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Effects of Potassium on Hearing

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ABSTRACT

The auditory system works with the outer ear, middle ear, inner ear and auditory nerve mechanisms working together in the periphery. Any disorder that may occur in the mechanisms can cause hearing loss. These structures work in harmony among themselves and this harmony is also seen between the perilymph and endolymph fluids in the inner ear. The ions in different concentrations in the perilymph fluid filling the bony labyrinth and the endolymph fluid filling the membrane labyrinths must create concentration changes between the hairy cells and the fluids in order for hearing to occur. Thus, the mechanical energy reaching the inner ear must be converted into electrical energy in the hairy cells and sent to the central nervous system. Concentration changes realise hearing by working with the mechanisms of entry into the cell or exit from the cell through the channels. The mechanism of intracellular and extracellular passage is directly related to the uptake, retention and excretion of potassium. Some diseases that occur with disorders in these mechanisms are associated with a history of hearing loss. This study aimed to review the studies evaluating the effects of potassium ion, which is found in different concentrations in the body and in the inner ear fluids, on hearing and to change the quality of life with potassium ion control in some hearing losses and to emphasise the importance of early diagnosis in potassium-related syndromic hearing losses. Studies in which dietary intake, endocochlear concentrations, canal functions and gene mutations of potassium ion were studied were included. The studies included in the review are in agreement that potassium mechanism affects hearing. However, the majority of the studies were animal experiments and studies on humans were rare. The studies report that the potassium mechanism indirectly affects hearing, but do not establish a direct effect relationship of the mechanism of potassium. Therefore, more studies on humans are needed to directly say that potassium deficiency or excess may cause hearing loss or may have a therapeutic effect.

Potasyum'un İşitme Üzerindeki Etkileri

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ÖZET

İşitme sistemi periferde dış kulak, orta kulak, iç kulak ve işitme siniri mekanizmanın beraber çalışması ile işlemektedir. Mekanizmalarda meydana gelebilecek herhangi bir bozukluk işitme kaybına neden olabilmektedir. Dış kulak, orta kulak ve iç kulak yapıları kendi aralarında uyum içerisinde çalışmaktadır ve bu uyum iç kulakta bulunan perilenf sıvısı ile endolenf sıvısı arasında da görülmektedir. Kemik labirentin içini dolduran perilenf sıvısında ve zar labirentlerin içini dolduran endolenf sıvısında değişik konsantrasyonlarda bulunan iyonlar işitmenin gerçekleşebilmesi için tüylü hücreler ile sıvılar arasında konsantrasyon değişiklikleri meydana getirmelidir. Böylece iç kulağa kadar gelen mekanik enerji tüylü hücrelerde elektriksel enerjiye çevrilerek işitme siniriyle merkezi sinir sistemine gönderilmelidir. Konsantrasyon değişiklikleri kanallar aracılığıyla hücre içine giriş mekanizması ya da hücre dışına çıkış mekanizması ile çalışarak işitmeyi gerçekleştirmektedir. Hücre içi ve dışı geçiş mekanizması potasyumun vücuda alınması, vücutta tutulması ve vücuttan atılması ile doğrudan ilgilidir. Potasyumun alınması, tutulması, atılması mekanizmalarında bozukluk ile ortaya çıkan bazı hastalıklarla işitme kaybı öyküsü birlikte görülmektedir. Bu araştırma vücutta ve iç kulak sıvılarında farklı konsantrasyonlarda bulunan potasyum iyonunun işitme üzerindeki etkilerini değerlendiren çalışmaları derlemeyi ve bazı işitme kayıplarında potasyum iyonu kontrolü ile hayat kalitesini değiştirebilmeyi, potasyum ile bağlatılı sendromik işitme kayıplarında erken teşhisin önemini vurgulamayı amaçlamıştır. Potasyum iyonunun; diyetle alımının, endokoklear konsantrasyonlarının, kanal fonksiyonlarının ve gen mutasyonlarının işlendiği çalışmalar dahil edilmiştir. Derlemeye alınan çalışmalar potasyum mekanizmasının işitmeyi etkilediği konusunda fikir birliğindedir. Ancak yapılan çalışmaların çoğunluğu hayvan deneyleri olup insanlar üzerinde yapılan çalışmalara az rastlanmıştır. Çalışmalar, potasyum mekanizmasının işitmeyi dolaylı olarak etkilediğini bildirmekte ancak potasyumun mekanizmasının doğrudan etki ilişkisini kurmamaktadır. Bu nedenle doğrudan potasyum eksikliğinin ya da fazlalığının işitme kaybına yol açabileceği ya da terapötik bir etkisi olabileceğini söylemek için insanlar üzerinde yapılacak daha çok çalışmaya ihtiyaç duyulmaktadır.

