NIJZ Nacionalni inštitut za javno zdravje

Zvok, poslušanje in hrup v vrtcih

Sonja Jeram

Nacionalni inštitut za javno zdravje

NIJZ OE Ljubljana, marec, 2014

http://82.149.22.226/~mobcinamb/index.php?ptype=0&menu=0&id=176&echosub=1

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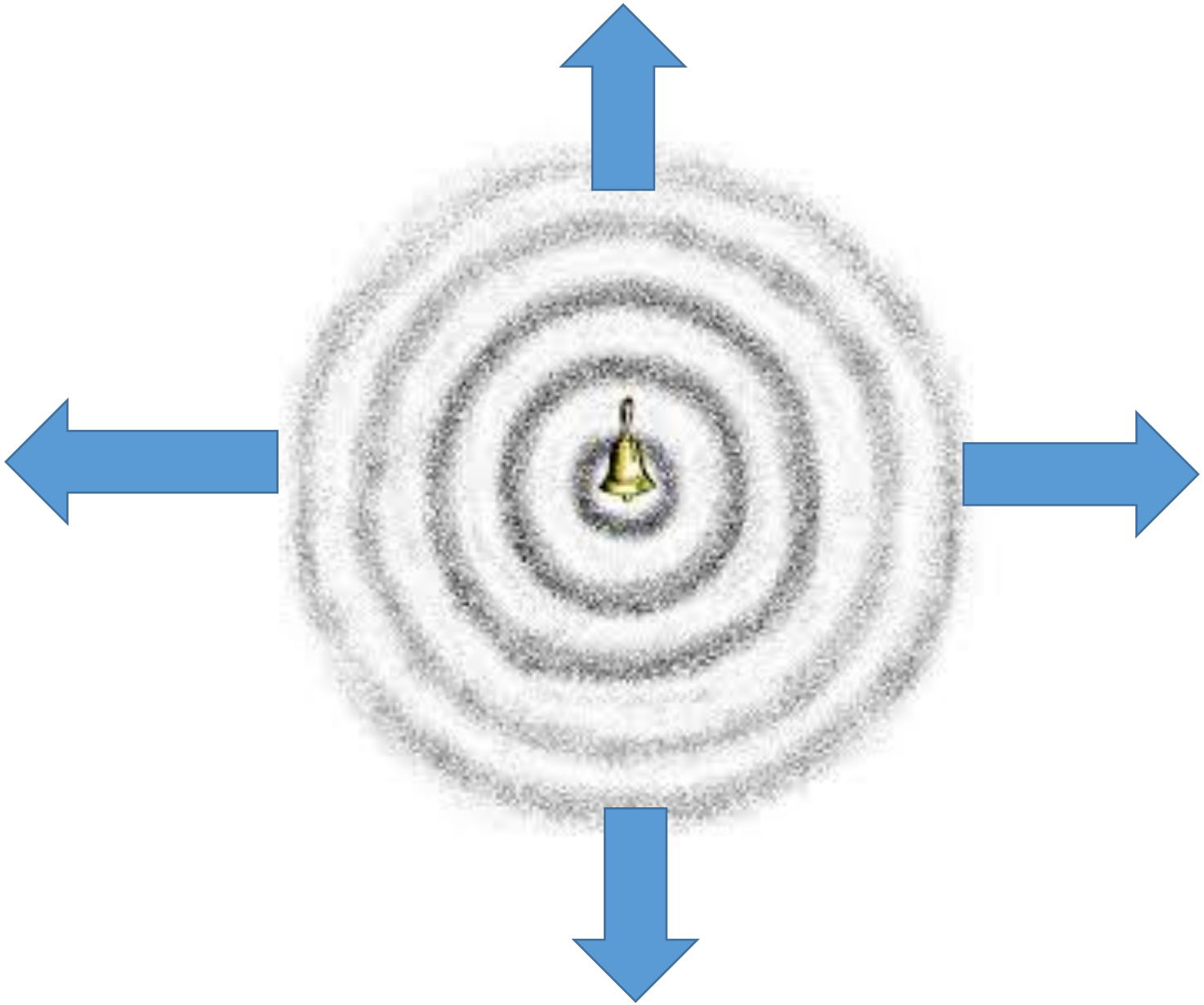
ZVOK



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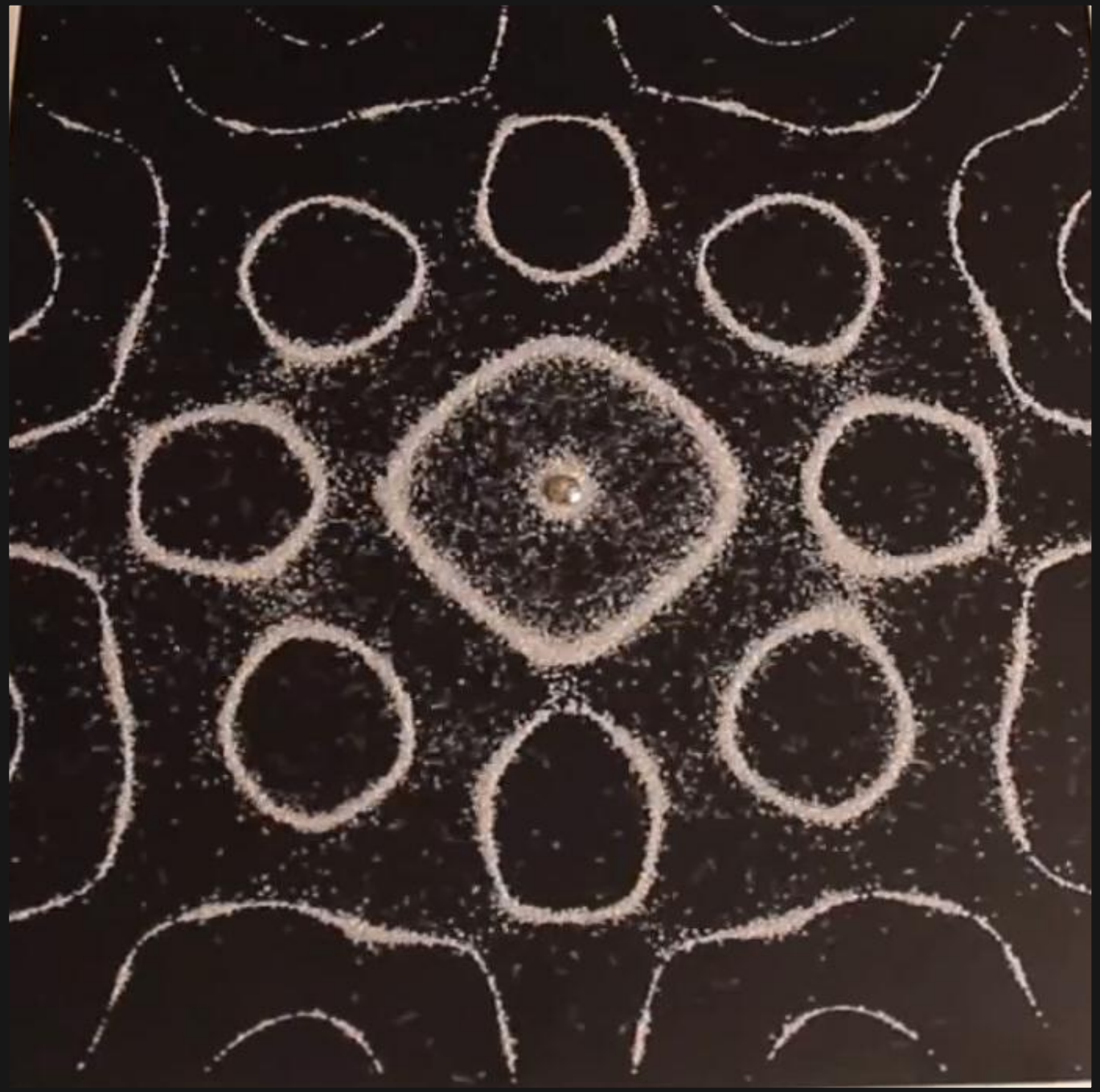

