

**Providing early auditory input in only one ear with a cochlear implant leaves unstimulated cortical pathways unprotected from deafness-induced abnormalities**

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**Objectives:** To explore the developmental consequences on the brain of missing a sensitive period for bilateral auditory input and driving development of the auditory cortex with stimulation from only one implant throughout childhood and adolescence.

**Background:** Hearing loss and recommended interventions for hearing loss in children comes in many forms. Sometimes, only a single hearing aid or single cochlear implant (CI) is recommended or is available even when hearing loss is bilateral. However, we have two ears and two auditory pathways by design, both of which are meant to be stimulated in order for the brain to receive optimal input and develop appropriately. Providing access to bilateral hearing in early development protects the auditory cortices in the brain from cortical reorganization in favour of the stimulated ear/pathways. It has been shown that stimulating the auditory system with only one CI and leaving the opposite ear/pathways deprived of input for longer than 1.5 years compromises bilateral auditory development.

**Methods:** We measured the development of electrically-evoked cortical responses in adolescents who had over a decade of unilateral CI experience before receiving a second implant in the opposite deprived ear. We then used novel imaging tools to localize underlying areas of cortical activity in the brain in response to sounds presented from each ear.

**Results:** Our results indicated that stimulating the auditory system with only one CI over the long-term promotes normal-like development of the auditory brain with good speech perception outcomes. However, abnormal responses were measured from the opposite deprived ear.

**Conclusions:** Our data suggests that leaving one ear without sound beyond the period of auditory maturation (>10 years) drives abnormal development in brain pathways and leaves that ear and cortical pathways unprotected from deafness-induced abnormalities.