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INTRODUCTION

There are three parts of the ear: outer, middle, and inner ear, and when a lession occurs in one of these parts, hearing loss occurs (Gelfand, 2016). Problems in the outer and middle ear cause conductive hearing loss, while problems in the inner ear result in sensorineural hearing loss. A disturbance in the concentration of minerals in the inner ear and the fluids that make hearing possible can also lead to hearing loss (Salvi et al., 2007). This investigation, conducted to determine the effects of the potassium ion, whose concentrations vary in the fluids of the inner ear, on the hearing mechanism and to compile studies on this topic, is intended to be a guide for further studies. This investigation highlights the importance of recognising and improving the quality of life that changes with a decrease or increase in potassium ions in the body.

Mechanism of Potassium

Potassium (K+) is a cation essential for cell function. The electrolyte balance, which results from intracellular and extracellular concentration differences, is crucial for the functioning of many mechanisms in the body. A disturbance in the balance of the intra-extracellular potassium concentration, which is also influenced by the sodium (Na+), hydrogen, ATPase, and magnesium concentration, affects many systems such as the cardiovascular system, the kidneys, and the musculoskeletal system, and can have fatal consequences (Erken & Arınsoy, 2017; Hall, 2016a).

An average adult has about 3500 mEq of potassium, with 98% of the total potassium in the cell, and only 2% in the extracellular fluid, and efforts are made to keep it within a very narrow range of 3.5-5.5 mEq/L. This balance is maintained by diffusion or active diffusion (Hall, 2016a). During intracellular and extracellular transport, Na+-K+ passes through specialized protein channels by diffusion. Protein channels in the cell membrane that control passage close the channels and stop diffusion when the action potential required by the cell is present (Hall, 2016b). The change in concentration caused by active diffusion is also balanced by the active Na+-K+-ATPase ion pump system. The diffusion mechanism works until the concentration in the intracellular and extracellular fluids is balanced, and this pumping system comes into play when more ion transfer is required. Thanks to this system, 2K+ ions enter the cell and 3Na+ ions leave the cell (Hall, 2016b; Insel et al., 2017). As more positive charge flows out of the cell, the inside of the cell becomes negative, creating a potential difference. In addition, this pump is also responsible for controlling the cell size (Hall, 2016c). The cell begins to depolarize due to the resulting potential difference. After this phase, the voltage-dependent Na+-K+ channels come into play. For conduction to take place in the cells, potential difference must start suddenly. The channels generating the sudden difference by influencing the ion flow rate are voltage-gated Na+-K+ channels. These channels control the permeability of membrane and prevent repolarization in a depolarized state and prevent depolarization in a repolarized state (Hall, 2016b).

Potassium level in blood can also fluctuate depending on intake or avoidance of foods with a high potassium content, such as potatoes, spinach, melon or bananas (Insel et al., 2017). The potassium level in blood, rising with potassium-rich meal, is balanced by absorbing sufficient amounts of it into the intracellular fluids and excreting it via the kidneys (Hall, 2016a). An imbalance of the total potassium level in the body is called hypokalemia or hyperkalemia. If the total potassium level in the blood is less than 3.5 mEq, it is called hypokalemia, and if it is greater than 5.5 mEq, called hyperkalemia (Onat et al., 2006).

