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EDITION 4 · OCTOBER 2026



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Dr Sukh Rashpal Sandhu | Subscribe free on LinkedIn

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INTRODUCTION

The HealthTech Herald: October 2026

By Dr Sukh Rashpal Sandhu

Edition 4 of The HealthTech Herald: 5 pieces for October 2026.

Your trusted source for the latest breakthroughs at the intersection of health and technology.

This edition brings together 5 new features written for this edition. New features are marked "Written for this edition".

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Thank you to every reader who subscribes, shares and responds.

Warm regards,
Dr Sukh Rashpal Sandhu
Author, The HealthTech Herald

THE HEALTHTECH HERALD

A Switch Made of Light: Why Optogenetics Won the 2026 Nobel Prize in Medicine

By Dr Sukh Rashpal Sandhu | Written for this edition

On 5 October 2026 the Nobel Assembly at Karolinska Institutet awarded the Nobel Prize in Physiology or Medicine to Karl Deisseroth, Peter Hegemann and Georg Nagel for the discoveries behind optogenetics. Their story runs from a green alga swimming towards the light to a method that lets scientists switch individual brain circuits on and off, and it is one of the clearest lessons in modern science about why curiosity-driven research matters.

The announcement

The 2026 Nobel season opened, as it always does, with the prize in physiology or medicine. The Nobel Assembly at Karolinska Institutet in Stockholm named three laureates, who share the prize equally:

Laureate	Born	Affiliation named by the Nobel Assembly
Karl Deisseroth	1971	Howard Hughes Medical Institute and Stanford University, USA
Peter Hegemann	1954	Humboldt University of Berlin, Germany
Georg Nagel	1953	University of Würzburg, Germany

The official citation reads that the prize was awarded "for their discoveries concerning light-gated ion channels and optogenetics". The prize amount is 12 million Swedish kronor, divided equally among the three.

The Nobel Assembly notes that Hegemann's prize-winning work was done at the Max Planck Institute for Biochemistry in Martinsried, and Nagel's at the Max Planck Institute for Biophysics in Frankfurt. Deisseroth, a psychiatrist as well as a bioengineer, did his work at Stanford.

The chair of the Nobel Committee for Physiology or Medicine, Per Svenningsson, summed up the significance in one line:

> *"Optogenetics provides opportunities for mapping the brain in a way that we could once only dream of." (Per Svenningsson, Nobel Committee)*

To understand why this prize matters for medicine, and why it has taken this long, it helps to go back to a basic problem that frustrated brain researchers for a century.

The problem: a brain we could watch but not control

The human brain weighs roughly 1.3 kilograms and contains, by the Nobel Committee's estimate, around 90 billion nerve cells, each linked to thousands of others. Nerve cells communicate by electrical impulses that last a few thousandths of a second. Cells with entirely different jobs can sit side by side, tangled together in the same small patch of tissue.

Through most of the twentieth century, neuroscience had two broad kinds of tools.

1. **Observation.** Researchers could record electrical activity, and later image blood flow or chemical signals, and see which regions were active during a task. But seeing a region light up during fear does not prove that region causes fear. It might be a bystander.
2. **Blunt intervention.** Researchers could damage an area, inject a drug or pass an electrical current through an electrode. These methods could show that a region mattered, but they affected every cell type in the area at once, and drugs act over minutes or hours, far slower than the brain's own signals.

The Nobel Committee describes the resulting picture of the brain as like a blurry photograph or a sketch map full of question marks. What was missing was a way to turn on or off one specific type of nerve cell, in a living animal, with millisecond timing.

The molecular biologist Francis Crick, who shared the 1962 Nobel Prize for the structure of DNA, had imagined exactly that. In his later career, while studying consciousness, he suggested that light might be the ideal control signal, because it is fast and can be delivered precisely. He admitted the idea sounded far-fetched. The 2026 laureates turned it into a working laboratory method.

Part one: an alga that swims towards light

The story starts not with the brain at all, but with *Chlamydomonas reinhardtii*, a single-celled green alga.

Chlamydomonas swims towards light so that it can photosynthesise. It senses light with an eyespot, a small orange patch on its surface that contains retinal, the same light-capturing molecule used in our own eyes.

In the early 1990s, Peter Hegemann, then at the Max Planck Institute for Biochemistry, measured the electrical response of the alga when its eyespot was illuminated. The response was astonishingly fast. According to the Nobel Committee's popular account, an electrical impulse appeared about half a millisecond after light reached the eyespot.

Human vision is much slower by comparison. In our eyes, light sets off a chemical chain reaction involving several steps before an ion channel finally opens and creates an electrical signal, and the whole process takes at least 10 milliseconds. Hegemann reasoned that the alga must be doing something simpler. His hypothesis was that a single

protein both captured the light and acted as the channel that let charged particles (ions) flow into the cell.

It was a bold idea. Scientists knew of many ion channels, but none that could respond to light on their own. Hegemann's proposal was met with scepticism, and for years his group struggled to isolate the protein, which fell apart when removed from its natural setting.

Part two: the frog egg experiment

The breakthrough came around the turn of the millennium, and it depended on open science. Japanese researchers had sequenced large parts of the *Chlamydomonas* genetic code and made thousands of gene sequences available. In that data, Hegemann's group found two genes that looked like they coded for light-capturing proteins.

To test them, Hegemann turned to Georg Nagel at the Max Planck Institute for Biophysics in Frankfurt. Nagel was expert in a classic technique: injecting genetic instructions into frog egg cells, which then manufacture the corresponding protein and place it in their outer membrane, where its behaviour can be measured.

When Nagel did this with the two algal genes and shone light on the eggs, the result confirmed Hegemann's decade-old hypothesis. The proteins were ion channels that opened in response to light. They were named channelrhodopsin-1 and channelrhodopsin-2.

Channelrhodopsin-2 proved to be the important one. The Nobel Committee reports that it opened within about 0.2 milliseconds of a light pulse, letting positively charged ions flow in and creating an electrical signal. The team then put the gene into human and hamster kidney cells grown in the laboratory, and those cells too became sensitive to light. The work was published in the early 2000s, with the key channelrhodopsin-2 paper appearing in 2003. In it, the authors suggested the protein could become a tool for triggering electrical impulses in cells using light.

That suggestion was the hinge of the whole story. A protein that evolved to help an alga find sunlight could, in principle, give a human scientist a light switch for any cell that carried its gene.

Part three: the psychiatrist who wanted better treatments

Karl Deisseroth came to the problem from the clinic. The Nobel Committee recounts that, during his medical training at Stanford, a placement in psychiatry left a deep impression on him. He saw how much patients with depression, schizophrenia and other conditions suffered, and how limited and side-effect-laden their treatments were. He also became fascinated by a basic question: why does the same organ work so differently in different people?

Deisseroth completed a PhD in neuroscience alongside his medical degree, but he realised that experiments on thin slices of brain tissue in a dish could only take him so far. To understand illness, he needed to study how nerve cells behave in a living brain.

When he set up his own laboratory, his team searched for a protein that could make nerve cells fire on command. Many researchers were sceptical that such a tool was achievable.

Deisseroth heard about channelrhodopsin-2, wrote to Nagel to request the DNA, and introduced it into rat nerve cells growing in a dish.

There was a real risk that sensitive nerve cells would not tolerate a foreign algal gene. They did. When blue light hit the cells, they fired a nerve signal immediately, and the signal passed on to neighbouring cells. Deisseroth's group published the result in 2005. The landmark paper, in *Nature Neuroscience*, listed Edward Boyden and Feng Zhang, both then at Stanford, alongside Ernst Bamberg, Georg Nagel and Deisseroth himself. It reported reliable, millisecond-by-millisecond control of nerve cell firing using light.

Part four: from a dish to a living brain

A switch that works in a dish is a proof of principle. A switch that works in a behaving animal is a research revolution. The method was given the name optogenetics in 2006, and in 2007 Deisseroth's group showed it working in the brains of living mice.

The approach had three essential parts, which are still the basis of the method today.

1. **Targeting.** Researchers deliver the channelrhodopsin gene in a way that means only one chosen type of nerve cell produces the protein. This is the "genetics" in optogenetics, and it is what makes the method so precise. Neighbouring cells of other types are not affected.
2. **Light delivery.** A thin optical fibre is placed in the brain through a small opening in the skull, so that light can reach the target area.
3. **Timing.** Pulses of light switch the targeted cells on, with millisecond timing that can match the brain's own rhythms.

In the 2007 experiment described by the Nobel Committee, light delivered to a particular cell type in the motor cortex made mice move their whiskers. The same year, working with other researchers, Deisseroth used optogenetics to wake sleeping mice by stimulating a recently discovered group of nerve cells suspected of controlling wakefulness. When the light came on, the mice woke, confirming that those cells were not just associated with waking but could cause it.

Other light-sensitive proteins were soon added to the toolkit, including some that switch nerve cells off rather than on, and some that respond to different colours of light. That made it possible to test not only whether a circuit could drive a behaviour, but whether the behaviour depended on it.

What optogenetics revealed

Over the following two decades, optogenetics spread to laboratories around the world. The Nobel Committee highlights several kinds of discovery that would have been very difficult without it.