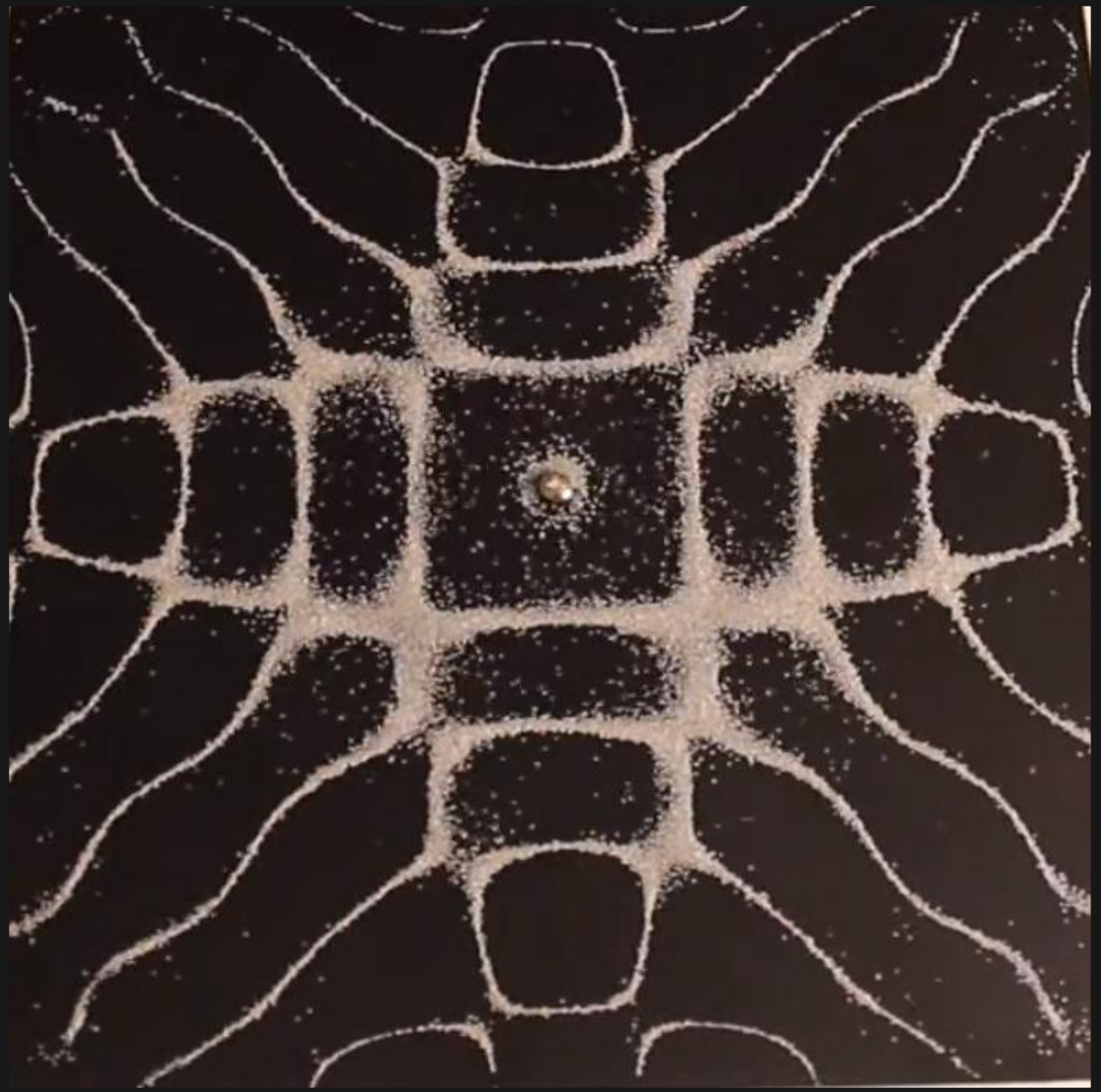

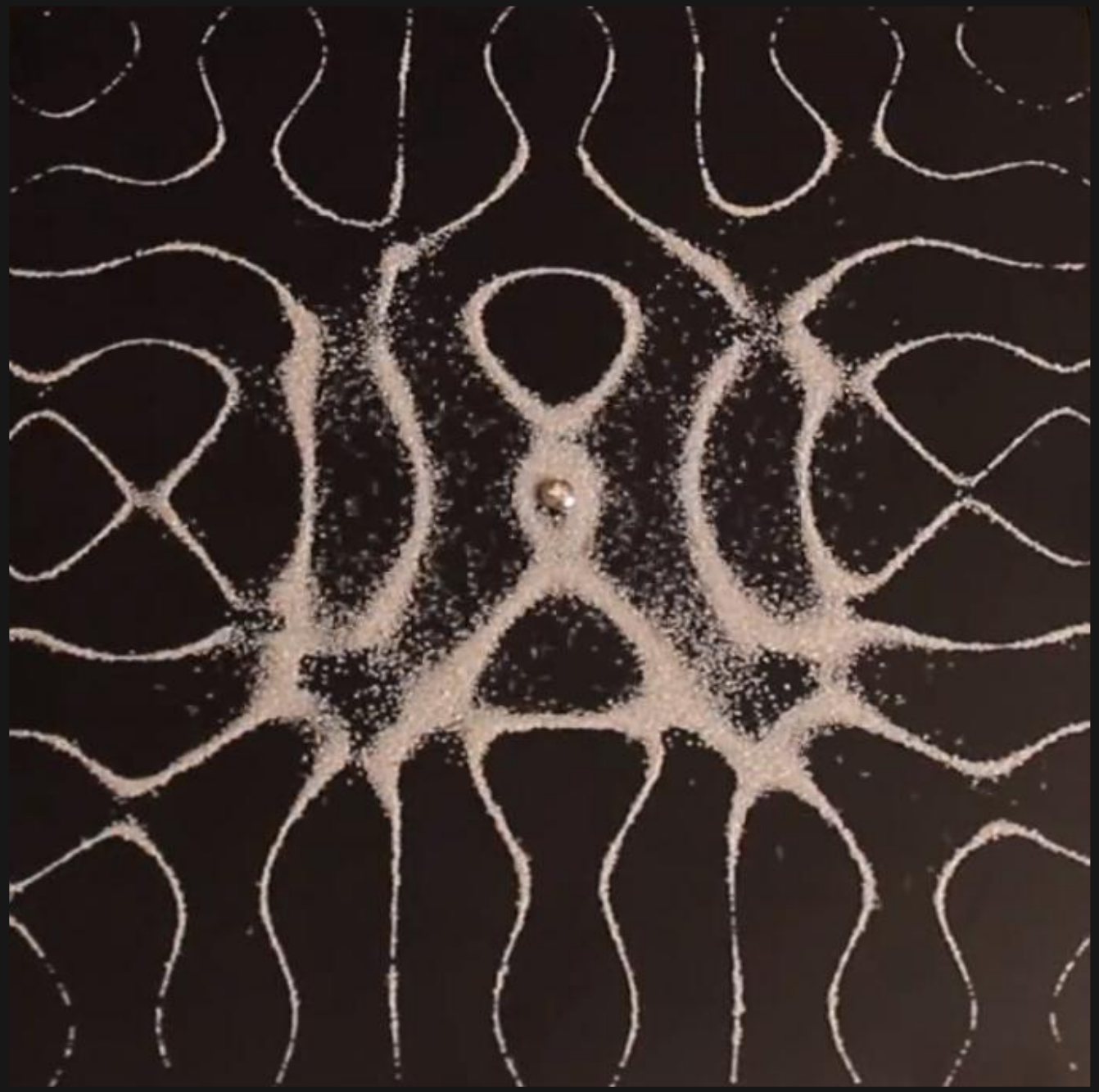

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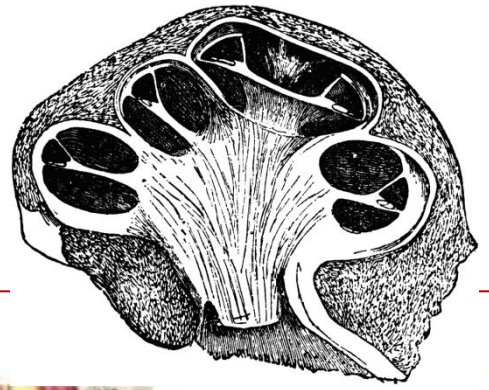
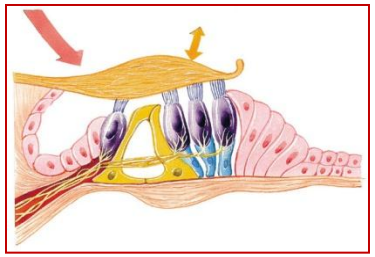
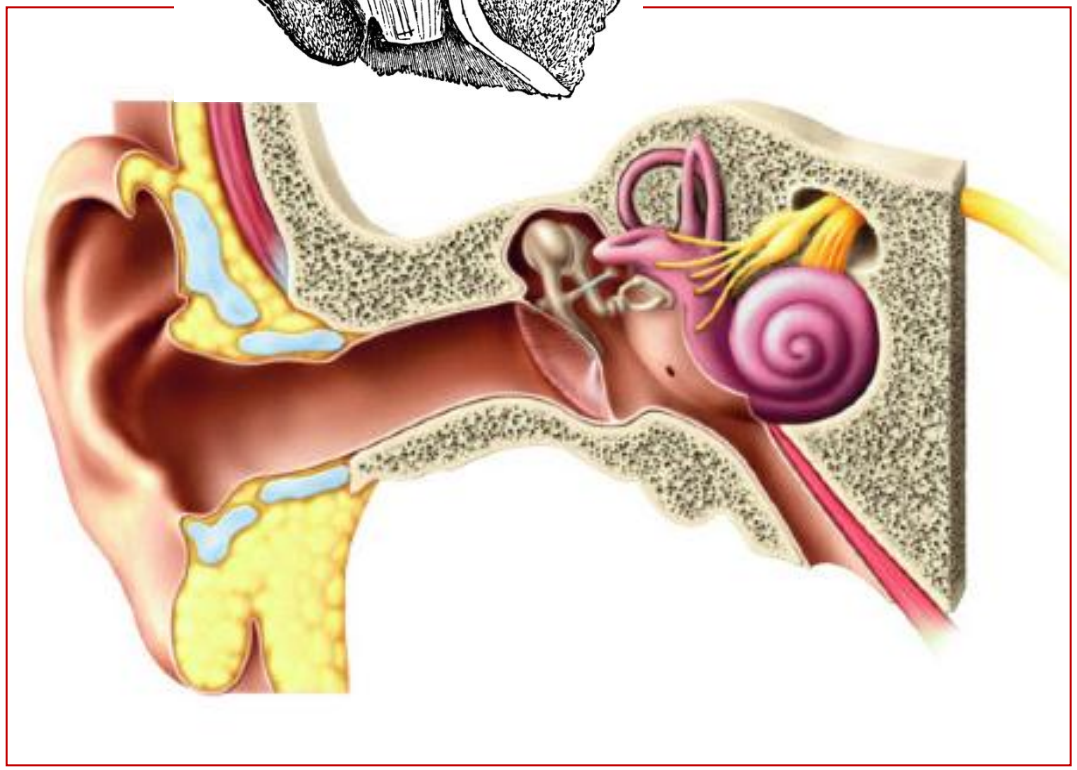
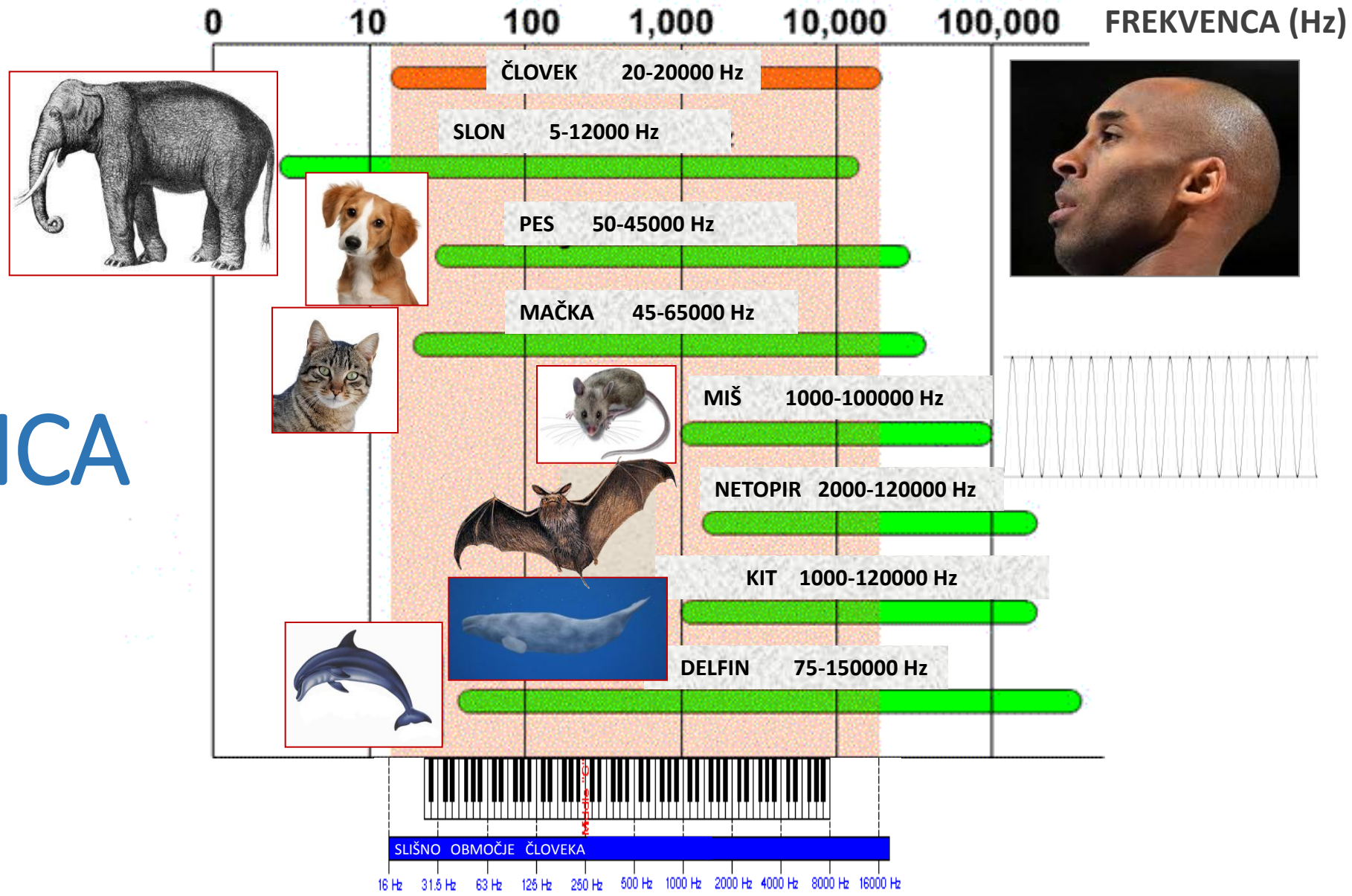






FREKVENCA ZVOKA



PSIHOAKUSTIKA

Kako zvok vpliva na nas?

Vpliv zvoka je odvisen od vrste zvoka, informacije, ki jo nosi s seboj, od ritma, okoliščin v katerih smo v trenutku poslušanja...

- **Alarm, pripravljenost**
- **Pomirjanje**
- **Pozornost, razmišljanje**
- **Vedenje, gibanje, ples**

ALARM, PRIPRAVLJENOST

ALARM, PRIPRAVLJENOST

POMIRJANJE

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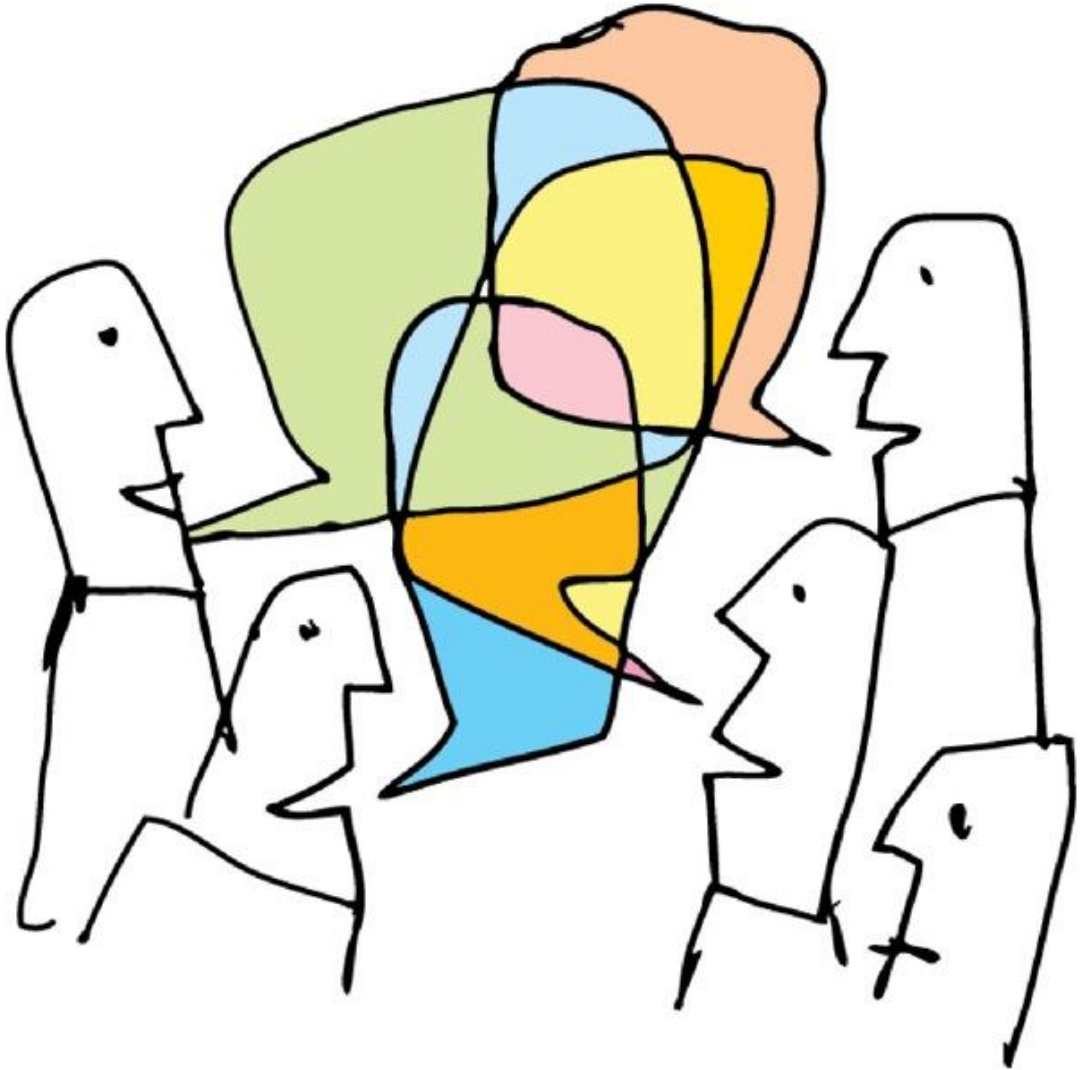
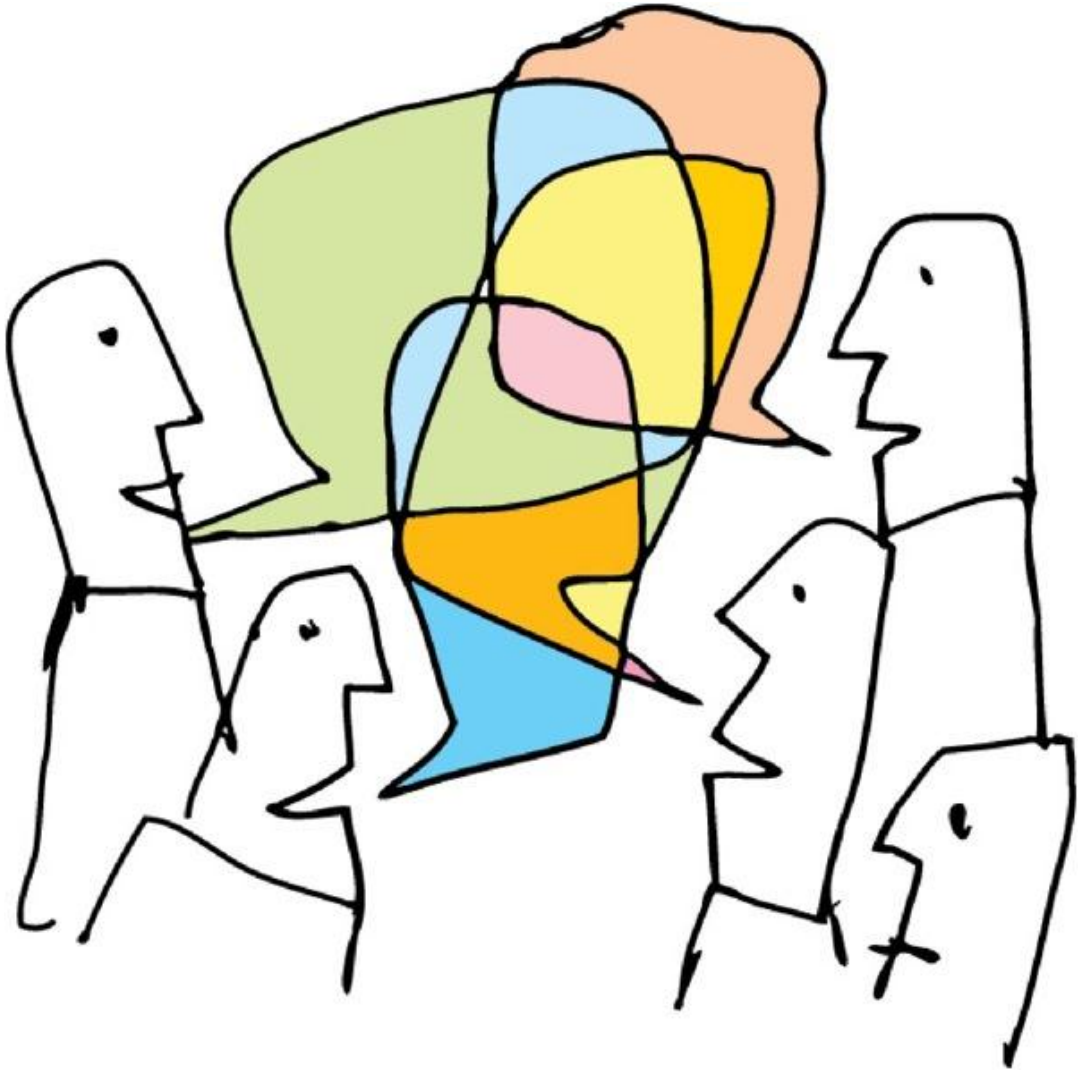
POZORNOST, RAZMIŠLJANJE



POZORNOST, RAZMIŠLJANJE



VEDENJE, GIBANJE, PLES

VEDENJE, GIBANJE, PLES

HRUP - NEZAŽELEN ZVOK

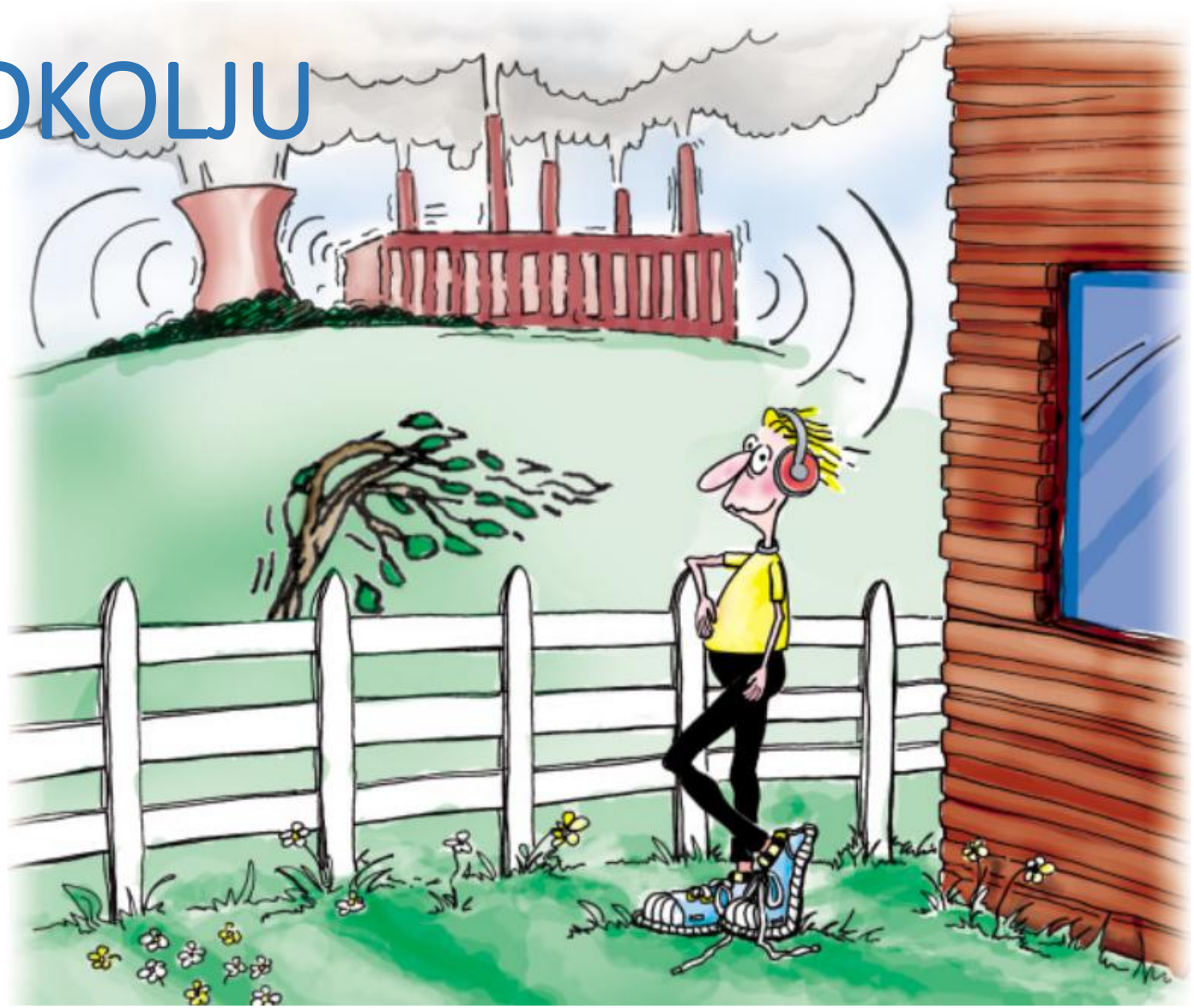
Viri hrupa

- Cestni promet
- Železniški promet
- Letalski promet
- Pomorski promet
- Industrija
- Gostinski lokali
- Javne prireditve
- Delovna orodja in naprave
- Zabava…

HRUP V OKOLJU

- **Bivalno okolje**
- **Šole**
- **Vrtci**
- **Igrišča**
- **Zdravilišča**
- **Zabavišča**
- **Parki**
- **Narava**

- **Delovno okolje**



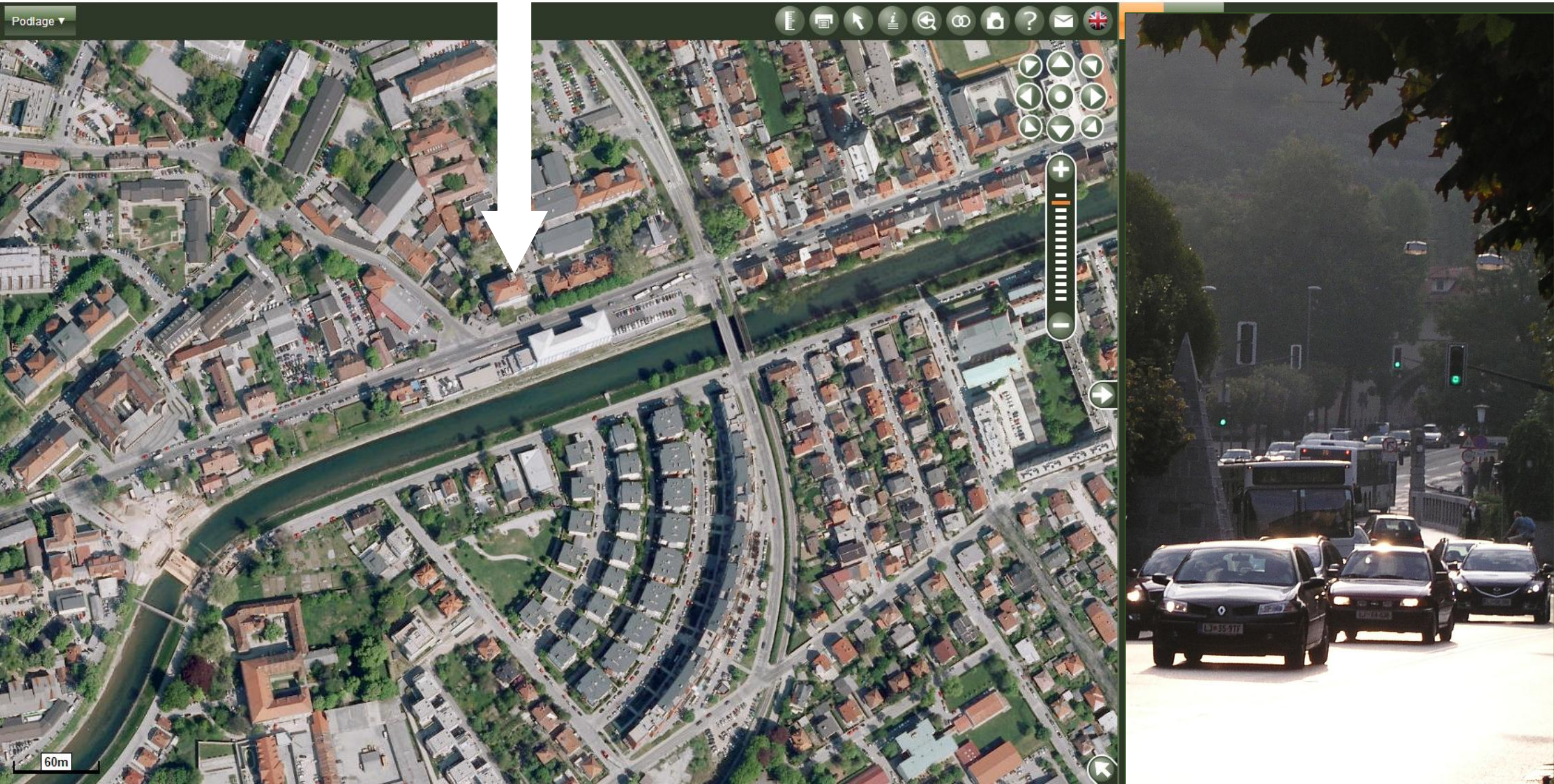
http://ims.si/documents/literatura/okoljski_hrup.pdf

