Hyperacusis: Diversity in Cause, Expression, and Advocacy

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Benjamin Auerbach

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Session Description Hyperacusis is a complex hearing disorder that encompasses a wide-range of reactions to sound, including excessive loudness, increased aversion/fear of sound, or even pain. While often associated with hearing loss and tinnitus, sound tolerance disturbances are observed across a broad spectrum of neurological disorders, including autism, chronic pain and post-traumatic stress disorder. Thus, hyperacusis is diverse in both its etiology and phenotypic expression, and it is imperative to consider this diversity when attempting to elucidate its physiological mechanisms and advance diagnostic and treatments. Bryan Pollard, founder and president of the Hyperacusis Research Ltd., was a champion for this cause. Bryan was a tireless advocate for hyperacusis research, working to connect researchers from diverse backgrounds, often through formal and informal workshops and dinners at the ARO midwinter meeting. He was the first non-researcher to present at ARO and was instrumental in creating a new diagnosis of pain hyperacusis. Bryan unfortunately passed away earlier this year.
This goal of this symposium is two-fold. First, we hope to update the hearing community on the status of the hyperacusis field and the progress that has been made towards diagnosing, treating, and modeling this disorder. Hyperacusis is a rapidly growing research field that intersects with many other aspects of hearing research, including central auditory processing and plasticity, tinnitus, and autism. As such, this symposium would have a broad target audience, from basic auditory neuroscientists interested in the neural mechanisms of perception and experience-dependent plasticity to audiologists seeking to understand and perhaps better treat a host of auditory processing disorders. In addition, we hope to use this symposium to commemorate the impact that Bryan Pollard had on the field, starting with a short introduction and in memoriam to highlight the ways he and the Hyperacusis Research foundation have helped move hyperacusis research forward.

Presenter Diversity The symposium will feature a diverse group of speakers, including several presenters from underrepresented minorities. The symposium will additionally include both up-and-coming young investigators and established leaders in the field, with speakers who employ

a variety of techniques and approaches, from animal models to human clinical research.

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Signature Benjamin D Auerbach

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Imaging hyperacusis: Past and Present

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Fatima Husain

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract Brain imaging of humans with hyperacusis remains one of the primary methods to identify neural networks sub serving this condition and dissociate them from those related to comorbid tinnitus or hearing loss. Despite the challenges associated with cost and noise of some tools, results of such studies have informed the existing experimental and theoretical framework of hyperacusis. In a pioneering fMRI study, Melcher and colleagues (2010) parsed out the contribution of co-occurring hyperacusis to brain imaging findings of tinnitus. In particular, they noted that while both subcortical and cortical auditory areas were responsive in those with hyperacusis (relative to controls), such an elevated response was only noted in the auditory cortex for those with tinnitus. Two recent studies (Koops and van Dijk, 2021; Hofmeier et al., 2021) further explored this dissociation. Koops and van Dijk confirmed the higher response in cortical and subcortical centers of auditory processing in response to external sounds but in addition found that there was reduced response to the frequencies of the internally-generated tinnitus sound. The Hofmeier study provided corroboration of the increased responsiveness of the tinnitus and hyperacusis group to external sounds in the cortical and subcortical auditory areas. Additionally they noted increased wave III amplitude in brain-stem response of the tinnitus and hyperacusis group relative to the control group; the tinnitus only group exhibited a prolonged and reduced wave V amplitude compared to the controls. These studies support the idea of central gain in the condition of hyperacusis but not necessarily if tinnitus occurs alone. They further point to the role played by attention in tinnitus, as noted in task-based fMRI studies in my own lab (Husain et al., 2011; 2015). What is not easily known is the contribution and interaction of clinically-significant hearing loss with hyperacusis. Koops and van Dijk noted the elevation of response at frequencies in the hearing loss as well as normal hearing range in those with hyperacusis. The Melcher studies were primarily in those with normal hearing. In an ongoing study, we are collecting both auditory brainstem response and fMRI data on young adults with hyperacusis and normal hearing, results of which will be reported at the meeting. In summary, current and future non-invasive brain imaging

studies continue to expand our understanding of pathophysiology of hyperacusis and eventually test therapies that help patients.

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Fatima *	Husain *	University of Illinois at Urbana-Champaign

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Signature Fatima T. Husain

Hyperacusis: Diversity in Cause, Expression, and Advocacy

A Need to Better Define and Characterize Loudness and Hyperacusis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Sylvie Hebert

Affiliation Universite de Montreal

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract Hyperacusis is a hearing disorder wherein everyday sounds are considered too loud, fearful, annoying, or painful. In its most accepted definition, hyperacusis designates sounds of moderate intensities that are judged louder than normal perception. A deeper understanding of what loudness is would contribute to a better definition of what hyperacusis is, and how it can be defined and diagnosed. Indeed, although loudness involves the perception of the intensity of sounds, i.e. its sensory dimension, loudness may also be modulated through its affective dimension. In this talk I will present recent data aiming at a better understanding of loudness, namely its sensory and its affective dimensions, and how these two dimensions can be dissociated within the same stimuli. I will also report the findings of a recent scoping review on the electrophysiological correlates of hyperacusis, in which we found a diversity in terms and definitions used to describe hyperacusis and several identifiable aetiologies, among which developmental disorders, neurological disorders, induced hearing damage, and idiopathic aetiology. Broader consensus around definitions and diagnostic criteria considering aetiologies may guide researchers to ask better questions and clinicians to more efficiently manage the patient who complain about hyperacusis.

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Sylvie *	Hebert *	Universite de Montreal

Charlotte	Bigras	Universite de Montreal
Ariane	Forget	Universite de Montreal
Berangere	Villatte	Universite de Montreal

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Signature Sylvie Hébert

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Stressful hearing: Hyperacusis Can Be Induced by Chronic Stressful Noise Exposure or Chronic Pharmacological Stress that Disrupts the HPA Axis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Richard Salvi

Affiliation Center for Hearing & Deafness, SUNY at Buffalo

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract We live in a noisy, stressful environment largely oblivious to its negative hearing-health consequences. One debilitating hearing impairment associated with noise-induced hearing loss is hyperacusis, a loudness intolerance disorder in which everyday sounds are perceived as excessively loud, aversive and stressful. To investigate the biological bases of hyperacusis, we developed a reaction time-intensity (RT-I) paradigm to test for loudness hyperacusis. To assess the aversive quality of hyperacusis, we developed an active sound avoidance paradigm (ASAP) to measure sound avoidance behaviors. To trigger the induction of hyperacusis, we exposed rats to intense high-frequency noise for several months; these conditions were expected to induce chronic noise-stress in addition to high-frequency loss. After this prolonged, stressful noise exposure, rats developed clear signs of sound avoidance hyperacusis as well as loudness hyperacusis with a low-frequency spectral profile. This prolonged, stressful noise exposure disrupted the hypothalamic-pituitary-adrenal (HPA) axis; it did not alter basal corticosterone (CORT) levels, but instead, greatly reduced the rise in corticosterone (CORT) triggered by restraint stress (i.e., it blunted the stress response). This noise exposure also chronically increased the expression of glucocorticoid receptors (GR) in the auditory cortex, part of the negative feedback network that suppressed the continued release of CORT. To determine the role of chronic stress independent of hearing loss, rats were chronically stressed by pharmacologically treating the rats with CORT-stress hormone. After chronic pharmacologic stress, rats developed behavioral evidence of loudness hyperacusis and sound avoidance hyperacusis. Consistent with the previous noise studies, basal CORT levels remained normal; however, rats exhibited a blunted CORT response to restraint stress and GR expression in the auditory cortex increased. Chronic pharmacologic CORT stress did not alter DPOAEs or the cochlear compound action potential; however, sound-evoked responses from chronically implanted electrodes on the auditory cortex were greatly enhanced. These results show for the first time that chronic

pharmacologic stress, in the absence of hearing loss, is sufficient to induce behavioral evidence of hyperacusis, which is associated with auditory cortex neural hyperactivity and enhanced central gain.

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Senthilvelan	Manohar	State University of New York at Buffalo
Guang-Di	Chen	Center for Hearing and Deafness, State University of New York at Buffalo

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Signature Richard Salvi

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Behavioral and Physiological Measures of Sound Intolerance

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Sarah Theodoroff

Affiliation Department of Veterans Affairs, NCRAR

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract The experience of individuals who have decreased tolerance to everyday sounds is heterogeneous. No consensus exists regarding what constitutes hyperacusis or noise sensitivity. A direct consequence of this is the lack of a “gold standard” regarding how to diagnose, assess, and treat these conditions. The purpose of this presentation is to address this gap in clinical knowledge.

Noise sensitivity refers to an increased reactivity to everyday sounds and encompasses a range of psychological attributes, often including annoyance or feeling overwhelmed by the sounds in the environment. An estimated 59% of patients with mild traumatic brain injury have noise sensitivity (Shepherd et al, 2019). Research is lacking both on the pathophysiology of noise sensitivity, its clinical treatment, and to what degree it may or may not be a similar phenomenon to hyperacusis. Hyperacusis describes a decreased sound tolerance driven by the perceived loudness of ordinary sounds. It often including physical discomfort or pain when listening to sounds that are at moderate or low intensity levels, which most people would find tolerable. Estimates are as high as 60-79% of tinnitus patients have comorbid hyperacusis (Andersson et al, 2001; Dauman & Bouscan-Faure, 2005).

When Veterans seek medical attention because ordinary sounds are painfully loud or there’s “too much noise to function” many clinicians are uncertain how best to meet their patients’ needs. This results in the focus being shifted to comorbid conditions hoping that the “hyperacusis” is a symptom of another condition. Research is needed to develop evidence-based tools that are capable of differentiating noise sensitivity from hyperacusis and from other health conditions that present with similar symptoms. This need motivated the ideas behind an on-going research project that is examining the relationship between auditory and psychological biomarkers to determine how well auditory and psychophysiological data predict self-reported decreased tolerance to everyday sounds. Behavioral and physiological measures are collected in order to detect where deficits exist in sensory and/or neurological structures associated with complaints of sound

intolerance.
Results from this work will elucidate aspects of the underlying pathophysiology of “hyperacusis” and “noise sensitivity.” Ultimately, outcomes from this avenue of research will guide the development of targeted rehabilitative treatments based on the etiology of these conditions and inform which disciplines should be working together to best meet the needs of this patient population (e.g., audiology, psychology, neurology).

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First Name	Last Name	Affiliation
Sarah *	Theodoroff *	Department of Veterans Affairs, NCRAR

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Signature Sarah Theodoroff

Hyperacusis: Diversity in Cause, Expression, and Advocacy

A Rodent Model of Acoustic Trauma to Study Neural and Inflammatory Mechanisms of Hyperacusis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Megan Wood

Affiliation Johns Hopkins University School of Medicine

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract The mechanism underlying painful hyperacusis is currently unknown. A subset of hyperacusis patients surveyed recently reported experiencing acoustic trauma before the onset of their symptoms. Therefore, we use acoustic trauma in rodents to induce neurological and inflammatory changes in the cochlea as a model for noise-induced hyperacusis. Acoustic trauma affects many cell types of the inner ear. We focus on the responses of type II auditory nerve fibers and immune cells as these cell types are the putative pain sensing neurons and chief responders to inflammation in the cochlea, respectively. Type II auditory nerve fibers exhibit changes in their calcium dynamics after acoustic trauma. New analysis of an existing dataset will be discussed to describe a sensitization of these fibers as a possible mechanism for hyperacusis. Another possible mechanism for hyperacusis is an enhanced inflammatory response following acoustic trauma. Neuroimmune crosstalk through neuropeptides found in the cochlea, such as CGRP, may play an important role in this possible mechanism. Recent studies following immune cell migration after blocking CGRP receptors during acoustic trauma will be discussed. Finally, the presentation will end with a discussion of the parameters needed to properly model painful hyperacusis in rodents including behavioral assays of pain perception to sound stimulation.

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First Name	Last Name	Affiliation

Megan *	Wood *	Johns Hopkins University School of Medicine
Viola	Monovich	Peabody Institute of Johns Hopkins University
Anda	Nyati	Johns Hopkins Bloomberg School of Public Health
Paul	Fuchs	Johns Hopkins University School of Medicine
Elisabeth	Glowatzki	Johns Hopkins University School of Medicine

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Signature Lara Beers Wood

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Investigating Hyperacusis in Rodent Models of Autism

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Benjamin Auerbach

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract Animal models are indispensable tools for identifying disease pathophysiology and developing objective biomarkers for hearing disorders like hyperacusis. However, care must be taken when attempting to recapitulate complex hearing disorders in model systems. Hyperacusis encompasses a wide range of reactions to sound and is observed across a broad spectrum of neurological disorders, and this diversity must be accounted for when attempting to measure and induce hyperacusis in animals. Recent progress has been made in developing rodent behavioral models that capture distinct aspects of sound perception disrupted in hyperacusis, with attempts to disentangle psychoacoustic (e.g. excessive loudness) from affective (e.g. decreased sound tolerance) aspects of the disorder. However, drug- and noise-induced hearing loss are still the primary methods used for inducing hyperacusis-like states in animals. Here we will discuss recent attempts to characterize sound tolerance disturbances in genetic models of neurodevelopmental disorders that present with high rates of hyperacusis, such as Fragile X syndrome and autism spectrum disorders (ASD). Comparing and contrasting hyperacusis associated with hearing loss and neurodevelopmental disruption has the potential to uncover convergent (or divergent) pathophysiological mechanisms across distinct forms of hyperacusis , which will help advance our ability to diagnose and treat this often devastating disorder.

