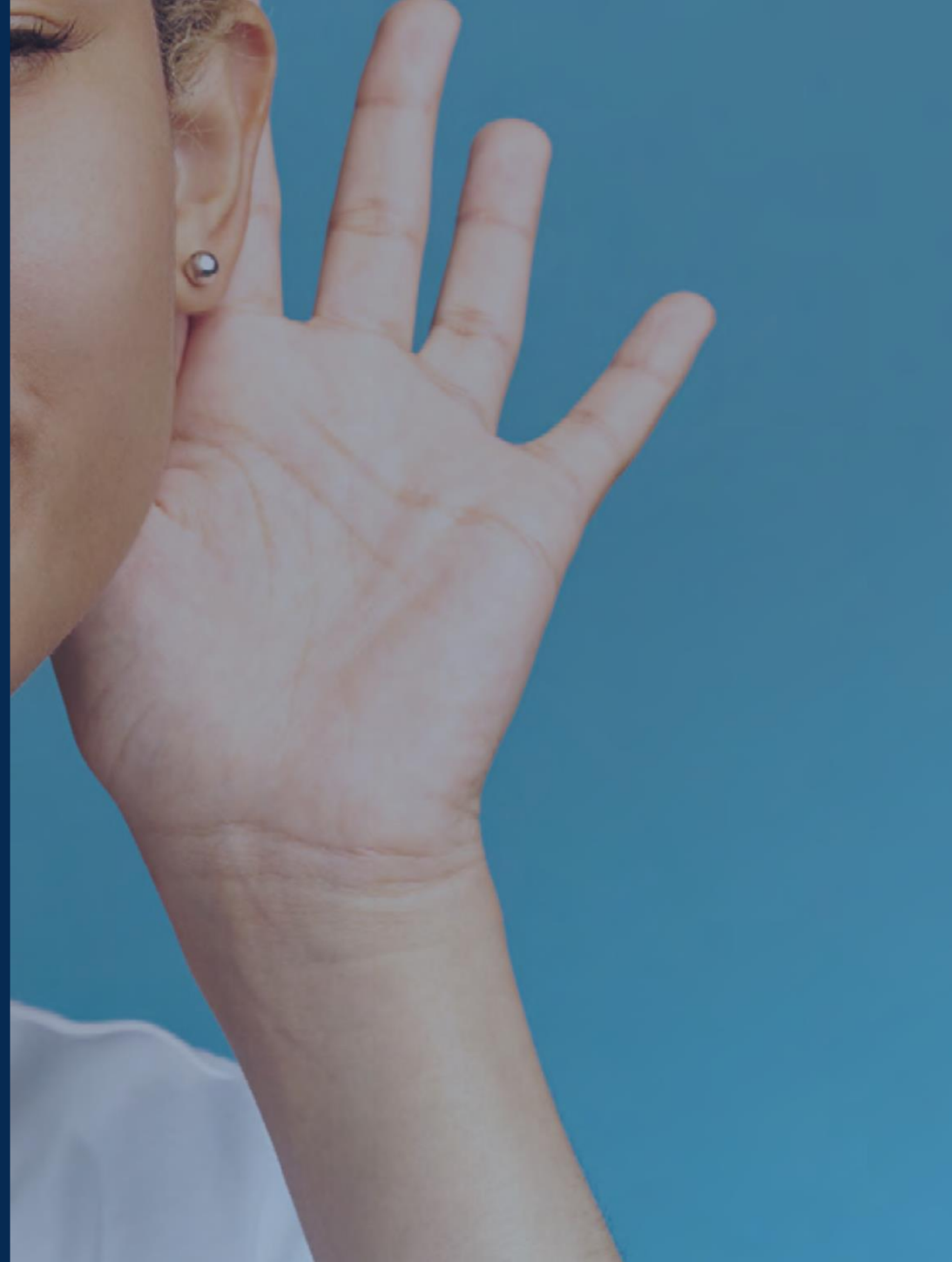
Sensorion

SENS-601 Program Day

Gene therapy for GJB2-related congenital hearing loss
From the biology to the clinic

22 SEPTEMBER 2026

Euronext Growth: ALSEN



Introduction

SENS-601 enters the clinic

Fred Chereau, Chief Executive Officer, Sensorion

Welcome to the SENS-601 Program Day

Listen-only mode

All participants are in listen-only mode for the presentations.