Memories with an address

In 2012, Deisseroth worked with Susumu Tonegawa, the 1987 Nobel laureate, on an experiment involving memory traces, sometimes called engrams. Researchers noted which nerve cells were active as mice formed a fearful memory. Later, when those same cells were reactivated with light, the mice behaved fearfully even though they were not in

danger. The Nobel Committee describes this as the first experiment to show exactly which nerve cells are necessary for a specific memory.

Circuits for everyday drives

Optogenetic experiments have identified circuits involved in pain, thirst, eating, reward, attention, social behaviour, body clocks and fever. They have shown that complex behaviours break down into components controlled by separate circuits. In studies of parenting in mice, for example, one circuit governs gathering young into the nest and another governs grooming them.

Beyond the brain

The method has also been used outside the brain. The Nobel Committee notes Deisseroth's work showing that heart rhythm can feed back into emotional state, so that a heart forced to work harder can reinforce anxiety. Other groups have used it to study how cells in the gut shape food preferences, including why some animals prefer sugar to artificial sweeteners.

A sharper picture of disease

Perhaps most importantly for medicine, optogenetics has given researchers a way to test ideas about what goes wrong in depression, anxiety, schizophrenia, Parkinson's disease and Alzheimer's disease in animal models. Instead of only observing which areas look different, scientists can switch specific circuits on and off and see whether symptoms appear or disappear.

From laboratory tool to treatment: where things stand

It is important to be clear about what optogenetics is today. It is overwhelmingly a research tool, used mostly in animals. Its greatest value has been in generating knowledge, which drug developers, neurosurgeons and psychiatrists can then use. But the first steps towards using it directly as a treatment are under way.

Restoring some sight

The most advanced clinical use is in the eye. In retinitis pigmentosa, an inherited condition, the light-sensing rods and cones of the retina die off, but other retinal cells survive. The idea is to give some of those surviving cells a light-sensitive protein so they can take over the job of detecting light.

The Nobel Committee notes that in one clinical trial, a channelrhodopsin-like protein was introduced into the retina of a blind person, who then regained some vision. Wearing special light-emitting goggles, the person could perceive, locate and touch objects on a table. That case was reported in 2021 in *Nature Medicine* by José-Alain Sahel, Botond Roska and colleagues.

The limits are significant. That report described a single patient, a man with retinitis pigmentosa, treated in one eye with a gene for a light-sensitive protein called ChrimsonR. The vision restored was partial and worked only while he wore engineered goggles that detect changes in the scene and project matching pulses of light onto the retina. Without

the goggles, he could not see the objects. It is a remarkable proof of concept, not a cure, and larger trials are needed to show how well and how safely it works across more people.

Better hearing implants

The Nobel Committee also mentions hopes for optogenetic cochlear implants. Current cochlear implants stimulate the auditory nerve electrically, and electrical current spreads through tissue, which limits how finely the implant can separate different pitches. Light can be focused more tightly, so an implant that stimulated light-sensitive auditory nerve cells might, in theory, deliver richer sound. This work is still at an early research stage.

Why the brain itself is harder

Using optogenetics to treat brain disorders in people faces bigger hurdles. It requires gene delivery into the brain, usually using modified viruses, plus a way to get light deep into tissue, which normally means implanted hardware. Each step carries risk, and regulators will rightly demand strong evidence of safety and benefit. For now, the main route from optogenetics to patients is indirect: it reveals which circuits matter, and that knowledge can guide treatments such as deep brain stimulation, focused drugs or targeted rehabilitation.

Why the prize took twenty years

The channelrhodopsin discoveries date from the early 2000s and the first nerve cell experiments from 2005. Optogenetics has been a contender for the Nobel Prize for well over a decade. Several reasons make a long wait common in medicine.

- **Impact takes time to prove.** The Nobel Committee generally looks for discoveries whose benefit to humankind is well established. Optogenetics had to show not only that it worked, but that it transformed a field.
- **Credit is complicated.** Major methods usually emerge from many hands. The Nobel Prize can be shared by no more than three people. Optogenetics drew on contributions from many scientists over many years, including co-authors on key papers and researchers who developed earlier light-based approaches. Choosing three laureates inevitably leaves out others whose work mattered, and the scientific community often debates such choices.
- **Clinical translation is still early.** Unlike some prizes for drugs already in wide use, this one honours a method whose medical applications are mostly still ahead.

The Nobel Assembly chose to recognise the discovery of the light-gated channel itself (Hegemann and Nagel) and its transformation into a tool for neuroscience (Deisseroth). That framing celebrates the chain from basic biology to application.

The lesson in the story: curiosity first

There is a striking pattern in this prize, and it echoes many before it. Nobody set out to build a brain-control tool by studying pond algae. Hegemann wanted to know how a single cell could react to light so quickly. That question had no obvious medical value at the time. His hypothesis was doubted. It took him around a decade to confirm it, and the

confirmation relied on genome data shared openly by other scientists and on a colleague's specialised skill with frog eggs.

Only then could a psychiatrist frustrated by the limits of his treatments see the potential and take the risk of building something new.

In my view, this is a powerful reminder for anyone who funds, governs or simply votes on research. Breakthroughs often travel through territory that looks irrelevant from the outside. Basic research on light-sensing microbes became one of the most important neuroscience methods of the century. Pressure to fund only research with an immediate, predictable payoff would likely have prevented it.

The story also illustrates the value of collaboration across disciplines. A biophysicist studying algal light sensing, a membrane physiologist expert in frog egg experiments and a psychiatrist-engineer each brought something the others lacked. The prize rewards that combination as much as any single individual.

What this means for general readers

For most people, the 2026 prize will not change anything they do next week. But it is worth understanding for three reasons.

1. **It underpins today's brain science.** Many of the brain discoveries you read about, from how memories form to how anxiety circuits work, rest on experiments that used optogenetics in animals. Knowing the method helps you read those stories critically.
2. **It points to future therapies.** Vision restoration is the first clinical frontier, with hearing implants and possibly some neurological conditions to follow. Progress will be slow and evidence-driven, and many early ideas will not succeed.
3. **It shows how medicine advances.** A treatment rarely appears from nowhere. It usually rests on decades of basic research, much of it publicly funded, most of it unglamorous.

Limits and cautions

A few cautions are worth keeping in mind whenever optogenetics appears in the news.

- **Mouse results are not human results.** Most optogenetic findings come from rodents. Brain circuits are often similar across mammals, but not identical, and behaviours such as "anxiety" in a mouse are simplified models of human experience.
- **Switching a circuit is not the same as treating a disease.** Showing that a circuit can trigger a symptom in an animal does not mean that switching it off will help a person with a complex illness.
- **Gene therapies carry real risks.** Clinical optogenetics depends on introducing genes into human cells. That requires careful trials, long-term monitoring and regulatory oversight.
- **Early clinical reports are small.** The vision restoration report involved a single patient. Encouraging early results need confirmation in larger, controlled studies.

This article is for general information and is not medical advice. Anyone with a vision, hearing or neurological condition who is interested in clinical trials should speak with their own specialist.

The bottom line

The 2026 Nobel Prize in Physiology or Medicine honours a chain of discovery that began with a simple question about how an alga senses light and ended with one of the most powerful tools in modern neuroscience. Hegemann and Nagel found a protein that turns light directly into an electrical signal. Deisseroth turned that protein into a precise switch for nerve cells in living brains. Together, their work has let researchers move from watching the brain to testing how it actually works.

The medical payoff is still mostly ahead of us, and it will need patience and rigorous trials. But the scientific payoff is already enormous. And the story is a lasting argument for curiosity, open data and collaboration across fields.

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THE HEALTHTECH HERALD

A Six-Month Course Toward Curing Hepatitis B: What the Bepirovirsen Approvals Mean

By Dr Sukh Rashpal Sandhu | Written for this edition

In October 2026 the US Food and Drug Administration approved bepirovirsen, sold as Hibsago, for some adults with chronic hepatitis B, two months after Japan became the first country to approve it. In two large trials, about one in five treated patients achieved a "functional cure" that lasted after all treatment stopped. Here is what that means, who it is for, and why the result is important but not the end of the story.

Why hepatitis B still matters

Hepatitis B is a virus that attacks the liver. For many adults who catch it, the infection clears on its own. But when it is caught at birth or in early childhood, it very often becomes chronic, which means lifelong.

The global scale is enormous. The World Health Organization estimates that 240 million people were living with chronic hepatitis B in 2024, with around 0.9 million new infections each year. In the same year, the WHO estimates, hepatitis B caused about 1.1 million deaths, mostly through cirrhosis (severe scarring of the liver) and liver cancer. The burden is heaviest in the WHO Western Pacific and African regions.

For Australian readers, this is not a distant problem. Our region carries the largest share of the global burden, and many Australians living with hepatitis B were born in countries where it is common. Chronic hepatitis B can be silent for decades, which is one reason testing matters.

A safe and highly effective vaccine has been available for decades, and it is the main reason new infections in children have fallen so sharply in many countries. But a vaccine prevents infection. It cannot help the hundreds of millions of people who already live with the virus.

The problem with today's treatment

Current standard treatment for chronic hepatitis B uses daily antiviral tablets known as nucleoside or nucleotide analogues. These drugs are effective at what they do. They block the virus from copying itself, push the amount of virus in the blood down to very low levels and reduce the risk of liver damage and cancer.