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Benjamin *	Auerbach *	University of Illinois at Urbana-Champaign

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Signature Benjamin D Auerbach

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Connecting Researchers to Clinicians and patients: Bryan Pollard's Roadmap to a Cure for Hyperacusis with Pain

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Michael Maholchic

Affiliation Hyperacusis Research, Ltx

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract In memoriam to Bryan Pollard, founder of Hyperacusis Research Ltd., and his impact on the field.

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Michael *	Maholchic *	Hyperacusis Research, Ltx

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Hyperacusis: Diversity in Cause, Expression, and Advocacy

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Signature Benjamin D Auerbach

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Imaging hyperacusis: Past and Present

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Status Submitted

Submitter Fatima Husain

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

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Signature Fatima T. Husain

Hyperacusis: Diversity in Cause, Expression, and Advocacy

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Submitter Sylvie Hebert

Affiliation Universite de Montreal

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Signature Sylvie Hébert

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Status Submitted

Submitter Richard Salvi

Affiliation Center for Hearing & Deafness, SUNY at Buffalo

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract We live in a noisy, stressful environment largely oblivious to its negative hearing-health consequences. One debilitating hearing impairment associated with noise-induced hearing loss is hyperacusis, a loudness intolerance disorder in which everyday sounds are perceived as excessively loud, aversive and stressful. To investigate the biological bases of hyperacusis, we developed a reaction time-intensity (RT-I) paradigm to test for loudness hyperacusis. To assess the aversive quality of hyperacusis, we developed an active sound avoidance paradigm (ASAP) to measure sound avoidance behaviors. To trigger the induction of hyperacusis, we exposed rats to intense high-frequency noise for several months; these conditions were expected to induce chronic noise-stress in addition to high-frequency loss. After this prolonged, stressful noise exposure, rats developed clear signs of sound avoidance hyperacusis as well as loudness hyperacusis with a low-frequency spectral profile. This prolonged, stressful noise exposure disrupted the hypothalamic-pituitary-adrenal (HPA) axis; it did not alter basal corticosterone (CORT) levels, but instead, greatly reduced the rise in corticosterone (CORT) triggered by restraint stress (i.e., it blunted the stress response). This noise exposure also chronically increased the expression of glucocorticoid receptors (GR) in the auditory cortex, part of the negative feedback network that suppressed the continued release of CORT. To determine the role of chronic stress independent of hearing loss, rats were chronically stressed by pharmacologically treating the rats with CORT-stress hormone. After chronic pharmacologic stress, rats developed behavioral evidence of loudness hyperacusis and sound avoidance hyperacusis. Consistent with the previous noise studies, basal CORT levels remained normal; however, rats exhibited a blunted CORT response to restraint stress and GR expression in the auditory cortex increased. Chronic pharmacologic CORT stress did not alter DPOAEs or the cochlear compound action potential; however, sound-evoked responses from chronically implanted electrodes on the auditory cortex were greatly enhanced. These results show for the first time that chronic

pharmacologic stress, in the absence of hearing loss, is sufficient to induce behavioral evidence of hyperacusis, which is associated with auditory cortex neural hyperactivity and enhanced central gain.

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First Name	Last Name	Affiliation
Richard *	Salvi *	Center for Hearing & Deafness, SUNY at Buffalo
Senthilvelan	Manohar	State University of New York at Buffalo
Guang-Di	Chen	Center for Hearing and Deafness, State University of New York at Buffalo

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Signature Richard Salvi

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Behavioral and Physiological Measures of Sound Intolerance

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Sarah Theodoroff

Affiliation Department of Veterans Affairs, NCRAR

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract The experience of individuals who have decreased tolerance to everyday sounds is heterogeneous. No consensus exists regarding what constitutes hyperacusis or noise sensitivity. A direct consequence of this is the lack of a “gold standard” regarding how to diagnose, assess, and treat these conditions. The purpose of this presentation is to address this gap in clinical knowledge.

Noise sensitivity refers to an increased reactivity to everyday sounds and encompasses a range of psychological attributes, often including annoyance or feeling overwhelmed by the sounds in the environment. An estimated 59% of patients with mild traumatic brain injury have noise sensitivity (Shepherd et al, 2019). Research is lacking both on the pathophysiology of noise sensitivity, its clinical treatment, and to what degree it may or may not be a similar phenomenon to hyperacusis. Hyperacusis describes a decreased sound tolerance driven by the perceived loudness of ordinary sounds. It often including physical discomfort or pain when listening to sounds that are at moderate or low intensity levels, which most people would find tolerable. Estimates are as high as 60-79% of tinnitus patients have comorbid hyperacusis (Andersson et al, 2001; Dauman & Bouscan-Faure, 2005).

When Veterans seek medical attention because ordinary sounds are painfully loud or there’s “too much noise to function” many clinicians are uncertain how best to meet their patients’ needs. This results in the focus being shifted to comorbid conditions hoping that the “hyperacusis” is a symptom of another condition. Research is needed to develop evidence-based tools that are capable of differentiating noise sensitivity from hyperacusis and from other health conditions that present with similar symptoms. This need motivated the ideas behind an on-going research project that is examining the relationship between auditory and psychological biomarkers to determine how well auditory and psychophysiological data predict self-reported decreased tolerance to everyday sounds. Behavioral and physiological measures are collected in order to detect where deficits exist in sensory and/or neurological structures associated with complaints of sound

intolerance.
Results from this work will elucidate aspects of the underlying pathophysiology of “hyperacusis” and “noise sensitivity.” Ultimately, outcomes from this avenue of research will guide the development of targeted rehabilitative treatments based on the etiology of these conditions and inform which disciplines should be working together to best meet the needs of this patient population (e.g., audiology, psychology, neurology).

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Sarah *	Theodoroff *	Department of Veterans Affairs, NCRAR

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Signature Sarah Theodoroff

Hyperacusis: Diversity in Cause, Expression, and Advocacy

A Rodent Model of Acoustic Trauma to Study Neural and Inflammatory Mechanisms of Hyperacusis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Megan Wood

Affiliation Johns Hopkins University School of Medicine

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract The mechanism underlying painful hyperacusis is currently unknown. A subset of hyperacusis patients surveyed recently reported experiencing acoustic trauma before the onset of their symptoms. Therefore, we use acoustic trauma in rodents to induce neurological and inflammatory changes in the cochlea as a model for noise-induced hyperacusis. Acoustic trauma affects many cell types of the inner ear. We focus on the responses of type II auditory nerve fibers and immune cells as these cell types are the putative pain sensing neurons and chief responders to inflammation in the cochlea, respectively. Type II auditory nerve fibers exhibit changes in their calcium dynamics after acoustic trauma. New analysis of an existing dataset will be discussed to describe a sensitization of these fibers as a possible mechanism for hyperacusis. Another possible mechanism for hyperacusis is an enhanced inflammatory response following acoustic trauma. Neuroimmune crosstalk through neuropeptides found in the cochlea, such as CGRP, may play an important role in this possible mechanism. Recent studies following immune cell migration after blocking CGRP receptors during acoustic trauma will be discussed. Finally, the presentation will end with a discussion of the parameters needed to properly model painful hyperacusis in rodents including behavioral assays of pain perception to sound stimulation.

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Megan *	Wood *	Johns Hopkins University School of Medicine
Viola	Monovich	Peabody Institute of Johns Hopkins University
Anda	Nyati	Johns Hopkins Bloomberg School of Public Health
Paul	Fuchs	Johns Hopkins University School of Medicine
Elisabeth	Glowatzki	Johns Hopkins University School of Medicine

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Signature Lara Beers Wood

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Investigating Hyperacusis in Rodent Models of Autism

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Benjamin Auerbach

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract Animal models are indispensable tools for identifying disease pathophysiology and developing objective biomarkers for hearing disorders like hyperacusis. However, care must be taken when attempting to recapitulate complex hearing disorders in model systems. Hyperacusis encompasses a wide range of reactions to sound and is observed across a broad spectrum of neurological disorders, and this diversity must be accounted for when attempting to measure and induce hyperacusis in animals. Recent progress has been made in developing rodent behavioral models that capture distinct aspects of sound perception disrupted in hyperacusis, with attempts to disentangle psychoacoustic (e.g. excessive loudness) from affective (e.g. decreased sound tolerance) aspects of the disorder. However, drug- and noise-induced hearing loss are still the primary methods used for inducing hyperacusis-like states in animals. Here we will discuss recent attempts to characterize sound tolerance disturbances in genetic models of neurodevelopmental disorders that present with high rates of hyperacusis, such as Fragile X syndrome and autism spectrum disorders (ASD). Comparing and contrasting hyperacusis associated with hearing loss and neurodevelopmental disruption has the potential to uncover convergent (or divergent) pathophysiological mechanisms across distinct forms of hyperacusis , which will help advance our ability to diagnose and treat this often devastating disorder.

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* Presenting Author

First Name	Last Name	Affiliation

Benjamin *	Auerbach *	University of Illinois at Urbana-Champaign

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Signature Benjamin D Auerbach

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Connecting Researchers to Clinicians and patients: Bryan Pollard's Roadmap to a Cure for Hyperacusis with Pain

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Michael Maholchic

Affiliation Hyperacusis Research, Ltx

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract In memoriam to Bryan Pollard, founder of Hyperacusis Research Ltd., and his impact on the field.

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Michael *	Maholchic *	Hyperacusis Research, Ltx

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Signature Michael Maholchic

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Benjamin Auerbach

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Session Description Hyperacusis is a complex hearing disorder that encompasses a wide-range of reactions to sound, including excessive loudness, increased aversion/fear of sound, or even pain. While often associated with hearing loss and tinnitus, sound tolerance disturbances are observed across a broad spectrum of neurological disorders, including autism, chronic pain and post-traumatic stress disorder. Thus, hyperacusis is diverse in both its etiology and phenotypic expression, and it is imperative to consider this diversity when attempting to elucidate its physiological mechanisms and advance diagnostic and treatments. Bryan Pollard, founder and president of the Hyperacusis Research Ltd., was a champion for this cause. Bryan was a tireless advocate for hyperacusis research, working to connect researchers from diverse backgrounds, often through formal and informal workshops and dinners at the ARO midwinter meeting. He was the first non-researcher to present at ARO and was instrumental in creating a new diagnosis of pain hyperacusis. Bryan unfortunately passed away earlier this year.
This goal of this symposium is two-fold. First, we hope to update the hearing community on the status of the hyperacusis field and the progress that has been made towards diagnosing, treating, and modeling this disorder. Hyperacusis is a rapidly growing research field that intersects with many other aspects of hearing research, including central auditory processing and plasticity, tinnitus, and autism. As such, this symposium would have a broad target audience, from basic auditory neuroscientists interested in the neural mechanisms of perception and experience-dependent plasticity to audiologists seeking to understand and perhaps better treat a host of auditory processing disorders. In addition, we hope to use this symposium to commemorate the impact that Bryan Pollard had on the field, starting with a short introduction and in memoriam to highlight the ways he and the Hyperacusis Research foundation have helped move hyperacusis research forward.

Presenter Diversity The symposium will feature a diverse group of speakers, including several presenters from underrepresented minorities. The symposium will additionally include both up-and-coming young investigators and established leaders in the field, with speakers who employ

a variety of techniques and approaches, from animal models to human clinical research.

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Signature Benjamin D Auerbach

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Imaging hyperacusis: Past and Present

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Fatima Husain

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract Brain imaging of humans with hyperacusis remains one of the primary methods to identify neural networks sub serving this condition and dissociate them from those related to comorbid tinnitus or hearing loss. Despite the challenges associated with cost and noise of some tools, results of such studies have informed the existing experimental and theoretical framework of hyperacusis. In a pioneering fMRI study, Melcher and colleagues (2010) parsed out the contribution of co-occurring hyperacusis to brain imaging findings of tinnitus. In particular, they noted that while both subcortical and cortical auditory areas were responsive in those with hyperacusis (relative to controls), such an elevated response was only noted in the auditory cortex for those with tinnitus. Two recent studies (Koops and van Dijk, 2021; Hofmeier et al., 2021) further explored this dissociation. Koops and van Dijk confirmed the higher response in cortical and subcortical centers of auditory processing in response to external sounds but in addition found that there was reduced response to the frequencies of the internally-generated tinnitus sound. The Hofmeier study provided corroboration of the increased responsiveness of the tinnitus and hyperacusis group to external sounds in the cortical and subcortical auditory areas. Additionally they noted increased wave III amplitude in brain-stem response of the tinnitus and hyperacusis group relative to the control group; the tinnitus only group exhibited a prolonged and reduced wave V amplitude compared to the controls. These studies support the idea of central gain in the condition of hyperacusis but not necessarily if tinnitus occurs alone. They further point to the role played by attention in tinnitus, as noted in task-based fMRI studies in my own lab (Husain et al., 2011; 2015). What is not easily known is the contribution and interaction of clinically-significant hearing loss with hyperacusis. Koops and van Dijk noted the elevation of response at frequencies in the hearing loss as well as normal hearing range in those with hyperacusis. The Melcher studies were primarily in those with normal hearing. In an ongoing study, we are collecting both auditory brainstem response and fMRI data on young adults with hyperacusis and normal hearing, results of which will be reported at the meeting. In summary, current and future non-invasive brain imaging

studies continue to expand our understanding of pathophysiology of hyperacusis and eventually test therapies that help patients.

