





Lisowski, Namyslowski, Morawski & Strojek (2001) evaluated DPOAEs in 42 normal hearing adults with Type I DM and 33 age and sex matched controls in two separ

with neuropathy when compared to controls ( $F=3.5$ ,  $p<0.05$ ). A significant difference in TEOAE amplitude levels was not found between diabetics without neuropathy and the control group. Analysis revealed lower DPOAE amplitudes in diabetics with signs of neuropathy at f2 values spanning 1306-5200Hz ( $p<0.05$ ). Lower DPOAE amplitudes were also found in diabetics without neuropathy at f2 values spanning 3284-5200Hz ( $p<0.01$ ). No correlations were found between TEOAE and/or DPOAE response amplitudes and metabolic control or duration of disease.

Overall, results from Ottaviani et al. (2002) and DiNardo et al. (1998) illustrate that DPOAE amplitude levels are reduced in adults with Type I DM when compared to healthy controls despite similar hearing thresholds. In comparing adults with and without diabetic neuropathy, Di Nardo et al. (1998) found that TEOAE and DPOAE amplitudes are lower in adults with signs of neuropathy than in controls. Where as DPOAE amplitude levels are also lower in diabetics without signs of neuropathy (across a smaller frequency range), TEOAE amplitude levels are not. These results also suggest that there may be reduced sensitivity between the two methodologies to the effects of diabetic neuropathy on an identified sub-clinical auditory impairment in the outer hair cells. Alternatively, the

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Taken together, these results demonstrate reduced TEOAE suppression in children with Type I DM when a pure tone or broadband noise is presented contralaterally in comparison to controls despite similar thresholds and no differences in initial TEOAE amplitude levels (Namyslowski et al. 2001, Ugar et al., 2009).

In a review of the literature three predominant themes immerge:

1. Adults with Type I DM have lower TEAOE and DPAOE amplitudes than healthy controls despite similar hearing thresholds. This suggests possible presenc

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