Q&A

Dial in on the conference number. Lines open for questions at the end.

90 minutes

The presentation runs ~ 90 minutes, and will be followed by a Q&A.

Slides and replay

Slides and a replay will be available at www.sensorion.com.

Forward-looking statements

This presentation contains certain forward-looking statements concerning the “Company” and its business, including statements regarding the Company’s clinical development plans, its regulatory submissions and the expected timing of clinical data. Such forward-looking statements are based on assumptions that Sensorion considers to be reasonable. However, there can be no assurance that such forward-looking statements will be verified, which statements are subject to numerous risks, including the risks set forth in the 2025 full year report published on March 18, 2026, and available on the Company’s website, and to the development of economic conditions, financial markets and the markets in which Sensorion operates. The forward-looking statements contained in this presentation are also subject to risks not yet known to Sensorion or not currently considered material by Sensorion. The occurrence of all or part of such risks could cause actual results, financial condition, performance or achievements of Sensorion to be materially different from such forward-looking statements.

Investigational product

SENS-601 is an investigational product candidate. It has not been approved by any regulatory authority, and its safety and efficacy have not been established. Data presented today are derived from preclinical studies and from natural history research. Preclinical results are not necessarily predictive of clinical outcomes.

No offer

This presentation and the information it contains do not constitute an offer to sell or subscribe for, or a solicitation of an offer to purchase or subscribe for, Sensorion shares in any country. The communication of this presentation in certain countries may constitute a violation of local laws and regulations. Any recipient of this presentation must inform themselves of any such local restrictions and comply therewith.

Unpublished data

Certain natural history data presented today are unpublished. They are presented for scientific discussion and are subject to further analysis.

Today’s agenda

Where we stand

- French ANSM authorized HearConnex on 31 August 2026
- Health Canada clinical trial application under review
- First patient dosing targeted by early 2027

Why this event, and why now

- Turning point for the program as SENS-601 enter the clinic
- Full review of GJB2-GT: the disease, the standard of care, the biology, the preclinical package and the trial design

What today covers

- Five sessions, from the patient journey to the design of the first-in-human study
- Panel discussion with all speakers to close

Session 1	Session 2	Session 3	Session 4	Session 5
The patient journey	**The science**	**The preclinical package**	**The clinical strategy**	**Panel and Q&A**

GJB2-GT: GJB2 Gene Therapy; targeted genetic treatment designed to restore hearing loss caused by mutations in the GJB2 gene (encoding Connexin 26).

Today’s speakers

Dr. Sharon Cushing

The Hospital for Sick Children (SickKids), Toronto

Pediatric otolaryngologist, Director of the Cochlear Implant Program. Coordinating Investigator, HearConnex.

Prof. Christine Petit

Institut Pasteur, Institut de l’Audition / Institut reConnect

Professor Emeritus at the Collège de France, laureate of several prizes incl. the Kavli Prize in Neuroscience.

Fred Chereau

Chief Executive Officer, Sensorion

Laurent Désiré

Head of Preclinical, Sensorion

Valérie Salentey

Head of Regulatory Affairs and Quality Assurance, Sensorion

SENS-601 is built on work that started long before the construct did

1

Foundation

- Inner-ear expertise across gene therapy and small molecules: biology, cochlear delivery, translational models and audiology endpoints
- Know-how that transfers across inner-ear modalities
- Strategic collaboration with Institut Pasteur on the genetics of hearing

2

Technology

- AAV vector design for cochlear targeting
- miR-based silencing to keep transgene expression out of hair cells
- Injection system built for intracochlear administration
- In-house process development and analytics

3

Clinical execution

- First-in-human experience of the surgical procedure in the pediatric population, through the Audiogene trial
- OTOCONEX, a natural history study feeding patient identification and recruitment

4

Where that leads

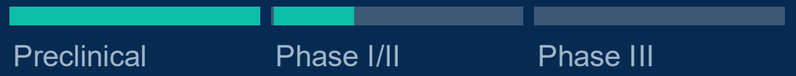
- HearConnex approved in France, Canadian CTA review ongoing. US IND and Australian CTA filings expected by year-end 2026
- First patient dosing by early 2027
- Clinical data to be generated throughout 2027

SENS-601 is entering the clinic in congenital GJB2-related HL, with plans to extend across the broader population

