specific properties for subcortical spontaneous neural activity cannot be identified.

The current research design should be continued on a larger scale for statistically significant findings.

Figure 1 & 2: Click ABR

Click ABR measurements were averaged in GI and GII. Table 1 compares GI measurements to the established norm (GIII). Table 2 compares GII measurements to the established norm. Statistical significant findings ($p<.05$) are labeled with an asterisk.

| ABR Measurement | Group I | Group III | t-Test | Sig. (2 tailed) |
|---|---|---|---|---|
| I Latency | 1.723 ± .10233 | 1.556 ± .10233 | -1.931 | .069 |
| III Latency | 3.699 ± .40534 | 3.761 ± .14746 | .455 | .655 |
| V Latency | 5.604 ± .32756 | 5.509 ± .14403 | -.84 | .412 |
| V Amplitude | .538 ± .368841 | .511 ± .15509 | -.214 | .833 |
| IPL I-III | 2.066 ± .50608 | 2.0206 ± .16628 | .831 | .417 |
| IPL III-V | 1.906 ± .44144 | 1.751 ± .19886 | -1.012 | .0331* |
| IPL I-V | 3.836 ± .17411 | 3.954 ± .18307 | 1.477 | .157 |

| ABR Measurement | Group II | Group III | t-Test | Sig. (2 tailed) |
|---|---|---|---|---|
| I Latency | 1.5809 ± .32164 | 1.556 ± .10233 | -.244 | .812 |
| III Latency | 3.6245 ± .3638 | 3.761 ± .14746 | 1.145 | .272 |
| V Latency | 5.8109 ± .38316 | 5.509 ± .14403 | -2.431 | .03* |
| V Amplitude | .4109 ± .14039 | .511 ± .15509 | 1.553 | .137 |
| IPL I-III | 2.0464 ± .25839 | 2.0206 ± .16628 | 1.664 | .113 |
| IPL III-V | 2.1864 ± .56197 | 1.751 ± .19886 | -2.409 | .032* |
| IPL I-V | 4.23 ± .48512 | 3.954 ± .18307 | -1.755 | .103 |

Figure 3, 4, & 5: Toneburst ABR

Toneburst ABR measurements were averaged in GI and GII and compared to the established norm. Three (total of six ears) of the seven frequencies tested for GII yielded significant differences ($p<.05$) for several of the ABR measurements. Statistically significant findings are labeled with an asterisk.

**GII: 6000 Hz Toneburst**

| ABR Measurement | Group I | Group III | t-Test | Sig. (2 tailed) |
|---|---|---|---|---|
| I Latency | 2.175 ± .2129 | 1.9 ± .43765 | -.829 | .434 |
| III Latency | 3.75 ± .35355 | 4.1643 ± .68266 | -.8 | .45 |
| V Latency | 5.585 ± .54447 | 6.5571 ± .33979 | -3.225 | .015* |
| V Amplitude | .19 ± .24042 | .3329 ± .12539 | -1.209 | .266 |
| IPL I-III | 1.575 ± .57276 | 2.2657 ± .84668 | -1.059 | .325 |
| IPL III-V | 1.835 ± .19092 | 2.3929 ± .74748 | -1 | .351 |
| IPL I-V | 3.415 ± .7566 | 4.6586 ± .73814 | -2.094 | .075 |

**GII: 12500 Hz Toneburst**

| ABR Measurement | Group I | Group III | t-Test | Sig. (2 tailed) |
|---|---|---|---|---|
| I Latency | 2.475 ± .45962 | 1.3857 ± .14909 | 3.302 | .176 |
| III Latency | 4.58 ± .42426 | 3.1243 ± .48675 | 3.769 | .007* |
| V Latency | 6.78 ± .14142 | 4.7914 ± .66702 | 4.001 | .005* |
| V Amplitude | .24 ± .07071 | .21 ± .13128 | .301 | .772 |
| IPL I-III | 2.105 ± .03536 | 1.7 ± .52243 | 1.044 | .331 |
| IPL III-V | 2.2 ± .56569 | 1.6643 ± .53935 | 1.23 | .258 |
| IPL I-V | 4.3 ± .59397 | 3.4057 ± .77724 | 1.48 | .182 |