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Fatima *	Husain *	University of Illinois at Urbana-Champaign

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Signature Fatima T. Husain

Hyperacusis: Diversity in Cause, Expression, and Advocacy

A Need to Better Define and Characterize Loudness and Hyperacusis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Sylvie Hebert

Affiliation Universite de Montreal

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract Hyperacusis is a hearing disorder wherein everyday sounds are considered too loud, fearful, annoying, or painful. In its most accepted definition, hyperacusis designates sounds of moderate intensities that are judged louder than normal perception. A deeper understanding of what loudness is would contribute to a better definition of what hyperacusis is, and how it can be defined and diagnosed. Indeed, although loudness involves the perception of the intensity of sounds, i.e. its sensory dimension, loudness may also be modulated through its affective dimension. In this talk I will present recent data aiming at a better understanding of loudness, namely its sensory and its affective dimensions, and how these two dimensions can be dissociated within the same stimuli. I will also report the findings of a recent scoping review on the electrophysiological correlates of hyperacusis, in which we found a diversity in terms and definitions used to describe hyperacusis and several identifiable aetiologies, among which developmental disorders, neurological disorders, induced hearing damage, and idiopathic aetiology. Broader consensus around definitions and diagnostic criteria considering aetiologies may guide researchers to ask better questions and clinicians to more efficiently manage the patient who complain about hyperacusis.

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Charlotte	Bigras	Universite de Montreal
Ariane	Forget	Universite de Montreal
Berangere	Villatte	Universite de Montreal

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Signature Sylvie Hébert

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Stressful hearing: Hyperacusis Can Be Induced by Chronic Stressful Noise Exposure or Chronic Pharmacological Stress that Disrupts the HPA Axis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Richard Salvi

Affiliation Center for Hearing & Deafness, SUNY at Buffalo

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract We live in a noisy, stressful environment largely oblivious to its negative hearing-health consequences. One debilitating hearing impairment associated with noise-induced hearing loss is hyperacusis, a loudness intolerance disorder in which everyday sounds are perceived as excessively loud, aversive and stressful. To investigate the biological bases of hyperacusis, we developed a reaction time-intensity (RT-I) paradigm to test for loudness hyperacusis. To assess the aversive quality of hyperacusis, we developed an active sound avoidance paradigm (ASAP) to measure sound avoidance behaviors. To trigger the induction of hyperacusis, we exposed rats to intense high-frequency noise for several months; these conditions were expected to induce chronic noise-stress in addition to high-frequency loss. After this prolonged, stressful noise exposure, rats developed clear signs of sound avoidance hyperacusis as well as loudness hyperacusis with a low-frequency spectral profile. This prolonged, stressful noise exposure disrupted the hypothalamic-pituitary-adrenal (HPA) axis; it did not alter basal corticosterone (CORT) levels, but instead, greatly reduced the rise in corticosterone (CORT) triggered by restraint stress (i.e., it blunted the stress response). This noise exposure also chronically increased the expression of glucocorticoid receptors (GR) in the auditory cortex, part of the negative feedback network that suppressed the continued release of CORT. To determine the role of chronic stress independent of hearing loss, rats were chronically stressed by pharmacologically treating the rats with CORT-stress hormone. After chronic pharmacologic stress, rats developed behavioral evidence of loudness hyperacusis and sound avoidance hyperacusis. Consistent with the previous noise studies, basal CORT levels remained normal; however, rats exhibited a blunted CORT response to restraint stress and GR expression in the auditory cortex increased. Chronic pharmacologic CORT stress did not alter DPOAEs or the cochlear compound action potential; however, sound-evoked responses from chronically implanted electrodes on the auditory cortex were greatly enhanced. These results show for the first time that chronic

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Richard *	Salvi *	Center for Hearing & Deafness, SUNY at Buffalo
Senthilvelan	Manohar	State University of New York at Buffalo
Guang-Di	Chen	Center for Hearing and Deafness, State University of New York at Buffalo

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Signature Richard Salvi

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Behavioral and Physiological Measures of Sound Intolerance

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Sarah Theodoroff

Affiliation Department of Veterans Affairs, NCRAR

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract The experience of individuals who have decreased tolerance to everyday sounds is heterogeneous. No consensus exists regarding what constitutes hyperacusis or noise sensitivity. A direct consequence of this is the lack of a “gold standard” regarding how to diagnose, assess, and treat these conditions. The purpose of this presentation is to address this gap in clinical knowledge.

Noise sensitivity refers to an increased reactivity to everyday sounds and encompasses a range of psychological attributes, often including annoyance or feeling overwhelmed by the sounds in the environment. An estimated 59% of patients with mild traumatic brain injury have noise sensitivity (Shepherd et al, 2019). Research is lacking both on the pathophysiology of noise sensitivity, its clinical treatment, and to what degree it may or may not be a similar phenomenon to hyperacusis. Hyperacusis describes a decreased sound tolerance driven by the perceived loudness of ordinary sounds. It often including physical discomfort or pain when listening to sounds that are at moderate or low intensity levels, which most people would find tolerable. Estimates are as high as 60-79% of tinnitus patients have comorbid hyperacusis (Andersson et al, 2001; Dauman & Bouscan-Faure, 2005).

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intolerance.
Results from this work will elucidate aspects of the underlying pathophysiology of “hyperacusis” and “noise sensitivity.” Ultimately, outcomes from this avenue of research will guide the development of targeted rehabilitative treatments based on the etiology of these conditions and inform which disciplines should be working together to best meet the needs of this patient population (e.g., audiology, psychology, neurology).

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* Presenting Author

First Name	Last Name	Affiliation
Sarah *	Theodoroff *	Department of Veterans Affairs, NCRAR

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Signature Sarah Theodoroff

Hyperacusis: Diversity in Cause, Expression, and Advocacy

A Rodent Model of Acoustic Trauma to Study Neural and Inflammatory Mechanisms of Hyperacusis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Megan Wood

Affiliation Johns Hopkins University School of Medicine

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract The mechanism underlying painful hyperacusis is currently unknown. A subset of hyperacusis patients surveyed recently reported experiencing acoustic trauma before the onset of their symptoms. Therefore, we use acoustic trauma in rodents to induce neurological and inflammatory changes in the cochlea as a model for noise-induced hyperacusis. Acoustic trauma affects many cell types of the inner ear. We focus on the responses of type II auditory nerve fibers and immune cells as these cell types are the putative pain sensing neurons and chief responders to inflammation in the cochlea, respectively. Type II auditory nerve fibers exhibit changes in their calcium dynamics after acoustic trauma. New analysis of an existing dataset will be discussed to describe a sensitization of these fibers as a possible mechanism for hyperacusis. Another possible mechanism for hyperacusis is an enhanced inflammatory response following acoustic trauma. Neuroimmune crosstalk through neuropeptides found in the cochlea, such as CGRP, may play an important role in this possible mechanism. Recent studies following immune cell migration after blocking CGRP receptors during acoustic trauma will be discussed. Finally, the presentation will end with a discussion of the parameters needed to properly model painful hyperacusis in rodents including behavioral assays of pain perception to sound stimulation.

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Viola	Monovich	Peabody Institute of Johns Hopkins University
Anda	Nyati	Johns Hopkins Bloomberg School of Public Health
Paul	Fuchs	Johns Hopkins University School of Medicine
Elisabeth	Glowatzki	Johns Hopkins University School of Medicine

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Signature Lara Beers Wood

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Investigating Hyperacusis in Rodent Models of Autism

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Benjamin Auerbach

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract Animal models are indispensable tools for identifying disease pathophysiology and developing objective biomarkers for hearing disorders like hyperacusis. However, care must be taken when attempting to recapitulate complex hearing disorders in model systems. Hyperacusis encompasses a wide range of reactions to sound and is observed across a broad spectrum of neurological disorders, and this diversity must be accounted for when attempting to measure and induce hyperacusis in animals. Recent progress has been made in developing rodent behavioral models that capture distinct aspects of sound perception disrupted in hyperacusis, with attempts to disentangle psychoacoustic (e.g. excessive loudness) from affective (e.g. decreased sound tolerance) aspects of the disorder. However, drug- and noise-induced hearing loss are still the primary methods used for inducing hyperacusis-like states in animals. Here we will discuss recent attempts to characterize sound tolerance disturbances in genetic models of neurodevelopmental disorders that present with high rates of hyperacusis, such as Fragile X syndrome and autism spectrum disorders (ASD). Comparing and contrasting hyperacusis associated with hearing loss and neurodevelopmental disruption has the potential to uncover convergent (or divergent) pathophysiological mechanisms across distinct forms of hyperacusis , which will help advance our ability to diagnose and treat this often devastating disorder.

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* Presenting Author

First Name	Last Name	Affiliation

Benjamin *	Auerbach *	University of Illinois at Urbana-Champaign

In-Person Participation I intend to participate in the MidWinter Meeting in-person for the entirety of the scheduled meeting.

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Signature Benjamin D Auerbach

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Connecting Researchers to Clinicians and patients: Bryan Pollard's Roadmap to a Cure for Hyperacusis with Pain

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Michael Maholchic

Affiliation Hyperacusis Research, Ltx

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract In memoriam to Bryan Pollard, founder of Hyperacusis Research Ltd., and his impact on the field.

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Michael *	Maholchic *	Hyperacusis Research, Ltx

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Signature Michael Maholchic

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Benjamin Auerbach

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Session Description Hyperacusis is a complex hearing disorder that encompasses a wide-range of reactions to sound, including excessive loudness, increased aversion/fear of sound, or even pain. While often associated with hearing loss and tinnitus, sound tolerance disturbances are observed across a broad spectrum of neurological disorders, including autism, chronic pain and post-traumatic stress disorder. Thus, hyperacusis is diverse in both its etiology and phenotypic expression, and it is imperative to consider this diversity when attempting to elucidate its physiological mechanisms and advance diagnostic and treatments. Bryan Pollard, founder and president of the Hyperacusis Research Ltd., was a champion for this cause. Bryan was a tireless advocate for hyperacusis research, working to connect researchers from diverse backgrounds, often through formal and informal workshops and dinners at the ARO midwinter meeting. He was the first non-researcher to present at ARO and was instrumental in creating a new diagnosis of pain hyperacusis. Bryan unfortunately passed away earlier this year.
This goal of this symposium is two-fold. First, we hope to update the hearing community on the status of the hyperacusis field and the progress that has been made towards diagnosing, treating, and modeling this disorder. Hyperacusis is a rapidly growing research field that intersects with many other aspects of hearing research, including central auditory processing and plasticity, tinnitus, and autism. As such, this symposium would have a broad target audience, from basic auditory neuroscientists interested in the neural mechanisms of perception and experience-dependent plasticity to audiologists seeking to understand and perhaps better treat a host of auditory processing disorders. In addition, we hope to use this symposium to commemorate the impact that Bryan Pollard had on the field, starting with a short introduction and in memoriam to highlight the ways he and the Hyperacusis Research foundation have helped move hyperacusis research forward.

