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**Conference or Workshop Item:**
Simultaneous suppression of tone burst-evoked otoacoustic emissions for two- and three-tone burst combinations

EC Killan and NJ Thyer
Academic Unit of Healthcare Science, Pharmacy and Radiography

Introduction
Simultaneous suppression of tone burst-evoked otoacoustic emissions (SSTBOAEs) has been explained in terms of a local nonlinear interaction (LNI)-based mechanism\(^1\). 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\(^2-4\) 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 LNI-based mechanism reported in the literature\(^1\). This model represents a single TBOAE generator channel via a gammachirp filter\(^3\) (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.

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

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 t-tests showed suppression for \(\Delta f = 1+2\) was significantly greater than suppression for \(\text{max}(\Delta f = 1, \Delta f = 2)\) (t = 3.52, p < 0.05) but the difference between \(\Delta f = 1+2\) and \(\text{sum}(\Delta f = 1, \Delta f = 2)\) 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\(^5\). 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.

References