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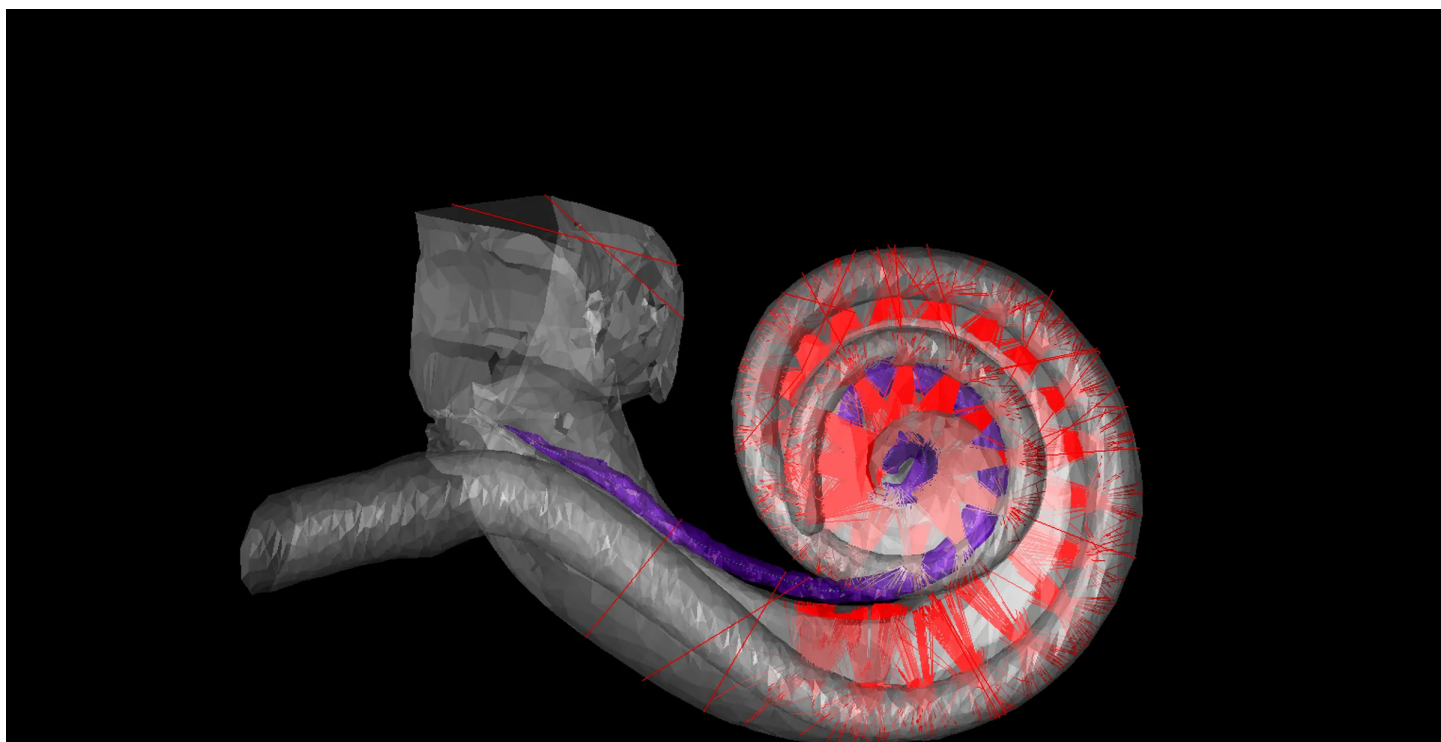
RESTORING HEARING WITH BEAMS OF LIGHT