Presenter Diversity The symposium will feature a diverse group of speakers, including several presenters from underrepresented minorities. The symposium will additionally include both up-and-coming young investigators and established leaders in the field, with speakers who employ

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Signature Benjamin D Auerbach

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Imaging hyperacusis: Past and Present

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Fatima Husain

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract Brain imaging of humans with hyperacusis remains one of the primary methods to identify neural networks sub serving this condition and dissociate them from those related to comorbid tinnitus or hearing loss. Despite the challenges associated with cost and noise of some tools, results of such studies have informed the existing experimental and theoretical framework of hyperacusis. In a pioneering fMRI study, Melcher and colleagues (2010) parsed out the contribution of co-occurring hyperacusis to brain imaging findings of tinnitus. In particular, they noted that while both subcortical and cortical auditory areas were responsive in those with hyperacusis (relative to controls), such an elevated response was only noted in the auditory cortex for those with tinnitus. Two recent studies (Koops and van Dijk, 2021; Hofmeier et al., 2021) further explored this dissociation. Koops and van Dijk confirmed the higher response in cortical and subcortical centers of auditory processing in response to external sounds but in addition found that there was reduced response to the frequencies of the internally-generated tinnitus sound. The Hofmeier study provided corroboration of the increased responsiveness of the tinnitus and hyperacusis group to external sounds in the cortical and subcortical auditory areas. Additionally they noted increased wave III amplitude in brain-stem response of the tinnitus and hyperacusis group relative to the control group; the tinnitus only group exhibited a prolonged and reduced wave V amplitude compared to the controls. These studies support the idea of central gain in the condition of hyperacusis but not necessarily if tinnitus occurs alone. They further point to the role played by attention in tinnitus, as noted in task-based fMRI studies in my own lab (Husain et al., 2011; 2015). What is not easily known is the contribution and interaction of clinically-significant hearing loss with hyperacusis. Koops and van Dijk noted the elevation of response at frequencies in the hearing loss as well as normal hearing range in those with hyperacusis. The Melcher studies were primarily in those with normal hearing. In an ongoing study, we are collecting both auditory brainstem response and fMRI data on young adults with hyperacusis and normal hearing, results of which will be reported at the meeting. In summary, current and future non-invasive brain imaging

studies continue to expand our understanding of pathophysiology of hyperacusis and eventually test therapies that help patients.

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Fatima *	Husain *	University of Illinois at Urbana-Champaign

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Signature Fatima T. Husain

Hyperacusis: Diversity in Cause, Expression, and Advocacy

A Need to Better Define and Characterize Loudness and Hyperacusis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Sylvie Hebert

Affiliation Universite de Montreal

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract Hyperacusis is a hearing disorder wherein everyday sounds are considered too loud, fearful, annoying, or painful. In its most accepted definition, hyperacusis designates sounds of moderate intensities that are judged louder than normal perception. A deeper understanding of what loudness is would contribute to a better definition of what hyperacusis is, and how it can be defined and diagnosed. Indeed, although loudness involves the perception of the intensity of sounds, i.e. its sensory dimension, loudness may also be modulated through its affective dimension. In this talk I will present recent data aiming at a better understanding of loudness, namely its sensory and its affective dimensions, and how these two dimensions can be dissociated within the same stimuli. I will also report the findings of a recent scoping review on the electrophysiological correlates of hyperacusis, in which we found a diversity in terms and definitions used to describe hyperacusis and several identifiable aetiologies, among which developmental disorders, neurological disorders, induced hearing damage, and idiopathic aetiology. Broader consensus around definitions and diagnostic criteria considering aetiologies may guide researchers to ask better questions and clinicians to more efficiently manage the patient who complain about hyperacusis.

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Sylvie *	Hebert *	Universite de Montreal

Charlotte	Bigras	Universite de Montreal
Ariane	Forget	Universite de Montreal
Berangere	Villatte	Universite de Montreal

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Signature Sylvie Hébert

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Stressful hearing: Hyperacusis Can Be Induced by Chronic Stressful Noise Exposure or Chronic Pharmacological Stress that Disrupts the HPA Axis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Richard Salvi

Affiliation Center for Hearing & Deafness, SUNY at Buffalo

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract We live in a noisy, stressful environment largely oblivious to its negative hearing-health consequences. One debilitating hearing impairment associated with noise-induced hearing loss is hyperacusis, a loudness intolerance disorder in which everyday sounds are perceived as excessively loud, aversive and stressful. To investigate the biological bases of hyperacusis, we developed a reaction time-intensity (RT-I) paradigm to test for loudness hyperacusis. To assess the aversive quality of hyperacusis, we developed an active sound avoidance paradigm (ASAP) to measure sound avoidance behaviors. To trigger the induction of hyperacusis, we exposed rats to intense high-frequency noise for several months; these conditions were expected to induce chronic noise-stress in addition to high-frequency loss. After this prolonged, stressful noise exposure, rats developed clear signs of sound avoidance hyperacusis as well as loudness hyperacusis with a low-frequency spectral profile. This prolonged, stressful noise exposure disrupted the hypothalamic-pituitary-adrenal (HPA) axis; it did not alter basal corticosterone (CORT) levels, but instead, greatly reduced the rise in corticosterone (CORT) triggered by restraint stress (i.e., it blunted the stress response). This noise exposure also chronically increased the expression of glucocorticoid receptors (GR) in the auditory cortex, part of the negative feedback network that suppressed the continued release of CORT. To determine the role of chronic stress independent of hearing loss, rats were chronically stressed by pharmacologically treating the rats with CORT-stress hormone. After chronic pharmacologic stress, rats developed behavioral evidence of loudness hyperacusis and sound avoidance hyperacusis. Consistent with the previous noise studies, basal CORT levels remained normal; however, rats exhibited a blunted CORT response to restraint stress and GR expression in the auditory cortex increased. Chronic pharmacologic CORT stress did not alter DPOAEs or the cochlear compound action potential; however, sound-evoked responses from chronically implanted electrodes on the auditory cortex were greatly enhanced. These results show for the first time that chronic

pharmacologic stress, in the absence of hearing loss, is sufficient to induce behavioral evidence of hyperacusis, which is associated with auditory cortex neural hyperactivity and enhanced central gain.

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Richard *	Salvi *	Center for Hearing & Deafness, SUNY at Buffalo
Senthilvelan	Manohar	State University of New York at Buffalo
Guang-Di	Chen	Center for Hearing and Deafness, State University of New York at Buffalo

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Signature Richard Salvi

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Behavioral and Physiological Measures of Sound Intolerance

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Sarah Theodoroff

Affiliation Department of Veterans Affairs, NCRAR

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract The experience of individuals who have decreased tolerance to everyday sounds is heterogeneous. No consensus exists regarding what constitutes hyperacusis or noise sensitivity. A direct consequence of this is the lack of a “gold standard” regarding how to diagnose, assess, and treat these conditions. The purpose of this presentation is to address this gap in clinical knowledge.

Noise sensitivity refers to an increased reactivity to everyday sounds and encompasses a range of psychological attributes, often including annoyance or feeling overwhelmed by the sounds in the environment. An estimated 59% of patients with mild traumatic brain injury have noise sensitivity (Shepherd et al, 2019). Research is lacking both on the pathophysiology of noise sensitivity, its clinical treatment, and to what degree it may or may not be a similar phenomenon to hyperacusis. Hyperacusis describes a decreased sound tolerance driven by the perceived loudness of ordinary sounds. It often including physical discomfort or pain when listening to sounds that are at moderate or low intensity levels, which most people would find tolerable. Estimates are as high as 60-79% of tinnitus patients have comorbid hyperacusis (Andersson et al, 2001; Dauman & Bouscan-Faure, 2005).

When Veterans seek medical attention because ordinary sounds are painfully loud or there’s “too much noise to function” many clinicians are uncertain how best to meet their patients’ needs. This results in the focus being shifted to comorbid conditions hoping that the “hyperacusis” is a symptom of another condition. Research is needed to develop evidence-based tools that are capable of differentiating noise sensitivity from hyperacusis and from other health conditions that present with similar symptoms. This need motivated the ideas behind an on-going research project that is examining the relationship between auditory and psychological biomarkers to determine how well auditory and psychophysiological data predict self-reported decreased tolerance to everyday sounds. Behavioral and physiological measures are collected in order to detect where deficits exist in sensory and/or neurological structures associated with complaints of sound

intolerance.
Results from this work will elucidate aspects of the underlying pathophysiology of “hyperacusis” and “noise sensitivity.” Ultimately, outcomes from this avenue of research will guide the development of targeted rehabilitative treatments based on the etiology of these conditions and inform which disciplines should be working together to best meet the needs of this patient population (e.g., audiology, psychology, neurology).

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First Name	Last Name	Affiliation
Sarah *	Theodoroff *	Department of Veterans Affairs, NCRAR

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Signature Sarah Theodoroff

Hyperacusis: Diversity in Cause, Expression, and Advocacy

A Rodent Model of Acoustic Trauma to Study Neural and Inflammatory Mechanisms of Hyperacusis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Megan Wood

Affiliation Johns Hopkins University School of Medicine

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract The mechanism underlying painful hyperacusis is currently unknown. A subset of hyperacusis patients surveyed recently reported experiencing acoustic trauma before the onset of their symptoms. Therefore, we use acoustic trauma in rodents to induce neurological and inflammatory changes in the cochlea as a model for noise-induced hyperacusis. Acoustic trauma affects many cell types of the inner ear. We focus on the responses of type II auditory nerve fibers and immune cells as these cell types are the putative pain sensing neurons and chief responders to inflammation in the cochlea, respectively. Type II auditory nerve fibers exhibit changes in their calcium dynamics after acoustic trauma. New analysis of an existing dataset will be discussed to describe a sensitization of these fibers as a possible mechanism for hyperacusis. Another possible mechanism for hyperacusis is an enhanced inflammatory response following acoustic trauma. Neuroimmune crosstalk through neuropeptides found in the cochlea, such as CGRP, may play an important role in this possible mechanism. Recent studies following immune cell migration after blocking CGRP receptors during acoustic trauma will be discussed. Finally, the presentation will end with a discussion of the parameters needed to properly model painful hyperacusis in rodents including behavioral assays of pain perception to sound stimulation.

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Megan *	Wood *	Johns Hopkins University School of Medicine
Viola	Monovich	Peabody Institute of Johns Hopkins University
Anda	Nyati	Johns Hopkins Bloomberg School of Public Health
Paul	Fuchs	Johns Hopkins University School of Medicine
Elisabeth	Glowatzki	Johns Hopkins University School of Medicine

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Signature Lara Beers Wood

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Investigating Hyperacusis in Rodent Models of Autism

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Benjamin Auerbach

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

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Benjamin *	Auerbach *	University of Illinois at Urbana-Champaign

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Signature Benjamin D Auerbach

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Connecting Researchers to Clinicians and patients: Bryan Pollard's Roadmap to a Cure for Hyperacusis with Pain

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Michael Maholchic

Affiliation Hyperacusis Research, Ltx

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

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Michael *	Maholchic *	Hyperacusis Research, Ltx

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Signature Michael Maholchic

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Benjamin Auerbach

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

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Presenter Diversity The symposium will feature a diverse group of speakers, including several presenters from underrepresented minorities. The symposium will additionally include both up-and-coming young investigators and established leaders in the field, with speakers who employ

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Signature Benjamin D Auerbach

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Imaging hyperacusis: Past and Present

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Fatima Husain

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

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Signature Fatima T. Husain

Hyperacusis: Diversity in Cause, Expression, and Advocacy

A Need to Better Define and Characterize Loudness and Hyperacusis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Sylvie Hebert

Affiliation Universite de Montreal

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract Hyperacusis is a hearing disorder wherein everyday sounds are considered too loud, fearful, annoying, or painful. In its most accepted definition, hyperacusis designates sounds of moderate intensities that are judged louder than normal perception. A deeper understanding of what loudness is would contribute to a better definition of what hyperacusis is, and how it can be defined and diagnosed. Indeed, although loudness involves the perception of the intensity of sounds, i.e. its sensory dimension, loudness may also be modulated through its affective dimension. In this talk I will present recent data aiming at a better understanding of loudness, namely its sensory and its affective dimensions, and how these two dimensions can be dissociated within the same stimuli. I will also report the findings of a recent scoping review on the electrophysiological correlates of hyperacusis, in which we found a diversity in terms and definitions used to describe hyperacusis and several identifiable aetiologies, among which developmental disorders, neurological disorders, induced hearing damage, and idiopathic aetiology. Broader consensus around definitions and diagnostic criteria considering aetiologies may guide researchers to ask better questions and clinicians to more efficiently manage the patient who complain about hyperacusis.