AGENCIJA RS ZA OKOLJE

ATLAS OKOLJA

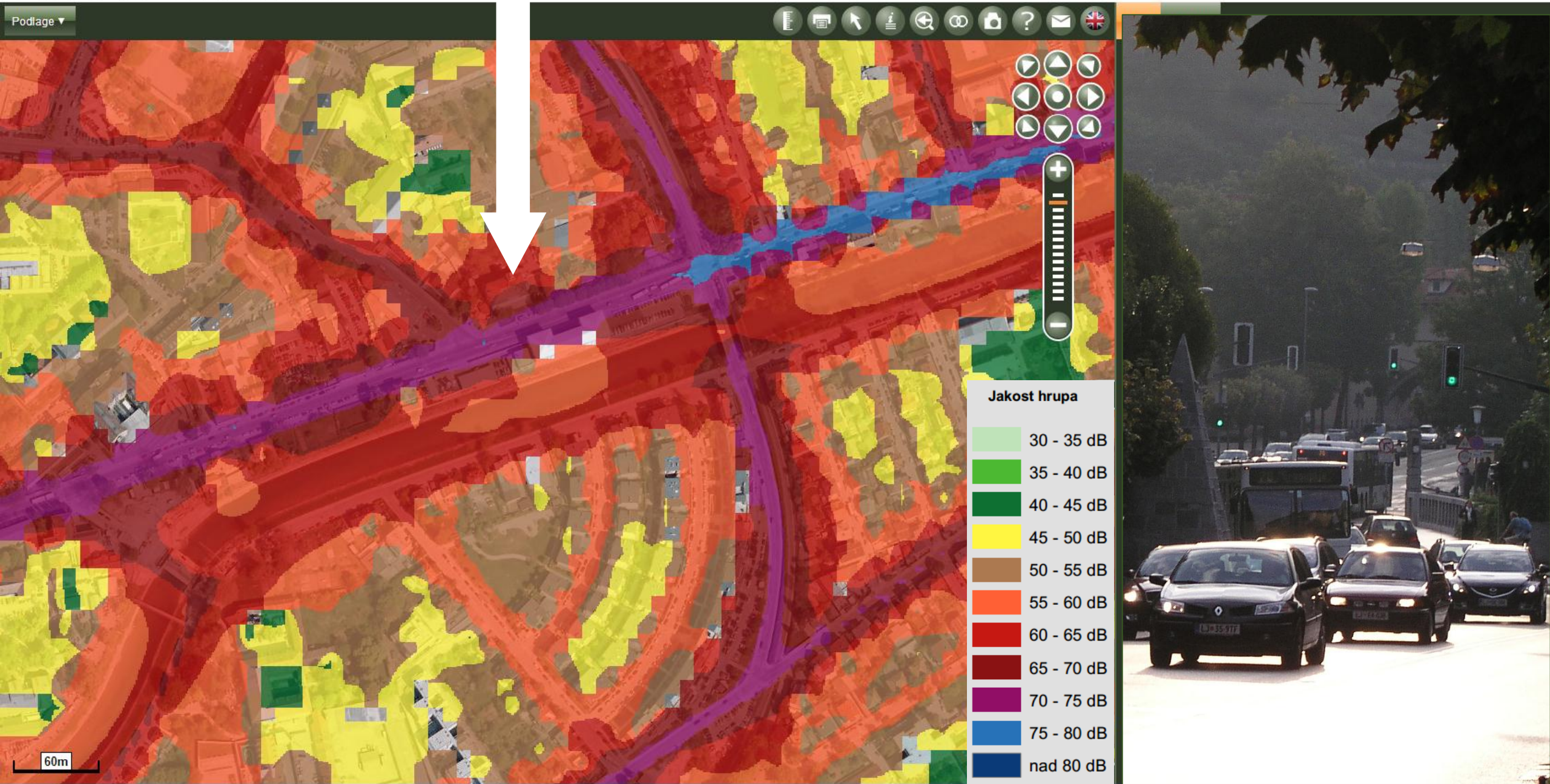
AGENCIJA RS ZA OKOLJE

ATLAS OKOLJA



AGENCIJA RS ZA OKOLJE

ATLAS OKOLJA



HRUP - NEZAŽELEN ZVOK

Zvočno okolje v predavalnici

- **Mejna vrednost ekvivalentne ravni hrupa L_{Aeq} = 35 dBA***
- **Mejna raven hrupa L_{AFmax} = 40 dBA***

- **Hrup na zunanji fasadi stavbe ob Zaloški ulici je L_{dvn} = 70 – 75 dBA**

Za primerno zvočno okolje v predavalnici je potrebna izolacija pred hrupom 40 dBA.

**Pravilnik o zaščiti pred hrupom v stavbah, Uradni list RS št. 10/2012 (Tehnična smernica)*

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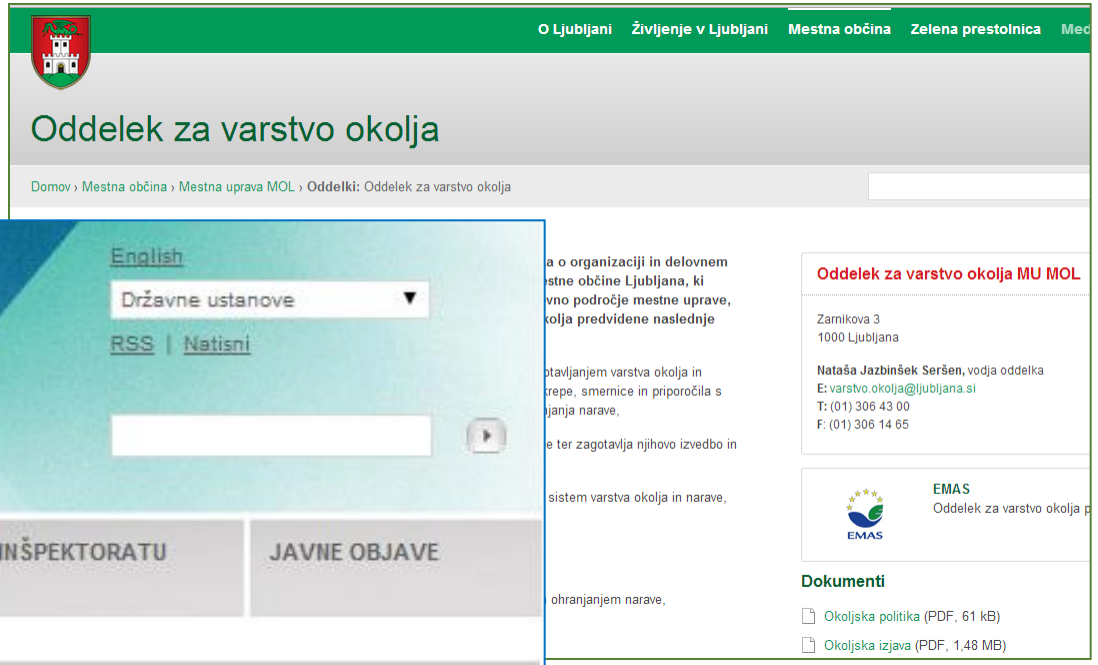
PRITOŽBE OBČANOV

Inšpektorat RS za kmetijstvo in okolje



HRUP - NEZAŽELEN ZVOK

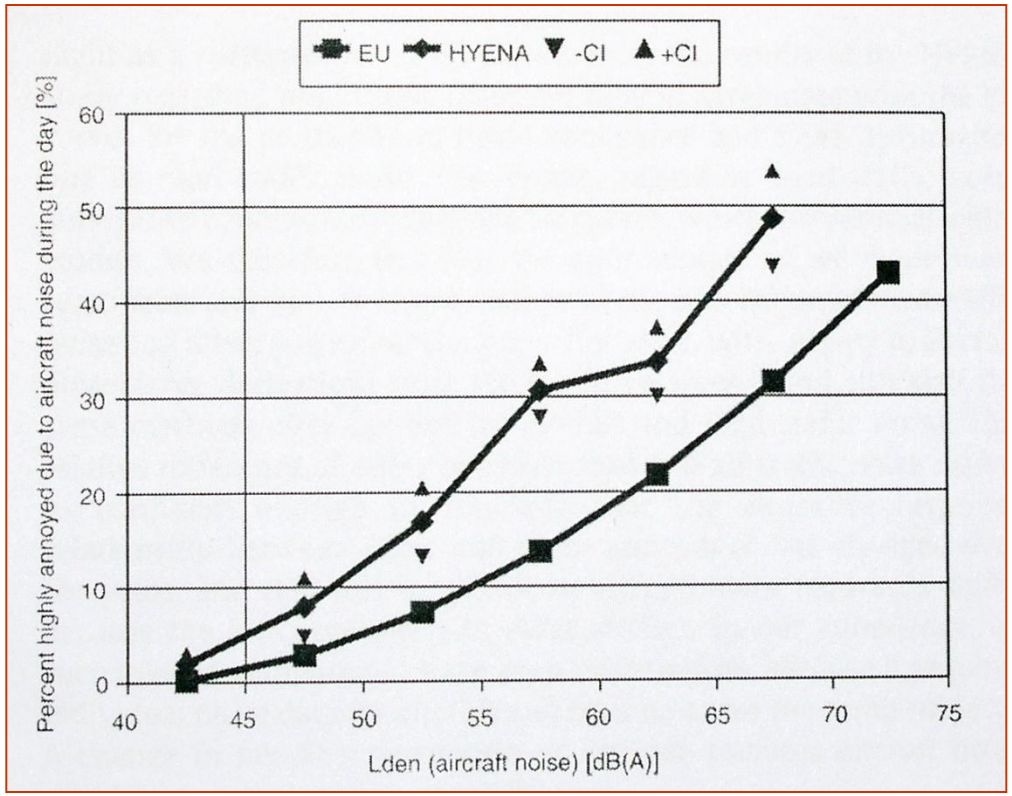
Vpliv na zdravje in počutje ljudi

- **Vznemirjenost**
- **Motnje spanja**
- **Motnje pri koncentraciji, delu, učenju…**
- **Povišan krvni tlak**
- **Bolezni srca in ožilja**

- **Poškodbe sluha**

VZNEMIRJENOST

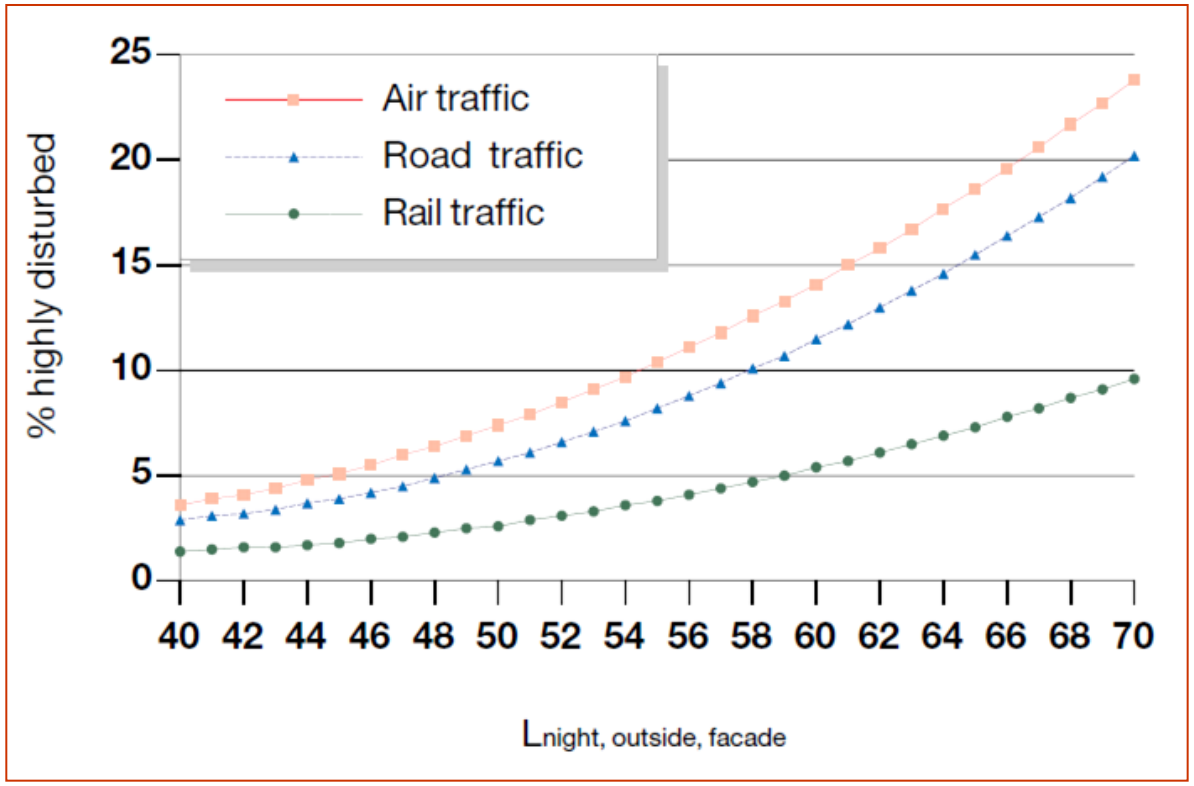
Odstotek vznemirjenih prebivalcev zaradi hrupa letal podnevi



EU krivulja iz leta 2002
HYENA študija iz leta 2009

MOTNJE SPANJA

Motnje spanja v nočnem času zaradi okoljskega hrupa (zračni, cestni in železniški promet)
(Position paper on dose-effect relationship for night time noise. EC, 2004)



MOTNJE KONCENTRACIJE

Učinek izpostavljenosti hrupu letal v šolah in sposobnost razumevanja branega teksta
The RANCH Project (Clark s sod., American Journal of Epidemiology, 163(1), 2005.

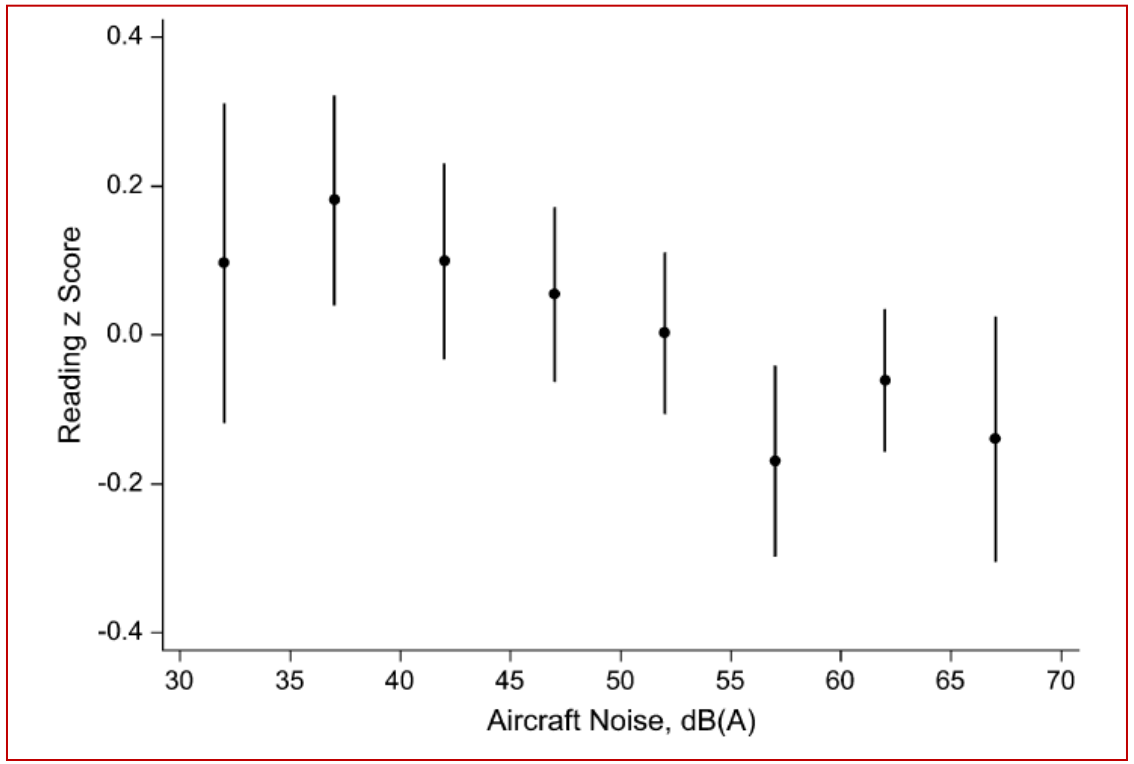


FIGURE 1. Adjusted mean reading *z* scores and 95% confidence intervals for 5-dB(A) bands of aircraft noise at school (adjusted for age, gender, and country), the RANCH project, 2001–2003. dB(A), a measure of sound level in decibels A-weighted to approximate the typical sensitivity of the human ear.