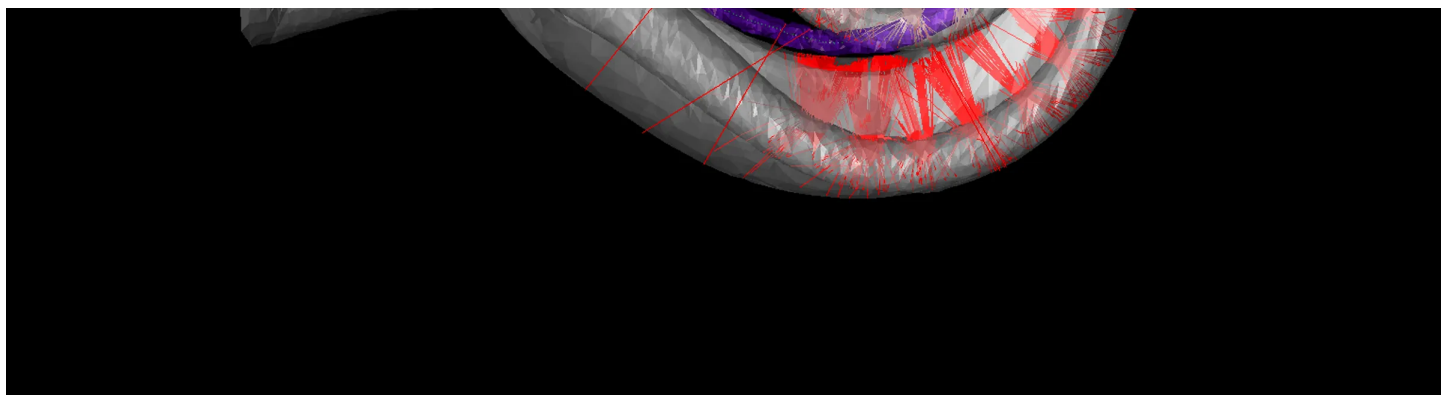
Gene therapy and optoelectronics could radically upgrade hearing for millions of people

BY [TOBIAS MOSER](#)

18 JUL 2022 | 13 MIN READ

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Human hearing depends on the cochlea, a snail-shaped structure in the inner ear. A new kind of cochlear implant for people with disabling hearing loss would use beams of light to stimulate the cochlear nerve. LAKSHAY KHURANA AND DANIEL KEPPELER

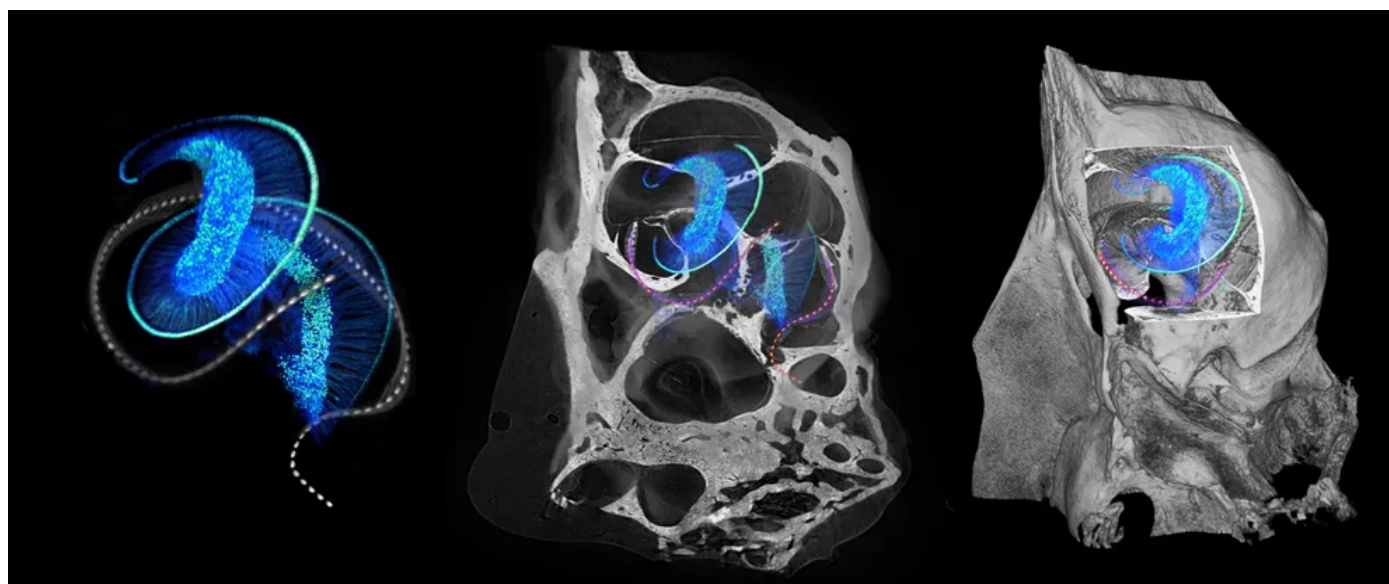
THERE'S A POPULAR misconception that cochlear implants restore natural hearing. In fact, these marvels of engineering give people a new kind of “electric hearing” that they must learn how to use.