**GII: 14000 Hz Toneburst**

| ABR Measurement | Group I | Group III | t-Test | Sig. (2 tailed) |
|---|---|---|---|---|
| I Latency | 1.485 ± .30406 | 1.0757 ± .173 | 2.59 | .036* |
| III Latency | 3.165 ± .12012 | 2.66 ± .39328 | 1.172 | .13 |
| V Latency | 6.6065 ± .33234 | 4.71 ± .6509 | 2.745 | .024* |
| V Amplitude | .55 ± .18385 | .2686 ± .2098 | 1.702 | 1.33 |
| IPL I-III | 1.68 ± .42426 | 1.5843 ± .3774 | .311 | .765 |
| IPL III-V | 2.9 ± .45255 | 1.9257 ± .42634 | 2.825 | .026* |
| IPL I-V | 4.575 ± .03536 | 3.5114 ± .65096 | 4.301 | .005* |

**PS 421**

## Longitudinal MRI Analysis of Volumetric Differences in Brainstem Neurons Following Noise Induced Temporary Threshold Shift

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### Background

Even exposure to loud noise resulting in temporary deafness often leads to conditions such as tinnitus. Noise over-exposure has been a major risk factor for chronic tinnitus for those in occupations associated with noisy environments. Currently there is no effective treatment for tinnitus. This emphasizes the need to determine metrics that will allow for longitudinal studies of tinnitus progression. Noise induced tinnitus has been associated with changes in neuronal activity. Magnetic resonance imaging (MRI) has been used to track longitudinal changes in tinnitus related neuronal activity using manganese enhanced MRI (MEMRI). In addition to activity, neuronal volume has also been suggested to change in people with chronic tinnitus. Temporal and spatial changes in brain volume were examined longitudinally in several brain regions, including the inferior colliculus (IC), following a single noise exposure.

### Methods

Hearing thresholds were determined (ABRs) in two groups (noise and no noise) of male Sprague Dawley rats. Noise animals were exposed to a 16 kHz, 106 dB SPL tone for 1 hour. For each group, $T_1$-weighted MRI images were obtained (7T Clinscan) before (baseline) and after noise exposure (1, 28, and 84 day(s)). Deficits in Gap detection were used as a measure of tinnitus. Morphology for regions of interest (ROIs) was identified on a single subject brain atlas using MRIStudio software. The atlas was registered to the subjects using Automated Image Registration (AIR) and Large Deformation Diffeomorphic Metric Mapping (LDDMM) in order to map the ROIs onto each subject. ROI volumes from the two groups were compared at each time point.

### Results

Differences in the volume of the IC occurred as early as 1 day after noise exposure, with significant decreases in the central nucleus of the IC (CIC) of noise exposed animals compared to control. Although hearing thresholds had returned to

normal, significant volumetric decreases were seen 84 days after noise exposure in the CIC of noise exposed animals. With the exception of the first week, Gap detection deficits were present at all time points (20 kHz, 60dB).

**Conclusions**
An outcome of our study is the production of a rat volumetric atlas with brain region coordinates that can be used as a template by others. Volumetric information may provide another crucial metric for understanding the effects of noise over-exposure on the progression of tinnitus.