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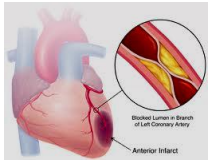
BOLEZNI SRCA IN OŽILJA



Odvisnost povečanega tveganja za pojav srčnega infarkta od izpostavljenosti hrupu cestnega prometa (Babisch, 2008)

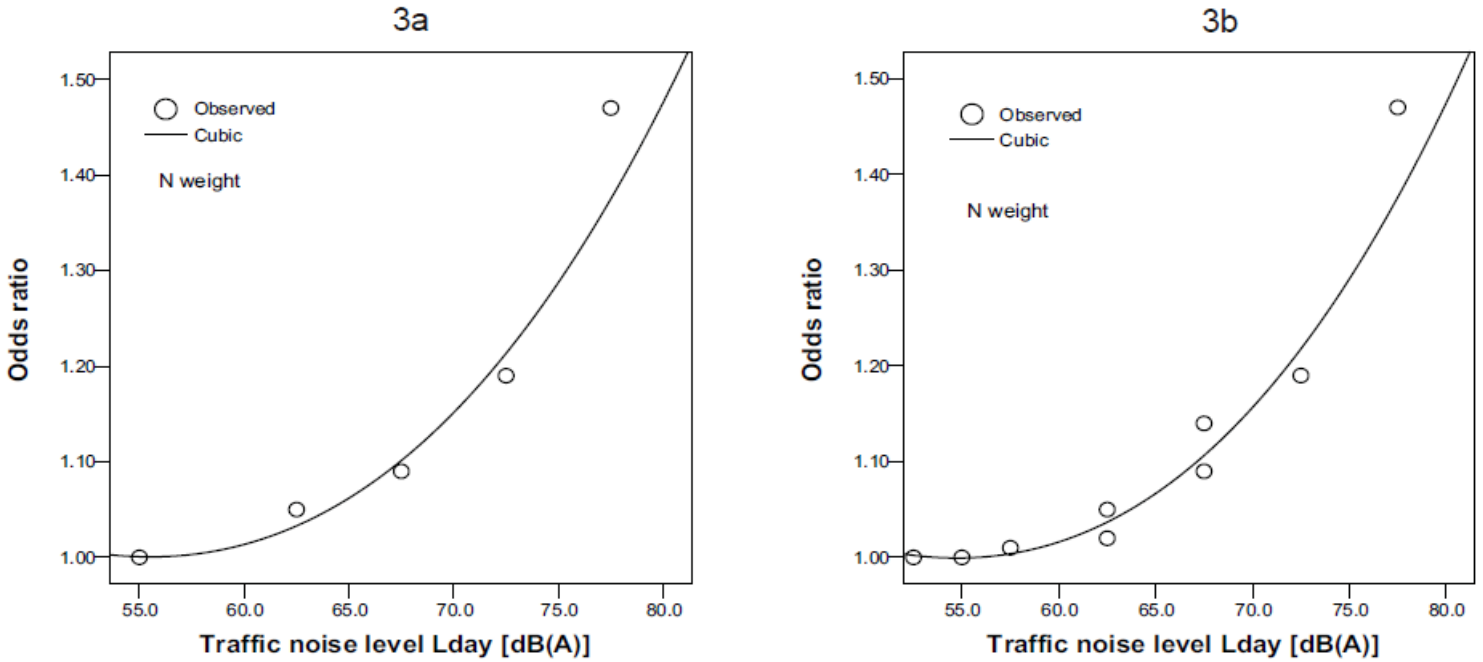
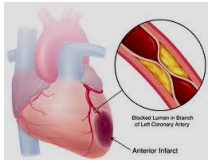
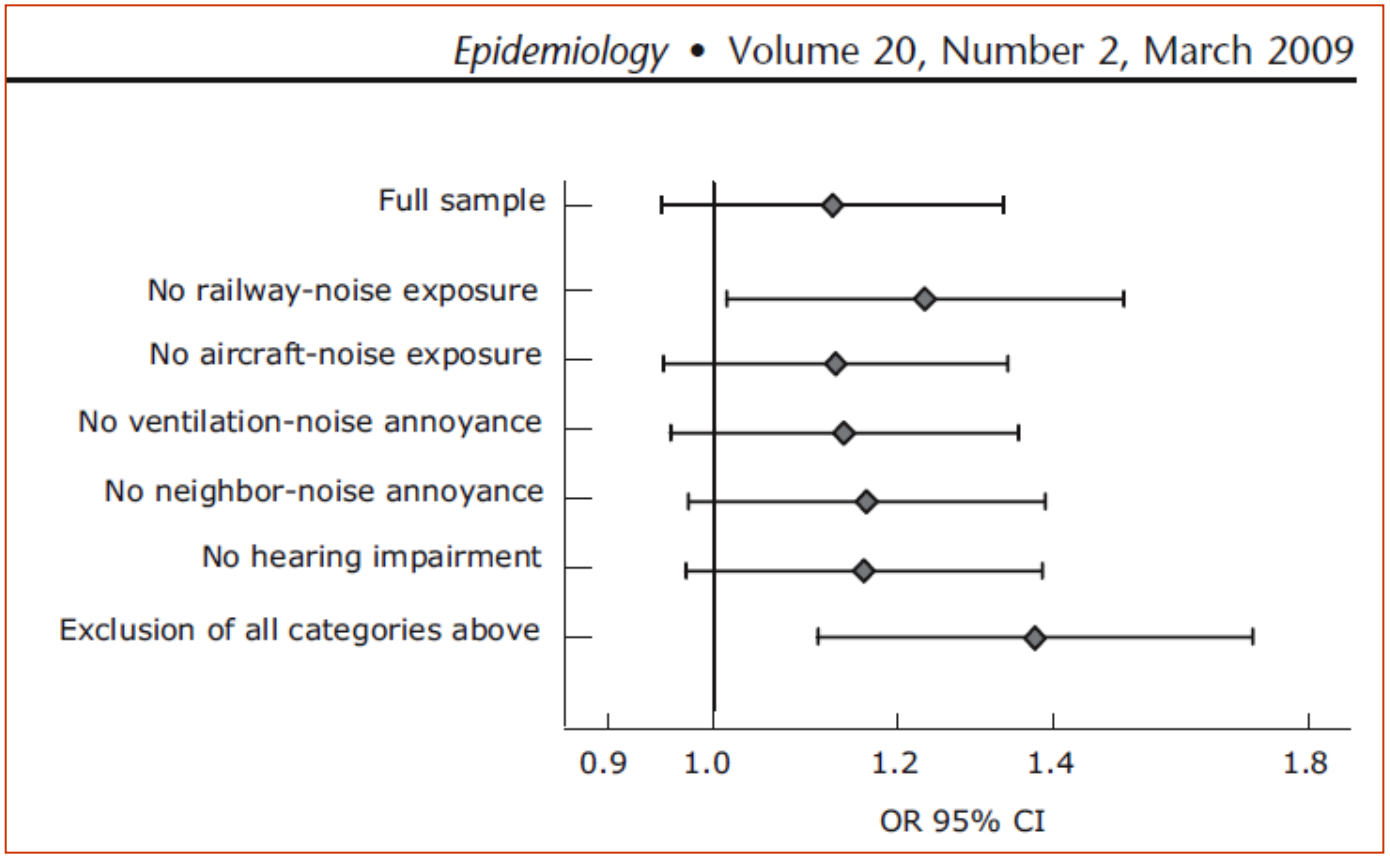


Figure 3 (a-b): Polynomial fits of the exposure-response relationship between road traffic noise and myocardial infarction. The left graph (3a) refers case-control or cohort studies (analytic studies), the right graph (3b) to cross-sectional, case-control or cohort studies (descriptive and analytic studies)

BOLEZNI SRCA IN OŽILJA



Odvisnost verjetnosti pojava srčnega infarkta od izpostavljenosti hrupu cestnega prometa.
Študija opravljena v Stockholmu. (Selander s sod., Epidemiology, 20:2, 2009)



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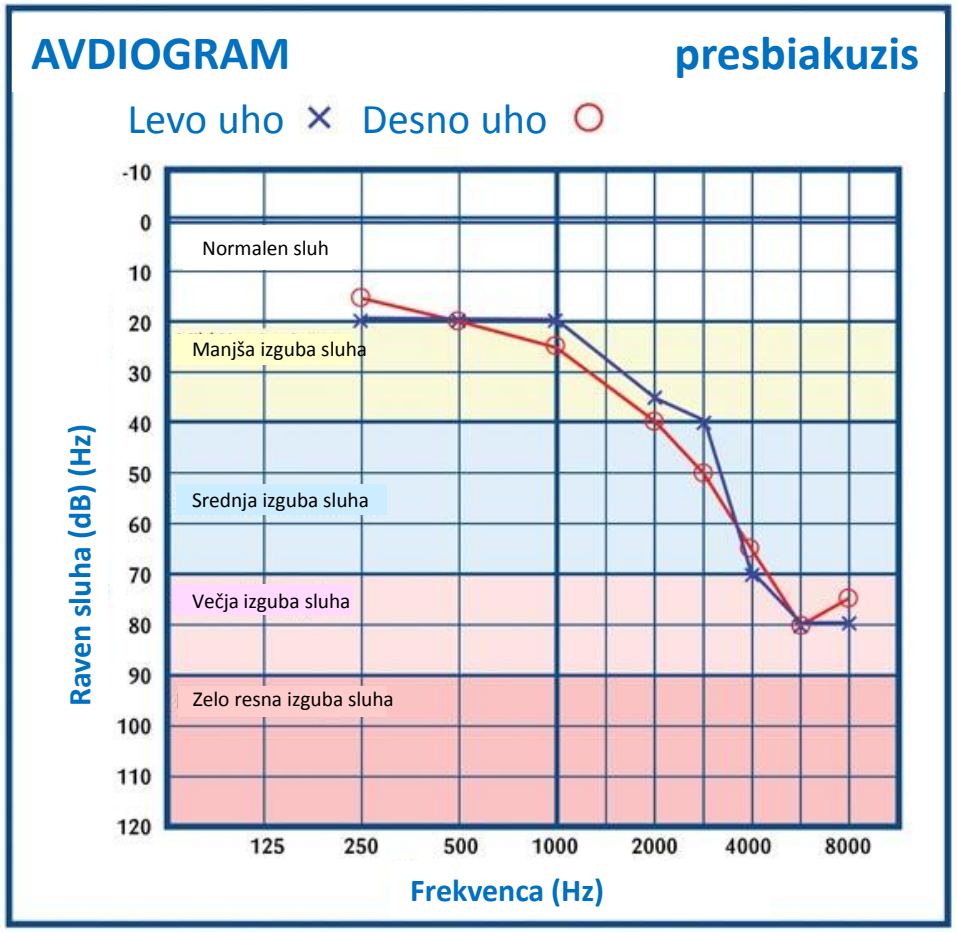
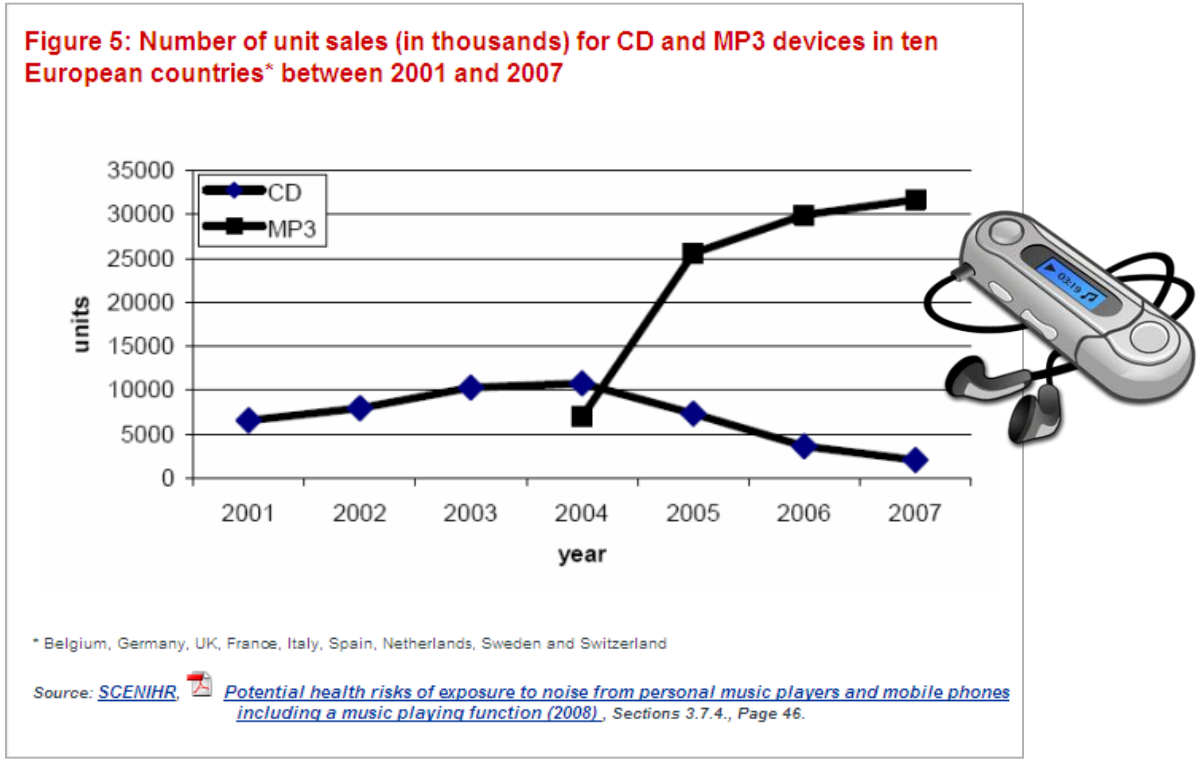
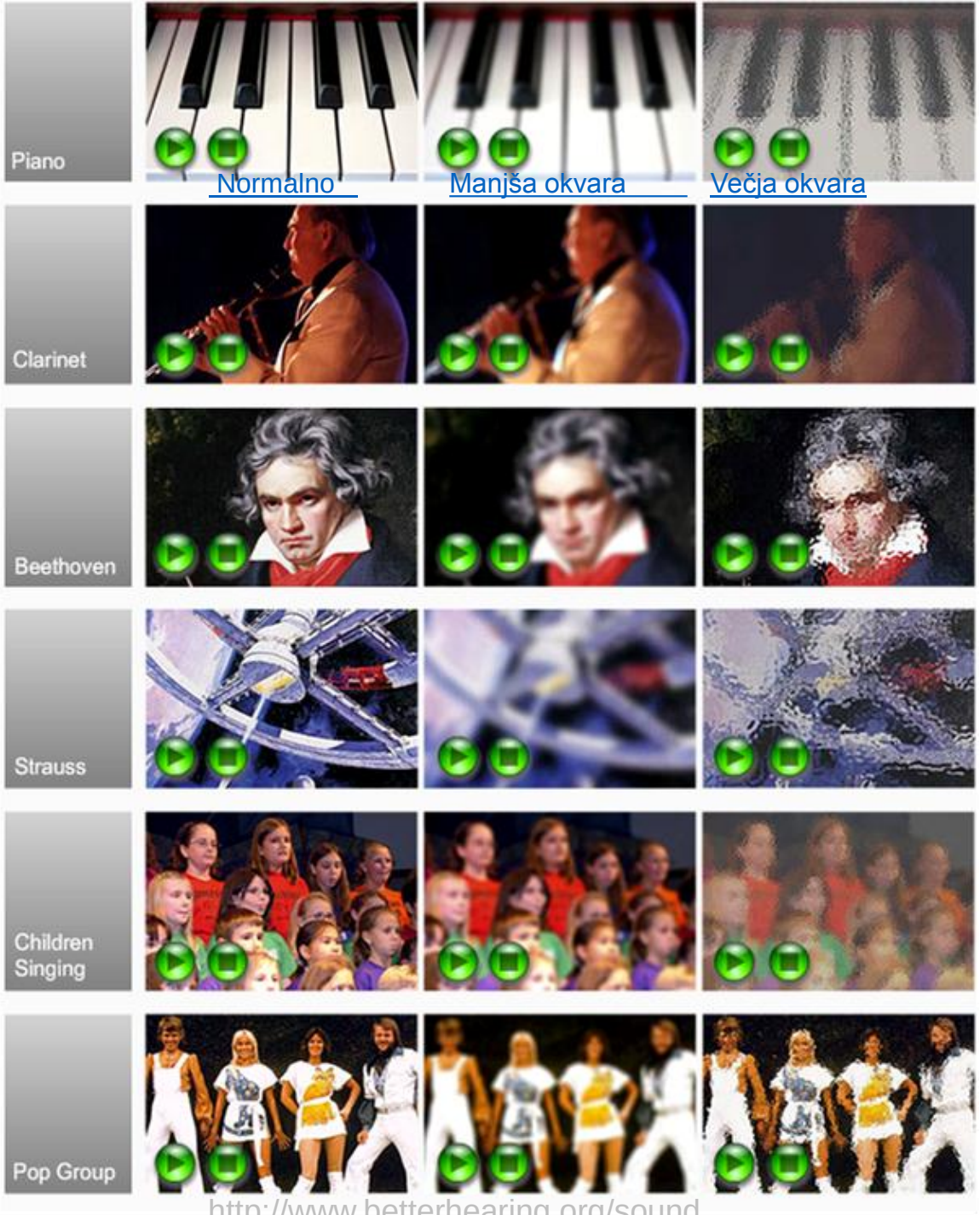
POŠKODBE SLUHA