But they have a crucial limitation. They suppress the virus without eliminating it. The hepatitis B virus keeps a stable copy of its genetic material inside infected liver cells, and some of its genetic material can also become integrated into the cell's own DNA. Those reservoirs keep producing viral proteins, including one called hepatitis B surface antigen, usually written as HBsAg.

Because of this, most people who start antiviral tablets stay on them indefinitely, often for life. If they stop, the virus commonly rebounds. GSK states that standard antiviral therapy typically achieves a functional cure in less than 1% of patients after a year of treatment.

That is the gap bepirovirsen was designed to address.

What "functional cure" means

The word "cure" in hepatitis B needs careful definition. Doctors and regulators generally distinguish between two ideas.

1. **Sterilising cure.** Every trace of the virus is gone from the body, including the hidden reservoirs in liver cells. No current treatment can reliably do this.
2. **Functional cure.** The virus is controlled by the person's own immune system without medication. In the bepirovirsen trials, this was defined as HBsAg becoming undetectable and the amount of virus in the blood staying below the lowest level that can be measured, for at least 24 weeks after all treatment stopped.

A functional cure is the main goal set by hepatitis B specialists for new treatments. It is linked to a better long-term outlook, although some viral genetic material can remain in the liver, and people who reach it still need follow-up.

What bepirovirsen is and how it works

Bepirovirsen is an antisense oligonucleotide, often shortened to ASO. That is a short, man-made strand of genetic material designed to latch onto a specific target in the cell's messenger RNA, the working copies of genetic instructions that cells use to build proteins.

In this case, the target is the RNA of the hepatitis B virus. GSK describes bepirovirsen as designed to recognise and reduce the production of viral RNA and viral proteins, lower the level of HBsAg in the blood and potentially help the immune system regain control. One idea behind the approach is that very high levels of viral surface protein may help to exhaust or confuse the immune response, so reducing them could give the immune system a better chance.

The drug was discovered by the US company Ionis Pharmaceuticals and licensed to GSK, which led late-stage development. It is given as an injection under the skin once a week for 24 weeks, alongside the patient's existing antiviral tablets. GSK states that, after the first dose, patients can have the option of self-administering it.

The FDA approval makes bepirovirsen the first antisense medicine approved in the United States for chronic hepatitis B, according to trade coverage of the decision.

The evidence: two large phase 3 trials

The approvals rest on two replicate phase 3 trials called B-Well 1 and B-Well 2. Their results were presented at the European Association for the Study of the Liver congress and published in the *New England Journal of Medicine* on 28 May 2026.

How the trials were designed

According to the published paper:

- The trials were randomised, double-blind and placebo-controlled, which is the strongest standard design for testing a medicine.
- They enrolled adults with chronic hepatitis B who did not have cirrhosis.
- All participants were already on stable antiviral tablets and had HBsAg levels above 100 and up to 3,000 international units per millilitre (IU/mL).
- Participants were randomly assigned in a 2:1 ratio to bepirovirsen 300 mg weekly or placebo injections for 24 weeks.
- Eligible patients then stopped their antiviral tablets at week 48.
- The main outcome was functional cure at week 72, that is, well after all treatment had ended.

Trade and public health coverage, including CIDRAP at the University of Minnesota, reported that the trials ran across 29 countries and enrolled more than 1,800 patients in total. The author list of the paper includes clinicians from Asia, Europe, the Americas, Australia and New Zealand.

The main results

Trial	Functional cure with bepirovirsen	Functional cure with placebo
B-Well 1	127 of 650 (20%)	0 of 328
B-Well 2	106 of 570 (19%)	0 of 286

Pooled, that is 233 of 1,220 treated patients, or 19%, compared with none of 614 on placebo. Both trials met their primary endpoint with statistically significant results.

The contrast with placebo is striking. Not a single placebo patient reached functional cure, which underlines how rarely it happens with standard care.

Results by starting HBsAg level

The level of surface antigen at the start mattered. GSK reports that in participants with HBsAg of 1,000 IU/mL or less, a key secondary group, the functional cure rate was 26% (200 of 768) compared with none of 393 on placebo. GSK estimates that this lower-antigen group represents roughly 45% of diagnosed chronic hepatitis B cases globally.

In exploratory analyses, which are less reliable than the main results, GSK reports that bepirovirsen reduced HBsAg to 100 IU/mL or less in 49% of all treated participants as of week 72. Lower antigen levels have been linked in the medical literature with better immune control, but this was not the trials' primary measure.

Safety

Bepirovirsen is not a gentle drug, and the safety data deserve careful reading. In the pooled analysis to week 72 reported in the journal:

- Adverse events of any kind were reported in 91% of patients on bepirovirsen and 73% on placebo.
- Serious adverse events were reported in 7% and 4% respectively.
- During the treatment period, adverse events of grade 3 or higher (severe) occurred in 16% on bepirovirsen and 3% on placebo.
- The most common severe event with bepirovirsen was a rise in the liver enzyme alanine aminotransferase (ALT), seen at grade 3 in 6% of treated patients.

GSK lists the three most frequent adverse events as redness at the injection site, local pain and a temporary rise in a liver enzyme.

Liver enzyme rises are, in part, an expected feature of this kind of treatment. When the immune system attacks infected liver cells, the liver becomes inflamed for a time, and that can show up as a "flare". Some flares may be part of how a cure is achieved. But flares can also be dangerous, which is why the prescribing information is so specific.

What the FDA approved

On 20 October 2026, GSK announced that the FDA had approved Hibsago (bepirovirsen). The approval was granted under Priority Review and Breakthrough Therapy designation.

Who it is for

The approved use is narrower than "everyone with hepatitis B". According to GSK's announcement, bepirovirsen is approved in combination with antiviral tablets for adults with chronic hepatitis B who:

- do not have cirrhosis,
- have been on antiviral tablet therapy for at least six months,
- have suppressed levels of virus in the blood, and
- have HBsAg levels of 3,000 IU/mL or less, as measured by an FDA-authorised test.

The need to measure HBsAg precisely is why diagnostic tests are part of the story. GSK notes that several tests are available, including a recently authorised FDA test, and trade coverage reported that a Roche quantitative surface antigen assay was authorised as a companion diagnostic.

The boxed warning

The US label carries a boxed warning, the FDA's most prominent safety alert, for two risks:

1. **Hepatic flares during treatment.** GSK's prescribing summary states that the drug may cause flares with ALT rising more than 20 to 40 times the upper limit of normal, associated with a fall in HBsAg. Most occurred in the first 12 weeks and resolved when treatment was interrupted or stopped. Liver tests must be checked before treatment and frequently during it.

2. **Low platelet counts (thrombocytopenia).** The drug may cause sudden drops in platelets, the blood cells that help clotting, which can be life-threatening. Platelets must be checked before starting and at least weekly during treatment. The drug must not be started if platelets are below a set threshold and must be stopped permanently if they fall too far.

The drug is contraindicated in people with cirrhosis or acute liver impairment.

In plain terms, this is a specialist medicine that needs careful monitoring. It is not a casual add-on.

Japan moved first

Japan's Ministry of Health, Labour and Welfare approved bepirovirsen in August 2026 (GSK announced it on 24 August), the first approval anywhere in the world. GSK said that review was accelerated by Japan's SENKU designation, which is given to innovative medicines that address a high unmet need. GSK also notes Breakthrough Therapy and Priority Review designations in China and says further regulatory submissions are under way in multiple jurisdictions.

Australian readers should not assume the drug is available here. GSK has said decisions in other countries are expected over the coming months, but availability, approval and subsidy are separate steps in each country. Anyone interested should talk to their liver specialist about current options and clinical trials.

Expert reaction

The response from hepatitis specialists has been positive but measured.

When the drug was approved, GSK quoted Dr Norah Terrault, Chief of Gastroenterology and Hepatology at USC Keck Medical Center and a co-author of the trial report:

> *"This is the start of a new era in treating people living with CHB." (Dr Norah Terrault)*

When the trials were published, the *New England Journal of Medicine* ran an accompanying editorial by Dr Anna Lok, a leading hepatitis researcher. As reported by CIDRAP, she described the results as impressive, while cautioning that they cannot be generalised to groups excluded from the trials, particularly people with cirrhosis.

That caution matters, because people with cirrhosis are among those at highest risk of serious liver complications.

Reading the result carefully

A 19% functional cure rate is, by the standards of hepatitis B, a genuine breakthrough. For decades the field has had almost nothing that achieved this after a fixed course of treatment. But a balanced reading needs to hold several facts at once.

Four in five do not reach functional cure

Most treated patients in the trials did not reach the primary goal. They still benefited from continued antiviral treatment, and many had large falls in HBsAg, but the headline result applies to a minority. Doctors will want better ways to predict who is most likely to respond. Starting HBsAg level is one clear predictor.

The eligible group is limited

The approval excludes people with cirrhosis, people with higher HBsAg levels and people not already well controlled on antiviral tablets. Worldwide, a large share of people with hepatitis B have never been diagnosed, let alone started treatment. A new drug does not reach people who do not know they are infected.

Durability needs longer follow-up

The primary outcome was measured at week 72, 24 weeks after antiviral tablets were stopped. That is a demanding test, but chronic hepatitis B unfolds over decades. Longer follow-up will show how durable the functional cures are and whether they translate into lower rates of cirrhosis and liver cancer over time.