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* Presenting Author

First Name	Last Name	Affiliation
Sylvie *	Hebert *	Universite de Montreal

Charlotte	Bigras	Universite de Montreal
Ariane	Forget	Universite de Montreal
Berangere	Villatte	Universite de Montreal

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Signature Sylvie Hébert

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Stressful hearing: Hyperacusis Can Be Induced by Chronic Stressful Noise Exposure or Chronic Pharmacological Stress that Disrupts the HPA Axis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Richard Salvi

Affiliation Center for Hearing & Deafness, SUNY at Buffalo

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract We live in a noisy, stressful environment largely oblivious to its negative hearing-health consequences. One debilitating hearing impairment associated with noise-induced hearing loss is hyperacusis, a loudness intolerance disorder in which everyday sounds are perceived as excessively loud, aversive and stressful. To investigate the biological bases of hyperacusis, we developed a reaction time-intensity (RT-I) paradigm to test for loudness hyperacusis. To assess the aversive quality of hyperacusis, we developed an active sound avoidance paradigm (ASAP) to measure sound avoidance behaviors. To trigger the induction of hyperacusis, we exposed rats to intense high-frequency noise for several months; these conditions were expected to induce chronic noise-stress in addition to high-frequency loss. After this prolonged, stressful noise exposure, rats developed clear signs of sound avoidance hyperacusis as well as loudness hyperacusis with a low-frequency spectral profile. This prolonged, stressful noise exposure disrupted the hypothalamic-pituitary-adrenal (HPA) axis; it did not alter basal corticosterone (CORT) levels, but instead, greatly reduced the rise in corticosterone (CORT) triggered by restraint stress (i.e., it blunted the stress response). This noise exposure also chronically increased the expression of glucocorticoid receptors (GR) in the auditory cortex, part of the negative feedback network that suppressed the continued release of CORT. To determine the role of chronic stress independent of hearing loss, rats were chronically stressed by pharmacologically treating the rats with CORT-stress hormone. After chronic pharmacologic stress, rats developed behavioral evidence of loudness hyperacusis and sound avoidance hyperacusis. Consistent with the previous noise studies, basal CORT levels remained normal; however, rats exhibited a blunted CORT response to restraint stress and GR expression in the auditory cortex increased. Chronic pharmacologic CORT stress did not alter DPOAEs or the cochlear compound action potential; however, sound-evoked responses from chronically implanted electrodes on the auditory cortex were greatly enhanced. These results show for the first time that chronic

pharmacologic stress, in the absence of hearing loss, is sufficient to induce behavioral evidence of hyperacusis, which is associated with auditory cortex neural hyperactivity and enhanced central gain.

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Richard *	Salvi *	Center for Hearing & Deafness, SUNY at Buffalo
Senthilvelan	Manohar	State University of New York at Buffalo
Guang-Di	Chen	Center for Hearing and Deafness, State University of New York at Buffalo

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Signature Richard Salvi

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Behavioral and Physiological Measures of Sound Intolerance

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Sarah Theodoroff

Affiliation Department of Veterans Affairs, NCRAR

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract The experience of individuals who have decreased tolerance to everyday sounds is heterogeneous. No consensus exists regarding what constitutes hyperacusis or noise sensitivity. A direct consequence of this is the lack of a “gold standard” regarding how to diagnose, assess, and treat these conditions. The purpose of this presentation is to address this gap in clinical knowledge.

Noise sensitivity refers to an increased reactivity to everyday sounds and encompasses a range of psychological attributes, often including annoyance or feeling overwhelmed by the sounds in the environment. An estimated 59% of patients with mild traumatic brain injury have noise sensitivity (Shepherd et al, 2019). Research is lacking both on the pathophysiology of noise sensitivity, its clinical treatment, and to what degree it may or may not be a similar phenomenon to hyperacusis. Hyperacusis describes a decreased sound tolerance driven by the perceived loudness of ordinary sounds. It often including physical discomfort or pain when listening to sounds that are at moderate or low intensity levels, which most people would find tolerable. Estimates are as high as 60-79% of tinnitus patients have comorbid hyperacusis (Andersson et al, 2001; Dauman & Bouscan-Faure, 2005).

When Veterans seek medical attention because ordinary sounds are painfully loud or there’s “too much noise to function” many clinicians are uncertain how best to meet their patients’ needs. This results in the focus being shifted to comorbid conditions hoping that the “hyperacusis” is a symptom of another condition. Research is needed to develop evidence-based tools that are capable of differentiating noise sensitivity from hyperacusis and from other health conditions that present with similar symptoms. This need motivated the ideas behind an on-going research project that is examining the relationship between auditory and psychological biomarkers to determine how well auditory and psychophysiological data predict self-reported decreased tolerance to everyday sounds. Behavioral and physiological measures are collected in order to detect where deficits exist in sensory and/or neurological structures associated with complaints of sound

intolerance.
Results from this work will elucidate aspects of the underlying pathophysiology of “hyperacusis” and “noise sensitivity.” Ultimately, outcomes from this avenue of research will guide the development of targeted rehabilitative treatments based on the etiology of these conditions and inform which disciplines should be working together to best meet the needs of this patient population (e.g., audiology, psychology, neurology).

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Sarah *	Theodoroff *	Department of Veterans Affairs, NCRAR

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Signature Sarah Theodoroff

Hyperacusis: Diversity in Cause, Expression, and Advocacy

A Rodent Model of Acoustic Trauma to Study Neural and Inflammatory Mechanisms of Hyperacusis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Megan Wood

Affiliation Johns Hopkins University School of Medicine

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract The mechanism underlying painful hyperacusis is currently unknown. A subset of hyperacusis patients surveyed recently reported experiencing acoustic trauma before the onset of their symptoms. Therefore, we use acoustic trauma in rodents to induce neurological and inflammatory changes in the cochlea as a model for noise-induced hyperacusis. Acoustic trauma affects many cell types of the inner ear. We focus on the responses of type II auditory nerve fibers and immune cells as these cell types are the putative pain sensing neurons and chief responders to inflammation in the cochlea, respectively. Type II auditory nerve fibers exhibit changes in their calcium dynamics after acoustic trauma. New analysis of an existing dataset will be discussed to describe a sensitization of these fibers as a possible mechanism for hyperacusis. Another possible mechanism for hyperacusis is an enhanced inflammatory response following acoustic trauma. Neuroimmune crosstalk through neuropeptides found in the cochlea, such as CGRP, may play an important role in this possible mechanism. Recent studies following immune cell migration after blocking CGRP receptors during acoustic trauma will be discussed. Finally, the presentation will end with a discussion of the parameters needed to properly model painful hyperacusis in rodents including behavioral assays of pain perception to sound stimulation.

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Megan *	Wood *	Johns Hopkins University School of Medicine
Viola	Monovich	Peabody Institute of Johns Hopkins University
Anda	Nyati	Johns Hopkins Bloomberg School of Public Health
Paul	Fuchs	Johns Hopkins University School of Medicine
Elisabeth	Glowatzki	Johns Hopkins University School of Medicine

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Signature Lara Beers Wood

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Investigating Hyperacusis in Rodent Models of Autism

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Benjamin Auerbach

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract Animal models are indispensable tools for identifying disease pathophysiology and developing objective biomarkers for hearing disorders like hyperacusis. However, care must be taken when attempting to recapitulate complex hearing disorders in model systems. Hyperacusis encompasses a wide range of reactions to sound and is observed across a broad spectrum of neurological disorders, and this diversity must be accounted for when attempting to measure and induce hyperacusis in animals. Recent progress has been made in developing rodent behavioral models that capture distinct aspects of sound perception disrupted in hyperacusis, with attempts to disentangle psychoacoustic (e.g. excessive loudness) from affective (e.g. decreased sound tolerance) aspects of the disorder. However, drug- and noise-induced hearing loss are still the primary methods used for inducing hyperacusis-like states in animals. Here we will discuss recent attempts to characterize sound tolerance disturbances in genetic models of neurodevelopmental disorders that present with high rates of hyperacusis, such as Fragile X syndrome and autism spectrum disorders (ASD). Comparing and contrasting hyperacusis associated with hearing loss and neurodevelopmental disruption has the potential to uncover convergent (or divergent) pathophysiological mechanisms across distinct forms of hyperacusis , which will help advance our ability to diagnose and treat this often devastating disorder.

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First Name	Last Name	Affiliation

Benjamin *	Auerbach *	University of Illinois at Urbana-Champaign

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Signature Benjamin D Auerbach

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Connecting Researchers to Clinicians and patients: Bryan Pollard's Roadmap to a Cure for Hyperacusis with Pain

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Michael Maholchic

Affiliation Hyperacusis Research, Ltx

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract In memoriam to Bryan Pollard, founder of Hyperacusis Research Ltd., and his impact on the field.

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Michael *	Maholchic *	Hyperacusis Research, Ltx

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Signature Michael Maholchic

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Benjamin Auerbach

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Session Description Hyperacusis is a complex hearing disorder that encompasses a wide-range of reactions to sound, including excessive loudness, increased aversion/fear of sound, or even pain. While often associated with hearing loss and tinnitus, sound tolerance disturbances are observed across a broad spectrum of neurological disorders, including autism, chronic pain and post-traumatic stress disorder. Thus, hyperacusis is diverse in both its etiology and phenotypic expression, and it is imperative to consider this diversity when attempting to elucidate its physiological mechanisms and advance diagnostic and treatments. Bryan Pollard, founder and president of the Hyperacusis Research Ltd., was a champion for this cause. Bryan was a tireless advocate for hyperacusis research, working to connect researchers from diverse backgrounds, often through formal and informal workshops and dinners at the ARO midwinter meeting. He was the first non-researcher to present at ARO and was instrumental in creating a new diagnosis of pain hyperacusis. Bryan unfortunately passed away earlier this year.
This goal of this symposium is two-fold. First, we hope to update the hearing community on the status of the hyperacusis field and the progress that has been made towards diagnosing, treating, and modeling this disorder. Hyperacusis is a rapidly growing research field that intersects with many other aspects of hearing research, including central auditory processing and plasticity, tinnitus, and autism. As such, this symposium would have a broad target audience, from basic auditory neuroscientists interested in the neural mechanisms of perception and experience-dependent plasticity to audiologists seeking to understand and perhaps better treat a host of auditory processing disorders. In addition, we hope to use this symposium to commemorate the impact that Bryan Pollard had on the field, starting with a short introduction and in memoriam to highlight the ways he and the Hyperacusis Research foundation have helped move hyperacusis research forward.

Presenter Diversity The symposium will feature a diverse group of speakers, including several presenters from underrepresented minorities. The symposium will additionally include both up-and-coming young investigators and established leaders in the field, with speakers who employ

a variety of techniques and approaches, from animal models to human clinical research.

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Signature Benjamin D Auerbach

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Imaging hyperacusis: Past and Present

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Fatima Husain

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract Brain imaging of humans with hyperacusis remains one of the primary methods to identify neural networks sub serving this condition and dissociate them from those related to comorbid tinnitus or hearing loss. Despite the challenges associated with cost and noise of some tools, results of such studies have informed the existing experimental and theoretical framework of hyperacusis. In a pioneering fMRI study, Melcher and colleagues (2010) parsed out the contribution of co-occurring hyperacusis to brain imaging findings of tinnitus. In particular, they noted that while both subcortical and cortical auditory areas were responsive in those with hyperacusis (relative to controls), such an elevated response was only noted in the auditory cortex for those with tinnitus. Two recent studies (Koops and van Dijk, 2021; Hofmeier et al., 2021) further explored this dissociation. Koops and van Dijk confirmed the higher response in cortical and subcortical centers of auditory processing in response to external sounds but in addition found that there was reduced response to the frequencies of the internally-generated tinnitus sound. The Hofmeier study provided corroboration of the increased responsiveness of the tinnitus and hyperacusis group to external sounds in the cortical and subcortical auditory areas. Additionally they noted increased wave III amplitude in brain-stem response of the tinnitus and hyperacusis group relative to the control group; the tinnitus only group exhibited a prolonged and reduced wave V amplitude compared to the controls. These studies support the idea of central gain in the condition of hyperacusis but not necessarily if tinnitus occurs alone. They further point to the role played by attention in tinnitus, as noted in task-based fMRI studies in my own lab (Husain et al., 2011; 2015). What is not easily known is the contribution and interaction of clinically-significant hearing loss with hyperacusis. Koops and van Dijk noted the elevation of response at frequencies in the hearing loss as well as normal hearing range in those with hyperacusis. The Melcher studies were primarily in those with normal hearing. In an ongoing study, we are collecting both auditory brainstem response and fMRI data on young adults with hyperacusis and normal hearing, results of which will be reported at the meeting. In summary, current and future non-invasive brain imaging

studies continue to expand our understanding of pathophysiology of hyperacusis and eventually test therapies that help patients.