Natural hearing results from vibrations hitting tiny structures called hair cells within the cochlea in the inner ear. A cochlear implant bypasses the damaged or dysfunctional parts of the ear and uses electrodes to directly stimulate the cochlear nerve, which sends signals to the brain. When my hearing-impaired patients have their cochlear implants turned on for the first time, they often report that voices sound flat and robotic and that background noises blur together and drown out voices. Although users can have many sessions with technicians to “tune” and adjust their implants’ settings to make sounds more pleasant and helpful, there’s a limit to what can be achieved with today’s technology.

I have been an otolaryngologist for more than two decades. My patients tell me they want more natural sound, more enjoyment of music, and most of all, better comprehension of speech, particularly in settings with background noise—the so-called cocktail party problem. For 15 years, my team at the University of Göttingen, in Germany, has been collaborating with colleagues at the University of Freiburg and beyond to reinvent the cochlear implant in a strikingly counterintuitive way: using light.

We recognize that today’s cochlear implants run up against hard limits of engineering and human physiology. So we’re developing a new kind of cochlear

full spectral nature of sounds and better mimic natural hearing. We aim to start clinical trials in 2026 and, if all goes well, we could get regulatory approval for our device at the beginning of the next decade. Then, people all over the world could begin to hear the light.



These 3D microscopic images of mouse ear anatomy show optical implants [dotted lines] twisting through the intricate structure of a normal cochlea, which contains hair cells; in deafness, these cells are lost or damaged. At left, the hair cells [light blue spiral] connect to the cochlear nerve cells [blue filaments and dots]. In the middle and right images, the bony housing of the mouse cochlea surrounds this delicate arrangement. DANIEL KEPPELER

How cochlear implants work

Some 466 million people worldwide suffer from disabling hearing loss that requires intervention, according to the World Health Organization. Hearing loss mainly results from damage to the cochlea caused by disease, noise, or age and, so far, there is no cure. Hearing can be partially restored by hearing aids, which essentially provide an amplified version of the sound to the remaining sensory

light.

Today's cochlear implants are the most successful neuroprosthetic to date. The first device was approved by the U.S. Food and Drug Administration in the 1980s, and nearly 737,000 devices had been implanted globally by 2019. Yet they make limited use of the neurons available for sound encoding in the cochlea. To understand why, you first need to understand how natural hearing works.

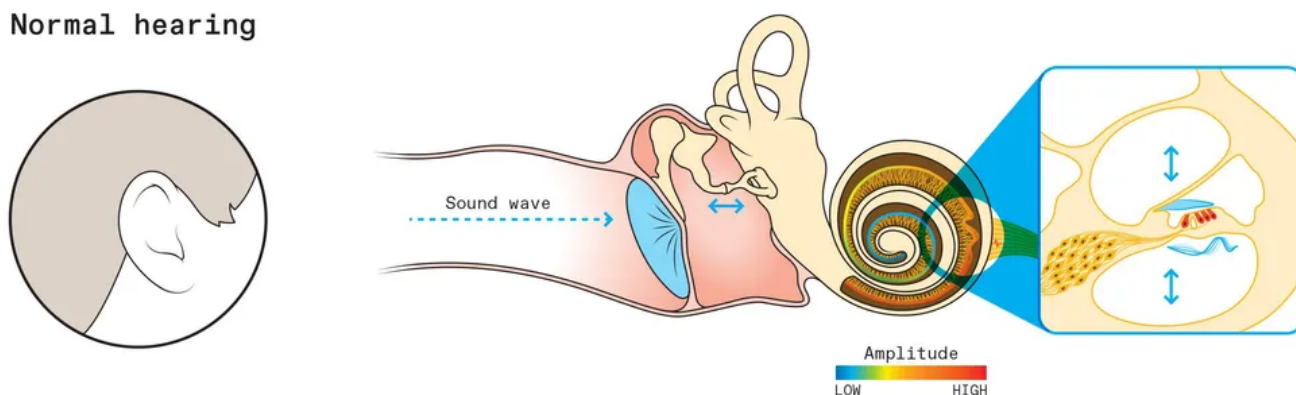
In a functioning human ear, sound waves are channeled down the ear canal and set the ear drum in motion, which in turn vibrates tiny bones in the middle ear. Those bones transfer the vibrations to the inner ear's cochlea, a snail-shaped structure about the size of a pea. Inside the fluid-filled cochlea, a membrane ripples in response to sound vibrations, and those ripples move bundles of sensory hair cells that project from the surface of that membrane. These movements trigger the hair cells to release neurotransmitters that cause an electrical signal in the neurons of the cochlear nerve. All these electrical signals encode the sound, and the signal travels up the nerve to the brain. Regardless of which sound frequency they encode, the cochlear neurons represent sound intensity by the rate and timing of their electrical signals: The firing rate can reach a few hundred hertz, and the timing can achieve submillisecond precision.

