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Figure 2 **Icy evidence in the Northern Hemisphere: a present-day ice-sheet on the Svalbard Islands.**

decrease in the nutrient availability, like that brought about by the sea surface being cut off from the deeper ocean, at least on a seasonal basis. At the same time, the reduction in vertical mixing allows the sea surface to warm. Thus the development of a seasonally layered, or stratified, surface ocean 2.7 million years ago, which was probably a regional response to the large-scale climatic changes at this time[12], allowed late summer/autumn warming of the sea surface and provided a moisture source for ice growth.

Haug *et al.*[8] test the interpretations of the geochemical records with a suite of numerical computer-model experiments. The simulated ocean is 'stratified' and 'destratified' to determine whether this mechanism can account for the geochemically derived changes in temperature. And it can. The stratified model state produces more extreme seasons and a larger North American ice sheet than does the destratified model.

This is an exemplary study. The individual climate indicators may not have withstood the uncertainties and assumptions that limit each of them, but put together by Haug *et al.* they tell a cogent story of the origin of the ice ages. ■

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Hearing

Aid from hair force

Corné Kros

Mammals hear with exquisite sensitivity and precision over a huge range of frequencies; tiny amplifiers in the inner ear make this possible. New results challenge current thinking on how these amplifiers work.

Our ability to hear relies on cells in the inner ear called hair cells — named after the bundle of 100 or so hair-like projections that protrudes from their upper surfaces. Sound bends the hair bundles, causing small electrical ('transducer') currents to flow, which in turn makes the hair cells signal the reception of sound to the brain. In mammals, the silent majority of the hair cells (the outer hair cells) do not talk to the brain, instead helping the inner hair cells — the true sensory receptors — to do so with more clarity than they could achieve by themselves. But how is this done?

For two decades scientists have sought the answer in the extraordinary ability of the outer hair cells to change their length rapidly when stimulated. Now, however, Kennedy, Crawford and Fettiplace[1] (page 880 of this issue) and Chan and Hudspeth[2] (in *Nature Neuroscience*) present provocative evidence that the main component of the elusive 'cochlear amplifier' may instead reside in the hair bundles of the outer hair cells.

Sound waves that reach the ear lead to vibrations inside the cochlea — a fluid-filled, coiled tube forming the auditory part of the inner ear (Fig. 1a). The sensory hair cells reside in a thin strip of tissue, the organ of Corti, that is wedged between two membranes of the cochlea. The vibrations cause a shearing motion between these two membranes, which bends the hair bundles. Like a rolled-up piano, one end of the organ of Corti vibrates best at low frequencies and the other at high frequencies. In normal ears, the vibration is boosted and sharpened for soft sounds by what has become known as the cochlear amplifier[3].

Twenty years ago, a remarkable discovery by Brownell and colleagues[4] seemed to show what the cochlear amplifier is made of: they found that electrical stimulation of the outer hair cells made them lengthen and shorten their cell bodies. The idea is that, *in vivo*, the electricity produced by bending the hair bundles would drive this lengthening and shortening, or electromotility, as fast as sound could vibrate the bundles. The strategic position of the outer hair cells would locally boost the vibration of the organ of Corti, and in this way stimulate, by fluid coupling, the bundles on inner hair cells.

At a molecular level, this mechanism is thought to rely on a motor protein called prestin, named from the musical term for a very fast tempo. The basolateral membranes of the outer hair cells are packed with this protein[5], which changes shape as fast as you can change the voltage across the membrane, over a range of frequencies up to at least 100 kHz (ref. 6). But there is a snag: although prestin is quick, it is not clear whether the transmembrane voltage *in vivo* changes much over the period of the sound wave, at sound frequencies greater than a few kilohertz. This is because the receptor potential due to the transducer currents is severely attenuated at higher frequencies by the electrical impedance of the cell[7].

An alternative source of force that is not voltage-dependent may thus be needed to power the cochlear amplifier. Kennedy and colleagues[1] report large forces generated by the hair bundles of rat outer hair cells *in vitro*, when stimulated by a flexible glass fibre. You would expect the tip of the fibre to move less when attached to the bundle than when it is freely moving in the fluid. So there must have been disbelief in the lab when, in some cases, the fibre moved further when coupled to the hair bundles, implying that a force in the bundle drags the fibre along, instead of the other way round.

This force — an order of magnitude larger than the force that is a necessary by-product of opening the ion channels through which the transducer current enters hair cells[8] — is not there at the moment the hair bundle is moved by the fibre, but develops within a fraction of a millisecond. Its time course is closely coupled to that over which the transducer current adapts to a steady

