



Acute stress modulates neural dynamics in the auditory cortex

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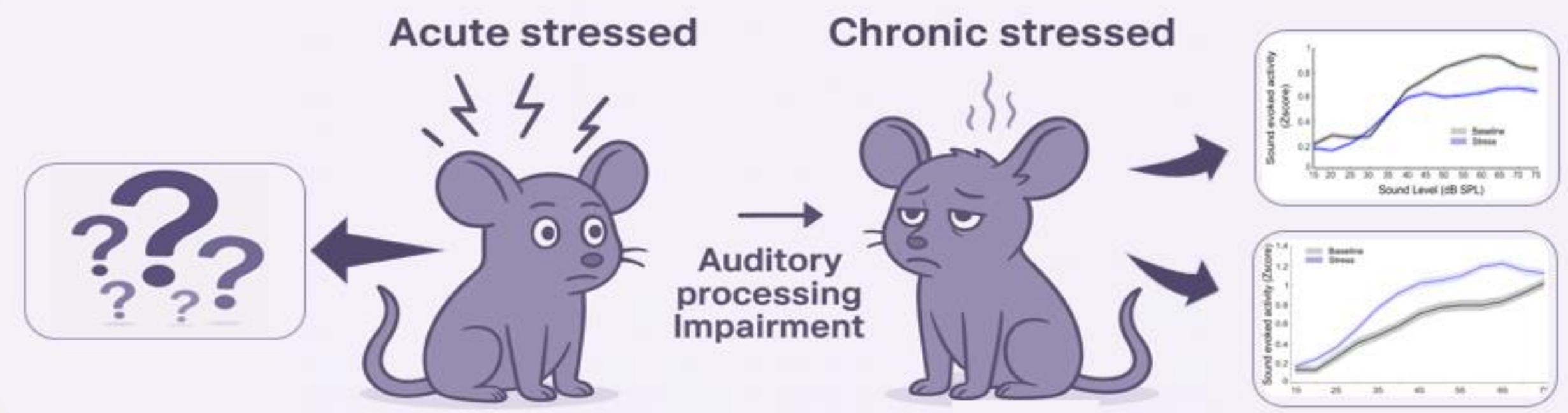
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Introduction

