A preventive role of autophagy in the cochlea to noise-exposure

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Sensorineural hearing loss is due to congenital abnormality of lateral wall, the organ of Corti or spiral ganglion, as well as acquired damage of the cochlea induced by exposure to noise and ototoxic drugs. On the other hand, autophagy, a self-digestion intracellular catabolic process, plays a crucial role in cellular homeostasis. Our previous studies demonstrated that noise at 110-dB sound pressure level (SPL), but not at 90-dB SPL, produced permanent hearing impairment. To elucidate the preventive role of autophagy in the cochlea to noise-exposure, we evaluated the effect of chloroquine (CQ, autophagy inhibitor) on hearing ability after the noise-exposure at 90-dB SPL as well as on the expression of LC3-II (LC3 = LC3-II/LC3-I, autophagy marker) in the cochlea. Immunoblot analysis revealed that CQ dramatically increased the LC3-II level in the lateral wall, but not in the organ of corti and modiolus (spiral ganglion). Moreover, CQ significantly exacerbated the noise-induced shift in the auditory brainstem response threshold after noise exposure at 90-dB SPL. Taken together, our data suggest that autophagy signals in the cochlear lateral wall exert a crucial role in maintain cochlear cellular homeostasis and represent a protective mechanism against noise exposure.