Monitoring is demanding

Weekly platelet checks and frequent liver tests during treatment require good access to clinics and laboratories. That could limit use in exactly the lower-resource settings where hepatitis B is most common, unless health systems plan for it.

Cost and access are unknown for most countries

Pricing and reimbursement decisions will shape how many people actually receive the drug. Those decisions are still to come in most places.

Why hepatitis B is harder than hepatitis C

Readers may remember that hepatitis C became curable for most people in the 2010s, with courses of antiviral tablets lasting a few months. It is natural to ask why hepatitis B has been so much harder.

The answer lies in how each virus lives inside the liver. Hepatitis C does not keep a long-lasting copy of its genetic material in the nucleus of liver cells, so once its copying is blocked for long enough, it can be eliminated. Hepatitis B does keep such a copy, and fragments of its genetic material can also become built into the cell's own DNA. Those reservoirs are why antiviral tablets suppress hepatitis B but rarely clear it, and why a functional cure, rather than complete elimination, is the realistic goal for now.

Bepirovirsen does not remove those reservoirs. Its approach is to cut the output of viral RNA and proteins and, in some patients, give the immune system the chance to take over control.

Why the science is still a milestone

Even with those limits, there are good reasons for specialists to be excited.

1. **It proves functional cure is achievable after a fixed course.** That shifts the goal of hepatitis B treatment from lifelong suppression towards finite therapy for at least some patients.
2. **It validates a new class of medicine for this disease.** Antisense drugs work differently from the antiviral tablets used now. That opens the door to combinations, for example with immune-boosting treatments or other drugs that lower viral proteins.
3. **It supports better testing.** Quantitative HBsAg testing becomes clinically important for deciding who is eligible, which may improve how patients are monitored more generally.
4. **It may reduce the burden of lifelong treatment.** For patients who achieve functional cure, stopping daily tablets is a meaningful change, although follow-up is still required.

What this means for people living with hepatitis B

If you or someone close to you has hepatitis B, a few practical points are worth considering. This is general information, not medical advice.

- **Do not stop your current treatment.** Stopping antiviral tablets without medical supervision can cause dangerous flares.
- **Ask about your HBsAg level.** Quantitative HBsAg is now a more important number. Your specialist can explain whether it is measured as part of your care.
- **Ask about trials and availability.** New treatments, including combinations, are being studied, and access varies by country.
- **Keep up regular monitoring.** Liver cancer surveillance and liver checks remain important whether or not new drugs become available.
- **Encourage testing and vaccination.** Family members and close contacts should be tested and, if not immune, vaccinated.

The bottom line

Bepirovirsen is the first medicine shown in large phase 3 trials to produce a functional cure in a meaningful share of people with chronic hepatitis B after a fixed six-month course. Japan approved it in August 2026, and the FDA followed on 20 October 2026, for a defined group of adults without cirrhosis whose virus is already suppressed and whose surface antigen levels are low enough.

About one in five treated patients in the trials reached functional cure, compared with none on placebo, and the rate was higher in those with lower starting antigen levels. The drug carries serious risks, including liver flares and drops in platelets, and needs close monitoring. Many patients will not be eligible, and most of those treated will not reach the cure threshold.

That is still a significant change. For a disease that has relied on lifelong suppression, the prospect of finite treatment for some is a real step forward. The next tests are durability, broader eligibility, combinations that lift the cure rate and, above all, access for the hundreds of millions of people who need it.

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THE HEALTHTECH HERALD

Speaking Through a Brain Implant: Where Communication Technology for Paralysis Stands in Late 2026

By Dr Sukh Rashpal Sandhu | Written for this edition

Between August and October 2026, brain-computer interfaces for people who have lost the ability to speak moved forward on three fronts: a peer-reviewed study showing one implant can decode speech and gestures together, new US regulatory approvals that widen a company's clinical trial, and a widely shared company video of a man with ALS speaking in a synthesised version of his own voice. Here is what each development shows, how much weight it can bear and what still stands between these devices and everyday use.

Why this technology matters

Imagine being fully alert and thinking clearly, but unable to speak, type or gesture. That is the reality for some people with advanced amyotrophic lateral sclerosis (ALS), also known as motor neurone disease, for some survivors of brainstem stroke, and for people with certain spinal cord injuries. Many communicate by moving their eyes across a letter board, one letter at a time, often at only a few words per minute.

A brain-computer interface, or BCI, aims to bypass the damaged pathway between brain and muscles. Electrodes record activity from the parts of the brain that plan movement. Software, increasingly based on machine learning, learns which patterns correspond to which intended sounds, words or actions. The output can be text on a screen, a synthesised voice or commands for a computer or phone.

The idea is decades old. What has changed in the past few years is performance. Speech BCIs have gone from laboratory curiosities to systems that, in a small number of research participants, can produce dozens of words per minute.

How a speech decoder learns

It helps to picture what a speech BCI is actually doing, because it is easy to imagine something closer to mind reading than the reality.

It reads intended movement, not thoughts

Most speech BCIs record from motor areas of the brain, the regions that plan and send commands to the muscles of the lips, tongue, jaw and voice box. When a person with

paralysis tries to say a word, those areas still produce patterns of activity, even though the commands never reach the muscles. The BCI captures those patterns.

That distinction matters. These systems are generally designed to decode what a person is attempting to say, not their private thoughts. The person has to make the effort to speak for the system to work.

Training takes time and effort

Before a system can decode, it must learn. In a typical research setup, the participant is shown words or sentences and asked to attempt to say them, again and again. The software records the brain activity for each attempt and learns which patterns go with which speech sounds or words.

Over many sessions, the decoder builds a model of the person's own brain activity. Modern systems use machine learning models for this, often combined with language models that predict likely word sequences, much as phone keyboards suggest the next word. That combination helps correct errors and speeds up output.

Output can take several forms

Once decoded, the intended speech can be shown as text, spoken aloud by a synthesised voice or used to animate an on-screen avatar. If a person banked recordings of their voice before losing speech, those recordings can be used to make the synthesised voice sound more like them.

Why performance varies

Performance depends on where electrodes are placed, how many there are, how stable the signals remain, how much training data is available and how the person's condition changes. That is one reason results from one participant cannot simply be assumed for another.

How we got here: the 2023 and 2024 milestones

To understand the 2026 news, it helps to recall a few earlier peer-reviewed landmarks.

- August 2023, *Nature*: A Stanford-led team, with Frank Willett as first author, reported a speech neuroprosthesis using small electrode arrays placed in the brain. A participant with ALS reached a decoding rate of 62 words per minute.
- August 2023, *Nature*: In the same issue, a University of California, San Francisco (UCSF) team led by neurosurgeon Edward Chang, with Sean Metzger as first author, reported a system using a thin sheet of electrodes laid on the brain's surface. It decoded speech to text, synthesised a voice and animated a digital avatar's face for a woman with severe paralysis after a brainstem stroke.
- August 2024, *New England Journal of Medicine*: A University of California, Davis team, with Nicholas Card as first author, described a speech neuroprosthesis that calibrated rapidly and achieved very high word accuracy for a man with ALS, who used it to communicate in daily life.

- 2025, *Nature Neuroscience*: The UCSF group, with Kaylo Littlejohn as first author, reported a streaming "brain-to-voice" system designed to reduce the delay between intended speech and synthesised sound.

Each of these studies involved one or a handful of participants. They proved the concept rather than establishing a treatment. But together they showed that useful, fast communication from brain signals is achievable.

Development one: speech and gestures from a single implant

What the study did

On 14 September 2026, *Nature Neuroscience* published an open-access paper from Edward Chang's group at UCSF, with Samantha Brosler, Jessie Liu and Alexander Silva as joint first authors. Its title captures the advance: simultaneous speech and gesture decoding for multimodal communication in paralysis.

Human communication is not only words. We nod, shrug, wave and point, often while speaking. People with paralysis frequently lose both speech and gesture. Earlier BCI research mostly decoded one behaviour at a time, either speech or a particular movement.

The researchers recorded from three participants with paralysis affecting both the vocal tract and limbs (two with severe limb paralysis after brainstem stroke, one with ALS and moderate limb paralysis). Each had a single high-density electrocorticography (ECoG) implant, a thin sheet of electrodes placed on the surface of the sensorimotor cortex, the strip of brain that controls movement.

According to the paper and the accompanying Nature press release:

1. The team first showed that each implant could reliably capture signals linked to a range of movements, including upper-limb movements and speech-related mouth and face movements, in all three participants.
2. In two participants, the team then ran a speech decoder and a gesture decoder in parallel to drive a personalised full-body virtual avatar.
3. Participants attempted specific gestures, responded to conversational prompts, or did both at once.
4. In conversational tasks, the system decoded speech and gestures accurately. One participant achieved median accuracies of 100% for both speech and gesture decoding across three conversational blocks.

The key finding

The most interesting result was a practical lesson. Decoders trained only on isolated behaviours, speech alone or gestures alone, did not automatically work well when participants tried both at the same time. The brain's activity patterns shift when two behaviours happen together. When the researchers trained their models on a mix of isolated and simultaneous attempts, performance improved across all situations.

That matters for real-world design. A future communication device will need to cope with the messy, overlapping way people actually communicate, not just tidy laboratory tasks.