Sensorion aims to build a leading global hearing loss franchise by expanding across indications

Congenital onset

In the clinic



CTA approved in France

CTA under review in Canada

U.S. IND and Australia CTA submission expected by year-end 2026

~2,000 newborns per year across the US, EU4 and UK

Pediatric progressive

Extension

Preclinical studies and population identification ongoing

Adult onset (presbycusis)

Extension

Preclinical studies and population identification ongoing

Throughout this presentation, GJB2-related HL refers to DFNB1A: autosomal recessive, non-syndromic hearing loss caused by biallelic GJB2 variants.
EU4: the four largest European Union markets: France, Germany, Italy and Spain. UK: United Kingdom, US: United-states.

Session 1

Hearing loss and DFNB1A: the patient journey and limitations of standard of care

Sharon Cushing, M.D., The Hospital for Sick Children (SickKids),Toronto
Coordinating Investigator, HearConnex

Estimated incidence

≈ 2,000

newborns each year across the USA, EU4 and UK

32 / 100,000

incidence in newborns in the US, EU4 and UK

Photo: Iuliia Bondarenko via Pixabay

GJB2 deficiency, congenital form

Severe to profound deafness at birth, with a narrow window for intervention

≥ 71 dB

Severe to profound deafness at birth in most patients

Diagnosis

Newborn hearing screening followed by GJB2 genotyping

Before 3 years

Management matters during cerebral plasticity, today with a cochlear implant

Consequences

Impact on language acquisition and development

Care pathway

Complex, involving screening, audiology, imaging, genetics and surgery

Population

32 per 100,000 newborns in the US, EU4 and UK: approximately 2,000 patients per year

How children are identified today

Permanent hearing loss screening in Ontario runs on two parallel tracks

Track 1

Physiologic hearing screening

Track 2

Risk factor screening

Congenital CMV

Genetic

Source: Newborn Screening Ontario

Genetic screening is the track that identifies DFNB1A

Track 1

Physiologic hearing screening

Track 2

Risk factor screening

Congenital CMV | **Genetic**

Source: Newborn Screening Ontario

The genetic risk factor panel screens three genes

Twenty variants identified in Ontario in total, half of them in GJB2.

GJB2

10 variants screened

c.35delG | c.235delC | c.167delT | c.71G>A | c.310_323del | c.139G>T | c.-23+1G>A | c.231G>A

c.427C>T | c.269T>C

GJB6

1 variant screened

D13S1830 (342 kb deletion)

SLC26A4

9 variants screened

c.707T>C | c.1001+1G>A | c.1246A>C | c.919-2A>G | c.2168A>G | c.1003T>C | c.1229C>T

c.1614+1G>A | c.1541A>G

ONTARIO, JULY 2019 – MARCH 2022

Universal newborn genetic risk factor screening works at population scale

Ontario
Ministry of Children, Community and Social Services

NEWBORN SCREENING ONTARIO
DÉPISTAGE NÉONATAL ONTARIO

422,943 babies born in Ontario	**412,424** received genetic screens (98.8%)	**93** positive genetic screens	**72** sensorineural hearing loss confirmed
98.8% consent rate	**79** referred	**3** passed	**11** missed