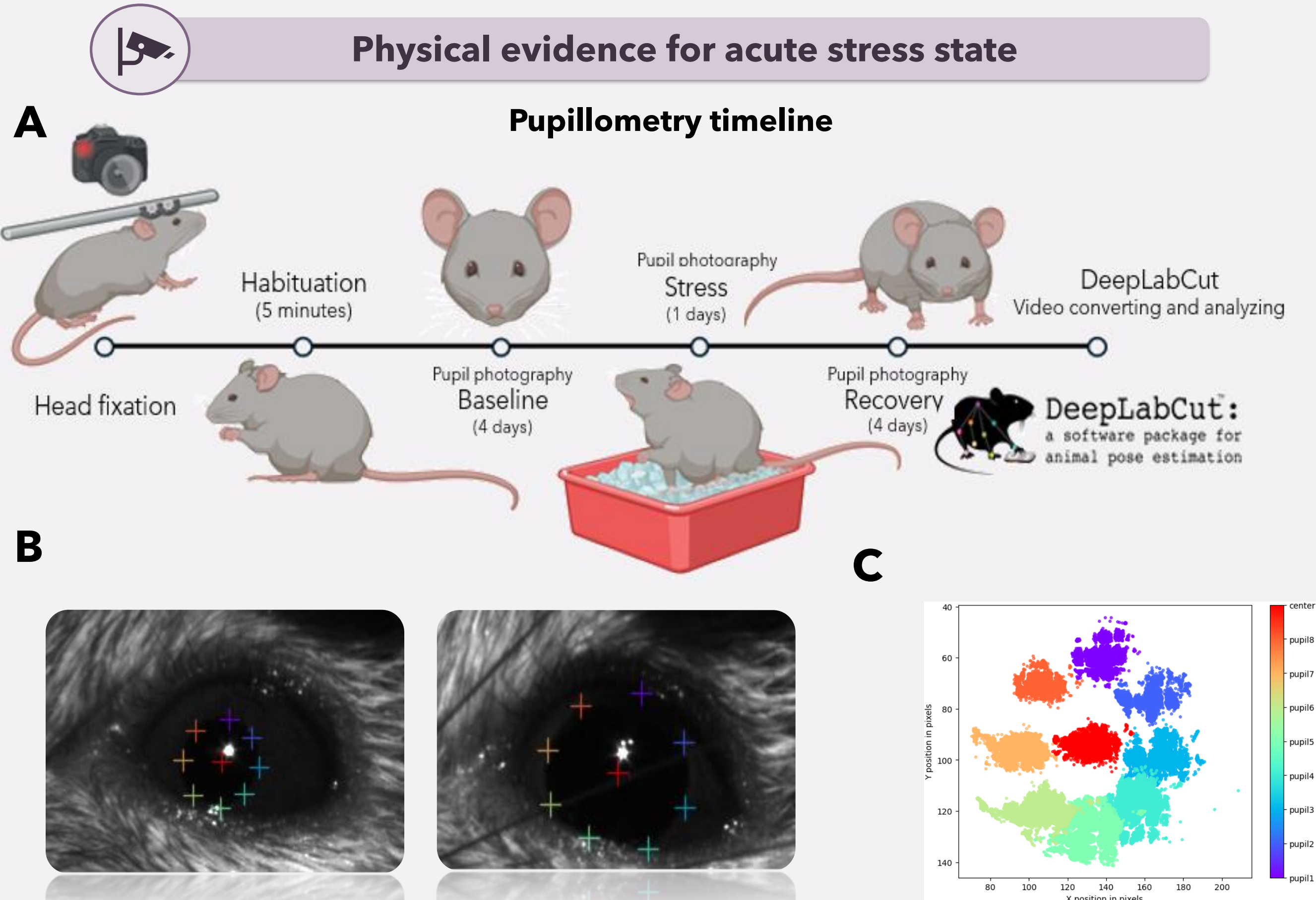
Stress is a fundamental biological response that rapidly recruits multiple brain systems to promote survival. Although its effects on cognition, memory, and emotional regulation are well established, much less is known about how acute stress shapes sensory processing. Here, we investigate how acute stress modulates auditory cortical activity in awake mice. Using pupillometry together with two-photon calcium imaging, we probe state-dependent changes in neural dynamics and coding of sound.