The limits

The authors and the Nature press release are clear about what remains to be done. Testing in more participants, with larger sets of words and gestures, will be needed before the technology can be used in practice. The study involved three people, and the avatar experiments two. Accuracy figures come from structured tasks with limited vocabularies, not open-ended daily conversation.

ECoG implants also require brain surgery. The electrodes in this research are connected to external equipment, which is a long way from a discreet device someone could use at home every day.

Development two: regulators widen a trial

Paradromics and the Connect-One study

While academic groups publish peer-reviewed results, several companies are trying to turn BCIs into approved medical devices. One of them, Paradromics, based in Austin, Texas, is developing a fully implantable, wireless system called Connexus for people with severe communication impairments.

According to coverage by HLTH and Medical Economics:

- Paradromics received an investigational device exemption from the US Food and Drug Administration in November 2025, which allowed it to begin a clinical study in people.
- The company completed its first long-term human implantation in June 2026, as part of an early feasibility study called Connect-One.
- The device records signals from individual nerve cells and sends them wirelessly to a receiver worn on the chest. A computer then converts them into text, synthesised speech or commands.
- In late August 2026, the FDA approved a change allowing a wider range of computing devices, including personal phones and laptops, to be used with the investigational system.
- In September 2026, the company reported that a participant with a progressive motor neurone disease had used the system to select words, answer open-ended questions and speak with her grandchildren on a phone call.
- In late October 2026 (reported on 27 October), the company announced FDA approval to remove an earlier requirement that participants live within four hours of a study site, allowing recruitment across the continental United States.

The study is designed to evaluate safety and function over six years. Coverage lists the clinical sites as UC Davis Health in California, Mass General Brigham in Massachusetts and University of Michigan Health.

How to read these approvals

It is important to be precise. These FDA decisions are approvals of changes to an investigational study, not approval of the device for sale. Connexus remains experimental.

The September communication report came from the company, not from a peer-reviewed journal. Early feasibility studies are designed mainly to test safety and basic function in a small number of people.

Still, the changes are meaningful. Allowing everyday consumer devices to connect to the system is a step towards practical home use. And removing the geographic restriction makes it easier for people far from a study centre, who may be among those most in need, to take part.

Development three: a company video that moved people

What Neuralink showed

In September 2026, Neuralink, the company co-founded by Elon Musk, shared a video from its VOICE clinical study. According to coverage by Notebookcheck and others, it showed a participant with ALS saying "I love you" to his wife through a brain implant, in a synthesised voice rebuilt from recordings made before his illness.

Coverage reports that:

- The VOICE study is registered on ClinicalTrials.gov and is recruiting people who have lost the ability to speak because of ALS, primary lateral sclerosis, stroke or spinal cord injury.
- The device has an FDA Breakthrough Device Designation, which is intended to speed development and review but is not an approval.
- Neuralink attaches an investigational disclaimer to participant videos.

How to read it

The video is powerful, and it is easy to see why it spread widely. But it is a company communication, not a scientific publication. No peer-reviewed data from the VOICE cohort appear to have been published by the end of October 2026. That means key questions cannot yet be answered from public evidence:

1. How accurate is the decoding across many sentences, not just a short phrase?
2. How fast is it, and how much delay is there before speech is produced?
3. How much calibration does each user need, and does performance hold up over months?
4. What were the surgical and device-related adverse events?

None of this is a criticism of the participants or the technology's potential. It is simply the difference between a demonstration and evidence. Peer-reviewed reports from academic groups, such as the 2023 and 2024 papers above and the September 2026 *Nature Neuroscience* study, are the benchmark against which company claims should eventually be measured.

Comparing the main approaches

BCIs for communication use different kinds of hardware, each with trade-offs.

Approach	Where electrodes sit	Strengths	Trade-offs

Approach	Where electrodes sit	Strengths	Trade-offs
Surface arrays (ECoG)	On the surface of the brain	Covers a wide area, signals are relatively stable over time	Requires opening the skull; records broader activity, not single cells
Penetrating microelectrodes	A few millimetres into brain tissue	Records individual nerve cells, high detail	Smaller coverage; tissue response can degrade signals over time
Fully implanted wireless systems	Varies by company	No wires through the skin, more practical for home use	Newer, with less long-term human data

Researchers are also exploring less invasive options, such as electrodes placed inside blood vessels near the brain, which avoid open brain surgery but record weaker signals.

The big questions still to answer

Brain-computer interfaces for communication have made extraordinary progress. Before they become routine treatments, several issues need solid answers.

Safety over years

Any device implanted in the brain carries risks, including infection, bleeding and the body's reaction to foreign material. Studies like Connect-One are designed to track safety over years, and regulators will want that evidence.

Durability of performance

Signals can change as the brain adapts, as the disease progresses or as tissue responds to the device. Systems need to keep working with minimal recalibration.

Usability at home

A system that works in a laboratory with engineers present is very different from one a person and their family can use independently. Wireless connections to everyday phones and laptops, as in the Paradromics change, are part of that challenge.

Privacy and consent

Devices that decode intended speech raise privacy questions. Researchers have explored safeguards so that systems decode only speech a person intends to share. As devices move towards wider use, clear rules on data ownership, security and consent will be essential.

Cost and access

BCIs involve neurosurgery, specialised hardware, software and ongoing support. Even if approved, they may be expensive. Health systems and insurers will need to decide who can access them, and how.

Equity in research

Many early participants are in the United States and near major academic centres. Removing geographic restrictions helps, but international access, including in Australia, will depend on future trials and regulatory decisions in each country.

What this means for people living with paralysis and their families

This is general information, not medical advice.

- **These systems are experimental.** No speech BCI discussed here is approved for routine clinical use. Access is through clinical trials.
- **Trials have strict eligibility.** Studies typically require specific diagnoses, levels of impairment and the ability to attend regular sessions.
- **Existing tools still matter.** Eye-tracking systems, speech-generating devices and voice banking before speech is lost are available now and can greatly improve communication. Voice banking, recording one's own voice early, may also allow more natural synthesised speech in future systems.
- **Ask your care team.** Neurologists and speech pathologists can advise on current communication options and on whether any relevant trials exist.

A reflection on hope and evidence

In my view, this field shows both the best and the most challenging sides of modern health technology. The best is obvious: people who had lost their voice are, in research settings, communicating with family again. Few medical advances are as directly human.

The challenge is that powerful stories can run ahead of evidence. A short video can create expectations that a device is nearly ready for everyone, when the reality is a small, carefully monitored trial. Responsible coverage, and responsible company communication, should keep the two clearly separated: celebrate the milestone, and be honest about how much is still unknown.

The healthiest pattern is the one visible in 2026. Academic groups publish detailed, peer-reviewed results that anyone can scrutinise. Companies run regulated trials and report results in time. Regulators approve changes step by step, based on safety data. And participants, who take real risks to help others, are treated as partners.

The bottom line

Brain-computer interfaces for communication took several steps forward between August and October 2026. A peer-reviewed *Nature Neuroscience* study from UCSF showed that one implant can decode speech and gestures together, with training on combined behaviour proving important. Paradromics gained FDA approval to connect its investigational system to everyday devices and to recruit participants nationwide. Neuralink shared a moving demonstration of a man with ALS speaking in a recreated version of his voice, although peer-reviewed data from that study had not yet been published.

None of these systems is yet an approved treatment. Small participant numbers, surgical risk, durability, privacy and cost all remain to be addressed. But the direction is clear, and for people who have lost the ability to speak, the prospect of a natural, fast and personal voice is closer than it has ever been.

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THE HEALTHTECH HERALD

A Vaccine Built From Your Own Tumour: The First Phase 3 Win for a Personalised mRNA Cancer Therapy

By Dr Sukh Rashpal Sandhu | Written for this edition

At the European Society for Medical Oncology Congress in Madrid in October 2026, an Australian-led team presented the first phase 3 trial to show that a personalised mRNA cancer therapy, designed from each patient's own tumour, can reduce the chance of melanoma coming back after surgery. It is a landmark for a technology most people first met through COVID-19 vaccines. It also comes with important caveats about survival data, cost and how widely the approach will work.

The trial in one paragraph

The trial is called INTerpath-001. It tested intismeran autogene, an investigational therapy developed jointly by Merck (known as MSD outside the United States and Canada) and Moderna, given alongside the established immunotherapy pembrolizumab (Keytruda). The patients had high-risk melanoma that had been completely removed by surgery. Compared with pembrolizumab alone, adding intismeran reduced the risk of the cancer returning, or of death, by 42% at a median follow-up of about two years. The results were presented on 24 October 2026 in the congress's opening Presidential Symposium by Professor Georgina Long, medical director of Melanoma Institute Australia, and the companies say they have been accepted for publication in the *New England Journal of Medicine*.

Why melanoma, and why Australia cares

Australia has one of the highest rates of melanoma in the world. Cancer Australia estimates that 17,443 new cases of melanoma of the skin would be diagnosed in Australia in 2025, making it the third most commonly diagnosed cancer, and that about 1,455 Australians would die from it that year. It estimates that one person in 19 will be diagnosed with melanoma by the age of 85.

Most melanomas are caught early and cured by surgery alone. But for people whose melanoma is thicker, has spread to nearby lymph nodes, or has reached other sites and been fully removed, the risk of it returning is substantial. When melanoma comes back, it often spreads to vital organs such as the lungs, liver or brain, where it becomes much harder to treat.

