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# Changes in distortion product otoacoustic emission (DPOAE) fine structure due to contralateral acoustic stimulation

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Contralateral acoustic stimulation (CAS) can cause changes in the amplitude of the $2f_1$-$f_2$ DPOAE in humans - most probably mediated by the medial olivocochlear reflex. DPOAE amplitude changes due to CAS show large interindividual variability and large changes from suppression to enhancement for small changes of the primary levels. The underlying mechanisms of these effects are still not fully understood. We hypothesize that the two interacting DPOAE sources might be differently affected by the CAS. If so, CAS will cause specific changes in DPOAE fine structure. Therefore, DPOAE fine structures were measured using frequency-modulated primaries ($f_2$: 1500-3000Hz, $f_2/f_1$: 1.2; $L_2$: 60dB SPL; $L_1$: 58, 63, 68dB SPL) without and with a broadband CAS (50 dB SPL). The fine structure changes and shifts according to CAS were analyzed in detail - including latency windowing to separate the contributions from the two interacting DPOAE sources. The results indicate, e.g., that there is no "true" enhancement in terms of enhanced cochlear activity during broadband CAS. Rather there are slight frequency shifts in DPOAE fine structure due to changes in the relative contribution of the two DPOAE sources. This shift can lead to observations of "pseudo" enhancement of up to 20dB for isolated frequencies.