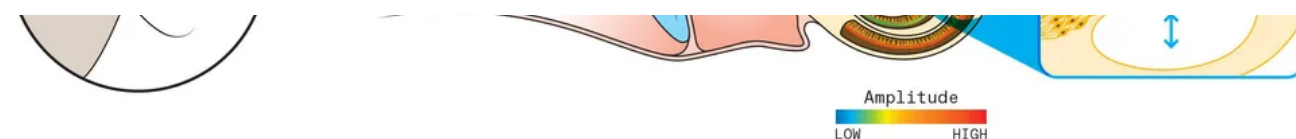
Hair cells in different parts of the cochlea respond to different frequencies of sound, with those at the base of the spiral-shaped cochlea detecting high-pitched sounds of up to about 20 kilohertz, and those at the top of the spiral detecting low-pitched sounds down to about 20 Hz. This frequency map of the cochlea is

within the cochlea is conductive, so the current from each electrode spreads out and causes broad activation of neurons across the frequency map of the cochlea. Because the frequency selectivity of electrical stimulation is limited, the quality of artificial hearing is limited, too. The natural process of hearing, in which hair cells trigger precise points on the cochlear nerve, can be thought of as playing the piano with your fingers; cochlear implants are more equivalent to playing with your fists. Even worse, this large stimulation overlap limits the way we can stimulate the auditory nerve, as it forces us to activate only one electrode at a time.