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Fatima *	Husain *	University of Illinois at Urbana-Champaign

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Signature Fatima T. Husain

Hyperacusis: Diversity in Cause, Expression, and Advocacy

A Need to Better Define and Characterize Loudness and Hyperacusis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Sylvie Hebert

Affiliation Universite de Montreal

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

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Signature Sylvie Hébert

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Stressful hearing: Hyperacusis Can Be Induced by Chronic Stressful Noise Exposure or Chronic Pharmacological Stress that Disrupts the HPA Axis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Richard Salvi

Affiliation Center for Hearing & Deafness, SUNY at Buffalo

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract We live in a noisy, stressful environment largely oblivious to its negative hearing-health consequences. One debilitating hearing impairment associated with noise-induced hearing loss is hyperacusis, a loudness intolerance disorder in which everyday sounds are perceived as excessively loud, aversive and stressful. To investigate the biological bases of hyperacusis, we developed a reaction time-intensity (RT-I) paradigm to test for loudness hyperacusis. To assess the aversive quality of hyperacusis, we developed an active sound avoidance paradigm (ASAP) to measure sound avoidance behaviors. To trigger the induction of hyperacusis, we exposed rats to intense high-frequency noise for several months; these conditions were expected to induce chronic noise-stress in addition to high-frequency loss. After this prolonged, stressful noise exposure, rats developed clear signs of sound avoidance hyperacusis as well as loudness hyperacusis with a low-frequency spectral profile. This prolonged, stressful noise exposure disrupted the hypothalamic-pituitary-adrenal (HPA) axis; it did not alter basal corticosterone (CORT) levels, but instead, greatly reduced the rise in corticosterone (CORT) triggered by restraint stress (i.e., it blunted the stress response). This noise exposure also chronically increased the expression of glucocorticoid receptors (GR) in the auditory cortex, part of the negative feedback network that suppressed the continued release of CORT. To determine the role of chronic stress independent of hearing loss, rats were chronically stressed by pharmacologically treating the rats with CORT-stress hormone. After chronic pharmacologic stress, rats developed behavioral evidence of loudness hyperacusis and sound avoidance hyperacusis. Consistent with the previous noise studies, basal CORT levels remained normal; however, rats exhibited a blunted CORT response to restraint stress and GR expression in the auditory cortex increased. Chronic pharmacologic CORT stress did not alter DPOAEs or the cochlear compound action potential; however, sound-evoked responses from chronically implanted electrodes on the auditory cortex were greatly enhanced. These results show for the first time that chronic

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Senthilvelan	Manohar	State University of New York at Buffalo
Guang-Di	Chen	Center for Hearing and Deafness, State University of New York at Buffalo

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Signature Richard Salvi

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Behavioral and Physiological Measures of Sound Intolerance

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Sarah Theodoroff

Affiliation Department of Veterans Affairs, NCRAR

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract The experience of individuals who have decreased tolerance to everyday sounds is heterogeneous. No consensus exists regarding what constitutes hyperacusis or noise sensitivity. A direct consequence of this is the lack of a “gold standard” regarding how to diagnose, assess, and treat these conditions. The purpose of this presentation is to address this gap in clinical knowledge.

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Sarah *	Theodoroff *	Department of Veterans Affairs, NCRAR

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Signature Sarah Theodoroff

Hyperacusis: Diversity in Cause, Expression, and Advocacy

A Rodent Model of Acoustic Trauma to Study Neural and Inflammatory Mechanisms of Hyperacusis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Megan Wood

Affiliation Johns Hopkins University School of Medicine

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract The mechanism underlying painful hyperacusis is currently unknown. A subset of hyperacusis patients surveyed recently reported experiencing acoustic trauma before the onset of their symptoms. Therefore, we use acoustic trauma in rodents to induce neurological and inflammatory changes in the cochlea as a model for noise-induced hyperacusis. Acoustic trauma affects many cell types of the inner ear. We focus on the responses of type II auditory nerve fibers and immune cells as these cell types are the putative pain sensing neurons and chief responders to inflammation in the cochlea, respectively. Type II auditory nerve fibers exhibit changes in their calcium dynamics after acoustic trauma. New analysis of an existing dataset will be discussed to describe a sensitization of these fibers as a possible mechanism for hyperacusis. Another possible mechanism for hyperacusis is an enhanced inflammatory response following acoustic trauma. Neuroimmune crosstalk through neuropeptides found in the cochlea, such as CGRP, may play an important role in this possible mechanism. Recent studies following immune cell migration after blocking CGRP receptors during acoustic trauma will be discussed. Finally, the presentation will end with a discussion of the parameters needed to properly model painful hyperacusis in rodents including behavioral assays of pain perception to sound stimulation.

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Viola	Monovich	Peabody Institute of Johns Hopkins University
Anda	Nyati	Johns Hopkins Bloomberg School of Public Health
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Elisabeth	Glowatzki	Johns Hopkins University School of Medicine

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Signature Lara Beers Wood

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Investigating Hyperacusis in Rodent Models of Autism

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Benjamin Auerbach

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract Animal models are indispensable tools for identifying disease pathophysiology and developing objective biomarkers for hearing disorders like hyperacusis. However, care must be taken when attempting to recapitulate complex hearing disorders in model systems. Hyperacusis encompasses a wide range of reactions to sound and is observed across a broad spectrum of neurological disorders, and this diversity must be accounted for when attempting to measure and induce hyperacusis in animals. Recent progress has been made in developing rodent behavioral models that capture distinct aspects of sound perception disrupted in hyperacusis, with attempts to disentangle psychoacoustic (e.g. excessive loudness) from affective (e.g. decreased sound tolerance) aspects of the disorder. However, drug- and noise-induced hearing loss are still the primary methods used for inducing hyperacusis-like states in animals. Here we will discuss recent attempts to characterize sound tolerance disturbances in genetic models of neurodevelopmental disorders that present with high rates of hyperacusis, such as Fragile X syndrome and autism spectrum disorders (ASD). Comparing and contrasting hyperacusis associated with hearing loss and neurodevelopmental disruption has the potential to uncover convergent (or divergent) pathophysiological mechanisms across distinct forms of hyperacusis , which will help advance our ability to diagnose and treat this often devastating disorder.

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Benjamin *	Auerbach *	University of Illinois at Urbana-Champaign

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Signature Benjamin D Auerbach

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Connecting Researchers to Clinicians and patients: Bryan Pollard's Roadmap to a Cure for Hyperacusis with Pain

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Michael Maholchic

Affiliation Hyperacusis Research, Ltx

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract In memoriam to Bryan Pollard, founder of Hyperacusis Research Ltd., and his impact on the field.

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Michael *	Maholchic *	Hyperacusis Research, Ltx

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Signature Michael Maholchic

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Benjamin Auerbach

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Session Description Hyperacusis is a complex hearing disorder that encompasses a wide-range of reactions to sound, including excessive loudness, increased aversion/fear of sound, or even pain. While often associated with hearing loss and tinnitus, sound tolerance disturbances are observed across a broad spectrum of neurological disorders, including autism, chronic pain and post-traumatic stress disorder. Thus, hyperacusis is diverse in both its etiology and phenotypic expression, and it is imperative to consider this diversity when attempting to elucidate its physiological mechanisms and advance diagnostic and treatments. Bryan Pollard, founder and president of the Hyperacusis Research Ltd., was a champion for this cause. Bryan was a tireless advocate for hyperacusis research, working to connect researchers from diverse backgrounds, often through formal and informal workshops and dinners at the ARO midwinter meeting. He was the first non-researcher to present at ARO and was instrumental in creating a new diagnosis of pain hyperacusis. Bryan unfortunately passed away earlier this year.
This goal of this symposium is two-fold. First, we hope to update the hearing community on the status of the hyperacusis field and the progress that has been made towards diagnosing, treating, and modeling this disorder. Hyperacusis is a rapidly growing research field that intersects with many other aspects of hearing research, including central auditory processing and plasticity, tinnitus, and autism. As such, this symposium would have a broad target audience, from basic auditory neuroscientists interested in the neural mechanisms of perception and experience-dependent plasticity to audiologists seeking to understand and perhaps better treat a host of auditory processing disorders. In addition, we hope to use this symposium to commemorate the impact that Bryan Pollard had on the field, starting with a short introduction and in memoriam to highlight the ways he and the Hyperacusis Research foundation have helped move hyperacusis research forward.

Presenter Diversity The symposium will feature a diverse group of speakers, including several presenters from underrepresented minorities. The symposium will additionally include both up-and-coming young investigators and established leaders in the field, with speakers who employ

a variety of techniques and approaches, from animal models to human clinical research.

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Signature Benjamin D Auerbach

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Imaging hyperacusis: Past and Present

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Fatima Husain

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract Brain imaging of humans with hyperacusis remains one of the primary methods to identify neural networks sub serving this condition and dissociate them from those related to comorbid tinnitus or hearing loss. Despite the challenges associated with cost and noise of some tools, results of such studies have informed the existing experimental and theoretical framework of hyperacusis. In a pioneering fMRI study, Melcher and colleagues (2010) parsed out the contribution of co-occurring hyperacusis to brain imaging findings of tinnitus. In particular, they noted that while both subcortical and cortical auditory areas were responsive in those with hyperacusis (relative to controls), such an elevated response was only noted in the auditory cortex for those with tinnitus. Two recent studies (Koops and van Dijk, 2021; Hofmeier et al., 2021) further explored this dissociation. Koops and van Dijk confirmed the higher response in cortical and subcortical centers of auditory processing in response to external sounds but in addition found that there was reduced response to the frequencies of the internally-generated tinnitus sound. The Hofmeier study provided corroboration of the increased responsiveness of the tinnitus and hyperacusis group to external sounds in the cortical and subcortical auditory areas. Additionally they noted increased wave III amplitude in brain-stem response of the tinnitus and hyperacusis group relative to the control group; the tinnitus only group exhibited a prolonged and reduced wave V amplitude compared to the controls. These studies support the idea of central gain in the condition of hyperacusis but not necessarily if tinnitus occurs alone. They further point to the role played by attention in tinnitus, as noted in task-based fMRI studies in my own lab (Husain et al., 2011; 2015). What is not easily known is the contribution and interaction of clinically-significant hearing loss with hyperacusis. Koops and van Dijk noted the elevation of response at frequencies in the hearing loss as well as normal hearing range in those with hyperacusis. The Melcher studies were primarily in those with normal hearing. In an ongoing study, we are collecting both auditory brainstem response and fMRI data on young adults with hyperacusis and normal hearing, results of which will be reported at the meeting. In summary, current and future non-invasive brain imaging

studies continue to expand our understanding of pathophysiology of hyperacusis and eventually test therapies that help patients.

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Fatima *	Husain *	University of Illinois at Urbana-Champaign

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Signature Fatima T. Husain

Hyperacusis: Diversity in Cause, Expression, and Advocacy

A Need to Better Define and Characterize Loudness and Hyperacusis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Sylvie Hebert

Affiliation Universite de Montreal

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract Hyperacusis is a hearing disorder wherein everyday sounds are considered too loud, fearful, annoying, or painful. In its most accepted definition, hyperacusis designates sounds of moderate intensities that are judged louder than normal perception. A deeper understanding of what loudness is would contribute to a better definition of what hyperacusis is, and how it can be defined and diagnosed. Indeed, although loudness involves the perception of the intensity of sounds, i.e. its sensory dimension, loudness may also be modulated through its affective dimension. In this talk I will present recent data aiming at a better understanding of loudness, namely its sensory and its affective dimensions, and how these two dimensions can be dissociated within the same stimuli. I will also report the findings of a recent scoping review on the electrophysiological correlates of hyperacusis, in which we found a diversity in terms and definitions used to describe hyperacusis and several identifiable aetiologies, among which developmental disorders, neurological disorders, induced hearing damage, and idiopathic aetiology. Broader consensus around definitions and diagnostic criteria considering aetiologies may guide researchers to ask better questions and clinicians to more efficiently manage the patient who complain about hyperacusis.

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Charlotte	Bigras	Universite de Montreal
Ariane	Forget	Universite de Montreal
Berangere	Villatte	Universite de Montreal

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Signature Sylvie Hébert

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Stressful hearing: Hyperacusis Can Be Induced by Chronic Stressful Noise Exposure or Chronic Pharmacological Stress that Disrupts the HPA Axis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Richard Salvi

Affiliation Center for Hearing & Deafness, SUNY at Buffalo

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract We live in a noisy, stressful environment largely oblivious to its negative hearing-health consequences. One debilitating hearing impairment associated with noise-induced hearing loss is hyperacusis, a loudness intolerance disorder in which everyday sounds are perceived as excessively loud, aversive and stressful. To investigate the biological bases of hyperacusis, we developed a reaction time-intensity (RT-I) paradigm to test for loudness hyperacusis. To assess the aversive quality of hyperacusis, we developed an active sound avoidance paradigm (ASAP) to measure sound avoidance behaviors. To trigger the induction of hyperacusis, we exposed rats to intense high-frequency noise for several months; these conditions were expected to induce chronic noise-stress in addition to high-frequency loss. After this prolonged, stressful noise exposure, rats developed clear signs of sound avoidance hyperacusis as well as loudness hyperacusis with a low-frequency spectral profile. This prolonged, stressful noise exposure disrupted the hypothalamic-pituitary-adrenal (HPA) axis; it did not alter basal corticosterone (CORT) levels, but instead, greatly reduced the rise in corticosterone (CORT) triggered by restraint stress (i.e., it blunted the stress response). This noise exposure also chronically increased the expression of glucocorticoid receptors (GR) in the auditory cortex, part of the negative feedback network that suppressed the continued release of CORT. To determine the role of chronic stress independent of hearing loss, rats were chronically stressed by pharmacologically treating the rats with CORT-stress hormone. After chronic pharmacologic stress, rats developed behavioral evidence of loudness hyperacusis and sound avoidance hyperacusis. Consistent with the previous noise studies, basal CORT levels remained normal; however, rats exhibited a blunted CORT response to restraint stress and GR expression in the auditory cortex increased. Chronic pharmacologic CORT stress did not alter DPOAEs or the cochlear compound action potential; however, sound-evoked responses from chronically implanted electrodes on the auditory cortex were greatly enhanced. These results show for the first time that chronic

pharmacologic stress, in the absence of hearing loss, is sufficient to induce behavioral evidence of hyperacusis, which is associated with auditory cortex neural hyperactivity and enhanced central gain.