Acute Vs. Chronic



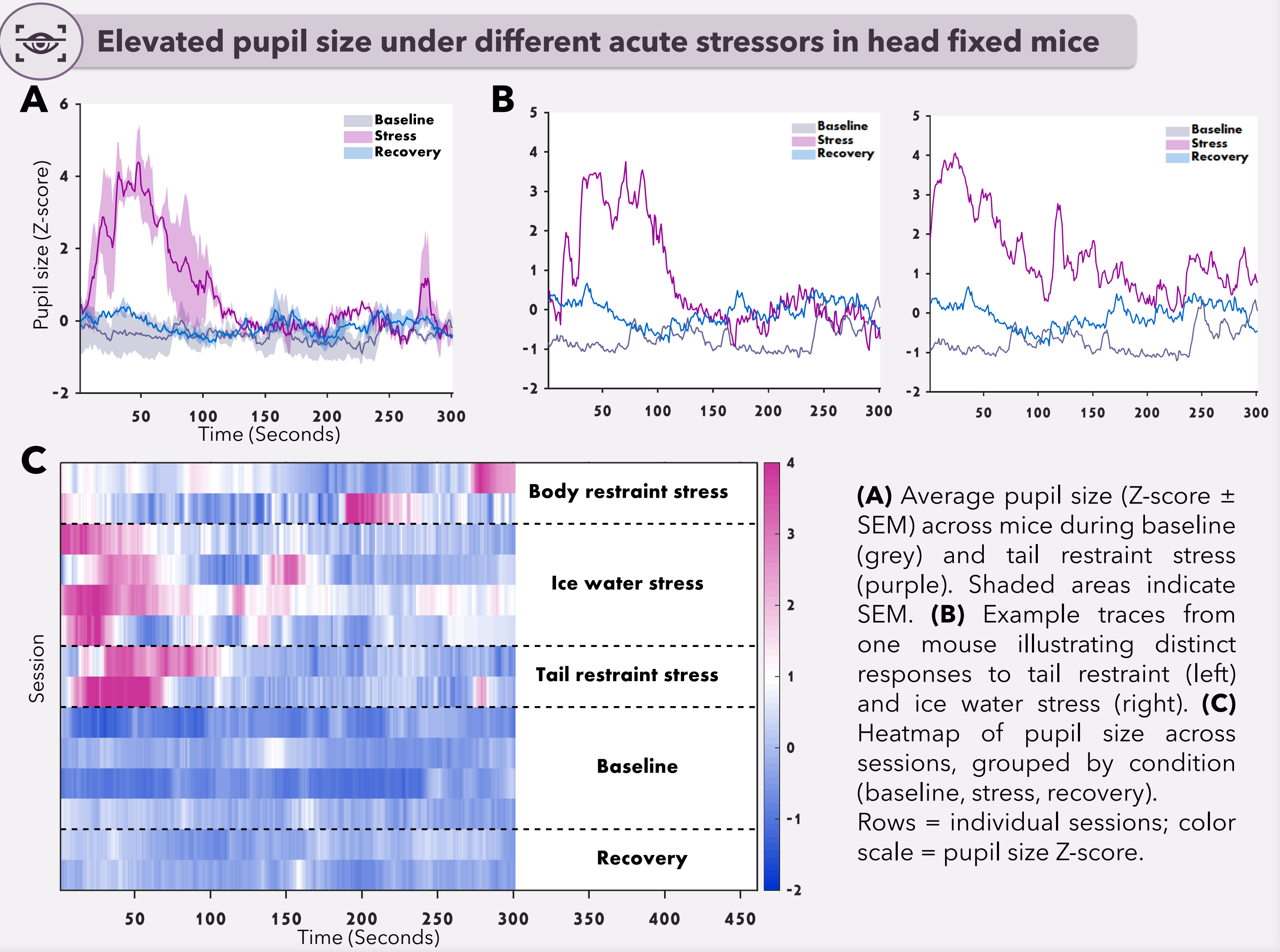
Pupillometry

Physical evidence for acute stress state



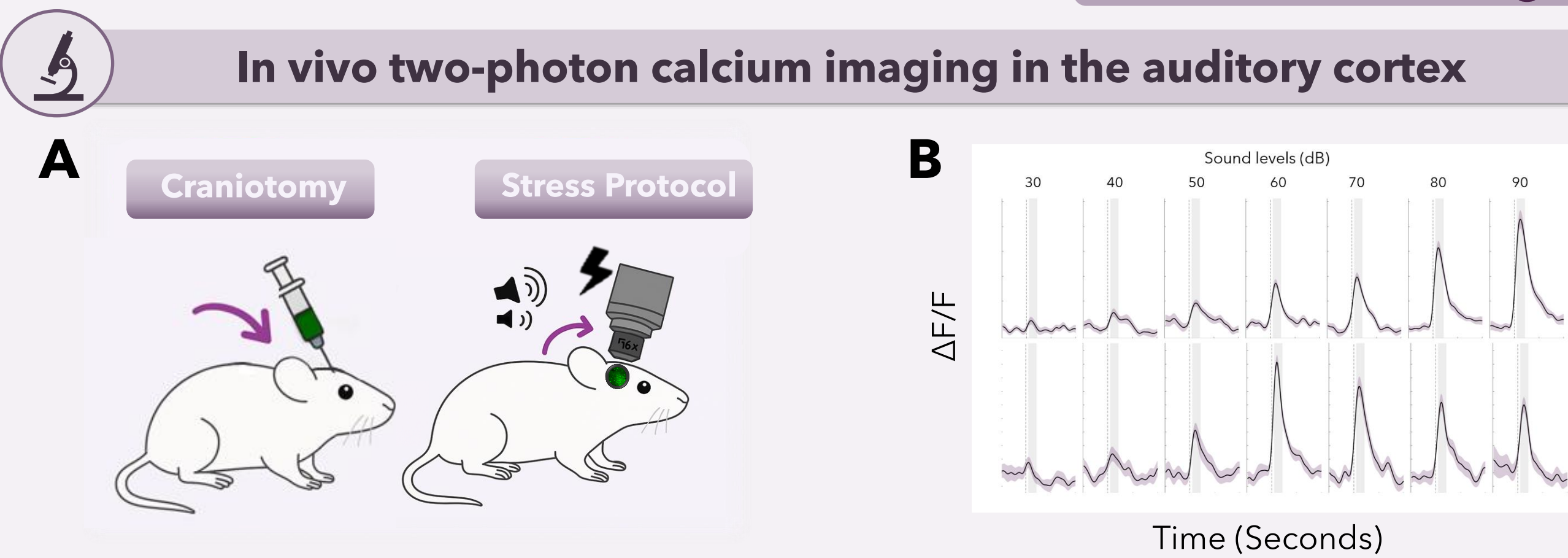
(A) Experimental scheme: schematic overview of the pupillometry setup and stress procedure. **(B)** Representative video frames illustrating DeepLabCut tracking of