Hypokalaemia is caused by reabsorption of potassium from the blood into the intracellular space, renal excretion, gastrointestinal losses, metabolic alcoholosis, loss through excessive sweating or inadequate intake, while hyperkalaemia is caused by tissue damage, metabolic acidosis, acute or chronic renal failure or syndromes that predominantly cause hypoaldesteronism (Erken and Arınsoy, 2017; Onat

et al., 2006). The level of insulin in the blood also affects the potassium level and insulin intake in diabetes can cause hyperkalaemia (Hall, 2016a).

The excretion or reabsorption of potassium from the body is ensured by the kidneys. Depending on the glomerular filtration rate of the kidneys, potassium is excreted and reabsorbed, and if this rate is impaired, the potassium mechanism is also disturbed. Potassium excretion and reabsorption, which is directly related to the sodium mechanism, varies under the influence of factors such as food intake, acidosis and aldosterone secretion. In order for intracellular and extracellular potassium levels to be at the required level, a hormonal, acidic and metabolic balance must be established (Hall, 2016a).

Mechanism of Hearing

The auditory system is divided into two areas: the system consisting of peripheral structures and the central auditory system (Belgin, 2017). While the peripheral auditory system is responsible for converting acoustic energy into electrochemical energy, the central auditory system is responsible for processing the electrical message (Seikel et al., 2010). The peripheral system consists of the structures of the outer, middle, and inner ear and the cochlear nerve, the auditory nerve, and is responsible for collecting, filtering and amplifying the received sound wave pressure and transmitting it to the central auditory system for further processing (O'Leary & Rowe, 2014). In the peripheral auditory system, sound is converted into electrical codes by the inner ear structures and the inner and outer hair cells in the cochlea, whereas the middle ear and outer ear use a mechanical way to convey sound (Gelfand, 2016; Seikel et al., 2010). The cochlea, a structure resembling a snail's shell, is in charge of hearing function and separated into two sections: membrane labyrinth running parallel to the bone labyrinth inside the cochlea, and the bone making up the outside portion of the cochlea (Hall, 2016d; Salvi et al., 2007).

After passing through the bony labyrinth, the membranous labyrinth is subdivided into three compartments: 'Scala vestibuli', which fits into the oval window (fenestra vestibuli); 'the Scala tympani', which ends at the round window (fenestra cochlea), and 'the Scala media' which is located in the middle area. Scala vestibuli and scala tympani contain perilymph, which is high in Na concentration. These ducts are separated from scala media by the Reissner's membrane above and the basilar membrane below. Scala media is filled with endolymph, an extracellular fluid that is high in K concentration. The organ of Corti, which contains the auditory hair cells, is located on the basilar membrane. It contains about 3,500 inner and 12,000 outer hair cells, as well as supporting cells. Hair cells are connected to a membrane structure called 'Reticular lamina', under which there is a cortilymph with a high Na concentration, similar to perilymph (O'Leary & Rowe, 2014; Salvi et al., 2007).

The hair cells have cilia that vary in length and bend over one another when the basilar membrane moves. Stimulation happens during this bending when short cilia bend toward long cilia, but not when long cilia bend toward short cilia. The cochlear nerve and inner hair cells synapse to transmit the audio stimulus to the central auditory centers. The cochlear nerve leaves the temporal bone, passes via the internal acoustic meatus canal, and travels in the direction of the brainstem. The spiral ganglion is the first location on this circuit where auditory stimulation is applied (Gelfand, 2016; Hall, 2016d).