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Signature Richard Salvi

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Behavioral and Physiological Measures of Sound Intolerance

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Sarah Theodoroff

Affiliation Department of Veterans Affairs, NCRAR

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract The experience of individuals who have decreased tolerance to everyday sounds is heterogeneous. No consensus exists regarding what constitutes hyperacusis or noise sensitivity. A direct consequence of this is the lack of a “gold standard” regarding how to diagnose, assess, and treat these conditions. The purpose of this presentation is to address this gap in clinical knowledge.

Noise sensitivity refers to an increased reactivity to everyday sounds and encompasses a range of psychological attributes, often including annoyance or feeling overwhelmed by the sounds in the environment. An estimated 59% of patients with mild traumatic brain injury have noise sensitivity (Shepherd et al, 2019). Research is lacking both on the pathophysiology of noise sensitivity, its clinical treatment, and to what degree it may or may not be a similar phenomenon to hyperacusis. Hyperacusis describes a decreased sound tolerance driven by the perceived loudness of ordinary sounds. It often including physical discomfort or pain when listening to sounds that are at moderate or low intensity levels, which most people would find tolerable. Estimates are as high as 60-79% of tinnitus patients have comorbid hyperacusis (Andersson et al, 2001; Dauman & Bouscan-Faure, 2005).

When Veterans seek medical attention because ordinary sounds are painfully loud or there’s “too much noise to function” many clinicians are uncertain how best to meet their patients’ needs. This results in the focus being shifted to comorbid conditions hoping that the “hyperacusis” is a symptom of another condition. Research is needed to develop evidence-based tools that are capable of differentiating noise sensitivity from hyperacusis and from other health conditions that present with similar symptoms. This need motivated the ideas behind an on-going research project that is examining the relationship between auditory and psychological biomarkers to determine how well auditory and psychophysiological data predict self-reported decreased tolerance to everyday sounds. Behavioral and physiological measures are collected in order to detect where deficits exist in sensory and/or neurological structures associated with complaints of sound

intolerance.
Results from this work will elucidate aspects of the underlying pathophysiology of “hyperacusis” and “noise sensitivity.” Ultimately, outcomes from this avenue of research will guide the development of targeted rehabilitative treatments based on the etiology of these conditions and inform which disciplines should be working together to best meet the needs of this patient population (e.g., audiology, psychology, neurology).

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First Name	Last Name	Affiliation
Sarah *	Theodoroff *	Department of Veterans Affairs, NCRAR

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Signature Sarah Theodoroff

Hyperacusis: Diversity in Cause, Expression, and Advocacy

A Rodent Model of Acoustic Trauma to Study Neural and Inflammatory Mechanisms of Hyperacusis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Megan Wood

Affiliation Johns Hopkins University School of Medicine

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

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Megan *	Wood *	Johns Hopkins University School of Medicine
Viola	Monovich	Peabody Institute of Johns Hopkins University
Anda	Nyati	Johns Hopkins Bloomberg School of Public Health
Paul	Fuchs	Johns Hopkins University School of Medicine
Elisabeth	Glowatzki	Johns Hopkins University School of Medicine

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Signature Lara Beers Wood

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Investigating Hyperacusis in Rodent Models of Autism

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Benjamin Auerbach

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

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Benjamin *	Auerbach *	University of Illinois at Urbana-Champaign

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Signature Benjamin D Auerbach

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Connecting Researchers to Clinicians and patients: Bryan Pollard's Roadmap to a Cure for Hyperacusis with Pain

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Michael Maholchic

Affiliation Hyperacusis Research, Ltx

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

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Michael *	Maholchic *	Hyperacusis Research, Ltx

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Signature Michael Maholchic

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Benjamin Auerbach

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Session Description Hyperacusis is a complex hearing disorder that encompasses a wide-range of reactions to sound, including excessive loudness, increased aversion/fear of sound, or even pain. While often associated with hearing loss and tinnitus, sound tolerance disturbances are observed across a broad spectrum of neurological disorders, including autism, chronic pain and post-traumatic stress disorder. Thus, hyperacusis is diverse in both its etiology and phenotypic expression, and it is imperative to consider this diversity when attempting to elucidate its physiological mechanisms and advance diagnostic and treatments. Bryan Pollard, founder and president of the Hyperacusis Research Ltd., was a champion for this cause. Bryan was a tireless advocate for hyperacusis research, working to connect researchers from diverse backgrounds, often through formal and informal workshops and dinners at the ARO midwinter meeting. He was the first non-researcher to present at ARO and was instrumental in creating a new diagnosis of pain hyperacusis. Bryan unfortunately passed away earlier this year.
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Presenter Diversity The symposium will feature a diverse group of speakers, including several presenters from underrepresented minorities. The symposium will additionally include both up-and-coming young investigators and established leaders in the field, with speakers who employ

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Signature Benjamin D Auerbach

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Imaging hyperacusis: Past and Present

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Fatima Husain

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract Brain imaging of humans with hyperacusis remains one of the primary methods to identify neural networks sub serving this condition and dissociate them from those related to comorbid tinnitus or hearing loss. Despite the challenges associated with cost and noise of some tools, results of such studies have informed the existing experimental and theoretical framework of hyperacusis. In a pioneering fMRI study, Melcher and colleagues (2010) parsed out the contribution of co-occurring hyperacusis to brain imaging findings of tinnitus. In particular, they noted that while both subcortical and cortical auditory areas were responsive in those with hyperacusis (relative to controls), such an elevated response was only noted in the auditory cortex for those with tinnitus. Two recent studies (Koops and van Dijk, 2021; Hofmeier et al., 2021) further explored this dissociation. Koops and van Dijk confirmed the higher response in cortical and subcortical centers of auditory processing in response to external sounds but in addition found that there was reduced response to the frequencies of the internally-generated tinnitus sound. The Hofmeier study provided corroboration of the increased responsiveness of the tinnitus and hyperacusis group to external sounds in the cortical and subcortical auditory areas. Additionally they noted increased wave III amplitude in brain-stem response of the tinnitus and hyperacusis group relative to the control group; the tinnitus only group exhibited a prolonged and reduced wave V amplitude compared to the controls. These studies support the idea of central gain in the condition of hyperacusis but not necessarily if tinnitus occurs alone. They further point to the role played by attention in tinnitus, as noted in task-based fMRI studies in my own lab (Husain et al., 2011; 2015). What is not easily known is the contribution and interaction of clinically-significant hearing loss with hyperacusis. Koops and van Dijk noted the elevation of response at frequencies in the hearing loss as well as normal hearing range in those with hyperacusis. The Melcher studies were primarily in those with normal hearing. In an ongoing study, we are collecting both auditory brainstem response and fMRI data on young adults with hyperacusis and normal hearing, results of which will be reported at the meeting. In summary, current and future non-invasive brain imaging

studies continue to expand our understanding of pathophysiology of hyperacusis and eventually test therapies that help patients.

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Fatima *	Husain *	University of Illinois at Urbana-Champaign

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Signature Fatima T. Husain

Hyperacusis: Diversity in Cause, Expression, and Advocacy

A Need to Better Define and Characterize Loudness and Hyperacusis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Sylvie Hebert

Affiliation Universite de Montreal

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract Hyperacusis is a hearing disorder wherein everyday sounds are considered too loud, fearful, annoying, or painful. In its most accepted definition, hyperacusis designates sounds of moderate intensities that are judged louder than normal perception. A deeper understanding of what loudness is would contribute to a better definition of what hyperacusis is, and how it can be defined and diagnosed. Indeed, although loudness involves the perception of the intensity of sounds, i.e. its sensory dimension, loudness may also be modulated through its affective dimension. In this talk I will present recent data aiming at a better understanding of loudness, namely its sensory and its affective dimensions, and how these two dimensions can be dissociated within the same stimuli. I will also report the findings of a recent scoping review on the electrophysiological correlates of hyperacusis, in which we found a diversity in terms and definitions used to describe hyperacusis and several identifiable aetiologies, among which developmental disorders, neurological disorders, induced hearing damage, and idiopathic aetiology. Broader consensus around definitions and diagnostic criteria considering aetiologies may guide researchers to ask better questions and clinicians to more efficiently manage the patient who complain about hyperacusis.

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Sylvie *	Hebert *	Universite de Montreal

Charlotte	Bigras	Universite de Montreal
Ariane	Forget	Universite de Montreal
Berangere	Villatte	Universite de Montreal

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Signature Sylvie Hébert

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Stressful hearing: Hyperacusis Can Be Induced by Chronic Stressful Noise Exposure or Chronic Pharmacological Stress that Disrupts the HPA Axis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Richard Salvi

Affiliation Center for Hearing & Deafness, SUNY at Buffalo

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract We live in a noisy, stressful environment largely oblivious to its negative hearing-health consequences. One debilitating hearing impairment associated with noise-induced hearing loss is hyperacusis, a loudness intolerance disorder in which everyday sounds are perceived as excessively loud, aversive and stressful. To investigate the biological bases of hyperacusis, we developed a reaction time-intensity (RT-I) paradigm to test for loudness hyperacusis. To assess the aversive quality of hyperacusis, we developed an active sound avoidance paradigm (ASAP) to measure sound avoidance behaviors. To trigger the induction of hyperacusis, we exposed rats to intense high-frequency noise for several months; these conditions were expected to induce chronic noise-stress in addition to high-frequency loss. After this prolonged, stressful noise exposure, rats developed clear signs of sound avoidance hyperacusis as well as loudness hyperacusis with a low-frequency spectral profile. This prolonged, stressful noise exposure disrupted the hypothalamic-pituitary-adrenal (HPA) axis; it did not alter basal corticosterone (CORT) levels, but instead, greatly reduced the rise in corticosterone (CORT) triggered by restraint stress (i.e., it blunted the stress response). This noise exposure also chronically increased the expression of glucocorticoid receptors (GR) in the auditory cortex, part of the negative feedback network that suppressed the continued release of CORT. To determine the role of chronic stress independent of hearing loss, rats were chronically stressed by pharmacologically treating the rats with CORT-stress hormone. After chronic pharmacologic stress, rats developed behavioral evidence of loudness hyperacusis and sound avoidance hyperacusis. Consistent with the previous noise studies, basal CORT levels remained normal; however, rats exhibited a blunted CORT response to restraint stress and GR expression in the auditory cortex increased. Chronic pharmacologic CORT stress did not alter DPOAEs or the cochlear compound action potential; however, sound-evoked responses from chronically implanted electrodes on the auditory cortex were greatly enhanced. These results show for the first time that chronic

pharmacologic stress, in the absence of hearing loss, is sufficient to induce behavioral evidence of hyperacusis, which is associated with auditory cortex neural hyperactivity and enhanced central gain.

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Richard *	Salvi *	Center for Hearing & Deafness, SUNY at Buffalo
Senthilvelan	Manohar	State University of New York at Buffalo
Guang-Di	Chen	Center for Hearing and Deafness, State University of New York at Buffalo

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Signature Richard Salvi

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Behavioral and Physiological Measures of Sound Intolerance

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Sarah Theodoroff

Affiliation Department of Veterans Affairs, NCRAR

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract The experience of individuals who have decreased tolerance to everyday sounds is heterogeneous. No consensus exists regarding what constitutes hyperacusis or noise sensitivity. A direct consequence of this is the lack of a “gold standard” regarding how to diagnose, assess, and treat these conditions. The purpose of this presentation is to address this gap in clinical knowledge.

Noise sensitivity refers to an increased reactivity to everyday sounds and encompasses a range of psychological attributes, often including annoyance or feeling overwhelmed by the sounds in the environment. An estimated 59% of patients with mild traumatic brain injury have noise sensitivity (Shepherd et al, 2019). Research is lacking both on the pathophysiology of noise sensitivity, its clinical treatment, and to what degree it may or may not be a similar phenomenon to hyperacusis. Hyperacusis describes a decreased sound tolerance driven by the perceived loudness of ordinary sounds. It often including physical discomfort or pain when listening to sounds that are at moderate or low intensity levels, which most people would find tolerable. Estimates are as high as 60-79% of tinnitus patients have comorbid hyperacusis (Andersson et al, 2001; Dauman & Bouscan-Faure, 2005).

When Veterans seek medical attention because ordinary sounds are painfully loud or there’s “too much noise to function” many clinicians are uncertain how best to meet their patients’ needs. This results in the focus being shifted to comorbid conditions hoping that the “hyperacusis” is a symptom of another condition. Research is needed to develop evidence-based tools that are capable of differentiating noise sensitivity from hyperacusis and from other health conditions that present with similar symptoms. This need motivated the ideas behind an on-going research project that is examining the relationship between auditory and psychological biomarkers to determine how well auditory and psychophysiological data predict self-reported decreased tolerance to everyday sounds. Behavioral and physiological measures are collected in order to detect where deficits exist in sensory and/or neurological structures associated with complaints of sound

intolerance.
Results from this work will elucidate aspects of the underlying pathophysiology of “hyperacusis” and “noise sensitivity.” Ultimately, outcomes from this avenue of research will guide the development of targeted rehabilitative treatments based on the etiology of these conditions and inform which disciplines should be working together to best meet the needs of this patient population (e.g., audiology, psychology, neurology).