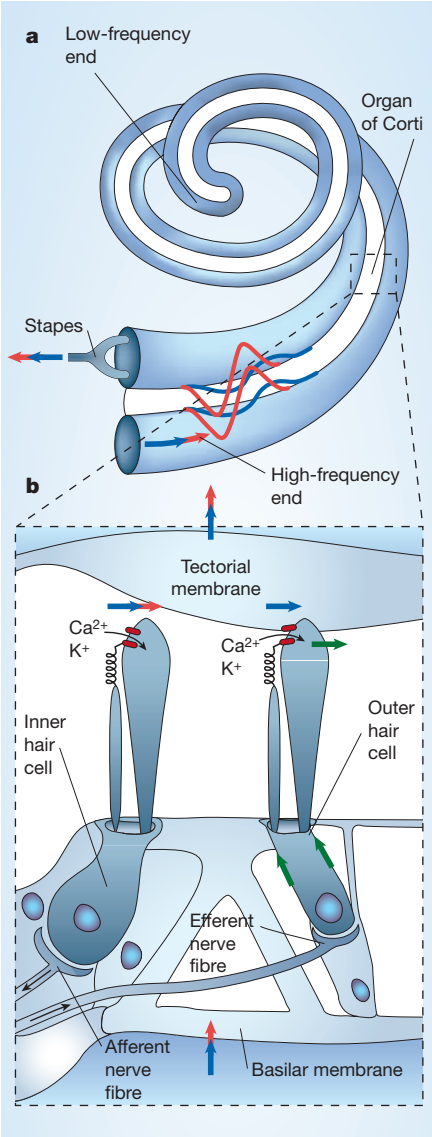

Figure 1 **Cochlear amplification.** a, **The cochlea. Sound hitting the eardrum is transformed into vibrations of the middle ear bones. The smallest, the stapes, couples the vibration into the cochlea. The organ of Corti is found along the length of the cochlea, sandwiched between two membranes; which part vibrates depends on the frequency. The cochlear amplifier increases and sharpens the vibrations in response to soft sounds (blue curve, passive vibration; red, cochlear amplifier working).** b, **Cross-section through part of the organ of Corti. Shearing between tectorial and basilar membranes opens channels in the bundles of outer hair cells, causing K^+ and Ca^{2+} ions to flow in. The cochlear amplifier in these cells (green arrows, force generator in the hair bundle and/or prestin protein in the cell membrane) enhances the vibrations in different parts of the organ of Corti (blue arrows, no amplification; red arrows, amplifier active). This boosts the membranes' motion and, via fluid coupling, the hair bundles of the inner hair cells. Afferent nerve fibres contact the inner hair cells and signal sound reception to the brain; efferent nerve fibres allow the brain to control outer hair cells. Arrows indicate motion in the excitatory direction.**

displacement. This calcium-dependent adaptation is extremely fast in mammalian outer hair cells[9], and may help them to respond best to sound frequencies appropriate for their position along the cochlear spiral.

This work shows that the hair bundles of outer hair cells contain a fast force generator that does not suffer from the speed limitations of being voltage dependent. But could it really provide amplification *in vivo*? This is where Chan and Hudspeth's study[2] comes in. Their experimental approach was to take a turn of the gerbil cochlea and put it in an *in vitro* environment that painstakingly recreated the normal, *in vivo*, situation. It was thus possible to stimulate the organ of Corti with sound and to record the motion of the hair bundles of the inner hair cells.

The authors observed evidence for amplification of the bundle motion at low sound intensities. The amplification disappeared when the transducer current was pharmacologically blocked. This by itself does not prove that the amplification is in the hair bundle: no transducer current also means no receptor potential and hence no prestin-driven electromotility. However, when Chan and Hudspeth then prevented most of the transducer current (normally carried by potassium ions), just leaving the small fraction that is carried by calcium ions, the receptor potentials of the outer hair cells would have been much attenuated (although these were not measured). But the amplification in the motion of the inner-hair-cell bundles remained. The conclusion is that calcium-dependent force generation by the outer-hair-cell bundles may be sufficient to drive the cochlear amplifier (Fig. 1b), without the need for electromotility.

So what of prestin? Genetically engineered mice lacking this protein do not have normal cochlear amplification, to which it must therefore make some contribution[10]. Prestin might work at a more leisurely pace, keeping the hair bundles at their most sensitive position and mediating the brain's ability to turn the noise down, through activation of the nerve fibres that signal to the outer hair cells. Perhaps it should be renamed 'andantin'.

These first demonstrations of force generation by the hair bundles of outer hair cells[1] and their effects on cochlear amplification[2] were obtained with cells from the low-frequency part of the cochlea, and it needs to be shown that they also apply at the speed limit of mammalian hearing. Other evidence is still missing, too. Do the forces measured by Kennedy and colleagues[1] disappear when transduction is blocked? Why do Chan and Hudspeth's results[2] not quite match the *in vivo* performance, even at low frequencies? What are needed now are experiments and models that lead to a precise, quantitative understanding of the balance of power between prestin and hair-bundle forces in cochlear amplification. ■

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Photonics

Expect more delays

Joe T. Mok and Benjamin J. Eggleton

Slow light research has been a fast-moving topic in recent years, with potential applications from quantum computing to telecommunications. Techniques are now emerging that can slow down light in optical fibres.

Light travels at a speed c of 300 million metres per second in a vacuum, but can be slowed down to cycling speed (around 17 metres per second)[1] or can even be brought to a halt[2,3], when the right medium is used. Although we may not see commercial applications appearing immediately, there is a clear potential for making practical use of slow light. Recently, Song *et al.*[4] reported a technique to delay light in optical fibres, aiming at applications in fibre-optic communication networks.

An example of a device where slow light could be particularly useful is an all-optical router. Routers are used in communication systems to direct information from one point to another. Whereas today's routers function by first converting the information sent in optical form into electronic form, all-optical routers use all-optical switching schemes, eliminating the optical–electronic–optical conversion and are therefore inherently fast. The realization of an all-optical router requires an optical buffer — a component that functions as temporary optical storage, to effectively synchronize data packets. This