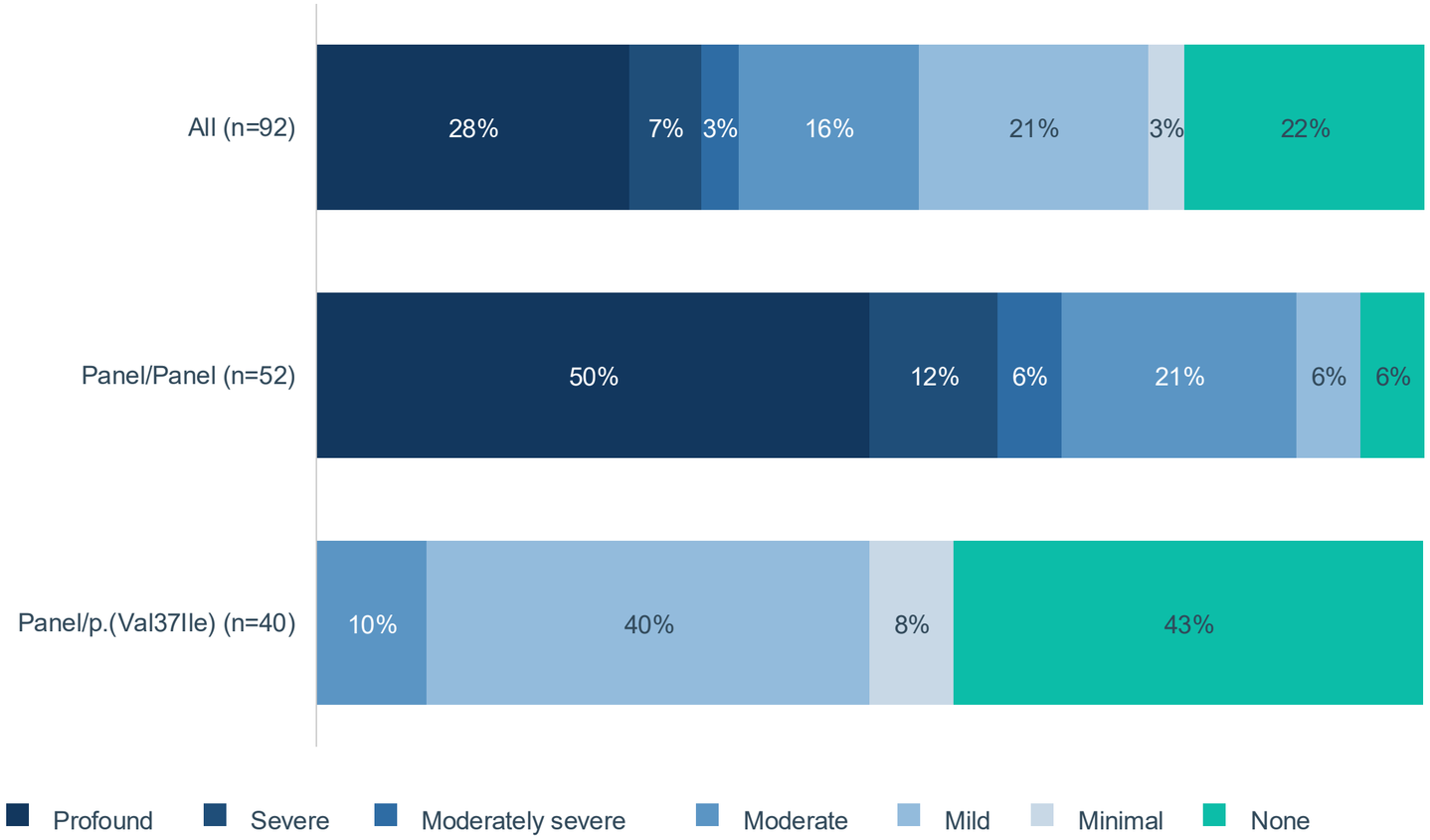
Ontario
Population ~15 M
Area 1.076 M km²

Kernohan K, Genetics in Medicine 2024

Genetic screening identifies the full severity spectrum, not only profound hearing loss

Hearing phenotypes identified through universal genetic screening (n=93)

- **Three quarter of screened infants fall outside profound loss**; only 28% fall into the profound category typically eligible for cochlear implantation
- **Biallelic panel variants carry the severe end**: 50% profound and 67% severe or worse, 35 of 52 infants
- **p.(Val37Ile) sits at the other end**: no profound or severe loss, 43% hearing normally at screening and 40% mild