The Effects of Potassium in the Hearing Mechanism

Perilymph contains a high concentration of Na (~150 mmol/L) and a low concentration of K (~3 mmol/L), while endolymph has a high concentration of K (~160 mmol/L) and a low concentration of Na (~1.5 mmol/L), and the concentrations of cortilymph are similar to perilymph. However, calcium (Ca) and chloride (Cl-) concentrations in perilymph and endolymph are also similar (Hall, 2016d; Salvi et al., 2007). Na+ and K+ ions cannot move between hair cells making up the embedded reticular lamina

because of their highly tight intercellular connections. The stria vascularis of the lateral wall contains energy-dependent adenosine triphosphate (ATP) electrogenic channels, which are responsible for the endolymph's distinct ionic composition. In the scale environment, these pumps raise the voltage and K concentration while decreasing the Na concentration. Each pump pulse in the canal causes a net gain of +1 in charge since it removes three Na+ ions every cycle and adds two K+ ions to the scale media (Insel et al., 2017; Onat et al., 2006).

According to Hudspeth (as cited in Salvi et al., 2007), there are models of hair cell transduction in which a mechanically gated ion canal located close to each cilia converts sound vibration into neuronal activity. The canal coverings open and the canal connections joined at the cilia tips extend as the cilia bend from short through long. Consequently, K+ ion flow of cortilymph to the hair cell is started. The cell is depolarized as a result. In contrast, the movement of K+ ions are obstructed when bending takes place in the opposite direction; this leads to the outer hair cell becoming hyperpolarized (Salvi et al., 2007).

METHOD

Approximately 50 studies were obtained from the searches made in Pubmed, Dergipark, Scholar Google databases, especially Pubmed. The 26 relevant studies were included in the review. The search was made to cover the last 10 years, and two older studies were added exceptionally because they were considered to be important in terms of results. The literature review was conducted for a total of 8 months in 2023 and 2024 to be examined at certain intervals.

DISCUSSION AND RESULTS

A retrospective cross-sectional study with 5925 participants examined the correlation between Korean individuals' intake of potassium and their hearing thresholds. Numerous factors were assessed, including potassium laboratory results, potassium levels in the food, data from chronic diseases like diabetes and hypertension, smoking, and the degree of physical activity of the subjects. High potassium consumption was found to be inversely correlated with hearing thresholds and hearing loss based on the data gathered and the group-based analyses. The group that consumed more potassium was shown to have lower hearing thresholds and a reduced prevalence of hearing loss. It has been stated that additional research is necessary to determine whether potassium directly affects hearing (Jung et al., 2019a).

Sudden Hearing Loss

A male patient with thyrotoxic hypokalemic periodic paralysis experienced acute hearing loss; blood values were taken, and the potassium level was 1.5 mEq/L. Although the patient's loss of muscle strength was reported to have improved while he was in the hospital, his severe hearing loss, which ranged from 60 to 105 dB, was reported to have not improved. It has been reported that it is unclear whether thyrotoxic-related electrolyte losses – particularly potassium ion losses – are the primary or indirect cause of hearing loss. While it has been suggested that renal tubular acidosis may be the cause of hearing loss in children, hearing loss is not seen in certain diseases, such as nephropathy, where the potassium mechanism is compromised. Consequently, when other parameters are assessed and found to be unaffected, it has been suggested that an electrolyte imbalance, particularly potassium ion imbalance, may be the cause of sudden hearing loss (Moriyama et al., 1988).

Gene Mutations

Measurements of endocochlear potential, distortion product otoacoustic emission, light and electron microscopy, and mutations in the genes of the channels involved in the potassium mechanism and auditory brainstem response (ABR) were performed in an experimental study on mice. The effect

of these canals on hearing was examined in the study, which involved measuring the mice's hearing thresholds at regular intervals. Consequently, it has been reported that the ear canals are linked to hearing and that mutation-related hearing loss increases with the age of the mice. It has been stated that the study would serve as a guide for future studies on improving hearing loss and protecting hearing (Diaz et al., 2007).