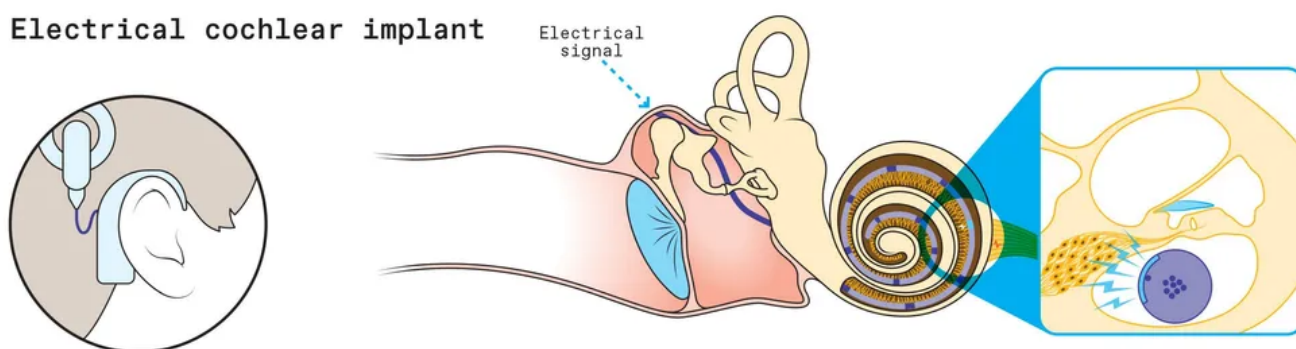
Three Ways to Hear

Normal hearing

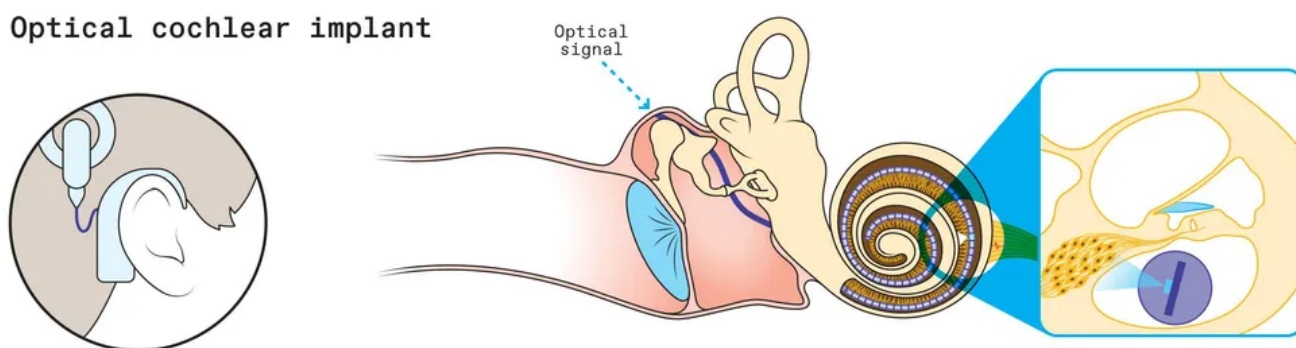




Electrical cochlear implant



Optical cochlear implant



CHRIS PHILPOT

In normal hearing, sound waves travel down the ear canal and vibrate the ear drum and tiny bones in the middle ear. Those vibrations then reach the spiral-shaped cochlea and move bundles of sensory hair cells. When the hair cells respond, it triggers a neural signal that travels up the cochlear nerve to the brain. Hair cells at the base of the spiral respond to high-pitched sounds; those at the tip respond to low-pitched sounds.

With an electrical cochlear implant, a microphone, processor, and transmitter are worn behind the ear. The processor translates a sound's pattern of frequencies into a crude stimulation pattern, which is transmitted to an implanted receiver and then to an electrode array that spirals through the cochlea. A limited number of electrodes (12 are shown here) directly stimulate the cells of the cochlear nerve. But each electrical pulse spreads out and stimulates off-target nerve cells, which results in muddier sound.

In a future optical cochlear implant, the external hardware could remain the same, though the processor could break up the sound into narrower frequency bands and transmit a more sophisticated stimulation pattern. The light source, either a flexible micro-LED array or optical fibers, would spiral through the cochlea, and the implant could have many more stimulation sites, because light is more easily confined in space than electrical current is. The user would have a gene-therapy treatment to make the cells of the cochlear nerve responsive to light, which would trigger precise signals that travel up the nerve to the brain.

How optogenetics works

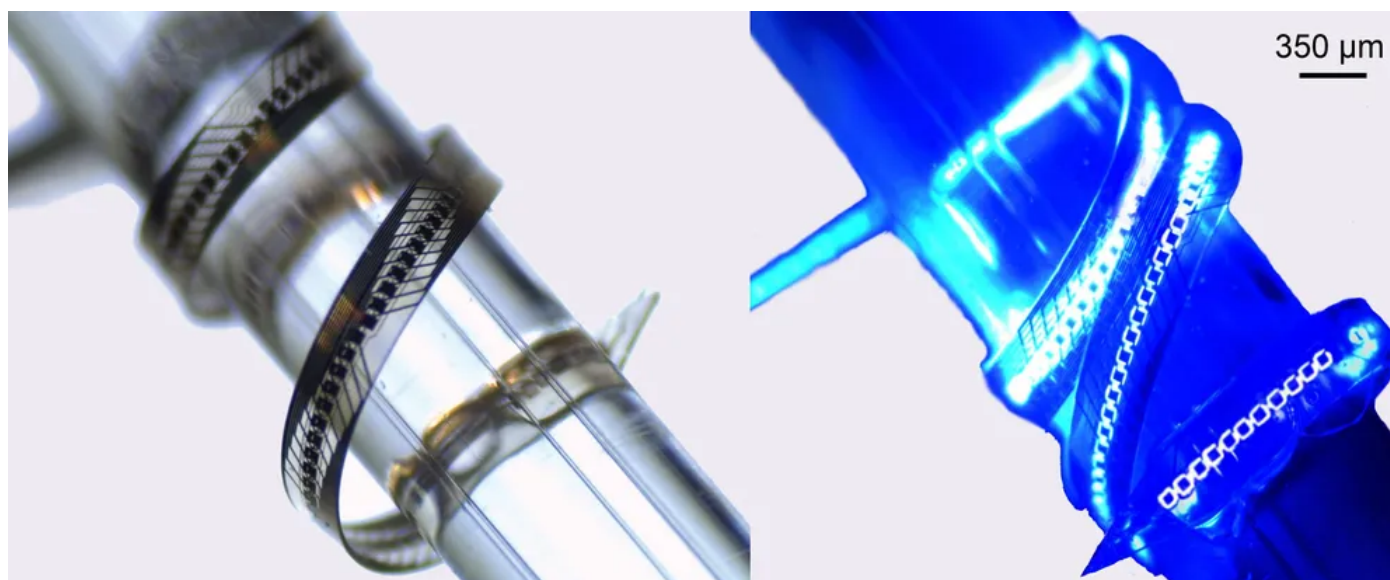
We are proposing a new type of implanted medical device that will be paired with a new type of gene therapy.

