. The increasing depolarization facilitates opening of a growing number of sodium channels. A virtuous circle occurs, whereby the inflow of sodium ions increases depolarization and depolarization triggers opening of new ion channels, thus the auditory cell is swiftly depolarized. The membrane potential nears the equilibrium potential for sodium ions, whereas electric potential – which is the driving force for these ions – nears zero. The driving force for potassium ions increases. The rising number of opening voltage-dependent potassium channels hinders depolarization. An inhibitor that has an abrupt effect on depolarization is the phenomenon of Na^+ ion channel inactivation. After about 1-2 ms from the opening of the channel, they become inactivated – in that situation they are closed and non-sensitive to excitation. Potassium influx via potassium channels of the side wall of the cell and the inactivation of sodium channels quickly leads to a reversed situation, whereby depolarization of the cell takes place and, eventually, hyperdepolarization occurs, which is a stimulus that causes potassium channels to close and brings back the state of equilibrium. Repolarization and hyperpolarization alter the state of sodium channels from inactive to closed, sensitive to a new excitatory stimulus.

The membrane potential returns to equilibrium.

The full cycle of a sodium ion channel operation (activation, opening, closing, inactivation) takes about 4-5 ms, allowing the information carried by a sound wave of frequency up to 200 Hz to be received assuming that the entire auditory cell is depolarized at the same time. Receiving higher-frequency sounds requires a higher number of ion channels that are activated

at different times, transferring the information to the synapse, without any simultaneous depolarization and contraction of the entire auditory cell. An influx of Ca^{++} ions enter the auditory cell via calcium channels in the side wall. The calcium level in the auditory cell is ca. 100 nM/l, whereas in the interstitial fluid it is ca. 1,200,000 nM/l. In the cell, calcium is mostly stored in the endoplasmic reticulum and the organelles. Cell depolarization and calcium influx are a signal for releasing calcium from the intracellular calcium deposits. The calcium that flows in via the ion channel together with the calcium released from the cell deposits spreads inside the auditory cell rapidly. As a result of the rise in the cytoplasmic calcium ions, they are bound by specific proteins that are activated in this manner, thus increasing the activity of various protein kinases.

In order for this mechanism to work, there needs to be a mechanism responsible for decreasing the calcium level in the cell very rapidly following the information transfer. This is the task of a pump that moves the Ca^{2+} ion outside the cell in exchange for 2 H^+ ions imported into the cell. $Ca^{2+}H^+ATPase$ also works, which draws energy from ATP. The other mechanism for lowering the calcium level in the cell is counter-transport that depends on the concentration of Na^+ ions, which exchanges two Na^+ ions from the extra-cellular space for a single Ca^{2+} ion that is discharged outside the cell. The third mechanism is ion pumps that move calcium ions from the fluids of the cell to the organelles, mainly the mitochondria, the endoplasmic reticulum, and the nucleus of the cell.

Although calcium regulates numerous intracellular mechanisms, its key task is to participate in the transfer, amplification and dissemination of intracellular information. This consists in Ca^{2+} ions affecting enzymes, namely, adenylate cyclase, phosphodiesterase, phospholipase A_2 , protein kinase

A. Calcium ions are a second messenger and take part in the forming of other second messengers such as cAMP, cGMP, IP_3 , and DAG. Intracellular amplification works by means of second messengers and receptor proteins for Ca^{2+} . Once bound with calcium, these proteins change their biological properties, affect other proteins by means of influencing cellular reactions. There are many Ca^{2+} receptor proteins; particular role is played by calmodulin, which has four binding sites for Ca^{2+} ions. The binding of subsequent ions to binding domains causes conformational changes in a calmodulin particle that increase its ability to bind enzyme particles. Enzyme activation takes place after at least three Ca^{2+} ions bind to them. At the calcium level in the cell at 10^{-6} mol/l, the activity of the calmodulin-calcium complex is 10,000 times higher than the activity of calmodulin alone. It is one of the more important elements of intracellular amplification. This complex acts directly on enzymes or indirectly by stimulating calmodulin-dependent protein kinases that transform enzymatic proteins into their active form by means of phosphorylation.

The calmodulin-related stage of transferring information inside the auditory cell leads to the splitting of the signal that is propagated in different directions. The amplified signal runs forward towards the centre; however, at the same time, calmodulin activates many so-called constitutive processes, that is, processes that occur in a non-excited cell.

Other cellular proteins whose action is dictated by the level of calcium in the cell are gelsolin, troponin C, and parvalbumin. Once bonded with calcium, they activate other cellular proteins. The activity of some enzymes increases in the presence of calcium ions. These include mitochondrial enzymes such as pyruvate dehydrogenase and alpha-ketoglutarate dehydrogenase, as well as the calpain-calpastatin proteolytic system responsible for proteolysis of many cell proteins.

The second system activated by the calcium-calmodulin complex consists in regulating the cooperation of all cell organelles. The third one, relatively slow-paced, consists in regulating protein production, particularly that of enzymatic proteins. Changes can pertain to the enzyme production process or the rate of proteolysis. The speed of a given reaction is impacted also by enzyme activators and enzyme inhibitors. These three process regulation systems work jointly to coordinate constitutive and regulated processes.