Review research asserts that hearing loss is caused by mutations in a potassium channel (Kir4.1), which is known to be encoded by the KCNJ10 gene and is present in syndromes like SeSAME and EAST syndromes. Having a primary potassium ion channel role in the satellite glial cells is shown that surround spiral ganglion neurons as well as a potassium concentration function in the production of endocochlear potentials. It has been stated that hearing loss results from the disruption of the potassium system by functions that are not completed as a result of growing degenerations (Chen & Zhao, 2014).

In both syndromic and non-syndromic sensorineural hearing loss, a cross-sectional investigation observed the expression of many genes known to be involved in the intricate embryonic development of the lateral wall in the fetal cochlea. Specific mutations in various genes (e.g. Pendred syndrome, which is involved in cochlear K+ ion transport) can cause hereditary sensorineural hearing loss. The primary function of melanocytes that develop between 9 and 18 fetal weeks in the stria vascularis and are responsible for K+ production and the formation of the endocochlear potential has been emphasised (Locher et al., 2015).

In a study conducted in Korea in patients with a history of ATP1A2 gene mutation, it was reported that this gene was associated with migraine and sensorineural hearing loss. It has been reported that this mutated gene is associated with the NA+/ K+-ATPase protein, one of the membrane proteins in the hearing mechanism, and causes an imbalance in its homeostasis. This study suggests that NA+/K+-ATPase dysfunction is associated with both migraine and sensorineural type hearing loss (Oh et al., 2015).

In another study on mice, the Gjb2 gene mutation encoding connexin26, which causes non-hereditary hearing loss in humans, was evaluated. It was reported that Gjb2 gene mutation causes hearing loss by disrupting potassium recycling in the cochlea. In gene mutation, the possibility of treatment with early intervention before the cochlea completes its development has been suggested (Chang et al., 2015).

In an experiment on 12 mice, hearing impairments were evaluated by modulating the voltage-dependent potassium channel Kv3.1 channel. It was reported that the high-voltage and low-voltage firing action occurring in the auditory brainstem can be modified and shifted to positive or negative by the mentioned AUT1 and AUT2 modulators. This emphasises the importance and therapeutic potential of potassium channels (Brown et al., 2016). Another experimental study also reported hearing loss due to mutation of the Kv7.4 potassium channel. It has been reported that it may lead to progressive and high frequency hearing loss (Shin et al., 2016).

The effects of mutations in the genes encoding the potassium channels in cell membranes—which permit potassium to flow through—are discussed in a review study. Hearing mechanism contains the channels known as "KCNQ," specifically the KCNQ3 and KCNQ4 subunits, and that gene mutations causing hearing loss are associated with syndromic disorders like Jervell and Lange-Nielsen syndrome. According to reports, hearing loss may result indirectly from disruption of the channels that control the potassium level in the auditory system (Wang & Li, 2016).

The mechanism underlying sensorineural hearing loss is yet unknown. However it is believed that the metabolic mistake known as "Propionic Acidemia," which is brought on by a deficit of the mitochondrial enzyme propionyl-CoA carboxylase, as in Jervell and Lange-Nielsen syndrome, may

possibly be responsible. Hearing loss can result from disruption of the potassium channel mechanism known as 'KvLQT1/KCNE1', which in turn disrupts the potassium's intercellular transport mechanism (Grünert et al., 2017).

In a study conducted by searching health databases in Korea, the relationship between KCNQ4 channel variants and hearing loss was evaluated. One of the variants (DFNA2 variant) was found to have a risk of late-onset hearing loss. The non-syndromic mutation of the KCNQ4 gene disrupts potassium channel activation and causes hearing loss. In this study using Korean public databases, gene screening is recommended for early diagnosis and intervention of hearing loss caused by KCNQ4 gene mutation (Jung et al., 2019b).

Endocochlear potential impairment and irreversible hearing loss have been linked to abnormalities in potassium channels brought on by gene mutations brought on by hypothyroidism (Mustapha et al., 2009). Also the impact of calcium ions on hearing was examined in a mouse study. Although the direct efficacy of potassium ions has not been documented, it has been asserted that calcium ions are required for hearing and that the proper progression of the potassium ion process is important for the calcium ion mechanism (Du et al., 2020).

