Hearing lessons from flies

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Abstract

Studying the auditory system of the fruit fly can reveal how hearing works in mammals.

Related research article Li T, Giagtzoglou N, Eberl D, Nagarkar-Jaiswal S, Cai T, Godt D, Groves AK, Bellen HJ. 2016. The E3 ligase Ubr3 regulates Usher syndrome and MYH9 disorder proteins in the auditory organs of Drosophila and mammals. *eLife* **5**:e15258. doi: 10.7554/eLife.15258**Image** A montage of scolopidia – the structures that fruit flies require for hearing

The myosin motor proteins play a variety of roles inside cells, such as transporting cargo around the cell and maintaining the structure of the cell's internal skeleton. Myosins also make important contributions to our sense of hearing, which can be revealed by studying conditions such as Usher syndrome (a severe sensory disorder that causes congenital deafness and late-onset blindness). In humans and other mammals, two myosin proteins called myosin VIIa and myosin IIa have been linked to deafness, but we do not understand how these proteins interact.

Now, in eLife, Andrew Groves, Hugo Bellen and co-workers – including Tongchao Li of Baylor College of Medicine as first author – report evidence of a conserved molecular machinery in the auditory organs of mammals and the fruit fly *Drosophila* (Li et al., 2016). Furthermore, the screen identified an enzyme called Ubr3 that regulates the interaction of the two myosins in *Drosophila*.

Auditory organs convert the mechanical energy in sound waves into electrical signals that can be interpreted by the brain. In mammals, this conversion happens in "hair cells" in the inner ear. These cells have thin protrusions called stereocilia on their surface, and the tips of these stereocilia contain ion channels called MET channels (which is short for mechanoelectrical transduction channels).

Five proteins associated with the most serious form of Usher syndrome – known as USH1 – are key components of the molecular apparatus that enables the MET channels to open and close in response to mechanical force. The USH1 proteins are restricted to the tips of the stereocilia, where they form a complex (Figure 1; Prosser et al., 2008; Weil et al., 1995). Two of the USH1 proteins work together to join the tip of each stereocilium to its next-highest neighbor, forming bundles of stereocilia (Kazmierczak et al., 2007). Deflecting these bundles stretches the stereocilial bundles, which opens the MET channels and

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