For these high-risk patients, doctors already offer "adjuvant" treatment, meaning extra treatment after surgery to lower the chance of recurrence. Since the late 2010s, that has

usually meant about a year of immunotherapy with a checkpoint inhibitor such as pembrolizumab. It works, but many patients still relapse. That is the gap INTerpath-001 set out to close.

It is fitting that Australian researchers played a leading role. Melanoma Institute Australia, based in Sydney, is one of the world's major melanoma research centres, and several Australian hospitals contributed to the earlier phase 2b study that led to this trial.

How a personalised mRNA cancer therapy works

The idea behind intismeran is elegant, and surprisingly intuitive once the pieces are laid out.

Cancer cells carry unique "typos"

As a cancer grows, its DNA picks up mutations. Some of those mutations change the proteins the cancer cells make. Fragments of these altered proteins can appear on the surface of the cancer cell, where the immune system may be able to spot them. These fragments are called neoantigens, meaning "new" antigens that are not present on healthy cells.

Every patient's cancer has its own set of mutations, so every patient's neoantigens are different. That is why a single, off-the-shelf vaccine struggles to target them.

Reading the tumour and designing a blueprint

Intismeran is made individually for each patient. According to Merck and Moderna:

1. A sample of the patient's tumour, taken during surgery, is sequenced alongside a sample of their normal DNA.
2. Computer analysis compares the two to identify the mutations unique to the cancer and predicts which neoantigens are most likely to provoke a strong immune response.
3. A personalised "blueprint" is created, and a synthetic mRNA molecule is manufactured that codes for up to 34 of that patient's neoantigens.

Teaching the immune system

When the mRNA is injected, the body's cells read its instructions and make the neoantigen fragments. The immune system is then exposed to these fragments and can generate T cells, a type of white blood cell, that recognise and attack cancer cells carrying the same markers.

This is the same basic mRNA principle used in COVID-19 vaccines, where mRNA instructs cells to make a harmless piece of the virus. Here, the "target" is the patient's own cancer.

Why combine it with pembrolizumab?

Pembrolizumab is a checkpoint inhibitor. It blocks a brake on the immune system called PD-1, which cancers can exploit to switch off T cells. The logic of the combination is that intismeran trains T cells to recognise the cancer, while pembrolizumab helps keep those T cells active.

How this differs from a preventive vaccine

The word "vaccine" can be misleading here, which is why the companies describe intismeran as an "individualised neoantigen therapy". Several differences are worth understanding.

1. **It treats, rather than prevents.** COVID-19 and influenza vaccines are given to healthy people to prevent infection. Intismeran is given to people who have already had cancer, to lower the chance it returns.
2. **It is made for one person only.** A preventive vaccine is mass-produced and identical for everyone. Each course of intismeran is designed and manufactured for a single patient, from their own tumour.
3. **It is given alongside another treatment.** In this trial, intismeran was always combined with pembrolizumab. The trial did not test it on its own.
4. **It is given after surgery, when little or no visible cancer remains.** The aim is to mop up microscopic cancer cells that might otherwise grow back. That setting, where the immune system faces a small number of cancer cells, may be where this kind of therapy has its best chance.

The approach also differs from earlier cancer vaccine attempts, many of which targeted proteins shared by many tumours and found on some healthy cells. Those vaccines largely disappointed in large trials. Targeting neoantigens unique to the cancer, and choosing dozens of them per patient, is designed to give the immune system clearer, more specific targets and to make it harder for the cancer to escape by losing any single one.

The road to phase 3

INTerpath-001 did not come out of nowhere. It followed a randomised phase 2b study called KEYNOTE-942, published in *The Lancet* in 2024, in 157 patients with resected stage IIIB to IV melanoma. Patients were randomly assigned in a 2:1 ratio to intismeran plus pembrolizumab or pembrolizumab alone.

A five-year update, published in the *Journal of Clinical Oncology* in 2026, reported that the combination continued to reduce the risk of recurrence (hazard ratio 0.51) and of distant spread (hazard ratio 0.41) compared with pembrolizumab alone, with a favourable but not statistically conclusive trend in overall survival. The authors described the five-year analyses as descriptive. Australian clinicians from Perth, Sydney and Brisbane were among the study's authors.

Phase 2b results are encouraging but involve relatively small numbers. A phase 3 trial, large and designed to test the effect definitively, was the necessary next step.

How INTerpath-001 was designed

According to Merck and Moderna:

- **Type of trial:** Randomised, double-blind, placebo-controlled and active-comparator-controlled, run globally.
- **Patients:** 1,137 people with high-risk (stage IIB to IV) cutaneous melanoma that had been completely removed by surgery.

- **Randomisation:** 2:1, to intismeran plus pembrolizumab, or placebo plus pembrolizumab.
- **Intismeran:** 1 mg every three weeks, for up to nine doses.
- **Pembrolizumab:** 400 mg every six weeks, for about one year.
- **Main outcome:** Recurrence-free survival, meaning the time until the cancer returns anywhere or the patient dies.
- **Key secondary outcomes:** Distant metastasis-free survival, overall survival, safety and quality of life.

The use of a placebo injection means neither patients nor doctors knew who was receiving the personalised therapy, which reduces bias.

What the trial found

Fewer recurrences

At the primary analysis, after a median follow-up of 24.6 months:

Measure	Intismeran plus pembrolizumab	Pembrolizumab alone
Recurrence or death (events)	136 patients (18.0%)	111 patients (29.2%)
Two-year recurrence-free survival	82.2%	70.0%
Distant metastasis or death (events)	89 patients (11.8%)	71 patients (18.7%)
Two-year distant metastasis-free survival	88.5%	80.2%

Event numbers are as reported by CancerNetwork from the ESMO presentation.

The hazard ratio for recurrence or death was 0.58 (95% confidence interval 0.45 to 0.75), a 42% relative reduction. The hazard ratio for distant metastasis or death was 0.62 (0.45 to 0.85), a 38% relative reduction. Both results were statistically significant.

In plain terms, about 82 in every 100 patients on the combination were alive and free of recurrence at two years, compared with about 70 in every 100 on pembrolizumab alone.

The benefit appeared after six months

One of the most interesting findings concerns timing. In the first six months, the two groups did about equally well. In a pre-specified analysis, after six months the combination reduced the risk of recurrence or death by 58% (hazard ratio 0.42) and the risk of distant spread or death by half (hazard ratio 0.50).

That pattern makes biological sense. The personalised therapy has to be manufactured after surgery; CancerNetwork reported that the median time to the first dose was 64 days. Then the immune system needs time to respond to a course of injections. Any benefit would be expected to show only after that.

Overall survival is not yet proven

This is the most important caveat. Overall survival, meaning whether patients actually live longer, is the result that matters most to many people. According to CancerNetwork's

report of the presentation, the overall survival data were immature, with a hazard ratio of 0.77 (95% confidence interval 0.45 to 1.31), which did not meet the pre-specified threshold for statistical significance. The companies describe a favourable trend. Follow-up continues.

That is common at this stage of an adjuvant trial, because deaths take longer to accumulate than recurrences. But it means the claim that the therapy saves lives has not yet been demonstrated.

Safety

According to Merck and Moderna:

- Treatment-related adverse events occurred in 95.8% of patients on the combination and 90.0% on pembrolizumab alone.
- Severe (grade 3 to 5) treatment-related adverse events occurred in 14.2% and 13.4% respectively, a very similar rate.
- The most common adverse events in the combination arm were fever (52.2%), pain at the injection site (51.0%) and fatigue (38.5%).

The companies say the safety profile was consistent with earlier studies and that no new safety signals were seen. Fever and injection site pain are familiar reactions to mRNA vaccines.

Can every patient's therapy actually be made?

A personalised therapy raises a practical question: can it be manufactured reliably and quickly enough for everyone who needs it? CancerNetwork's report of the presentation offers some answers.

- Of 1,749 patients screened, 302 (17.3%) could not enter the trial because their tumour sample was inadequate.
- Among those randomised to the combination, intismeran was successfully manufactured for more than 99% of patients.
- For 91% of randomised patients, the maximum of 34 neoantigens was identified.
- The median time to the first dose was 64 days.

These are strong manufacturing results for a bespoke product. But the screening figure is a reminder that the approach depends on having good-quality tumour tissue, which is not always available.

What experts are saying

Professor Long described the approach as a shift away from treating cancers by where they start in the body. In the companies' announcement, she said:

> *"For decades, cancer treatment has largely been defined by where a tumor originates in the body." (Professor Georgina Long, Melanoma Institute Australia)*

She went on to describe a more individualised approach that tailors treatment to the unique mutations of each patient's tumour.

Not every reaction was celebratory. Some financial coverage reported that the results landed below some investors' expectations, and the overall survival result is not yet mature. That contrast is useful. A result can be scientifically important and clinically meaningful while still leaving big questions open.

Reading the result carefully

What INTerpath-001 shows

- A personalised mRNA therapy, added to standard immunotherapy, significantly reduced melanoma recurrence and distant spread over about two years in a large, randomised, blinded phase 3 trial.
- The benefit was seen across most pre-specified subgroups, including different disease stages and age groups.
- Serious side effects were not meaningfully more common than with pembrolizumab alone.
- The therapy could be manufactured for almost all patients who were randomised to receive it.