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Sarah *	Theodoroff *	Department of Veterans Affairs, NCRAR

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Signature Sarah Theodoroff

Hyperacusis: Diversity in Cause, Expression, and Advocacy

A Rodent Model of Acoustic Trauma to Study Neural and Inflammatory Mechanisms of Hyperacusis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Megan Wood

Affiliation Johns Hopkins University School of Medicine

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract The mechanism underlying painful hyperacusis is currently unknown. A subset of hyperacusis patients surveyed recently reported experiencing acoustic trauma before the onset of their symptoms. Therefore, we use acoustic trauma in rodents to induce neurological and inflammatory changes in the cochlea as a model for noise-induced hyperacusis. Acoustic trauma affects many cell types of the inner ear. We focus on the responses of type II auditory nerve fibers and immune cells as these cell types are the putative pain sensing neurons and chief responders to inflammation in the cochlea, respectively. Type II auditory nerve fibers exhibit changes in their calcium dynamics after acoustic trauma. New analysis of an existing dataset will be discussed to describe a sensitization of these fibers as a possible mechanism for hyperacusis. Another possible mechanism for hyperacusis is an enhanced inflammatory response following acoustic trauma. Neuroimmune crosstalk through neuropeptides found in the cochlea, such as CGRP, may play an important role in this possible mechanism. Recent studies following immune cell migration after blocking CGRP receptors during acoustic trauma will be discussed. Finally, the presentation will end with a discussion of the parameters needed to properly model painful hyperacusis in rodents including behavioral assays of pain perception to sound stimulation.

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Megan *	Wood *	Johns Hopkins University School of Medicine
Viola	Monovich	Peabody Institute of Johns Hopkins University
Anda	Nyati	Johns Hopkins Bloomberg School of Public Health
Paul	Fuchs	Johns Hopkins University School of Medicine
Elisabeth	Glowatzki	Johns Hopkins University School of Medicine

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Signature Lara Beers Wood

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Investigating Hyperacusis in Rodent Models of Autism

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Benjamin Auerbach

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract Animal models are indispensable tools for identifying disease pathophysiology and developing objective biomarkers for hearing disorders like hyperacusis. However, care must be taken when attempting to recapitulate complex hearing disorders in model systems. Hyperacusis encompasses a wide range of reactions to sound and is observed across a broad spectrum of neurological disorders, and this diversity must be accounted for when attempting to measure and induce hyperacusis in animals. Recent progress has been made in developing rodent behavioral models that capture distinct aspects of sound perception disrupted in hyperacusis, with attempts to disentangle psychoacoustic (e.g. excessive loudness) from affective (e.g. decreased sound tolerance) aspects of the disorder. However, drug- and noise-induced hearing loss are still the primary methods used for inducing hyperacusis-like states in animals. Here we will discuss recent attempts to characterize sound tolerance disturbances in genetic models of neurodevelopmental disorders that present with high rates of hyperacusis, such as Fragile X syndrome and autism spectrum disorders (ASD). Comparing and contrasting hyperacusis associated with hearing loss and neurodevelopmental disruption has the potential to uncover convergent (or divergent) pathophysiological mechanisms across distinct forms of hyperacusis , which will help advance our ability to diagnose and treat this often devastating disorder.

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Benjamin *	Auerbach *	University of Illinois at Urbana-Champaign

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Signature Benjamin D Auerbach

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Connecting Researchers to Clinicians and patients: Bryan Pollard's Roadmap to a Cure for Hyperacusis with Pain

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Michael Maholchic

Affiliation Hyperacusis Research, Ltx

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract In memoriam to Bryan Pollard, founder of Hyperacusis Research Ltd., and his impact on the field.

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Michael *	Maholchic *	Hyperacusis Research, Ltx

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Signature Michael Maholchic

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Benjamin Auerbach

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Session Description Hyperacusis is a complex hearing disorder that encompasses a wide-range of reactions to sound, including excessive loudness, increased aversion/fear of sound, or even pain. While often associated with hearing loss and tinnitus, sound tolerance disturbances are observed across a broad spectrum of neurological disorders, including autism, chronic pain and post-traumatic stress disorder. Thus, hyperacusis is diverse in both its etiology and phenotypic expression, and it is imperative to consider this diversity when attempting to elucidate its physiological mechanisms and advance diagnostic and treatments. Bryan Pollard, founder and president of the Hyperacusis Research Ltd., was a champion for this cause. Bryan was a tireless advocate for hyperacusis research, working to connect researchers from diverse backgrounds, often through formal and informal workshops and dinners at the ARO midwinter meeting. He was the first non-researcher to present at ARO and was instrumental in creating a new diagnosis of pain hyperacusis. Bryan unfortunately passed away earlier this year.
This goal of this symposium is two-fold. First, we hope to update the hearing community on the status of the hyperacusis field and the progress that has been made towards diagnosing, treating, and modeling this disorder. Hyperacusis is a rapidly growing research field that intersects with many other aspects of hearing research, including central auditory processing and plasticity, tinnitus, and autism. As such, this symposium would have a broad target audience, from basic auditory neuroscientists interested in the neural mechanisms of perception and experience-dependent plasticity to audiologists seeking to understand and perhaps better treat a host of auditory processing disorders. In addition, we hope to use this symposium to commemorate the impact that Bryan Pollard had on the field, starting with a short introduction and in memoriam to highlight the ways he and the Hyperacusis Research foundation have helped move hyperacusis research forward.

Presenter Diversity The symposium will feature a diverse group of speakers, including several presenters from underrepresented minorities. The symposium will additionally include both up-and-coming young investigators and established leaders in the field, with speakers who employ

a variety of techniques and approaches, from animal models to human clinical research.

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Signature Benjamin D Auerbach

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Imaging hyperacusis: Past and Present

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Fatima Husain

Affiliation University of Illinois at Urbana-Champaign

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

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Signature Fatima T. Husain

Hyperacusis: Diversity in Cause, Expression, and Advocacy

A Need to Better Define and Characterize Loudness and Hyperacusis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Sylvie Hebert

Affiliation Universite de Montreal

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

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Signature Sylvie Hébert

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Stressful hearing: Hyperacusis Can Be Induced by Chronic Stressful Noise Exposure or Chronic Pharmacological Stress that Disrupts the HPA Axis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Richard Salvi

Affiliation Center for Hearing & Deafness, SUNY at Buffalo

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

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Guang-Di	Chen	Center for Hearing and Deafness, State University of New York at Buffalo

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Signature Richard Salvi

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Behavioral and Physiological Measures of Sound Intolerance

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Sarah Theodoroff

Affiliation Department of Veterans Affairs, NCRAR

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract The experience of individuals who have decreased tolerance to everyday sounds is heterogeneous. No consensus exists regarding what constitutes hyperacusis or noise sensitivity. A direct consequence of this is the lack of a “gold standard” regarding how to diagnose, assess, and treat these conditions. The purpose of this presentation is to address this gap in clinical knowledge.

Noise sensitivity refers to an increased reactivity to everyday sounds and encompasses a range of psychological attributes, often including annoyance or feeling overwhelmed by the sounds in the environment. An estimated 59% of patients with mild traumatic brain injury have noise sensitivity (Shepherd et al, 2019). Research is lacking both on the pathophysiology of noise sensitivity, its clinical treatment, and to what degree it may or may not be a similar phenomenon to hyperacusis. Hyperacusis describes a decreased sound tolerance driven by the perceived loudness of ordinary sounds. It often including physical discomfort or pain when listening to sounds that are at moderate or low intensity levels, which most people would find tolerable. Estimates are as high as 60-79% of tinnitus patients have comorbid hyperacusis (Andersson et al, 2001; Dauman & Bouscan-Faure, 2005).

When Veterans seek medical attention because ordinary sounds are painfully loud or there’s “too much noise to function” many clinicians are uncertain how best to meet their patients’ needs. This results in the focus being shifted to comorbid conditions hoping that the “hyperacusis” is a symptom of another condition. Research is needed to develop evidence-based tools that are capable of differentiating noise sensitivity from hyperacusis and from other health conditions that present with similar symptoms. This need motivated the ideas behind an on-going research project that is examining the relationship between auditory and psychological biomarkers to determine how well auditory and psychophysiological data predict self-reported decreased tolerance to everyday sounds. Behavioral and physiological measures are collected in order to detect where deficits exist in sensory and/or neurological structures associated with complaints of sound

intolerance.
Results from this work will elucidate aspects of the underlying pathophysiology of “hyperacusis” and “noise sensitivity.” Ultimately, outcomes from this avenue of research will guide the development of targeted rehabilitative treatments based on the etiology of these conditions and inform which disciplines should be working together to best meet the needs of this patient population (e.g., audiology, psychology, neurology).

Enter all co-authors. You may search for any co-authors by using the search box. If you cannot find a co-author in this database, you may type them into the grid below. Please use the arrows to move authorship into the correct order. This is the order that will be printed in any program materials.

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Signature Sarah Theodoroff

Hyperacusis: Diversity in Cause, Expression, and Advocacy

A Rodent Model of Acoustic Trauma to Study Neural and Inflammatory Mechanisms of Hyperacusis

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Megan Wood

Affiliation Johns Hopkins University School of Medicine

Participant(s) Benjamin Auerbach (Chair), Fatima Husain (Co-chair), Fatima Husain (Presenter), Sylvie Hebert (Presenter), Richard Salvi (Presenter), Sarah Theodoroff (Presenter), Megan Wood (Presenter), Benjamin Auerbach (Presenter), Michael Maholchic (Presenter)

SUBMISSION DETAILS

Individual Abstract The mechanism underlying painful hyperacusis is currently unknown. A subset of hyperacusis patients surveyed recently reported experiencing acoustic trauma before the onset of their symptoms. Therefore, we use acoustic trauma in rodents to induce neurological and inflammatory changes in the cochlea as a model for noise-induced hyperacusis. Acoustic trauma affects many cell types of the inner ear. We focus on the responses of type II auditory nerve fibers and immune cells as these cell types are the putative pain sensing neurons and chief responders to inflammation in the cochlea, respectively. Type II auditory nerve fibers exhibit changes in their calcium dynamics after acoustic trauma. New analysis of an existing dataset will be discussed to describe a sensitization of these fibers as a possible mechanism for hyperacusis. Another possible mechanism for hyperacusis is an enhanced inflammatory response following acoustic trauma. Neuroimmune crosstalk through neuropeptides found in the cochlea, such as CGRP, may play an important role in this possible mechanism. Recent studies following immune cell migration after blocking CGRP receptors during acoustic trauma will be discussed. Finally, the presentation will end with a discussion of the parameters needed to properly model painful hyperacusis in rodents including behavioral assays of pain perception to sound stimulation.

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Signature Lara Beers Wood

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Investigating Hyperacusis in Rodent Models of Autism

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Benjamin Auerbach

Affiliation University of Illinois at Urbana-Champaign

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SUBMISSION DETAILS

Individual Abstract Animal models are indispensable tools for identifying disease pathophysiology and developing objective biomarkers for hearing disorders like hyperacusis. However, care must be taken when attempting to recapitulate complex hearing disorders in model systems. Hyperacusis encompasses a wide range of reactions to sound and is observed across a broad spectrum of neurological disorders, and this diversity must be accounted for when attempting to measure and induce hyperacusis in animals. Recent progress has been made in developing rodent behavioral models that capture distinct aspects of sound perception disrupted in hyperacusis, with attempts to disentangle psychoacoustic (e.g. excessive loudness) from affective (e.g. decreased sound tolerance) aspects of the disorder. However, drug- and noise-induced hearing loss are still the primary methods used for inducing hyperacusis-like states in animals. Here we will discuss recent attempts to characterize sound tolerance disturbances in genetic models of neurodevelopmental disorders that present with high rates of hyperacusis, such as Fragile X syndrome and autism spectrum disorders (ASD). Comparing and contrasting hyperacusis associated with hearing loss and neurodevelopmental disruption has the potential to uncover convergent (or divergent) pathophysiological mechanisms across distinct forms of hyperacusis , which will help advance our ability to diagnose and treat this often devastating disorder.

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Signature Benjamin D Auerbach

Hyperacusis: Diversity in Cause, Expression, and Advocacy

Connecting Researchers to Clinicians and patients: Bryan Pollard's Roadmap to a Cure for Hyperacusis with Pain

Submission ID 3003155

Submission Type Symposia

Topic Hearing Loss: Consequences and Adaptation

Status Submitted

Submitter Michael Maholchic

Affiliation Hyperacusis Research, Ltx

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SUBMISSION DETAILS

Individual Abstract In memoriam to Bryan Pollard, founder of Hyperacusis Research Ltd., and his impact on the field.

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