

HYPERACUSIS



Abstract

Hyperacusis is characterized by an intolerance to everyday sounds perceived as painfully loud, significantly disrupting daily life (1). This condition affects nearly 9% of the population (2,3) and is often accompanied by hearing loss. Studies suggest hyperacusis may result from the central nervous system's adaptation to peripheral sensory loss, leading to unregulated amplification of auditory stimuli. This process would maintain neural activity levels after sensory impairment, but can distort auditory perception (4, 5). Imaging studies also shows increased sound-evoked activity in several auditory regions in hyperacusis patients, indicating a disruption in the balance of neuronal excitation and inhibition (6, 7). Despite extensive research, effective treatments for hyperacusis remain limited due to the complexity of its neural mechanisms and the lack of preclinical models. Our study aimed to correlate behavioral changes in animals, measured by startle response tests, with **neurophysiological changes** indicative of hyperacusis. We utilized techniques to measure both peripheral and central auditory function in awake animals (7, 8), proposing that increased central activity compensates for peripheral sensory loss.

Objectives

Our goal is to identify a combination of neural biomarkers and behavioral changes reliably characterizing the presence of hyperacusis.



MAKING HEARING A PRIORITY

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Development of an awake animal model for hyperacusis screening

Dejean C.^{1,2,4}, Postal O.^{1,2}, Dupont T.¹, Michalski N.¹, Pucheu S.⁴, Gourévitch B.^{1,3} ¹Université Paris Cité, Institut Pasteur, AP-HP, Inserm, Fondation Pour l'Audition, Institut de l'Audition (IDA), IHU reConnect, Paris, France ²Sorbonne Universités, Paris, France • ³CNRS • ⁴Cilcare, Montpellier, France

- 1/ Subjective measure: startle reflex Intensity function 70 → 110 dB G Auditory Startle Response Auditory Startle Normal Startle **Putative** Reflex hyperacusis \sim 2/ Objective measures: threshold, amplitude, latencies, Histology amplitude-intensity function in auditory pathways (CN) (SOC) (LL & IC) III IV V (VIIIN) (VIIIN) P35 100 ms Auditory Brainstem Response Cortical Auditory Evoked (ABR) Potentials (CAEP)

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behavioral correlates of hyperacusis.



- inferior colliculus and the auditory cortex.

ABRs Wave IV at 15 kHz, 70 dB



- auditory system after a noise induced temporary hearing loss.

(1) Fackrell K. et al. 2022; (2) Hall, A. J et al. 2016; (3) Smit, A. L. et al 2021; (4) Schaette, R. & et al. 2006; (5) Ransdell, J. L. 2012; (6) Knipper et al. 2013; (7) Auerbach et al. 2021 ; (8) Postal. et al. 2022 ; (8) Dejean C. et al. 2023





Results

• The noise exposure caused an increase of the startle reflex amplitude in some animals, known to be possible



• Positive correlations between the Hyperacusis Index and the amplitude of the evoked response to sounds in the

• The correlation appeared between **5 to 10 weeks** after the noise exposure.



Conclusion

 An increase of the startle reflex amplitude after an acoustic trauma seems to correlate well with an increased central auditory evoked response, even in the absence of peripheral changes.

• Following a temporary hearing loss, a subset of animals could be identified as possibly suffering from hyperacusis based on their **simultaneous increase** of their behavioral and neural response to sounds.

• These results open the door to a better characterization of **pathophysiological mechanisms** which impact the central

Bibliography





CNIT Paris La Défens