pupil landmarks. Left - baseline; Right - stress; same mouse. **(C)** Landmark XY coordinates extracted by DeepLabCut, used to calculate pupil size across sessions.

Elevated pupil size under different acute stressors in head fixed mice



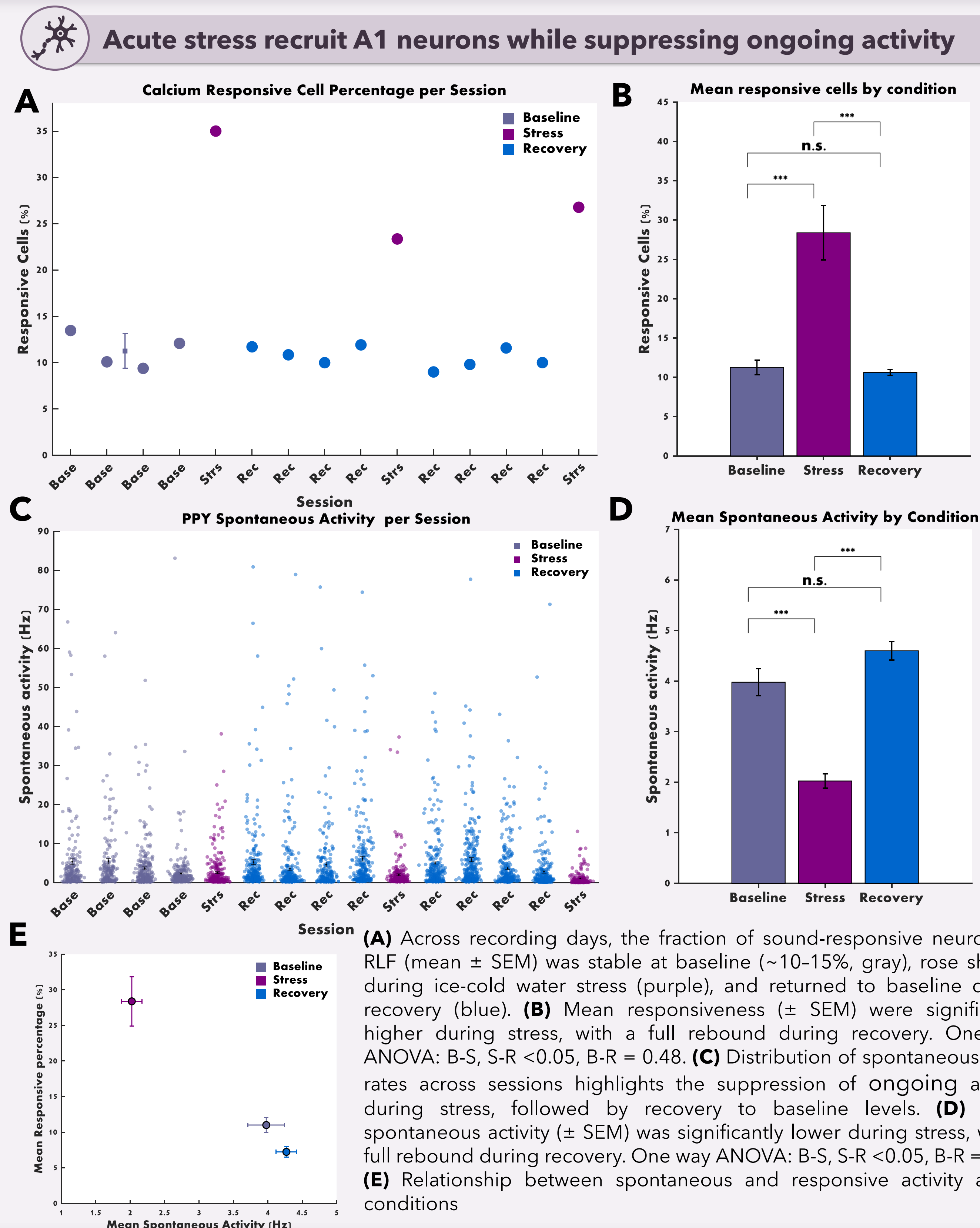
2P calcium imaging of primary auditory cortex