What it does not yet show

- That patients live longer. Overall survival is immature and not statistically significant.
- How durable the benefit is beyond two years in this population, although the phase 2b five-year data are encouraging.
- Whether the approach works in other cancers. Trials in lung cancer and other tumours are under way, but each must be proven separately.
- How it compares with other ways of strengthening adjuvant treatment.

A note on subgroups

CancerNetwork reported that confidence intervals crossed "no effect" in several smaller subgroups, including a US subgroup analysis of distant spread based on only 10 events. Small subgroups often produce unstable results, and readers should be cautious about drawing conclusions from them in either direction.

Big practical questions

Regulatory approval

Merck and Moderna say they are engaging with regulatory authorities. Intismeran is not approved anywhere. Any approval will depend on regulators' review of the full data, possibly including more mature survival results. In Australia, that would mean a Therapeutic Goods Administration decision, followed by a separate decision on public subsidy.

Cost and manufacturing capacity

Each dose course is made for one patient. That requires tumour sequencing, computational design and individual manufacturing at scale. The costs are not yet public, but bespoke therapies are generally expensive. Health systems will need to weigh the benefit against the price.

Time and logistics

The trial showed that a median of about two months to the first dose is achievable in a large, well-organised study. Routine care would require tight coordination between surgeons, pathologists, laboratories and oncologists. That is easier in major cancer centres than in regional areas.

Equity

Patients who live far from major centres, or whose tumour samples are inadequate, may be less able to benefit. Planning for fair access should start early.

What this means for patients and families

This is general information, not medical advice.

- **If you have had high-risk melanoma removed,** talk to your oncologist about current adjuvant options. Intismeran is not yet approved, but your specialist can tell you about clinical trials.
- **Tumour samples matter.** Personalised therapies depend on good tissue samples. That is one more reason to have surgery and pathology done in experienced centres where possible.
- **Prevention and early detection still come first.** Sun protection, regular skin checks and early review of changing spots remain the most powerful ways to reduce melanoma deaths in Australia.

Why this result matters beyond melanoma

In my view, INTerpath-001 is significant less for the specific numbers and more for what it proves is possible. For decades, the idea of a vaccine designed from a patient's own tumour sounded like science fiction or, at best, a niche experiment. This trial shows that such therapies can be designed, manufactured for almost every patient in a global trial, and deliver a clear, statistically robust reduction in recurrence.

It also reflects how much mRNA technology matured during the COVID-19 pandemic. The ability to manufacture mRNA quickly and flexibly is exactly what personalised cancer therapy needs.

The next tests are demanding: survival benefit, durability, success in other cancers, affordability and fair access. But the principle has now passed its first major test.

The bottom line

In INTerpath-001, adding intismeran autogene, a personalised mRNA therapy made from each patient's tumour, to pembrolizumab after surgery for high-risk melanoma reduced

the risk of recurrence or death by 42% and the risk of distant spread or death by 38%, compared with pembrolizumab alone, over about two years. The benefit emerged after the first six months, consistent with the time needed to make the therapy and for the immune system to respond. Serious side effects were similar between groups.

Overall survival has not yet been shown to improve, and the therapy is not approved anywhere. Cost, manufacturing at scale and access are open questions. Even so, this is the first phase 3 trial to show that a personalised mRNA cancer therapy can change outcomes, and it was led by Australian melanoma researchers. That is a milestone worth understanding.

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THE HEALTHTECH HERALD

Turning Down the Source of Kidney Damage: New Treatments Reshape IgA Nephropathy

By Dr Sukh Rashpal Sandhu | Written for this edition

In late October 2026, IgA nephropathy, a common cause of kidney failure in young adults, was the centre of attention at the world's largest kidney meeting. A phase 3 trial of povetacicept, published in the *New England Journal of Medicine* on 23 October, roughly halved protein leakage into the urine, and the US regulator granted accelerated approval to another drug for the condition on 21 October. Here is what these advances mean, and why kidney specialists are excited but still waiting for the most important answer.

A disease many people have never heard of

IgA nephropathy, sometimes called Berger's disease, is a kidney disease driven by the immune system. Its name comes from immunoglobulin A, or IgA, an antibody that normally helps protect the lining of the gut and airways.

In people with IgA nephropathy, an abnormal form of IgA is produced. It lacks some of the sugar molecules normally attached to it, which is why it is called galactose-deficient IgA1, shortened to Gd-IgA1. The immune system makes antibodies against this abnormal IgA, and the two clump together into immune complexes. These complexes lodge in the glomeruli, the tiny filtering units of the kidney, and trigger inflammation.

Over time, that inflammation damages the filters. Two signs follow:

1. **Proteinuria**, meaning protein leaking into the urine.
2. **Haematuria**, meaning blood in the urine, which may be visible or detectable only by testing.

The disease can only be confirmed by a kidney biopsy. It is the most frequently diagnosed primary glomerular disease worldwide, meaning the most common disease that starts in the kidney's filters. It often appears in teenagers and young adults, and over a lifetime many patients progress to kidney failure, needing dialysis or a transplant, even with the best current supportive care.

For Australian readers, this is a familiar condition in kidney clinics. Two of the new trial's authors are based in Australia, at John Hunter Hospital and the University of Newcastle in New South Wales, and at the University of Sydney.

How IgA nephropathy is found

One of the difficult features of IgA nephropathy is how quietly it can progress. Many people feel well for years. The disease is often picked up in one of a few ways:

- **Visible blood in the urine**, sometimes turning it the colour of cola or tea, often during or just after a sore throat or other infection.
- **A routine urine test** that shows blood or protein, perhaps at a health check, a pregnancy check or an insurance medical.
- **High blood pressure** in a young person, which leads to kidney tests.
- **Reduced kidney function** on a blood test, sometimes found only when significant damage has already occurred.

A urine test showing protein or blood does not mean someone has IgA nephropathy. Many other conditions can cause those findings. A kidney specialist will usually arrange further tests and, if needed, a biopsy, in which a tiny sample of kidney tissue is examined under a microscope for IgA deposits.

Because early IgA nephropathy can be silent, simple checks matter. In Australia, kidney health checks that include blood pressure, a blood test for kidney function and a urine test for protein are recommended for people at higher risk of chronic kidney disease, and your GP can advise whether they apply to you.

Why targeting the source is different

The new treatments can be grouped by where they act in the chain of events that damages the kidney.

1. **Upstream: the antibody supply.** Drugs such as povetacicept and telitacicept block BAFF and APRIL, the signals that keep IgA-producing cells active. In principle, this reduces the abnormal Gd-IgA1 that starts the process.
2. **Midstream: the inflammatory response.** Drugs such as ravulizumab and sefaxersen act on the complement system, a set of immune proteins that amplifies inflammation once immune complexes have lodged in the kidney.
3. **Downstream: protecting the filters.** Blood pressure medicines and SGLT2 inhibitors reduce strain on the kidney's filters, whatever the cause.

A useful comparison is a leaking roof. Downstream treatments are like buckets and better gutters. Midstream treatments reduce the damage the water does. Upstream treatments try to stop the rain getting in at all. In practice, patients may benefit from more than one approach, and trials will need to show which combinations work best and which patients benefit most from each.

Why protein in the urine matters so much

Kidney doctors watch protein in the urine closely. It is usually measured as the urine protein-to-creatinine ratio, or UPCR, which adjusts for how concentrated the urine is.

In IgA nephropathy, higher proteinuria is strongly linked to faster loss of kidney function. Lowering it is associated with better long-term outcomes. International guidelines from Kidney Disease: Improving Global Outcomes (KDIGO) now point to a treatment target of

proteinuria below 0.5 grams a day, and ideally below 0.3 grams. Trials often express this as a UPCR below 0.5 grams per gram.

Because kidney failure can take many years to develop, regulators such as the US Food and Drug Administration have accepted reductions in proteinuria as a "surrogate" measure. A surrogate is a marker expected to predict a real clinical benefit. Drugs can receive accelerated approval on the basis of proteinuria, with the condition that longer trials then confirm they protect kidney function. The FDA has already granted accelerated approval to several IgA nephropathy therapies in this way.

Standard treatment today

For years, treatment focused on protecting the kidneys rather than tackling the immune cause. That typically includes:

- **Blood pressure medicines** called ACE inhibitors or angiotensin receptor blockers, which also reduce proteinuria.
- **SGLT2 inhibitors**, originally diabetes drugs, which have been shown to protect the kidneys in many forms of chronic kidney disease.
- **Lifestyle measures**, such as blood pressure control and not smoking.
- **Steroids or other immune-suppressing drugs** in selected patients, with significant side effects.

These approaches slow the disease but often do not stop it. The new generation of treatments aims higher: to reduce the production of the abnormal IgA, or to block the inflammation it triggers.

Povetacicept: switching off the supply

How it works

Povetacicept, developed by Vertex Pharmaceuticals, targets the immune signals that keep IgA-producing cells going. Two proteins, called BAFF (B-cell activating factor) and APRIL (a proliferation-inducing ligand), help sustain the B cells that make antibodies, including IgA. Both are often elevated in people with IgA nephropathy.