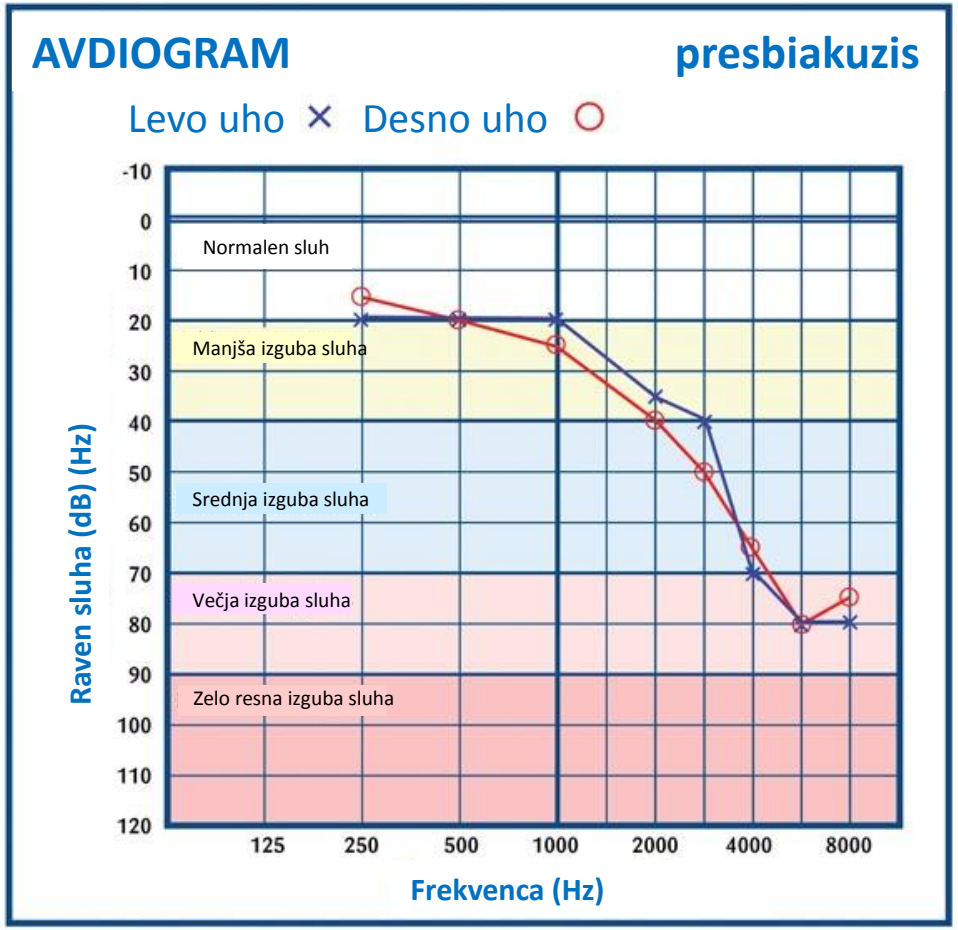




GLASBA



http://www.betterhearing.org/sound



http://auditoryneuroscience.com/acoustics/clinical_audiograms

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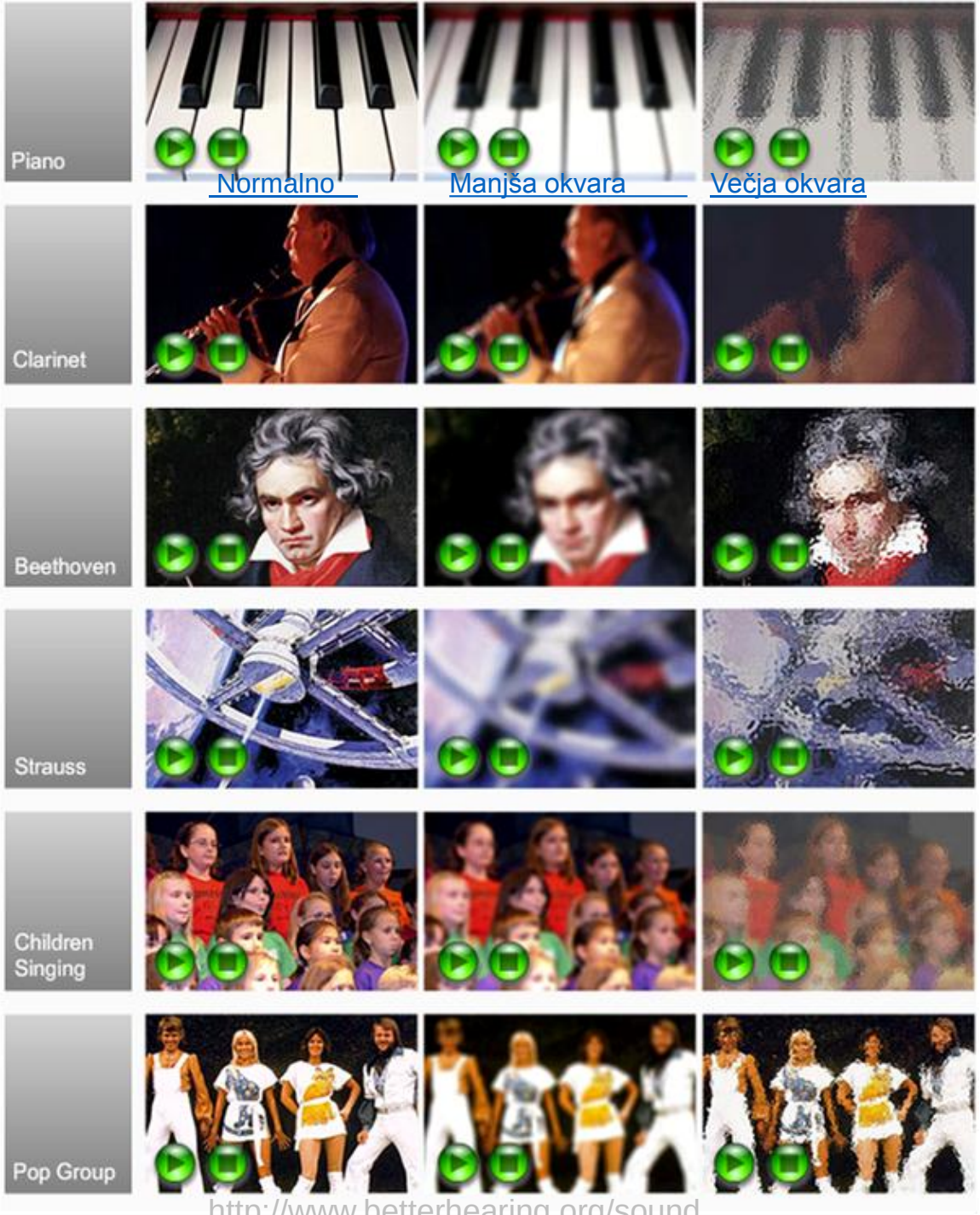

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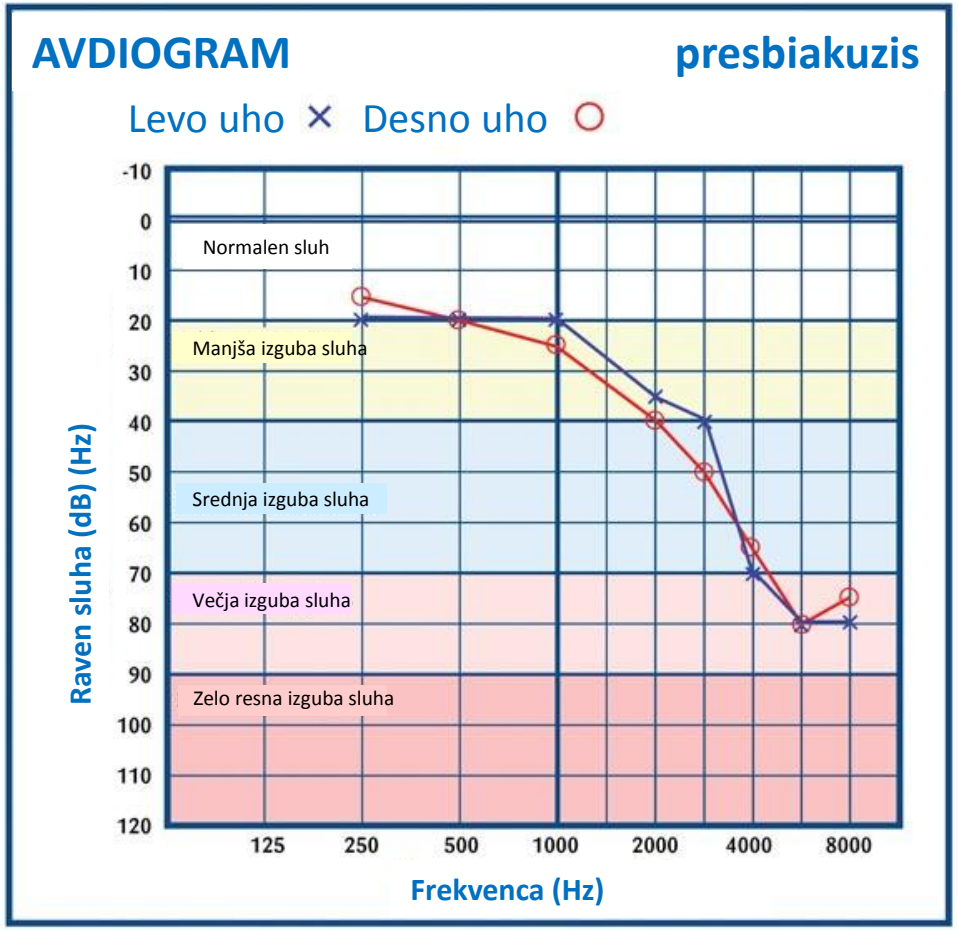

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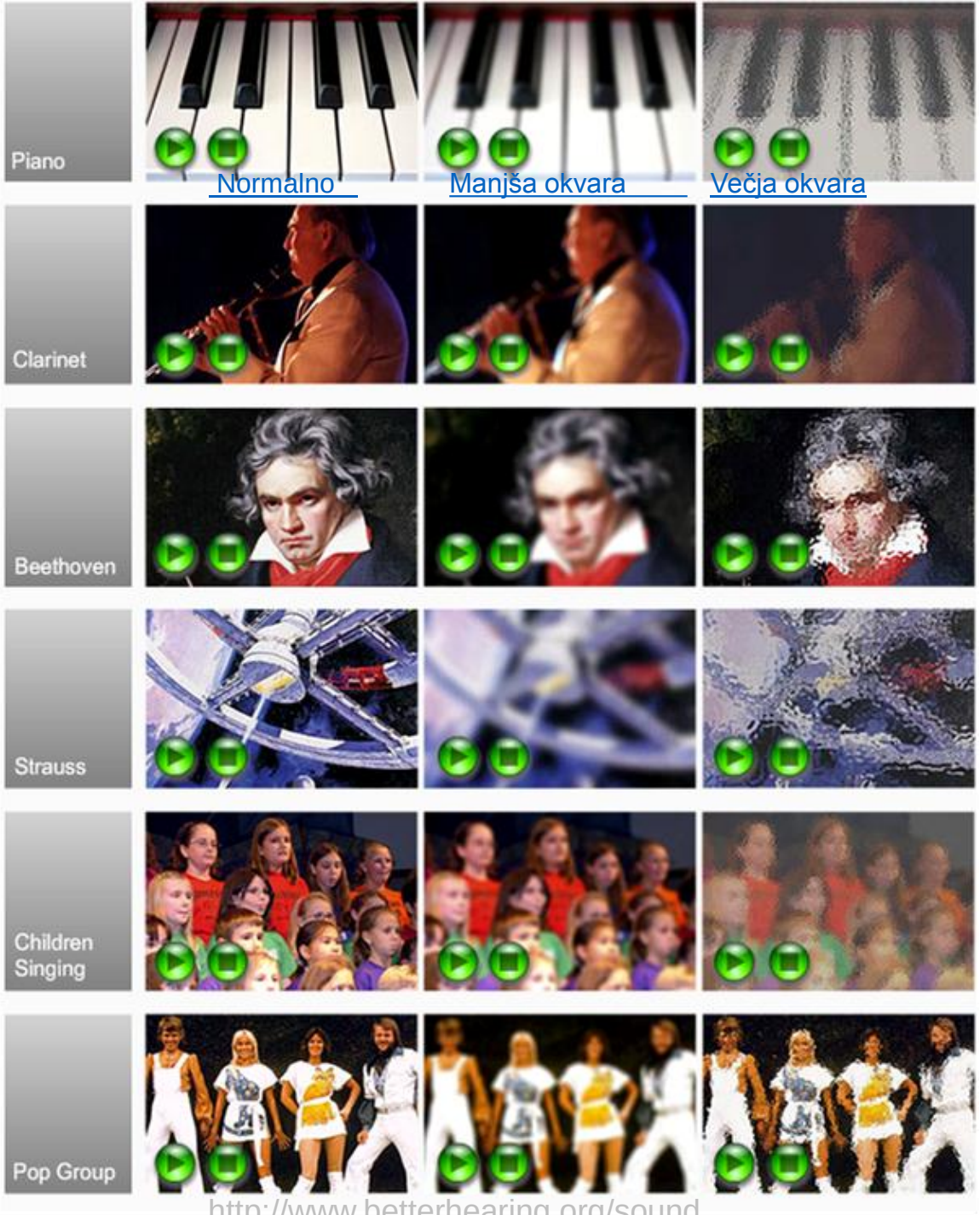

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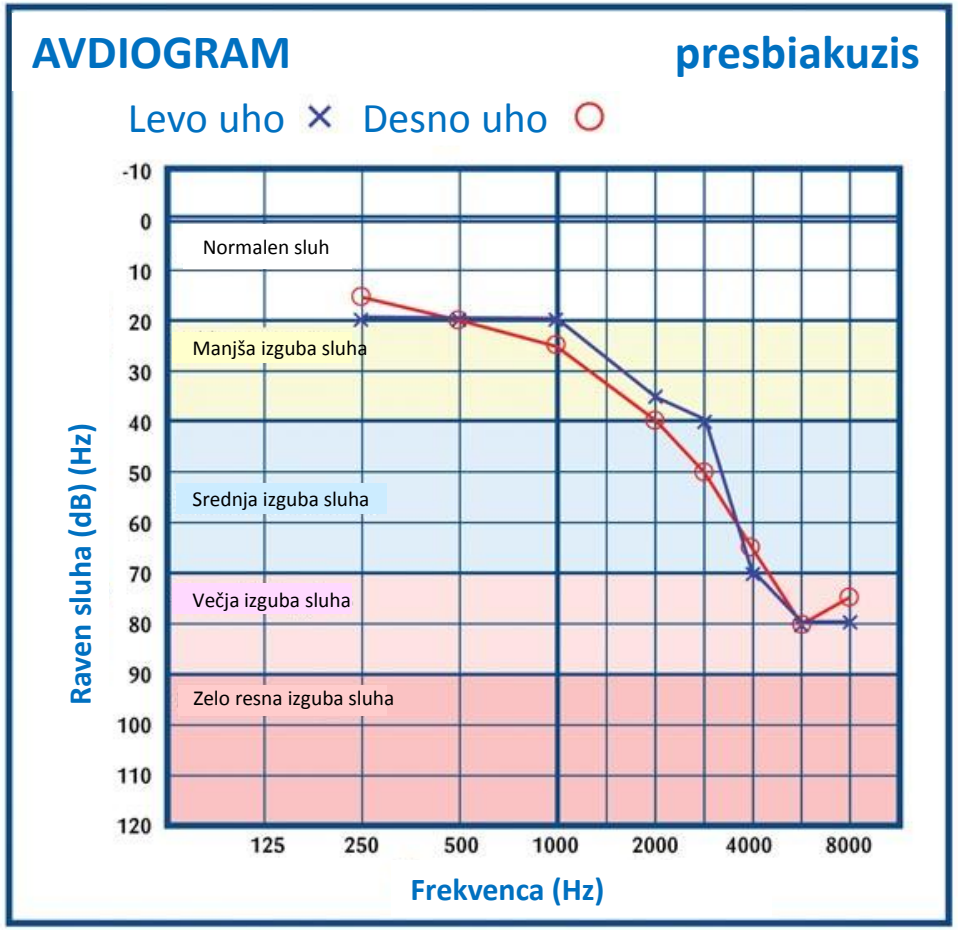

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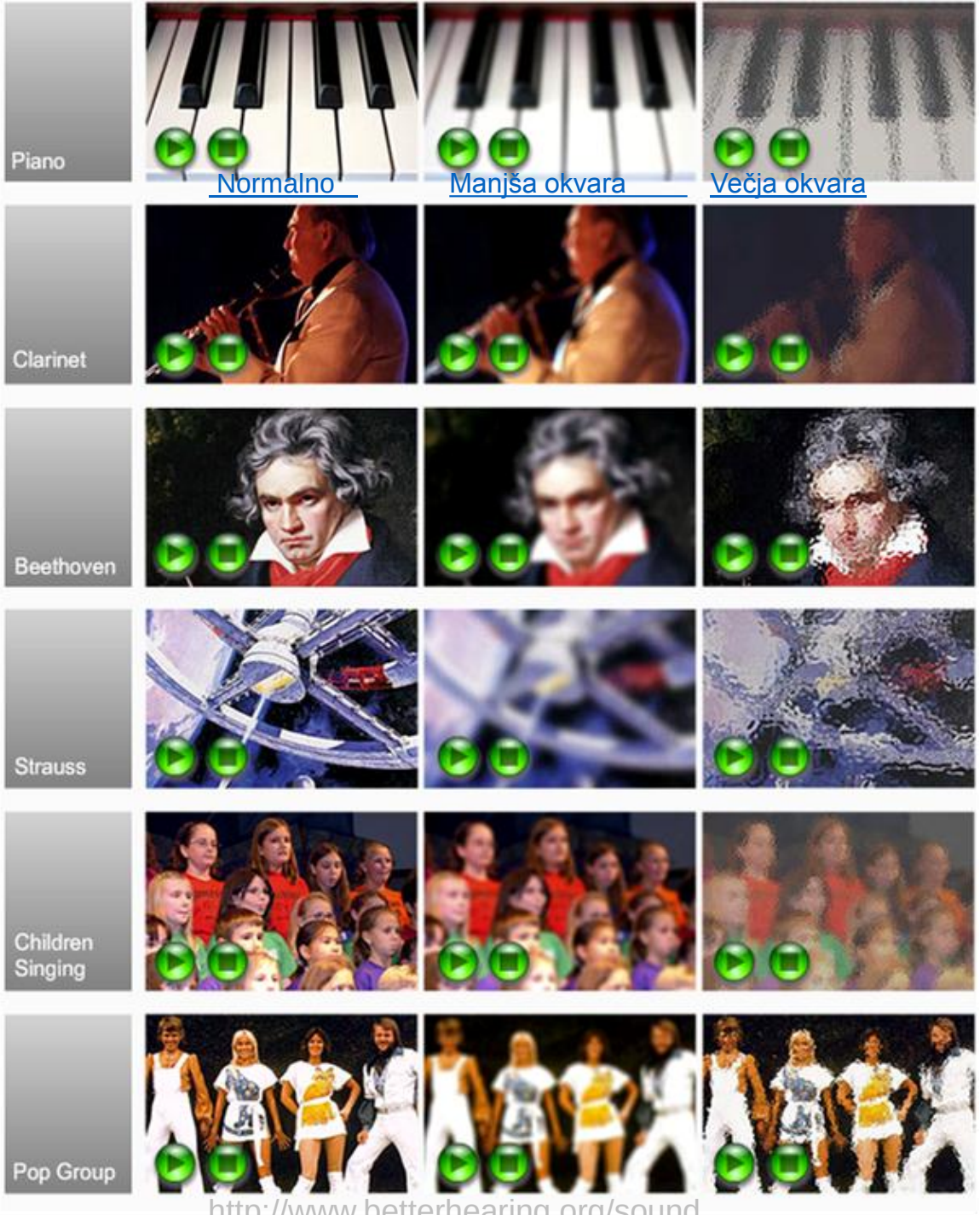