Age-related hearing loss

In a study evaluating Kv1.1 and Kv1.2 potassium channels of mice, hearing was first evaluated in the healthy state and then hearing was evaluated by blocking the channel with drugs. As a result of electrophysiological evaluations, it was reported that the mentioned channels had an effect on the speed of the response to the stimulus and could vary depending on the age of the mice. It was suggested that the therapeutic effect of channel modulators should also be evaluated (Smith et al., 2015).

In a study conducted on mice, hearing loss was evaluated and associated with the disruption of the homeostatic balance formed by potassium, sodium and ATPase ions with age. As a result of the study, the importance of this balance, including potassium ion, was emphasized. It was recommended to investigate its therapeutic effects (Ding et al., 2018).

In the study where the potassium channels in the auditory system were evaluated, the delays and insufficiencies that developed in the channels due to age, the degeneration of the stria vascularis in the cochlea, and the potassium mechanism disruptions that developed due to these reasons were examined. This was done because age-related hearing loss and the potassium mechanism are effective on each other. It has been underlined that both deterioration in the potassium mechanism and in the high frequency region of the cochlea contribute to age-related hearing loss (Peixoto Pinheiro et al., 2021).

In a study, the degeneration of ribbon synapses in the inner hair cells was mentioned with increasing extracellular potassium levels. It is reported that increased potassium levels in extracellular tissue, especially with age and noise exposure, cause swelling and degeneration of ribbon synapses (Zhao et al., 2021). This study shows that a decrease in potassium levels can cause hearing loss as well as an increase in potassium levels can cause hearing loss.

A potassium channel (Kv7.4 channel) agonist (ACOU085) was injected into the mouse tympanic cavity as part of an experimental study. The results showed that the agonist was absorbed by the perilymph, which delayed the degeneration of outer hair cells and decreased the likelihood of age-related hearing loss. According to reports, the goal of the in vivo investigation was to develop medication therapies (Peixoto Pinheiro et al., 2022).

Noise-induced hearing loss

Potential processes of noise-induced hearing loss were assessed in a review. Although there was

not enough research, it was noted in the study that gene variants and abnormalities in the potassium channels in the auditory system may also contribute to noise-induced hearing loss. However, it has been reported that it is not possible to say that it is the direct cause (Mao & Chen, 2021).

In an experimental study on mice, tinnitus was induced by giving noise after the mice were conditioned on sound. Then, it was tested whether this tinnitus would be reduced by KCNQ2/3 potassium channel activator. While no significant difference was observed in the 1-week result of the experiment, a significant decrease was observed in the tinnitus induced in mice and in the conditioned behaviour accordingly in the 2-week result. This shows the effect of potassium ion and its channels on hearing. The study is reported to be instructive for the use of potassium channel activators in tinnitus treatment (Marinos et al., 2021).

Meniere's Disease

In a different study, 22 rats were split up into groups and given injections of potassium chloride (KCl) solution into their tympanic canals at different concentrations. The ABR test and nystagmus direction change were used to assess the variable hearing thresholds associated with Meniere's disease. It was claimed that the potassium concentration of perilymph may fluctuate, generating Meniere's symptoms, and that Meniere's disease may occur with potassium toxicity (Kamakura et al., 2019).

Potassium Channels and Hearing

In a study of 21 adult male mice under anesthesia, mice were injected with the activator of certain potassium channels (KCNQ2/3 and KCNQ3/5) (ICA-105665). It showed that the active ingredient of the drug, sodium salicylate, reduced the deleterious effects of compound action potentials and the amplitudes of distortion products in otoacoustic emission. According to reports, ICA-105665 may shed light on future research on protection from ototoxicity (Sun et al., 2015).