In vivo two-photon calcium imaging in the auditory cortex



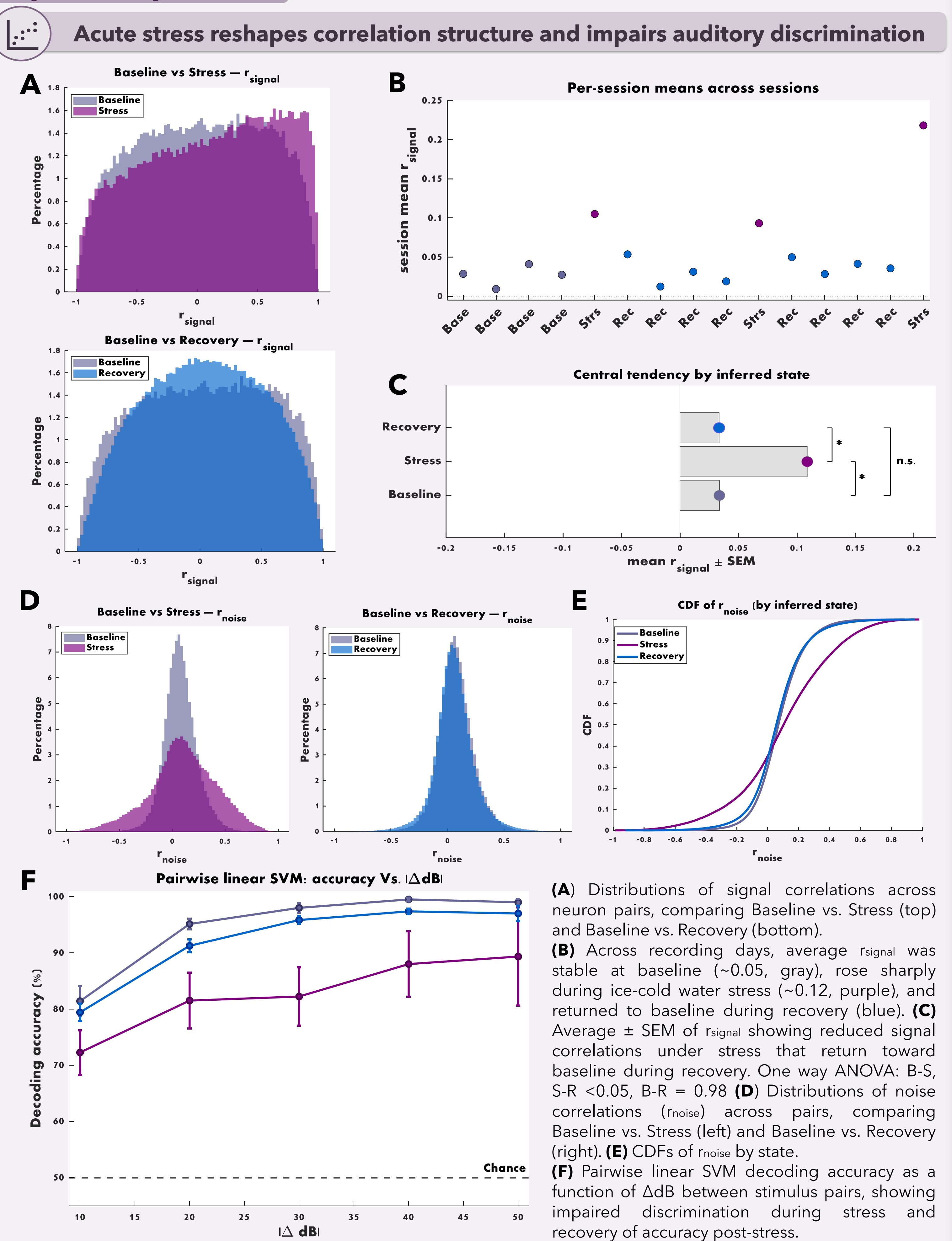
(A) Experimental scheme: Mice (8-16 weeks; SST-Cre \times Ai14) underwent craniotomy and virus injection (pGP-AAV-syn-jGCaMP8s-WPRE) to express GCaMP in the auditory cortex. Recordings were performed during baseline, stress, and recovery sessions following the stress protocol described above. **(B)** Example calcium traces from two neurons during baseline, showing graded responses to white noise (30-90 dB).