If we used optogenetics to make cochlear nerve cells light sensitive, we could then precisely hit these targets with beams of low-energy light to produce much finer auditory sensations than with the electrical implant. We could theoretically have more than five times as many targets spaced throughout the cochlea, perhaps as many as 64 or 128. Sound stimuli could be electronically split up into many more discrete frequency bands, giving users a much richer experience of sound. This general idea had been taken up earlier by [Claus-Peter Richter](#) from Northwestern University, who proposed directly stimulating the auditory nerve with high-energy infrared light, though that concept wasn't confirmed by other

channelrhodopsin, turned out to be a long process. Many early efforts in optogenetics used channelrhodopsin-2 (ChR2) that opens an ion channel in response to blue light. We used it in a proof-of-concept experiment in mice that demonstrated that optogenetic stimulation of the auditory pathway provided better frequency selectivity than electrical stimulation did.

In our continued search for the best channelrhodopsin for our purpose, we tried a ChR2 variant called calcium translocating channelrhodopsin (CatCh) from the Max Planck Institute of Biophysics lab of Ernst Bamberg, one of the world pioneers of optogenetics. We delivered CatCh to the cochlear neurons of Mongolian gerbils using a harmless virus as a vector. We next trained the gerbils

We were excited when a leader in optogenetics, Edward Boyden at MIT, discovered a faster-acting channelrhodopsin that his team called Chronos. Although it still required blue light for activation, Chronos was the fastest channelrhodopsin to date, taking about 3.6 milliseconds to close at room temperature. Even better, we found that it closed within about 1 ms at the warmer temperature of the body. However, it took some extra tricks to get



This flexible micro-LED array, fabricated at the University of Freiburg, is wrapped around a glass

when it goes from the laser diode to the fiber, when it travels down the fiber, and

Tobias Moser is a neuroscientist and otolaryngologist at the [Göttingen Campus](#) in Germany.

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**Keith Kumm**

Fascinating. Unclear how the "genetics" works - neurons don't regenerate, complicated, multi-process CRISPR probably ruled out nearterm, so possibly simple trans-wall mRNA? If so, endurance is a big question, just as in vaccines. Regarding the external DSP, modified DFT (mDFT) is chip-feasible, "channelizing" the pressure audio precisely with minimal overlap, and simultaneously in time and wavelength, supporting the idea of ultra-selective ON/OFF/pulsetrain stimuli to the disparate ion channels via some demultiplexer built into the optics. Multiple wavelength (WDM) optics might provide simultaneous, orthogonal transmission of overlapping pulsetrain channels to loci distributed across the optical cannula. This might involve annular layering or some reflection plane scheme, possibly interacting with multi-spectral wavelets cohering at differing propagation distances? Just a thought. On an entirely separate, but tightly related subject, tinnitus, is it possible that this same basic technology could be used to turn OFF (or otherwise defeat) neurochannels exciting this deleterious artifact of hearing loss? I think tinnitus could be as important an application regime as profound hearing loss, since so many more persons, and so often younger, suffer from it than from the near-total deafness that is associated with clinical cochlear implantation, applied particularly to elderly patients today. I would enjoy hearing from the author on these few ideas and questions. He and his team are to be complimented and encouraged for working on a very important problem in quality of life. I would like to listen (!) along for future developments here.

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