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INVITED LECTURE

Representation of vocal communication streams in the brain



From
Sound
to Mind

Exploring
the Neural
Language
of Communication



Dr. Sharba Bandyopadhyay

Ph.D., Johns Hopkins University, Baltimore, USA

Associate Professor

Indian Institute of Technology Kharagpur, India

Electronics and Electrical
Communication Engineering

This talk will explore how the brain represents and processes vocal communication streams—unraveling the neural coding, temporal dynamics, and circuit mechanisms underlying communication.

EVENT DETAILS



DATE

SEPTEMBER 17,
2026

Thursday



TIME

2:00 PM
Sharp



VENUE

Meghnad Saha Auditorium,
Rasbehari Prangan,
UCSTA (Rajabazar Science
College), Kolkata-700009

ABOUT THE SPEAKER

Dr. Sharba Bandyopadhyay is an Associate Professor at the Department of Electronics and Electrical Communication Engineering with a Ph.D. from Johns Hopkins University, Baltimore, USA. His research focuses on neural representation and plasticity of auditory signals particularly vocal communication signals - in normal and disease models.



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Representation of vocal communication streams in the brain

Sharba Bandyopadhyay

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Mouse ultrasonic vocalization streams (USVs) are of communicative significance and context specific. Analyses of USVs are used to understand neurodevelopmental aspects related to communication. Mouse models of Autism Spectrum Disorders (ASDs) show deficits in USVs. Studies of USV production and neuronal coding primarily consider single tokens. We show that pup (pup isolation calls, PICs) and adult USVs (courtship and mating calls, CMCs) contain sequential structure, quantified through predictability, as in birdsong and speech. We use two mouse models of ASDs – a genetic model, 16p11.2del, and an environmental model, in utero exposure to valproate (VPA). In both models, structured PIC sequences are altered. Wild type mother mice in playback experiments preferentially approach PICs of typically developing pups over ASD-model pups. Similarly, adult male VPA mouse CMC sequences have altered structure, and naïve female mice preferentially approach CMCs of typically developed mice over VPA mice. Thus, altered call sequences in ASD models lead to communication deficits akin to human ASDs. Electrophysiological recordings further show altered coding of sequences at the single-unit level in VPA mice. Importantly, recordings from primary auditory cortex show that neural activity is not restricted to individual USV tokens but extends into the inter-syllable/token interval, with interval-period activity reflecting sequential context. This indicates that cortical processing can bridge successive vocalization elements and represent their temporal relationships. While these findings establish USV sequence production and representation as an appropriate tool to investigate ASD-related communication deficits, an important question remains. Mouse USVs have been considered innate, with the periaqueductal gray (PAG) sufficient for USV production and no involvement of primary motor cortex (M1). Pharmacological inactivation of M1 shows that production of vocalization sequences requires M1. Simultaneous 2-photon Ca^{2+} imaging and glutamate uncaging show altered developmental microcircuitry in VPA M1, which may underlie the loss of sequential structure in ASD-model mice. Thus, mouse USV sequences provide a realistic framework for investigating the neural mechanisms of communication disorders.