**COMPARISON OF SALICYLATE & NOISE-INDUCED TINNITUS RAT MODELS**


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OBJECTIVE

The objective of this study is to compare the salicylate-induced & the noise-induced tinnitus rat models using three robust and validated experimental in vivo techniques: behavioral test, auditory cortex electrophysiology, and brain in vivo imaging.

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**RESULTS**

1/ Gap Prepulse Inhibition Acoustic Startle (GPIAS)

**STATE OF THE ART**

In normal animals, a silent gap before the startle stimulus will reliably inhibit the reflex. Animals with tinnitus exhibit deficits detecting the silent gap. Because the gap is not as easily detected, the reflex is not inhibited to the same degree. (Turner 2007)

**Salicylate-induced tinnitus model**

- 1-way ANOVA: p<0.05
- m = no significant differences
- n = 8-9 ears / group

**Noise-induced tinnitus model**

- 1-way ANOVA: p<0.05
- m = no significant differences
- n = 8-9 rats / group

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2/ Manganese Enhanced MRI

**STATE OF THE ART**

MRI system for small animal

- 24 h before MRI session, animals received 30 µl transynaptic injection of manganese, MnCl2 8.2 mmol/kg, under anesthesia
- Salicylate is administrated intraperitoneally at 300 mg/kg b.w. after manganese injection

**Salicylate-induced tinnitus model**

**Noise-induced tinnitus model**

We observed an increase of MEMRI signals indifferent auditory brain structures compared to controls 24 hours after salicylate administration and 30 days after acoustic trauma. An increase of cochlea signal without differences in interior cochlea is observed in salicylate model whereas opposite pattern is observed in noise-induced tinnitus model.

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3/ Electrophysiology

**STATE OF THE ART**

A single microelectrode of 17 µm of platinum 10% iridium contact surface is placed into the layer 4 of the primary auditory cortex in order to measure the spontaneous spikes before and 2h after salicylate administration or 30 days after acoustic trauma.

**Salicylate model**

Before salicylate

- 2.5 spikes/min

1h after salicylate

- 6.8 spikes/min

**Noise model**

Control (sham)

- 2 spikes/min

18 days after acoustic trauma

- 11.2 spikes/min

The combination of behavioral test, electrophysiology recording, and in vivo imaging allows to measure putative signs of tinnitus in both rat models. Similar results were observed in electrophysiology and MEMRI imaging read-outs for salicylate and noise induced tinnitus model. However, using gap prepulse inhibition test, we observed that salicylate induced tinnitus at the BBN whereas the acoustic trauma induced tinnitus at 12, 16, and 24 kHz but not at the BBN. Taken together, these data open the door for screening and characterization of new drug efficacy on tinnitus disorder.

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