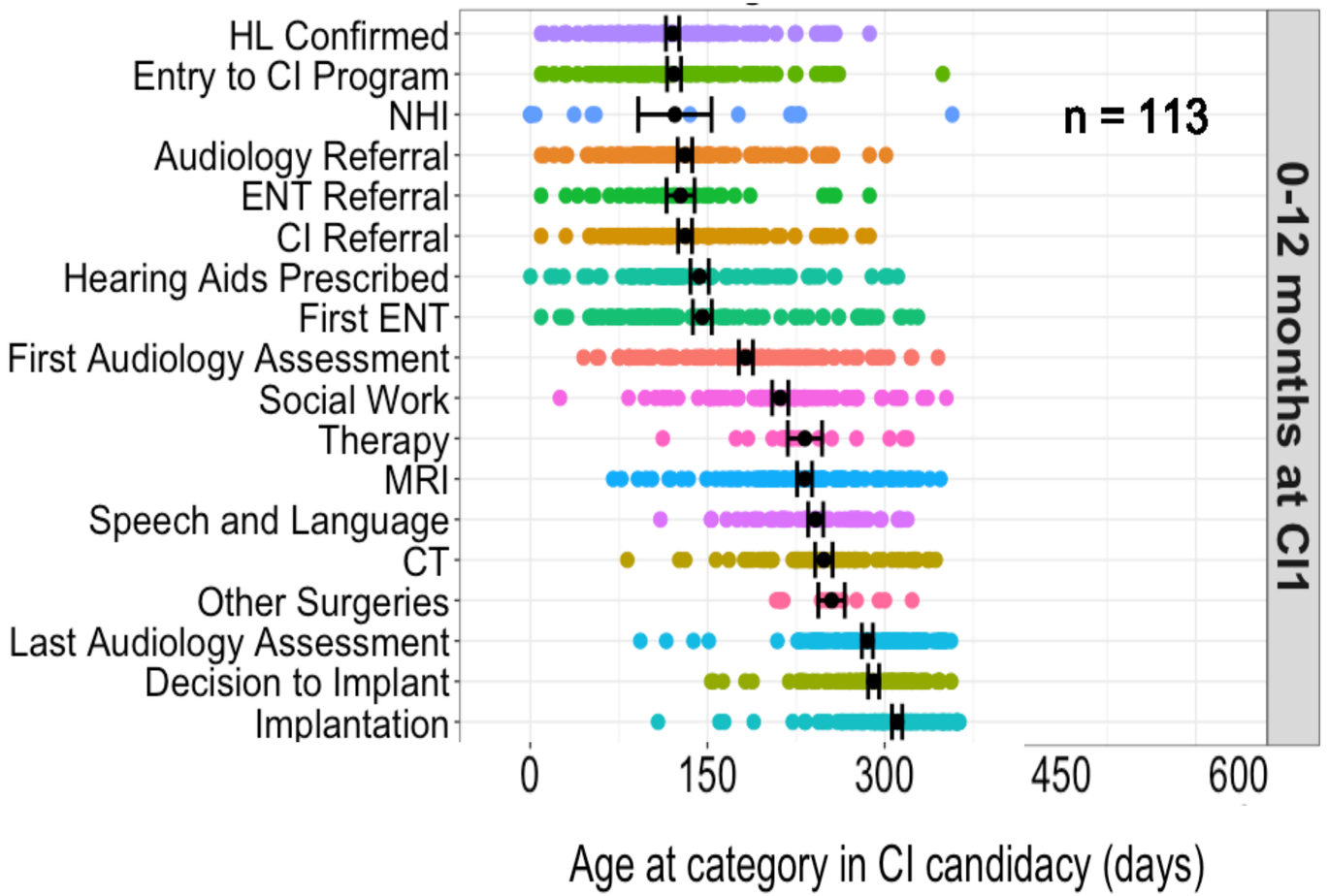


Kernohan K, Genetics in Medicine 2024

Reaching implantation takes many steps, each adding delay

Age at each category in the cochlear implant candidacy process (days), n=113

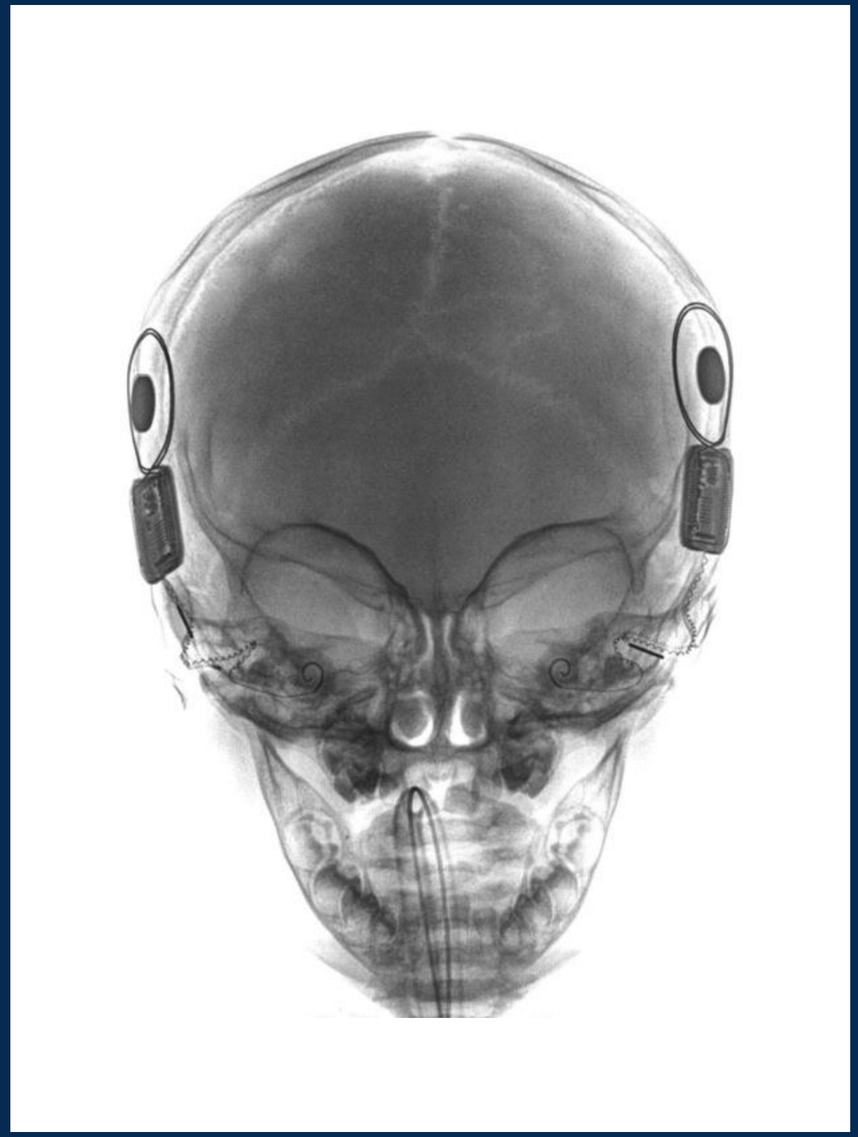
- Eighteen distinct steps separate newborn screening from implantation
- Each dot is one child; 113 children followed through the full pathway
- The median age rises at every step, and the spread widens as the pathway lengthens



Timeline of implant candidacy process
CI: Cochlear implant

The cochlear implant journey, and where it stops

Two implants, two sound processors,
and a lifetime of device management.



Cochlear implantation commits a child to lifelong device management

Note: time periods shown reflect age at each step of implantation process; does not reflect incremental time between stages

Birth	Referral	Diagnosis	Work-up	Surgery	Activation