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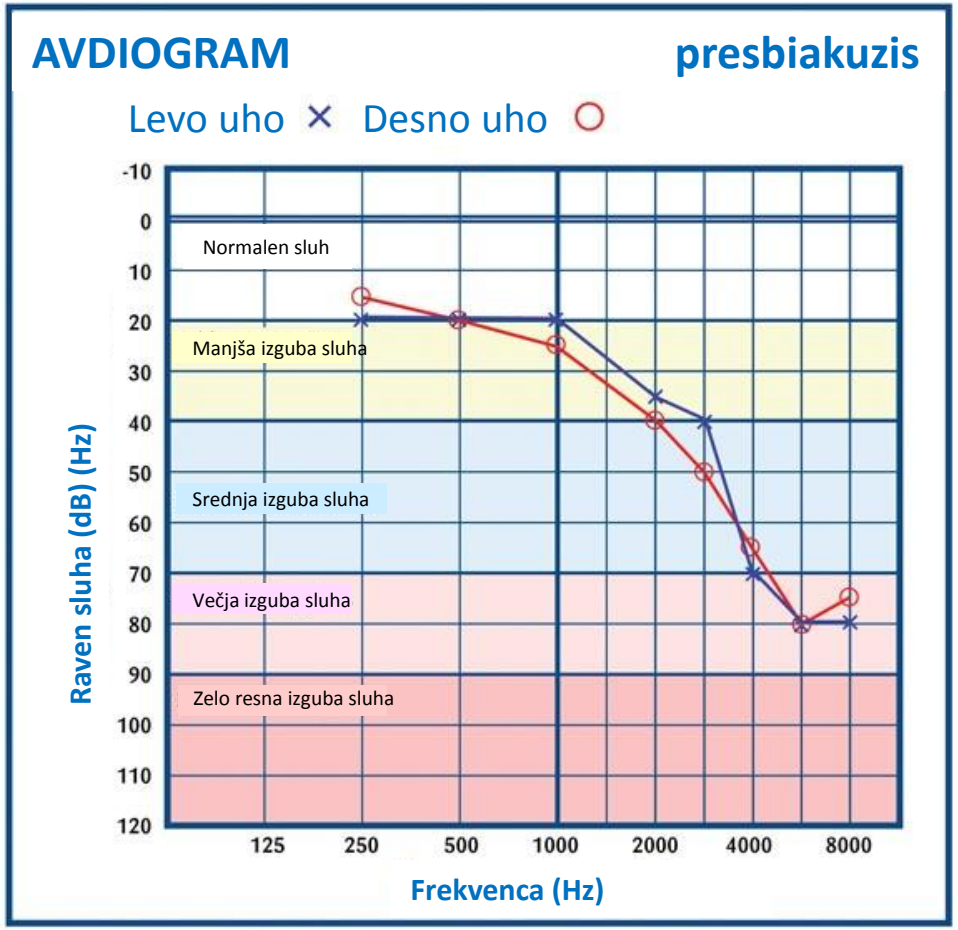

http://auditoryneuroscience.com/acoustics/clinical_audiograms

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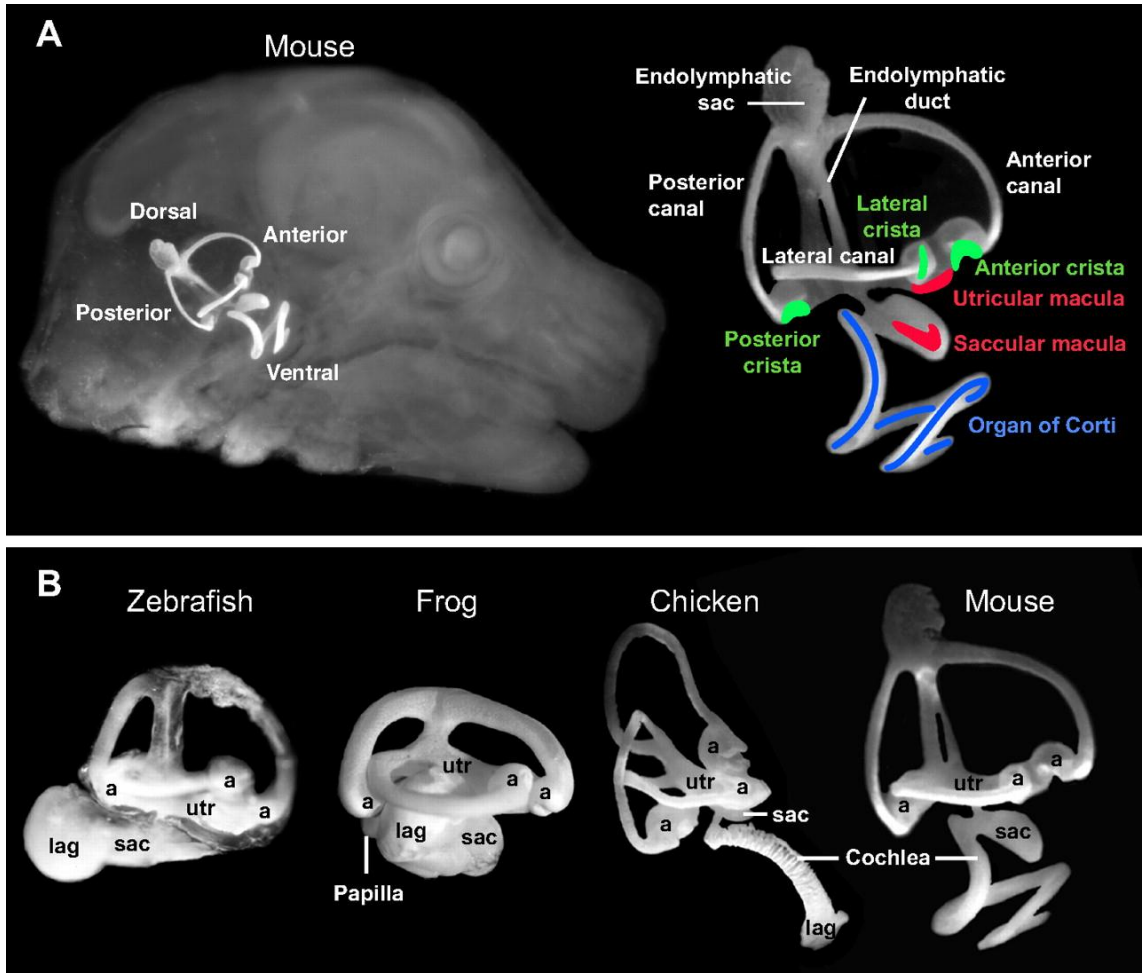
ANATOMIJA UŠESA

ribe, dvoživke, ptice, sesalci



Groves A K , and Fekete D M Development 2012;139:245-257

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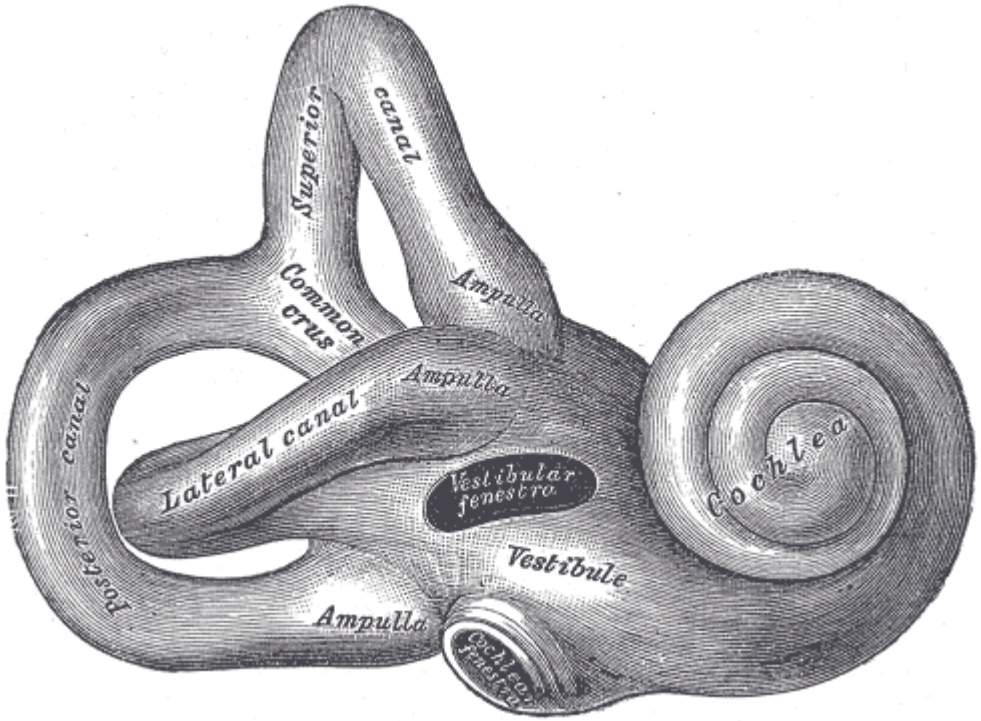

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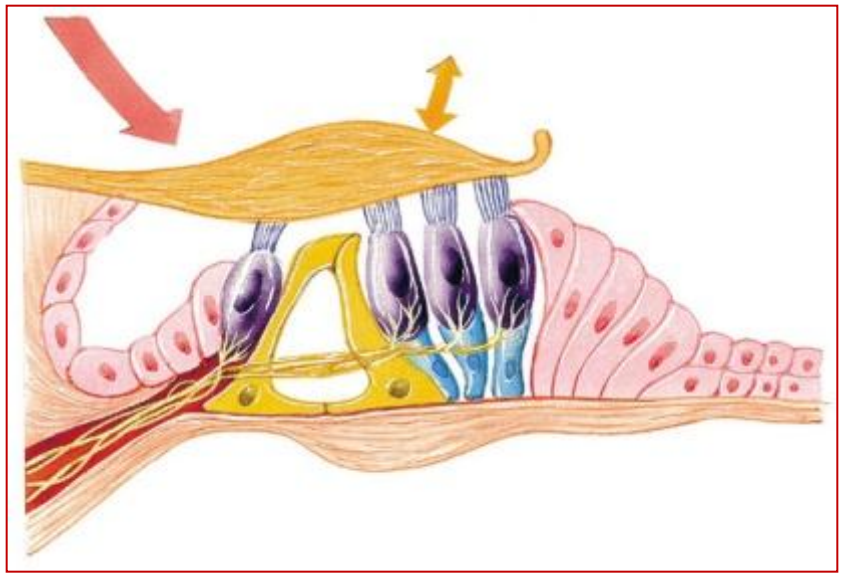
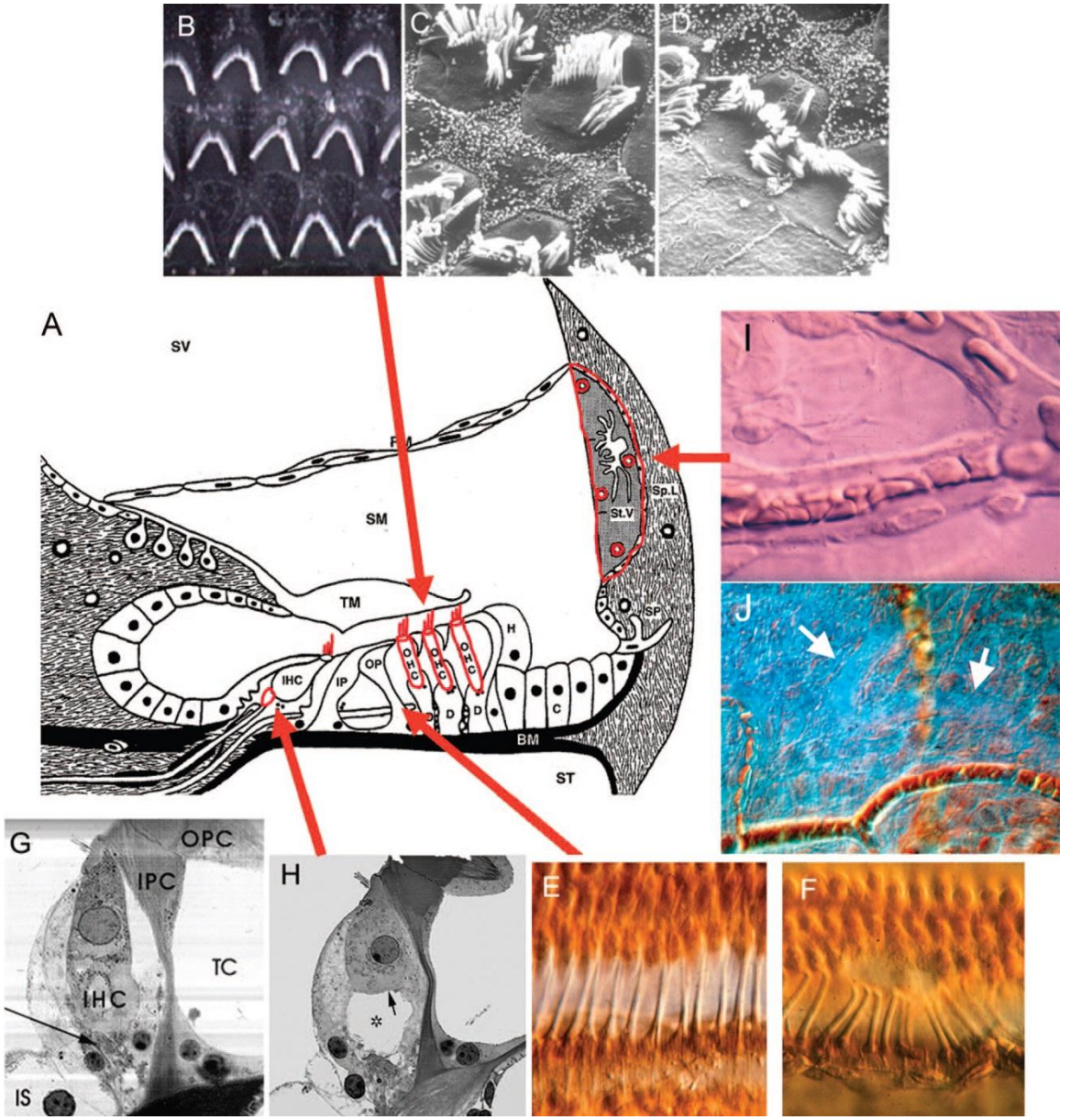
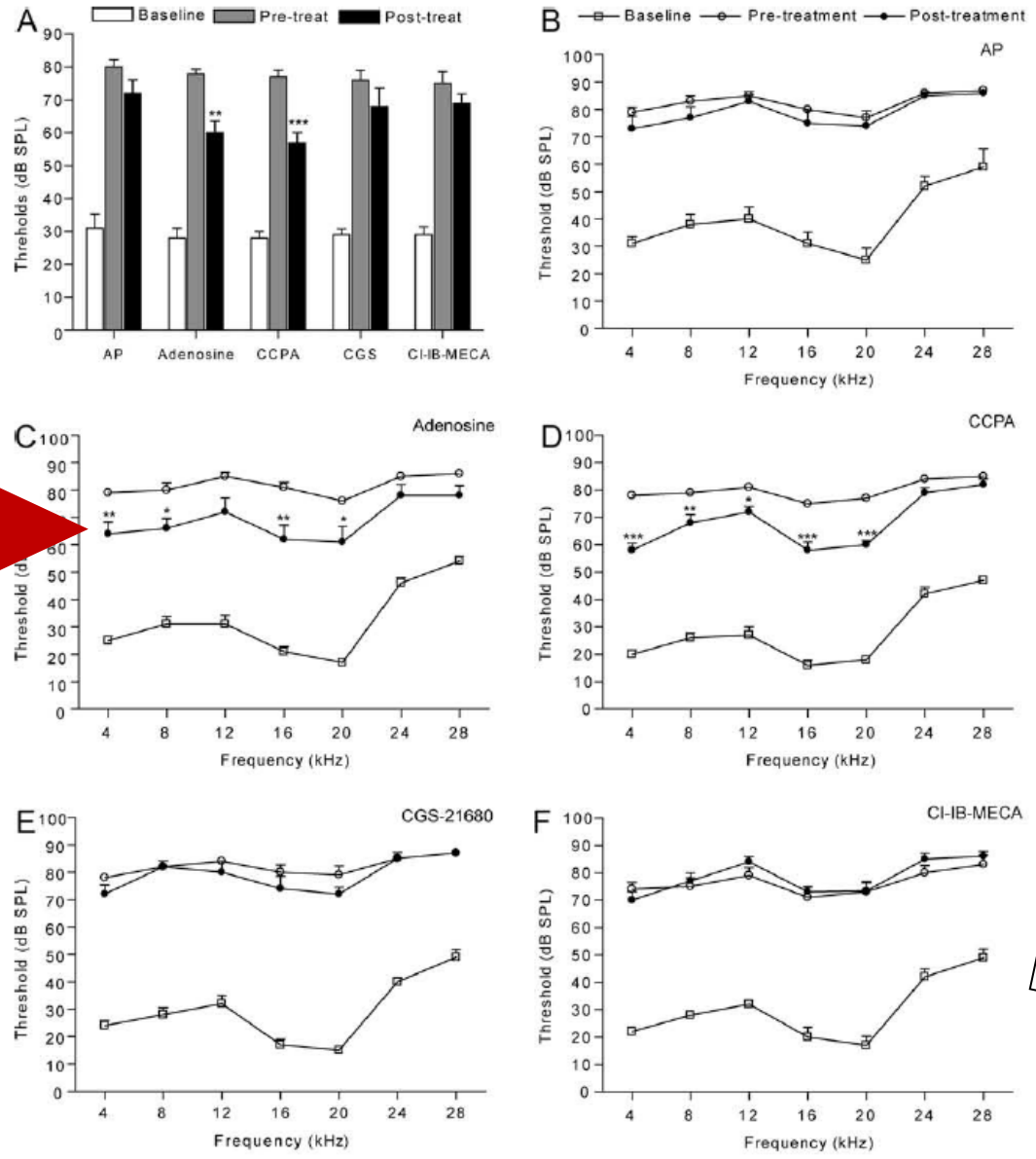




Činčila

Možne okvare sluha zaradi previsoke jakosti zvoka

D. Henderson, E. C. Bielefeld, K. Carney Harris, B. Hua Hu. The Role of Oxidative Stress in Noise-Induced Hearing Loss. EAR & HEARING, VOL. 27 NO. 1



Hearing Research 260 (2010) 81–88
Contents lists available at ScienceDirect
Hearing Research
journal homepage: www.elsevier.com/locate/heares

Post exposure administration of A_1 adenosine receptor agonists attenuates noise-induced hearing loss

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[a] Department of Physiology, Faculty of Medical and Health Sciences, The University of Auckland, New Zealand
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ABSTRACT

Adenosine is a constitutive cell metabolite with a putative role in protection and regeneration in many tissues. This study was undertaken to determine if adenosine signalling pathways are involved in protection against noise injury. A_1 adenosine receptor expression levels were altered in the cochlea exposed to loud sound, suggesting their involvement in the development of noise injury. Adenosine and selective adenosine receptor agonists (CCPA, CGS-21680 and Cl-IB-MECA) were applied to the round window membrane of the cochlea 6 h after noise exposure. Auditory brainstem responses measured 48 h after drug administration demonstrated partial recovery of hearing thresholds (up to 20 dB) in the cochleae treated with adenosine (non-selective adenosine receptor agonist) or CCPA (selective A_1 adenosine receptor agonist. In contrast, the selective A_{2A} adenosine receptor agonist CGS-21680 and A_3 adenosine receptor agonist Cl-IB-MECA did not protect the cochlea from hearing loss. Sound-evoked cochlear potentials in control rats exposed to ambient noise were minimally altered by local administration of the adenosine receptor agonists used in the noise study. Free radical generation in the cochlea exposed to noise was reduced by administration of adenosine and CCPA. This study pinpoints A_1 adenosine receptors as attractive targets for pharmacological interventions to reduce noise-induced cochlear injury after exposure.