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**PS 422**

## Transdermal Stimulus-Timing Dependent Plasticity in Dorsal Cochlear Nucleus Reduces Tinnitus in Guinea Pigs

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**Introduction**
Nearly two-thirds of tinnitus patients can modulate their tinnitus with somatic maneuvers of the face and/or neck (Levine et al, 2007), which is likely mediated by somatosensory connections to the cochlear nucleus (CN). The dorsal cochlear nucleus (DCN) integrates auditory inputs with somatosensory inputs from trigeminal and dorsal column systems. Fusiform cells, the principle output neurons of the DCN, exhibit stimulus-timing dependent plasticity (STDP) in response to bimodal auditory-somatosensory stimulation: Hebbian-like plasticity occurs when neurons increase their activity responding to somatosensory-preceding auditory stimulation, but anti-Hebbian rules occur when auditory- precedes somatosensory stimulation (Koehler and Shore, 2013a). In tinnitus animals, Hebbian-like rules invert to anti-Hebbian-like rules with broadened temporal windows (Koehler and Shore, 2013b). Our recent work demonstrated that fusiform cell STDP can be induced non-invasively using transdermal stimulation (Wu et al., 2015). To test whether STDP can be harnessed to treat tinnitus, we applied paired auditory-somatosensory stimulation, via transdermal neck stimulation, to noise-damaged guinea pigs with behavioral and neural evidence of tinnitus. Preliminary data shows decrements in both behavioral and neural correlates of tinnitus after one month of 30 min bimodal stimulation.

**Methods**
Guinea pigs were noise-exposed with a 7 kHz-centered, half-octave band at 97 dB SPL for 2 hours to induce a temporary threshold shift. Gap-Prepulse Inhibition of Acoustic Startle (GPIAS) was used to assess tinnitus. Somatosensory stimulation was provided via transdermal stimulating electrodes placed near the C2 cervical vertebrae. Short electrical stimuli were presented within 20 ms of 40 dB SL sounds matching the tinnitus spectra. Tinnitus animals were treated for four weeks concurrently with biweekly tinnitus assessments. Following cessation of treatment, single unit recordings from DCN were obtained with recording electrodes placed stereotaxically into the DCN fusiform cell layer after ketamine/xylazine anesthesia. Unit responses to tones and noise were assessed for units with best frequencies ranging from 4 kHz to 32 kHz.

**Results**
Animals with tinnitus showed elevated spontaneous activity and neural synchrony at the measured tinnitus frequencies. In contrast, exposed animals without tinnitus animals did not show elevated spontaneous rates or synchrony compared to unexposed animals. Animals receiving bimodal STDP induction showed decreases in spontaneous rates and neural synchrony, which correlated with a reduction in behavioral evidence of tinnitus.

**Conclusions**
Our results demonstrate that non-invasive, long-term alteration of DCN neural activity through bimodal STDP can be used to alleviate behavioral and neural correlates of tinnitus. This treatment strategy may provide relief in human tinnitus sufferers.

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**PS 423**

## Analysis of Auditory and Non-Auditory Input in the Dorsal Cochlear Nucleus in a Rat Model of Tinnitus & Noise-Induced Hearing Loss

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**Background**
Hair cell damage resulting from acoustic trauma not only alters hearing thresholds but may also initiate a cascade of maladaptive plastic changes in central auditory nuclei. Increasing evidence has implicated such plastic changes in the dorsal cochlear nucleus (DCN) in the induction of chronic tinnitus. The DCN consists of 3 layers (molecular, fusiform, and deep), which create a circuit. Fusiform cells in the DCN are the main output cells of the DCN circuit, and they are also the first site of multi-sensory integration in the auditory system. Input to fusiform cells can be identified as auditory or non-auditory by the distribution of vesicular glutamate transporters (VGlut). Fusiform cell basal dendritic fields receive auditory input via the auditory nerve (molecular and fusiform layers) and their apical dendritic field receives non-auditory input via parallel fiber projections (molecular and fusiform layers) from granule cells. We sought to quantify changes in auditory and non-auditory input to the DCN following acoustic trauma.

**Methods**
Adult male, Long-Evans rats were unilaterally exposed to a 118 dB, 16 kHz pure-tone for 4 hours (n=8) while anesthetized using isoflurane, and allowed to recover for 2 weeks. Pre-

ABSTRACT BOOK

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