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Killan, EC and Thyer, NJ (2014) Simultaneous suppression of tone burst-evoked otoacoustic emissions for two- and three-tone burst combinations. In: Fourth Joint Annual Conference, Experimental and Clinical Short Papers meetings of the British Society of Audiology, Sep 2013, Keele University.

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# Simultaneous suppression of tone burst-evoked otoacoustic emissions for two- and three-tone burst combinations

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# Introduction

Simultaneous suppression of tone burst-evoked otoacoustic emissions (SSTBOAEs) has been explained in terms of a local nonlinear interaction (LNI)-based mechanism<sup>[1]</sup>. This states that SSTBOAEs results from local interactions between the basilar membrane vibration patterns (caused by each of *two* tone bursts) governed by TBOAE generator channel compressive nonlinearity. However, it is not clear whether this mechanism can account for SSTBOAEs caused by *three* additional tone bursts. Whilst a simple additive effect is predicted by the LNI-based mechanism (so that more suppression is caused by the three-tone burst combination compared to the two-tone burst combination), comparison of suppression values reported across three separate studies<sup>[2-4]</sup> suggests that the opposite is true. There are two possible explanations for this difference:

- (i) it simply reflects methodological and subject differences across the three studies (none of which tested both two- and three-tone burst combinations), or
- (ii) it implies that more complex interactions, different to those assumed in the LNI-based mechanism, are involved for the three-tone burst combination.

# Method

In order to understand which alternative is most likely, SSTBOAEs for two- and three-tone burst combinations were measured from 13 normal human ears and predicted by a simple mathematical model of the LNIbased mechanism reported in the literature<sup>[1]</sup>. This model represents a single TBOAE generator channel via a gammachirp filter<sup>[5]</sup> (see Fig. 1A) in series with a mathematical compressive nonlinearity (see Fig. 1B). For the model and normal ears, SSTBOAEs was measured using the test paradigm shown in Fig. 2 for combinations of tone bursts 1 and 2 kHz (i.e.  $\Delta f = 1$ ), 1 and 3 kHz (i.e.  $\Delta f = 2$ ) and 1, 2 and 3 kHz (i.e.  $\Delta f = 1+2$ ). Tone bursts were presented at 60 dB p.e. SPL, i.e. when the cochlear response characteristic is assumed to be nonlinear.



**Fig. 1A**. Magnitude response for gammachirp filter with peak frequency at 1.2 kHz. **B**. Level function obtained for  $y = Ax^{0.5}$ .

# References

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Fig. 2. lest paradigm tor measuring SS1BOAEs. The case tor two-tone bursts (TB<sub>1</sub> and TB<sub>2</sub>) is shown, though the principle is the same for three tone bursts.

# Results

Fig 3A shows the predictions obtained from the model of the LNI-based mechanism. As expected, a simple additive effect is observed, so that:

- Suppression for  $\Delta f = 1+2 > \max(\Delta f = 1, \Delta f = 2)$  and
- Suppression for  $\Delta f = 1+2 > sum(\Delta f = 1, \Delta f = 2)$ .

Though greater suppression was obtained from human ears across all combinations, the same pattern was seen. Fig 3B shows the mean results (+1 standard error (SE)) obtained across all 13 ears. Paired ttests showed suppression for  $\Delta f = 1+2$  was significantly greater than suppression for max( $\Delta f = 1$ ,  $\Delta f = 2$ ) (t = 3.52, p < 0.05) but the difference between  $\Delta f = 1+2$  and sum( $\Delta f = 1$ ,  $\Delta f = 2$ ) was not significant (t = 0.77, p = 0.46).





# Conclusions

The findings of this study suggest that SSTBOAE caused by three-tone burst combinations is governed by the same LNI-based mechanism that has been used to account for SSTBOAE caused by two-tone burst combinations. In light of this, it is further suggested that the unexpected differences apparent across the previous studies (i.e. that greater suppression was observed for two-tone burst combinations compared to three tone-burst combinations) were most likely accounted for by methodological and subject differences.

A primary role for the LNI-based mechanism in SSTBOAEs has been used to argue in favour of the frequency specificity of TBOAEs<sup>[1]</sup>. Such arguments cannot be made where alternative, more complex interactions are thought to occur. Given that the findings of this experiment indicate that the LNI-based mechanism is sufficient to account for SSTBOAEs and alternative interactions are not required, they therefore provide support for the frequency specific use of TBOAEs in the clinical setting.