Monash University researchers have identified a new protease target for the development of treatments for acquired hearing loss. We seek a partner to develop novel inhibitors of the target and drug delivery mechanisms.

**THE TECHNOLOGY**

Individuals that lack the protease inhibitor SERPINB6 suffer severe and progressive hearing loss (DFNB91). Using their SERPINB6 knockout mouse model, Monash researchers have shown that absence of SERPINB6 results in degeneration of the Organ of Corti in the inner ear (cochlea) as discrete cell types die. Sensory hair cells die first, followed by neurons and fibrocytes.

The researchers have now identified a target protease that is responsible for this damage. The protease is normally present in the cochlea, but has not previously been implicated in inner ear function or pathology. Animals that lack the protease have better than normal baseline hearing (Fig.1).

The researchers have also established that intense noise increases the levels of the protease in the inner ear. Notably, animals lacking the protease show significantly less noise-induced hearing loss and cochlear damage (Fig.2).

It therefore appears that irreversible damage to the inner ear by this protease is responsible for noise-induced hearing loss, and possibly age-related hearing degeneration.

Developing inhibitors of the protease could lead to a first in class drug treatment for the preservation of hearing following trauma or during ageing.

**THE FUTURE**

Monash seeks a partner to develop novel inhibitors of the protease (including monoclonal antibodies and/or small molecules) and to explore delivery mechanisms for targeting the cochlear and inner ear. The Monash team has extensive experience in mouse auditory anatomy and physiology, and protease biochemistry, including a biochemical assay for testing inhibitors that can be adapted for HTS.