Calcium is the second information transmitter that precedes the formation of the other second messengers (cAMP, cGMP, DAG, IP_3) that are produced

by enzymes whose excitation is caused by an increased calcium level or activated by G proteins. The stage of producing second messengers is one of the several mechanisms of intracellular amplification. One enzyme particle can produce as many as several hundreds of second messengers.

The mechanical energy of the external signal, which acts solely as a trigger for a cascade of intracellular reactions, causes constitutive and regulated processes inside the cell. Their intensity is proportionate to the energy of the external signal. Intracellular information transfer pathways are launched. Second messengers are water-soluble and can swiftly move inside the cell. The processing of information in the cell and forwarding it is related to the reversible creation and hydrolysis of phosphor-ester bonds. The forming of bonds takes place owing to kinases, whereas hydrolysis is the task of phosphatases. Each cell comes with a set of ca. 1,000 different kinases, suggesting that these kinases play a key role in intracellular signalling. Kinases are responsible for protein phosphorylation that change their conformation, become active and cause excitation of other proteins, thus triggering a protein activation cascade of the signalling pathway. Phosphorylation is a “turn on and hand it forward” type of action whereas phosphatases which are just as numerous in the cell as kinases are, always operate on the “turn off, end of information” principle. Information transfer is an endergonic process that requires energy from the breakdown of the high-energy compound of ATP or GTP. Two types of hydrolyzing enzymes are operating here, namely, ATPases and GTPases, proteins that are intracellular molecular switchers. They take active part in the transfer of intracellular information.

An auditory cell acts in accordance with two programmes; the first one is related to the life of a cell as the basic unit of an organism whereas the other is linked to transferring auditory information. Both these programmes cooperate, oftentimes using the same information transmission pathways, the same substrates and the same enzymes. The functioning of the latter depends on the proper functioning of the former.

The mechanical energy of the external signal, transformed into the electrical energy of the membrane potential and then to the chemical energy of ionic and covalent bonds, intracellular transmitters, is then amplified and split into the two systems. The smaller the energy of the external signal, the greater the amplification. High-intensity sounds are accompanied by the phenomena of adaptation and dampening. The final product of these transformations (a transmitter) is a tool in the information transfer system. The production and storage of this product is regulated by the first (constitutive) system, whereas the discharge of the transmitter to the synapse is part of the second (regulated) system. If the external signal of a threshold amperage creates a potential of 10^{-9} V on the membrane of a receptor cell, that energy must be amplified multiple times so that it can reach the central nervous system.

Inside the cell, the signal – a portion of energy – moves in the form of a wave at the speed of ca. 0.5 millimetre per second. The frequency of calcium waves codes the information related to sound wave frequency whereas the transferred energy corresponds to the intensity of the sound. An increase in the calcium level in the presynaptic area signals a release of a portion of the transmitter into the synapse. The amount of the transmitter is proportionate to the intensity of the sound – to the energy that releases synaptic vesicles. These vesicles are moved by means of anterograde transport at the moment when calcium-activated proteins break down the protein bonds that fix the vesicles to the cytoskeleton. The molecular force that shifts the vesicles towards the presynaptic membrane is kinesin. A protein complex in the presynaptic area facilitates contact between the vesicles and the presynaptic membrane, makes it easier for them to bind and form a channel that links the interior of a vesicle with a synapse. Retrograde transport is the responsibility of a protein called dynein. These membranes that go back are used for creating new synaptic vesicles. It is the so-called cell membrane recycling. The release of a transmitter into the synapse is linked to a transfer of a bit of information whose intensity and frequency matches that of the signal. About 50 nm wide, the synaptic cleft is filled with a liquid in which the transmitter

moves from the presynaptic membrane to the postsynaptic membrane and then connects with specific ion channels causing them to open. The transmitter is active only for about one millisecond and then becomes separated from the ion channel and is broken down by enzymes present in the synaptic cleft.

The level of the transmitter falls abruptly, after which ion channels become sensitive to its influx. On the postsynaptic membrane depolarization potential arises, called excitatory postsynaptic potential. If a certain depolarization threshold is exceeded, which is ca. 15 mV, then this depolarization moves along the afferent nerve to the next synapse, to a cell of the auditory ganglion.

Synaptic transmission is related to many regulatory mechanisms such as pre- and post-synaptic inhibition and aggregation, spatial and temporal aggregation, enzymatic degradation, and transmitter reabsorption. In the synapse, the energy of the chemical bonds of the transmitter is converted into the electrical energy of the post-synaptic potential transferred to the central nervous system. In the synapse, the information that is being transferred undergoes an encoding process. The encoding entails organizing impulses in a nerve fibre or a bundle of fibres with respect to the quantity and size depending on the intensity and frequency of the sound. In each subsequent synapsis, the information is then decoded, the electrical signal is converted into chemical energy of the transmitter, the basic information that reaches the synapse is integrated with the additional information from interneurons, the chemical energy is converted to the electrical energy of the excitatory postsynaptic potential with simultaneous encoding of this information. Having crossed several synapses and fragments of the pathway between the synapses, the information in the form of energy pulses reaches the central nervous system. It is then decoded, subjected to an analysis similar to Fourier analysis and compared to the information recoded in the non-volatile memory. An image of the audible sound experienced is formed and then recoded in the memory, which can be then recreated even after long years.

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