Acute stress recruit A1 neurons while suppressing ongoing activity



(A) Across recording days, the fraction of sound-responsive neurons to RLF (mean \pm SEM) was stable at baseline (\sim 10-15%, gray), rose sharply during ice-cold water stress (purple), and returned to baseline during recovery (blue). **(B)** Mean responsiveness (\pm SEM) were significantly higher during stress, with a full rebound during recovery. One way ANOVA: B-S, S-R < 0.05 , B-R = 0.48. **(C)** Distribution of spontaneous firing rates across sessions highlights the suppression of ongoing activity during stress, followed by recovery to baseline levels. **(D)** Mean spontaneous activity (\pm SEM) was significantly lower during stress, with a full rebound during recovery. One way ANOVA: B-S, S-R < 0.05 , B-R = 0.57. **(E)** Relationship between spontaneous and responsive activity across conditions

Acute stress reshapes correlation structure and impairs auditory discrimination



(A) Distributions of signal correlations across neuron pairs, comparing Baseline vs. Stress (top) and Baseline vs. Recovery (bottom). **(B)** Across recording days, average r_{signal} was stable at baseline (\sim 0.05, gray), rose sharply during ice-cold water stress (\sim 0.12, purple), and returned to baseline during recovery (blue). **(C)** Average \pm SEM of r_{signal} showing reduced signal correlations under stress that return toward baseline during recovery. One way ANOVA: B-S, S-R < 0.05 , B-R = 0.98 **(D)** Distributions of noise correlations (r_{noise}) across pairs, comparing Baseline vs. Stress (left) and Baseline vs. Recovery (right). **(E)** CDFs of r_{noise} by state. **(F)** Pairwise linear SVM decoding accuracy as a function of Δ dB between stimulus pairs, showing impaired discrimination during stress and recovery of accuracy post-stress.

Conclusions and ongoing work

Acute stress reshapes auditory cortical dynamics by engaging more neurons, dampening ongoing activity, and enhancing shared variability across the network. Such reorganization may limit coding precision, providing a neural basis for stress-related deficits in auditory perception. Ongoing work will test how these changes directly impact auditory perception and recovery dynamics.