Newborn hearing screening	GP or pediatrician referral to ENT	Hearing loss diagnosis, early intervention	Hearing aid fitting, SLP / AVT evaluation, imaging, genetic diagnosis	Cochlear implant surgery	Activation and switch-on 1, 3, 6 and 12 months post activation, annually thereafter
Age 1 – 30 days	Age 1 – 3 months		Age 4 – 6 months	Age 7 – 14 months	

Lifelong implications

Ongoing CI programming
Plus speech and language, and auditory verbal therapy appointments

Monitoring and repairs
Daily device management for the family

Processor upgrade
Every 5 years

Explant and re-implant
May occur once per ear during a lifetime

Hearing loss diagnosis, cochlear implantation and CI activation may occur months or years later, depending on the underlying etiology, newborn screening methodology, completion of diagnostic follow-up and local care pathways. Genetic diagnosis may occur at any point in the journey, or not at all.

An implant delivers a low-resolution version of what the cochlea does

Cochlear implantation is the current standard of care

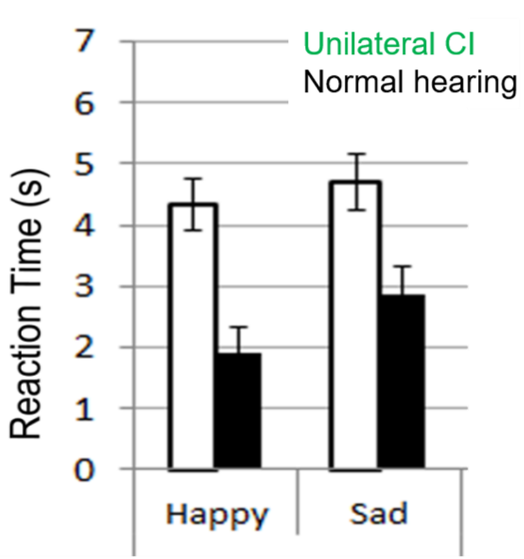
The signal provided by cochlear implants can be compared to a pixelated, **low-resolution image**.