1. Introduction

Noise-induced hearing loss (NIHL) refers to hearing impairment caused by sustained and repeated exposure to excessive sound levels. Hearing loss from noise exposure is a leading occupational injury with up to 5% of the population at risk world-wide. NIHL is commonly attributed to occupational hazards but there is concern that it is becoming increasingly prevalent with recreational activities such as loud music from portable MP3 players. Any form of sound exposure of sufficient intensity and exposure time can lead to NIHL. Exposure to sound levels around 85 dBA leads to a temporary elevation of auditory thresholds (temporary threshold shift, TTS), which can be reversed within a few days after the exposure. However, with either sustained or more intense exposure, the change in auditory thresholds becomes permanent (permanent threshold shift, PTS). There are virtually no treatments that can ameliorate the damage to the cochlea and reduce the impact of sensorineural hearing loss. Hearing aids and cochlear implants are currently the only management options offered to hearing impaired persons, whilst pharmacological therapies for NIHL have only recently been proposed (Yamashita et al., 2005).

Noise exposure drives mitochondrial activity and free radical production, causes excitotoxic swelling of afferent nerve terminals, and induces both necrotic and apoptotic cell death in the organ of Corti (Henderson et al., 2006). Cochlear injury and the loss of auditory function from noise exposure appears to be largely due to oxidative stress and glutamate excitotoxicity (Henderson et al., 2006; Le Prell et al., 2007). This implies that compounds targeting these mechanisms could offer considerable potential as therapies for NIHL. Adenosine is an endogenous neuromodulator with the ability to boost antioxidant defences, increase oxygen supply, inhibit presynaptically the release of glutamate, trigger anti-inflammatory responses and promote angiogenesis

Abbreviations: ABR, auditory brainstem response; AP, artificial perilymph; CAP, compound action potential; NIHL, noise-induced hearing loss; NT, nitrotyrosine; RWM, round window membrane; R-PIA, (R)-N⁶-phenylisopropyladenosine; SP, summating potential; SPL, sound pressure level
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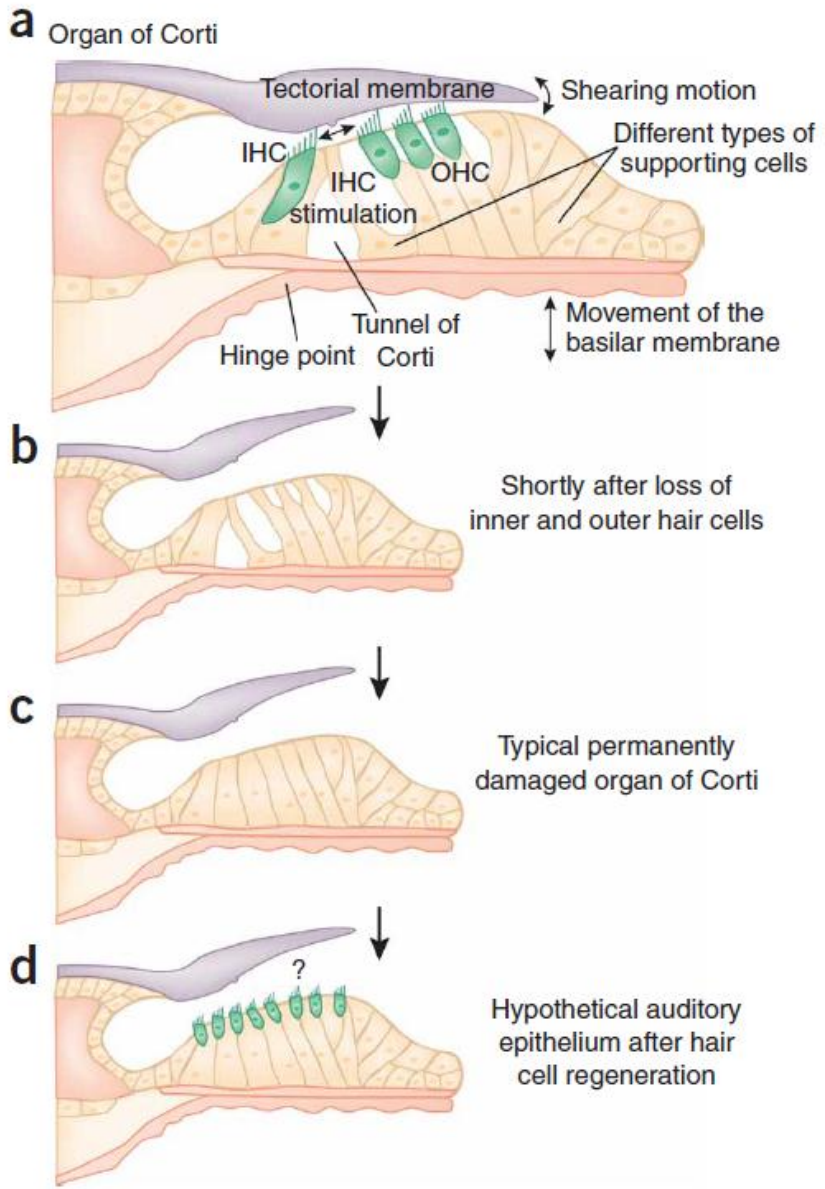

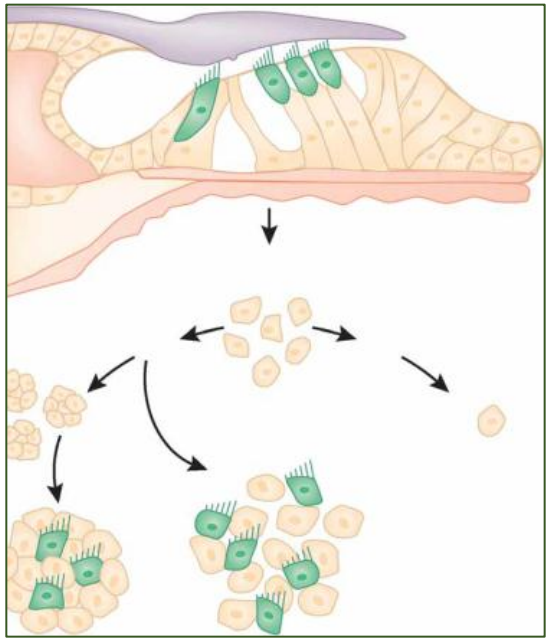


HEARING

nature neuroscience

PERSPECTIVE

Quo vadis, hair cell regeneration?

John V Brigande[1] & Stefan Heller[2]

Hearing loss is a global health problem with profound socioeconomic impact. We contend that acquired hearing loss is mainly a modern disorder caused by man-made noise and modern drugs, among other causes. These factors, combined with increasing lifespan, have exposed a deficit in cochlear self-regeneration that was irrelevant for most of mammalian evolution. Nevertheless, the mammalian cochlea has evolved from phylogenetically older structures, which do have the capacity for self-repair. Moreover, nonmammalian vertebrates can regenerate auditory hair cells that restore sensory function. We will offer a critical perspective on recent advances in stem cell biology, gene therapy, cell cycle regulation and pharmacotherapeutics to define and validate regenerative medical interventions for mammalian hair cell loss. Although these advances are promising, we are only beginning to fully appreciate the complexity of the many challenges that lie ahead.

Of all the cochlear structures and cell types, the sensory hair cells, fewer than 15,000 at birth, are the Achilles' heel of the auditory system. Loss of hair cells accounts for most hearing loss, affecting substantial proportions of the population (see **Box 1**). Generally, outer hair cells (OHCs) are more sensitive than inner hair cells (IHCs) to insults that damage hearing and to the effects of aging[1]. OHC loss, usually starting at the high-frequency basal region of the cochlea, leads to a substantial auditory threshold shift. More severe forms of hearing impairment generally involve the loss of IHCs. After hair cell loss, the organ of Corti undergoes gradual cytomorphological dedifferentiation of supporting cells; this is sometimes followed by a collapse of the tunnel of Corti, resulting in a structure that often features an unorganized mound of inconspicuous cells[2–4] (**Box 2** and **Fig. 1**).

Regeneration of cochlear hair cells is considered the ultimate remedy for hearing loss. Nevertheless, what would seem to be a simple replacement of a single cell type turns out to be a remarkably complex endeavor when one takes into account the very different functions of IHCs and OHCs, as well as their precise integration into accessory structures, such as the tectorial membrane, and the restoration of organ of Corti micromechanics. This situation is aggravated by the morphological dedifferentiation of the cellular components of the organ of Corti, particularly in cases of progressive hearing loss. Consequently, hair cell replacement cannot be viewed as simply seeding new hair cells and getting them connected to the afferent auditory nerve. For proper restoration, hair cell regeneration needs to be conducted in the context of extensive cochlear restoration, either back to the original morphological configuration or into an alternative design featuring sensitivity and tonotopy, combined with longevity of the newly introduced cells (**Fig. 1d**).

We divide this perspective into two parts: in the first part, we discuss current basic research in inner ear cell regeneration, as well as the unraveling of key genes involved in cochlear development and cell proliferation control. In the second part, we focus on therapeutic approaches and roadblocks, including delivery of cells, genes and small compounds.

Laboratory-based results

Nonmammalian vertebrate hair cell regeneration reveals existence of inner ear stem cells. In 1988, Corwin and Cotanche[5] and, independently, Ryals and Rubel[6] reported that cochlear hair cells regenerate after acoustic trauma in birds. The two reports hypothesized similarly: "that regenerated hair cells originate from mitotic divisions of supporting cells or some unidentified latent stem cells"[5] and "that the regenerative potential is retained in adult animals, suggesting that a dormant stem cell population is retained throughout life"[6]. In contrast, the mammalian cochlea is unable to regenerate hair cells after ototoxic insults[7,8]. However, in 1993 it was reported that hair cell regeneration in response to aminoglycoside ototoxicity occurs in the vestibular sensory epithelia of adult mammals, albeit to a far less impressive extent than had been seen in birds[9,10]. Hair cell regeneration in all these instances seems to originate from supporting cells that reenter the cell cycle when neighboring hair cells are dying. Mitotic supporting cells subsequently divide asymmetrically, generating new hair cells and supporting cells. In some instances, a phenotypic conversion also described as transdifferentiation into hair cells has also been observed. This mechanism is potentially faster in generating hair cells than the asymmetric division of supporting cells, but it also requires subsequent divisions to replenish the supporting cell pool.

Asymmetric division of supporting cells to generate replacement hair cells and to make identical copies of themselves is a defining feature of somatic stem cells. Other regenerating sensory systems—for example, the olfactory neuroepithelium—use resident precursors for cell replenishment during natural turnover; in addition, they harbor a quiescent adult stem cell population for complete regeneration after massive injury[11]. It is compelling to argue that with the exception of the mammalian organ of Corti, hair cell-bearing organs too are bona fide stem cell systems in which all or a subpopulation of supporting cells serves as dormant or latent stem cells.

This concept was explicitly tested by Li and colleagues[12], who demonstrated that the adult vestibular sensory epithelia of mice do harbor cells that proliferate *in vitro*. When subjected to low density and

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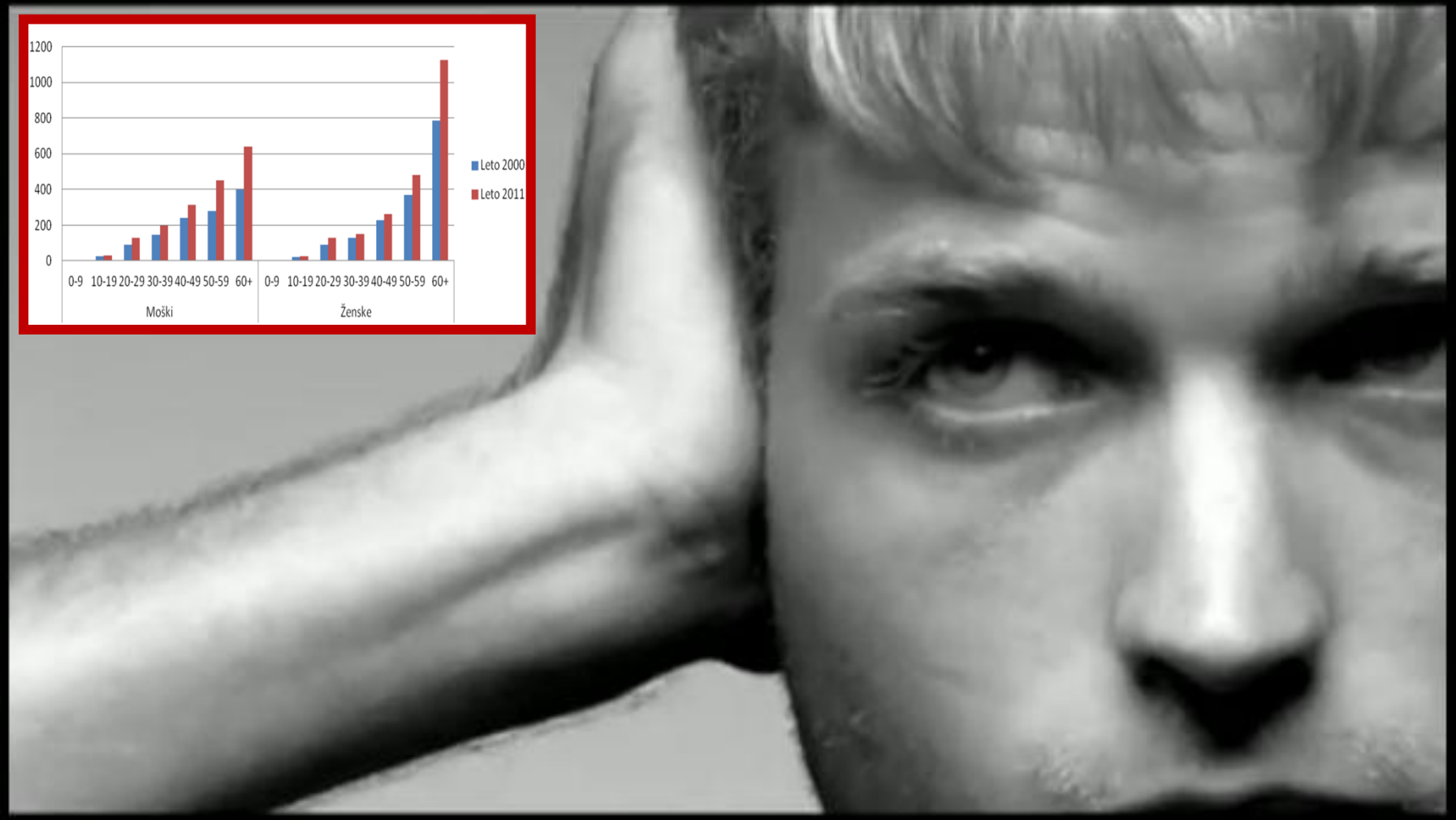
TINITUS – ŠUMENJE V UŠESIH



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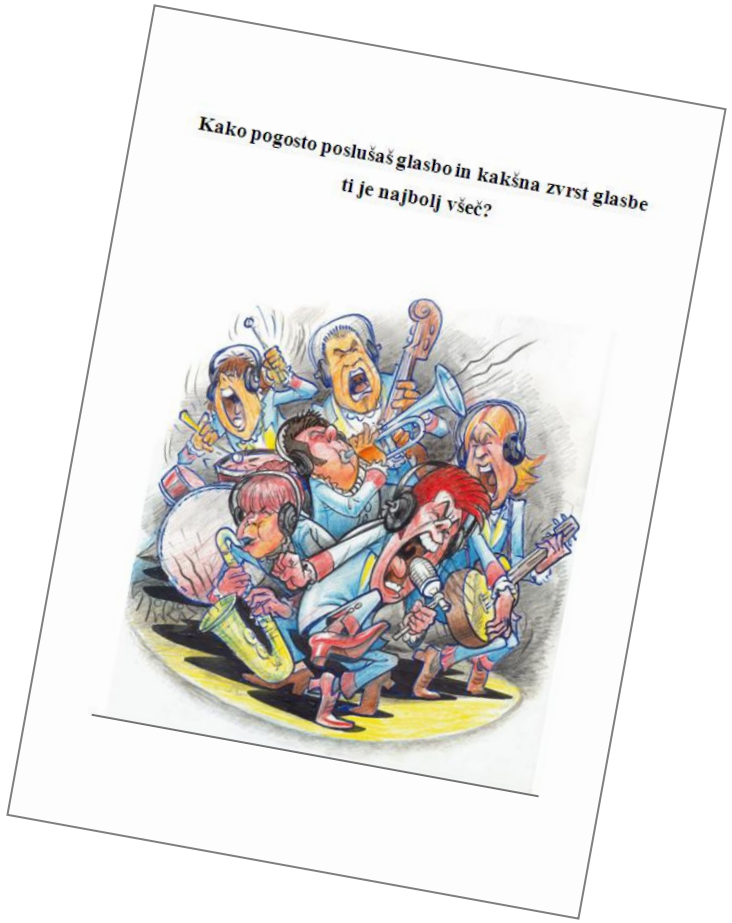
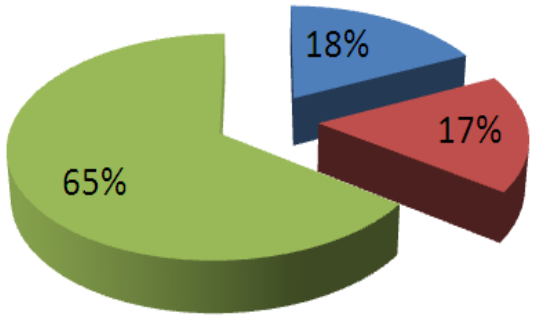




Tveganje obstaja

Tveganje je malo verjetno

Tveganja ni

Odstotek dijakov drugega letnika srednje šole (n=420), ki so zaradi pogostosti, trajanja in nastavitve nivoja intenzitete zvoka na prenosnih predvajalnikih glasbe izpostavljeni tveganju za pojav trajnih poškodb sluha. (Jeram in Breznikar, 2012)