Tinnitus

In an experiment on mice, 4 different potassium channel openers (retigabine, flupirtinin, NS1619 and isopimaric acid) were used to investigate the therapeutic effect after hyperacusis-induced tinnitus was induced. As a result of the study, retigabine was found to be significantly therapeutically useful. Isopyramaric acid also has similar data, but it is reported that more research is needed with the other two drugs (Wu et al., 2014).

In an experimental study on adult male mice, healthy ABR measurements were followed by exposure to 110 dB SPL noise for 1 hour. In the study, which was concluded by analysing the mRNA level of Na+, K+-ATPase enzyme activity, it was reported that the enzyme activity decreased significantly within 2 hours following noise exposure and remained low for at least 7 days. The importance of ion mechanism in terms of noise-induced hearing loss is emphasised (Yamaguchi et al., 2014).

A mouse trial revealed how potassium channel activators, such as the sodium salicylate antagonist Maxipost and Retigabine (RTG), affected hearing. When combined with sodium salicylate, activators have been shown to protect against peripheral damage and lower the incidence of tinnitus caused by ototoxicity. However, they had no effect on distortion product otoacoustic emissions or compound action potentials when used alone (Sheppard et al., 2015).

Nutrient Intake

In an experimental study on White Leghorn chickens, deafness-causing proteins were found in channels in cholesterol-rich membranes. Although the study associates hearing loss with cholesterol, it

is noteworthy that one of these channels is KCNQ1, the potassium channel. It also reports that cholesterol has an important role in the recycling of potassium in the ear (Thomas et al., 2014).

In a study published in 2024, the relationship between nutrient intake and hearing loss was investigated and the relationship between hearing loss and many macro and micronutrients such as energy, fluid intake, protein, fatty acids, dietary fibre, potassium, magnesium, iron, zinc vitamin A, vitamin D B group vitamins was evaluated. As a result of the study, potassium intake was found to be significantly lower in the group with unilateral hearing loss compared to the group with bilateral hearing loss. It has been reported that especially women experience these nutrient deficiencies more than men (Han et al., 2024).

CONCLUSION AND RECOMMENDATIONS

There are numerous mechanisms at work in the auditory system. Any malfunction that could arise in the mechanisms will impair hearing and lower standard of living. It is worthwhile to research the impact of the potassium process on the auditory system, as it is still not entirely understood. There is general agreement that potassium is necessary for hearing to occur, even though the collected research do not directly reflect on the impact of potassium on the hearing mechanism. Most reports have focused on potassium channel activity and gene alterations. Research has linked gene mutations to hearing loss. Some, but not all, syndromic diseases are mentioned in the discussion section as being associated with hearing loss due to defects in potassium ion channels. In this context, it is thought that the precursors of diseases can be determined by evaluating blood potassium levels, even in the embryonic period. However, blood potassium levels may or may not give information about hearing. In addition to animal trials, human cross-sectional and experimental studies are needed to evaluate blood potassium levels as a precursor of hearing loss. Although more studies on this subject are needed, this conclusion will support early treatment and early use of hearing aids in hearing loss due to potassium ion. Early use of hearing aids is important for individuals with hearing loss not to be separated from their social life. In line with the information we have compiled in our study, it is thought that the detection of individuals who have or may have hearing loss due to potassium ion and the rapidity of the treatment or device process will positively affect the mental and well-being of individuals. Changes in the concentration of potassium ions, especially dietary intake and excretion, reduce quality of life. Research on this topic is recommended to carry out research on this subject and to investigate treatment methods in hearing loss due to potassium ion, especially to explain the function of blood tests in the determination of hearing loss.

Ethical Statement

This article abstract was produced by developing and partially modifying the content of the paper titled 'The Effects of Potassium Ion on Hearing', which was presented online at the 3rd International Symposium on Scientific Research and Innovative Studies but the full text was not published.

Ethics Committee Approval

This article does not require ethics committee approval.

Author Contributions

Research Design (CRediT 1) Author 2 (30%) - Author 3 (70%)

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