According to the published paper, povetacicept is an engineered fusion protein designed to block both BAFF and APRIL at the same time. The aim is to reduce the production of Gd-IgA1 upstream, before the immune complexes can form and damage the kidneys.

It is given as an injection under the skin once every four weeks.

The RAINIER trial

The results come from a pre-specified interim analysis of RAINIER, a phase 3 trial. According to the *New England Journal of Medicine* paper and AJMC's report:

- **Design:** Double-blind and placebo-controlled, planned to run for two years, at 224 sites in 29 countries.
- **Participants:** Adults with biopsy-proven IgA nephropathy, significant proteinuria and kidney function (eGFR) of at least 30 mL/min/1.73 m².

- **Background therapy:** All had at least 12 weeks of stable, maximally tolerated ACE inhibitor or angiotensin receptor blocker treatment, and 97% were on one. In the interim efficacy group, 66% were also taking an SGLT2 inhibitor.
- **Randomisation:** 2:1 to povetacicept 80 mg or placebo every four weeks.
- **Numbers:** 557 participants received povetacicept (372) or placebo (185). The interim efficacy analysis included the first 199 participants randomised (131 povetacicept, 68 placebo).
- **Main outcome for this analysis:** Change in 24-hour UPCR at week 36.

What it found

Measure at week 36	Povetacicept	Placebo
Change in 24-hour UPCR (proteinuria)	52.0% decrease	4.3% decrease
Change in Gd-IgA1	77.4% decrease	9.1% increase
Haematuria resolved (those with haematuria at baseline)	85%	23%
Reached UPCR below 0.5 g/g (exploratory)	42% (54 of 128)	6% (4 of 65)

The adjusted difference in proteinuria between groups was 49.8 percentage points (95% confidence interval 39.2 to 58.6), and the result was highly statistically significant. AJMC reported that the effect was consistent across subgroups by age, sex, race, baseline proteinuria, baseline kidney function and region, and that spot urine measurements began to separate as early as week 4.

The large fall in Gd-IgA1, the abnormal antibody at the root of the disease, supports the idea that the drug is acting on the underlying biology rather than just masking symptoms.

Safety

According to the published paper, adverse events occurred in 75% of participants on povetacicept and 78% on placebo, and serious adverse events in 3% and 4%. AJMC reported that none of the serious adverse events was judged related to povetacicept and no deaths occurred.

Other findings reported by AJMC:

- Injection site reactions were more common with povetacicept (15% compared with 2%), though almost all were mild.
- Infections occurred in 46% compared with 41%, with serious or severe infections in 1% of each group and no opportunistic infections.
- Blood levels of IgG, another antibody type, fell by 38% by week 24, but no participant stopped treatment because of low IgG.

That last point deserves attention over the longer term. Any drug that dampens antibody production could, in principle, affect the body's ability to fight infection. So far, the infection data are reassuring, but longer follow-up is needed.

What the lead investigator said

The trial's senior author, Professor Richard Lafayette of Stanford University Medical Center, presented the data at Kidney Week in Denver. AJMC reported that he described the efficacy as "transformational" during his presentation.

What happens next

The FDA has accepted Vertex's application for accelerated approval, with a target action date of 30 November 2026. That decision had not been made by the end of October. The trial's final analysis will measure the change in kidney function, the eGFR slope, through week 104, which will show whether the fall in proteinuria translates into slower loss of kidney function.

A crowded and fast-moving field

Povetacicept is not the only new approach. Several were in the spotlight at the American Society of Nephrology's Kidney Week, held in Denver from 21 to 25 October 2026, according to HCPLive's coverage.

Ravulizumab: accelerated approval on 21 October

Ravulizumab (Ultomiris), a long-acting drug that blocks a part of the immune system called complement C5, takes a different route. Rather than reducing abnormal IgA, it targets inflammation downstream of the immune complexes. HCPLive reported that in the I CAN trial, ravulizumab reduced proteinuria by 43.4% compared with placebo at week 34, among 352 patients, and that the FDA granted it accelerated approval for IgA nephropathy on 21 October 2026 on the basis of those data. Ravulizumab was already approved for other rare complement-driven conditions.

Sefaxersen: a liver-directed antisense drug

Sefaxersen is an antisense medicine, a short strand of genetic material that reduces production of complement factor B in the liver. HCPLive reported that in the IMAgINATION trial's interim analysis, it reduced proteinuria by 43.1% compared with placebo at week 37.

Telitacicept: two-year kidney function data

Telitacicept, like povetacicept, blocks BAFF and APRIL. HCPLive reported that final two-year results from the TELIGAN trial, conducted in China among 318 adults, showed kidney function declining by 0.6 mL/min/1.73 m² per year with telitacicept compared with 5.7 per year with placebo. Proteinuria fell by 60.7% compared with placebo at week 104. Telitacicept holds conditional approval for IgA nephropathy in China, and HCPLive reported that no US filing for this condition has been announced.

The TELIGAN result is notable because it addresses the question the other trials have not yet answered: whether this class of drugs preserves kidney function over two years, not just proteinuria. It was a single-country trial, so results elsewhere may differ.

How the approaches compare

Approach	Example	Target	Key evidence reported in October 2026
Dual BAFF/APRIL blockade	Povetacicept	Production of abnormal IgA	49.8 percentage point placebo-adjusted fall in proteinuria at week 36 (NEJM)
Dual BAFF/APRIL blockade	Telitacicept	Production of abnormal IgA	Two-year kidney function benefit in China (TELIGAN)
Complement C5 inhibition	Ravulizumab	Downstream inflammation	43.4% fall in proteinuria versus placebo; FDA accelerated approval
Complement factor B reduction	Sefaxersen	Downstream inflammation	43.1% fall in proteinuria versus placebo at week 37

Comparisons across trials should be made cautiously. HCPLive noted that differences in trial design and timepoints limit direct comparison, and kidney function results from several of these trials are still pending.

Reading the results carefully

Proteinuria is a surrogate, not the end goal

A large fall in proteinuria is encouraging, and the link between proteinuria and long-term outcomes in IgA nephropathy is well established. But patients care about avoiding dialysis and transplant. Until two-year kidney function results arrive for povetacicept, the most important question remains open.

Interim analyses use part of the trial

The povetacicept efficacy results come from 199 of 557 participants at 36 weeks. Interim analyses are pre-specified and statistically rigorous, but the final analysis will be larger and longer.

Company involvement

AJMC noted that Vertex funded RAINIER and designed it with the trial's steering committee, and shared in data collection and analysis. That is standard for industry-sponsored trials and does not invalidate the results, but it is something readers should know. Independent replication and long-term data build confidence.

Long-term immune effects

Drugs that reduce antibody production may need careful monitoring for infections and vaccine response over years. The early data are reassuring, but chronic diseases require chronic treatment, and long-term safety matters.

Access outside the United States

These are US decisions. Approval and subsidy in Australia would require separate applications to the Therapeutic Goods Administration and the Pharmaceutical Benefits

Advisory Committee. Patients should ask their nephrologist about current options and clinical trials.

What this means for people living with IgA nephropathy

This is general information, not medical advice.

- **Know your numbers.** Ask your kidney specialist about your UPCR and eGFR, and what targets apply to you.
- **Keep up proven treatments.** Blood pressure control, ACE inhibitors or angiotensin receptor blockers, and SGLT2 inhibitors where appropriate remain the foundation of care.
- **Ask about trials.** Many new IgA nephropathy treatments are being tested, and trial participation may be an option.
- **Do not change medicines on your own.** New drugs are added to, not substituted for, established care in these trials.

Why this matters

In my view, October 2026 marks a turning point in how IgA nephropathy is treated. For a long time, care focused on slowing damage. Now there are several drugs that act on the disease's immune machinery, either by reducing the abnormal antibody at its source or by blocking the inflammation it triggers. Some patients may eventually take combinations, much as people with high blood pressure or heart failure take several complementary medicines.

The challenge for health systems will be cost and choice. With several new drugs arriving, doctors and regulators will need good evidence about which patients benefit most from which treatments, and in what order. Two-year and longer kidney function data will be the deciding test.

There is also a lesson here about how modern medicine advances. Each of these drugs depends on years of basic research into how IgA nephropathy actually develops: the discovery of the abnormal Gd-IgA1 molecule, the antibodies made against it, and the roles of BAFF, APRIL and complement. Understanding the mechanism made it possible to design drugs that act at specific points along the chain. That pattern, from careful biology to targeted treatment, is increasingly how progress happens across medicine.

The bottom line

IgA nephropathy is a common, immune-driven kidney disease that often affects young adults and can lead to kidney failure. In late October 2026, the phase 3 RAINIER trial, published in the *New England Journal of Medicine*, showed that povetacicept, which blocks the BAFF and APRIL signals that sustain antibody-producing cells, reduced proteinuria by about half compared with placebo at 36 weeks, lowered the abnormal IgA at the root of the disease and resolved blood in the urine in most patients who had it. Side effects were similar to placebo, apart from more injection site reactions.

The same week, the FDA granted accelerated approval to ravulizumab for IgA nephropathy, and other trials reported strong results for related drugs. The key caveat is that most of these results rest on proteinuria, a surrogate measure. Two-year kidney function data, expected for povetacicept from the final RAINIER analysis, will show whether these advances truly keep people off dialysis. For a disease that has long had limited options, the direction of travel is clearly positive.

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