UČINKI HRUPA NA ZDRAVJE OTROK

- **Avralni (avditorni)**
- **Ekstra-avralni (ne avditorni)**

UČINKI HRUPA NA ZDRAVJE OTROK

Avralni učinki

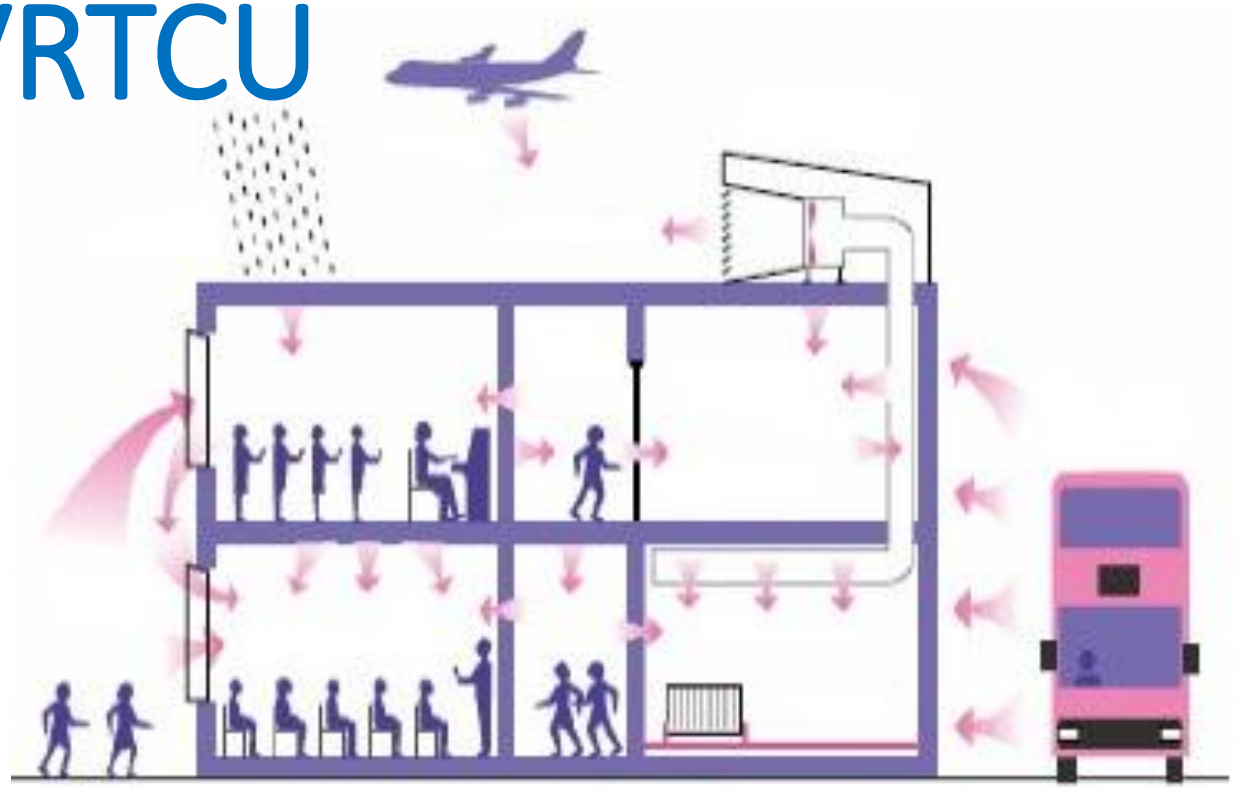
- Okvare sluha
- Začasni premik slušnega praga
- Šumenje v ušesih (tinitus)

UČINKI HRUPA NA ZDRAVJE OTROK

Ekstra-avralni učinki

- Vpliv na kognitivne funkcije (miselne sposobnosti). Hrup lahko negativno vpliva na učenje, **jezikovne sposobnosti**, motivacijo in koncentracijo ter posledično vpliva na spomin in uspešnost izvajanja bolj ali manj zahtevnih nalog.
- Hrup lahko pri otrocih izzove stres kar je povezano s povišanim srčnim utripom in hormonskim odgovorom.
- Motnje spanja
- Zmanjša možnost potrebne obnove telesnih in možganskih funkcij.
- Posredno hrup lahko vpliva na glasnejše govorjenje, kar lahko privede do hripavosti in tvorbe vozličkov na glasilkah.

ZVOČNO OKOLJE V VRTCU



- Hrup iz zunanjega okolja

- Hrup napeljav v stavbi
- Hrup naprav, ki se v vrtcu uporabljajo

- Zvok dejavnosti (igra, hoja, učenje, govor ipd.)
- Igrače, ki lahko povzročijo zvok

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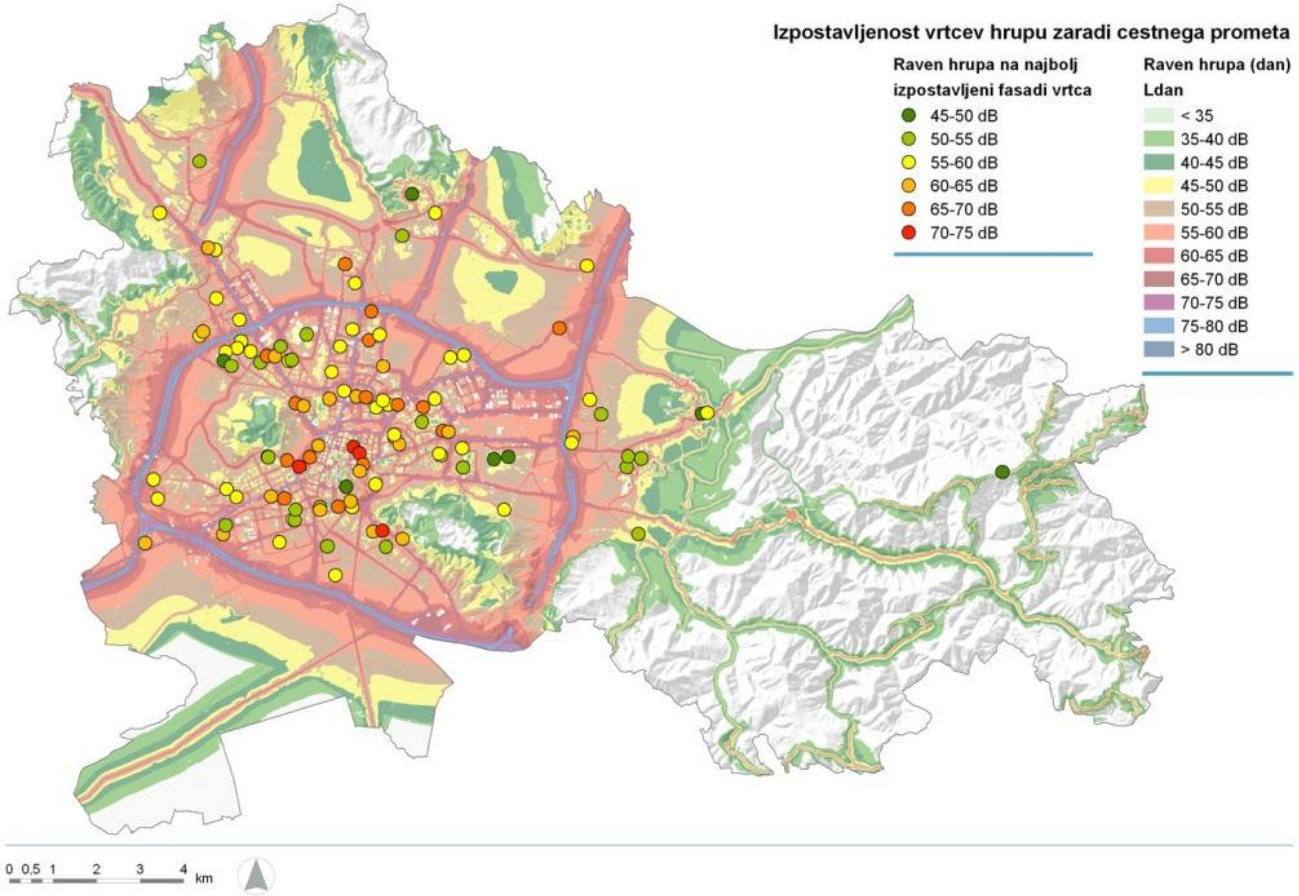
ZVOČNO OKOLJE V VRTCU

Priporočene vrednosti

- **Zvočno okolje igrišča na prostem (hrup v okolici naj ne presega 55 dBA)**
- **Zvočno okolje učilnice (ozadje hrupa naj bo pod 35 dBA)**
- **Odmevni čas (0,6 – 0,9 sekunde, odvisno od velikosti in namembnosti prostora)**

Priporočila Svetovne zdravstvene organizacije in slovenske Tehnične smernice zaščite pred hrupom v stavbah (TSG-1-005:2012)

ARSO – KAZALNIKI OKOLJA



http://kazalci.arso.gov.si/?data=indicator&ind_id=381

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ZVOČNO OKOLJE V VRTCU

UKREPI

- **Tehnični (vključno z akustiko)**
- **Organizacijski**
- **Pedagoški (vključno z dejavnostmi za obveščanje in ozaveščanje)**

ZVOČNO OKOLJE V VRTCU

Tehnični ukrepi

- Zagotovitev mehke, porozne cestne površine, ki lahko zmanjša hrup prometa do 9 dB
- Postavitev protihrupnih ograj med vrtcem in prometno cesto ali železniško progo
- Preusmeritev prometa na druge ceste in proge
- Izolacija streh in fasad ter vgraditev oken z odlično zvočno izolacijo
- Zmanjšanje hrupa naprav v prostoru: Ventilatorji, vodovodne napeljave, toplotne postaje, klimatske naprave
- Zmanjšanje prenatrpanosti prostora: Zmanjšanje števila otrok na kvadratni meter oziroma povečanje števila kvadratnih metrov na otroka, vpliva na zmanjšanje hrupa kot tudi na zmanjšanje bremena nalezljivih bolezni
- Uporaba prenosnih sten v večjih prostorih, da ustvarimo manjše delovne kotičke
- Namestitev lahkih mineralnih plošč za absorpcijo zvoka na stene in strop za zmanjšanje odmevnega časa

ZVOČNO OKOLJE V VRTCU

Organizacijski ukrepi

- Odstranitev glasnih igrač
- Polaganje debelih, trdnih in pralnih talnih oblog ali preprog
- Uporaba debelih, pralnih prtov na mizah
- Izključitev ali znižanje glasbe iz radia, televizije ali video predvajalnika
- Zagotovitev bolj tihih obrokov (manj otrok naenkrat)
- Zagotovitev priporočenega odmevnega časa tudi v prostorih kot so avle, hodniki in drugi prostori, kjer se otroci pogosto igrajo
- Ukinitev ali premestitev vrtcev, ki so izpostavljeni prekomernemu hrupu zaradi prometa
- Zagotoviti, da otroci obiščejo naravo, na primer oddelek vrtca, ki se nahaja v gozdu

ZVOČNO OKOLJE V VRTCU

Pedagoški ukrepi

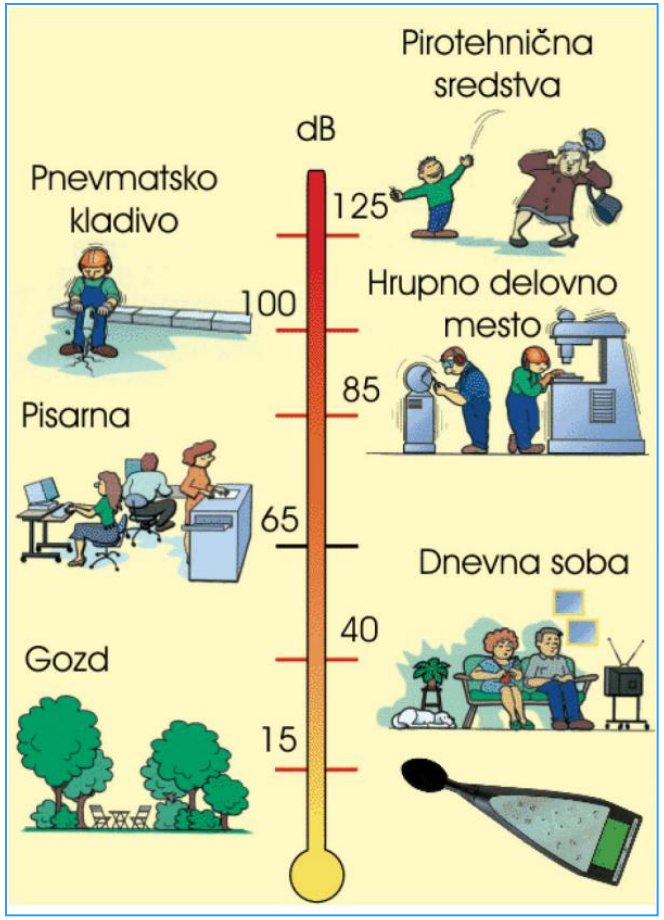
- Zmanjšanje hrupa, ki ga ustvarjajo odrasli. Otroci na splošno jemljejo odrasle za vzor, posnemajo njihovo vedenje. Če odrasli govorijo glasno, zato, da jih slišimo, otroci to dojemajo kot normalno vedenje
- Vaje z otroki, za prepoznavanje hrupa, ki ga ustvarjajo sami; "dobri" in " slabi" zvoki
- Izobraževanje in ozaveščanje oseb, ki delajo ali prihajajo v vrtec, da bodo primerno prilagodili svoje vedenje in s tem zmanjšali hrup
- Uporaba zvočnega ušesa: Naprava za merjenje hrupa, ki je oblikovana kot veliko uho in opremljena z rdečo in zeleno signalno lučko, ki opozarjata na prekomeren ali sprejemljiv hrup. Glede primernosti te naprave so še vedno deljena mnenja, saj je pomembno, da je naprava uporabljena v pravem vsebinskem kontekstu. Otroci zaradi rdeče barve ne smejo dobiti občutka krivde.
- Učenje poslušanja

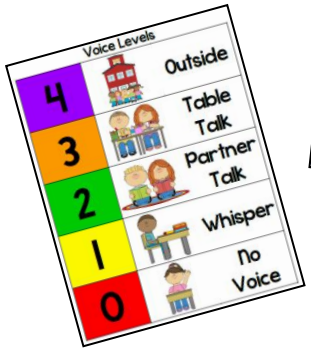
PRAVILA V VRTCU

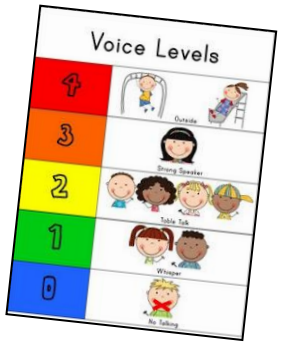
UPOŠTEVAJ PRAVILA

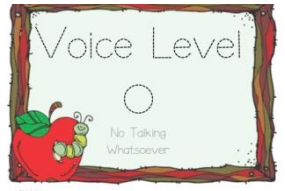
1. Vstopi mirno in tiho
2. Pripravi se na učenje
3. Če želiš govoriti, dvigni roko
4. Potrudi se čim bolj
5. Bodi prijateljski

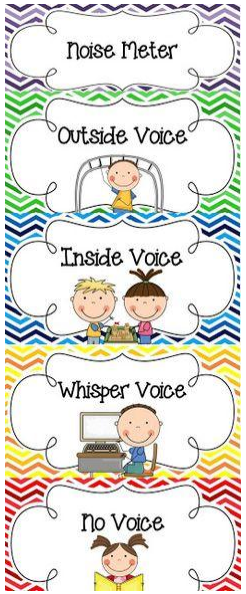
KAJ JE TIHO IN KAJ JE GLASNO?











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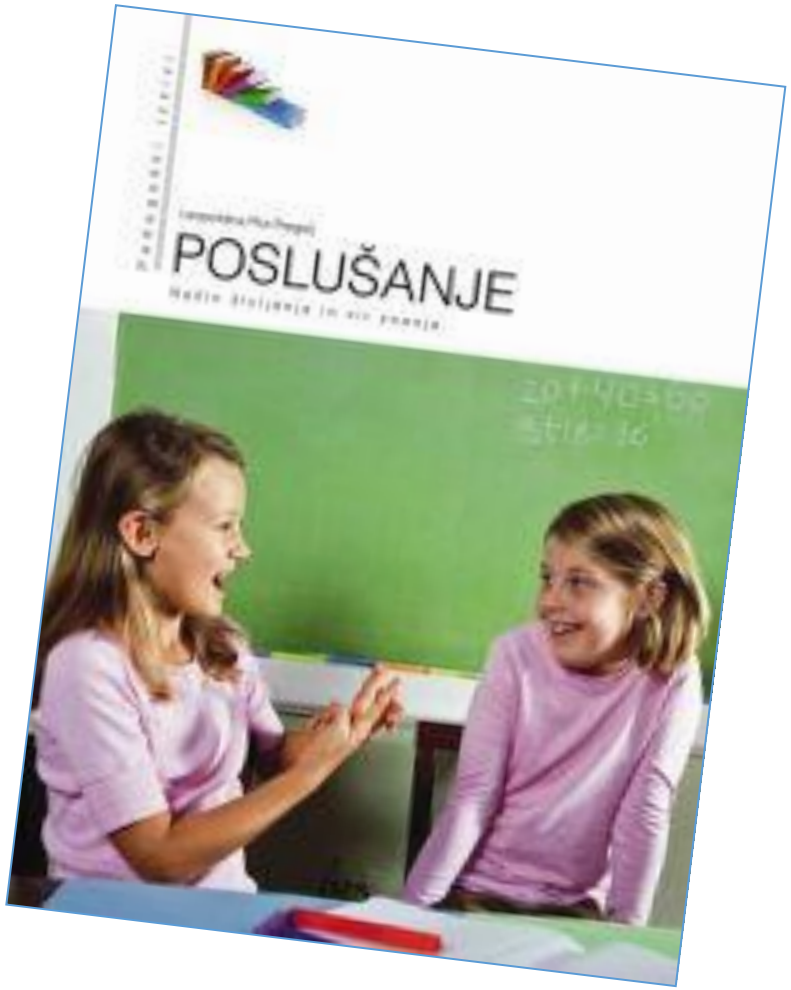
POSLUŠANJE



… Poslušanje uvrščamo med temeljne človekove potrebe. Poslušamo, da se naučimo govoriti, brati in pisati; poslušamo, da spoznamo svet okoli sebe; poslušamo, da sogovorce razbremenimo težav; poslušamo, da spoznamo sami sebe in svoje moči; poslušamo, da ustvarjamo čustveno bližino in priznamo druge ljudi, poslušamo, da ...

Leopoldina Plut Pregelj

Dobro, da imam ušesa! Dobro, da slišim!

Slikanica za otroke in odrasle

NIJZ Nacionalni inštitut za javno zdravje

Dobro, da imam ušesa! Dobro, da slišim!

Predlogi za igre

ZVOČNI SPOMIN

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Projekti in kontakti

»Kako pogosto poslušaš glasbo in kakšna zvrst glasbe ti je najbolj všeč?«

Projekt je namenjen ozaveščanju osnovnošolcev in srednješolcev o nevarnosti, ki jo pretirano poslušanje glasne glasbe predstavlja za poškodbe sluha. Anketa je bila izvedena v sklopu akcije ob Mednarodnem dnevu ozaveščanja o hrupu, aprila 2013. Kontakt: Nacionalni inštitut za javno zdravje. Trubarjeva 2, Ljubljana.

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