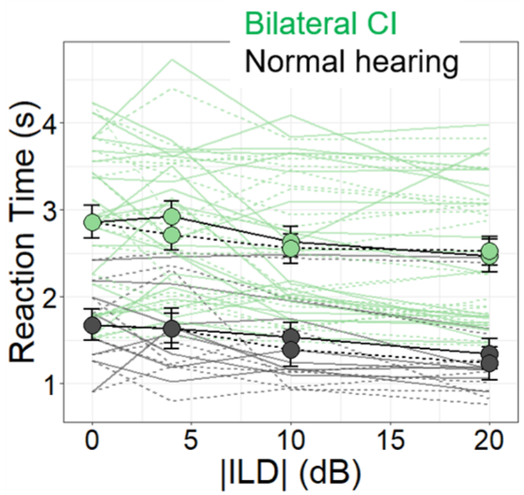
A cochlea with restored transmission of the signal can be compared to a **well-focused and clear image**.

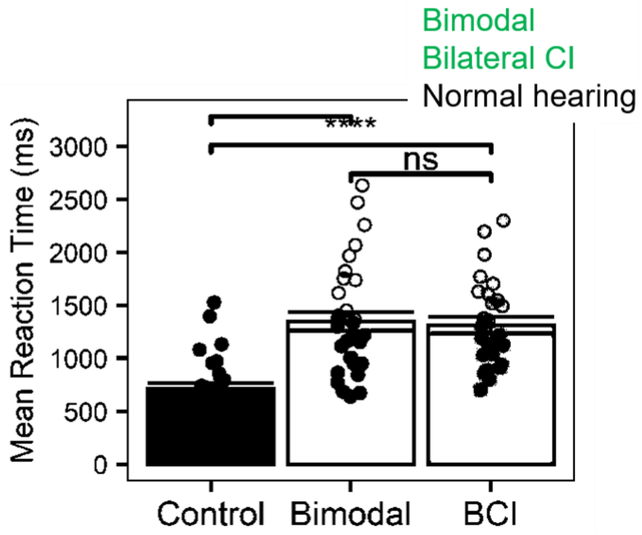
The lateral spread of electrical current from each electrode contact stimulates too many auditory neurons at the same time, rather than the physiological one-to-one connectivity between hair cell and auditory neuron

Listening with an implant stays effortful, even when it works

Reaction time is longer for implant users than for normal-hearing peers when discriminating emotion in music, lateralizing bilateral cues and localizing sound.







Hopyan et al., Child Neurophys 2014; Fung et al., Clinical Neurophys 2025; Alemu et al., Trends in Hearing 2025

In their own words

What families say about living with an implant

A short excerpt from the Hear Here podcast.

HEAR HERE

Scan to listen

In their own words

Hearing loss costs energy, every day

Two adults describe listening fatigue in the Hear Here podcast.

> *“I consider myself to be a very extroverted person, but if I’m at a restaurant with my family or my friends, after the first hour, I’m absolutely exhausted.”*

Sofia

> *“The hearing fatigue is always kind of in the background. It’s hard to identify if the fatigue I’m experiencing is just from a normal activity like exercise, or if it is something additional that’s being added because of my hearing loss.”*

May

A one-time administration changes what the rest of childhood looks like

Note: time periods shown reflect age at each step of gene therapy treatment process; does not reflect incremental time between stages

Birth

Newborn hearing screening

Age 1 – 30 days

Referral

GP or paediatrician referral to ENT

Diagnosis

Hearing loss diagnosis, genetic diagnosis, early intervention

Age 1 – 3 months

Work-up

SLP / AVT evaluation, imaging

4 – 6 months

One-time

Gene therapy surgery

One-time administration

Post-operative speech therapy, assessment and monitoring at 1, 3 and 6 months, semi-annually until age 6, annually thereafter

Potential long-term benefits

- Improvement in hearing without the need of a device
- Ease of communication in social situations
- Improvement in ability to understand in noise
- Development of independence and autonomy
- Improvement in social skill development
- Increased social engagement with peers
- Reduction of listening fatigue and listening effort
- Participation in sports and activities without fear of damage to the sound processor

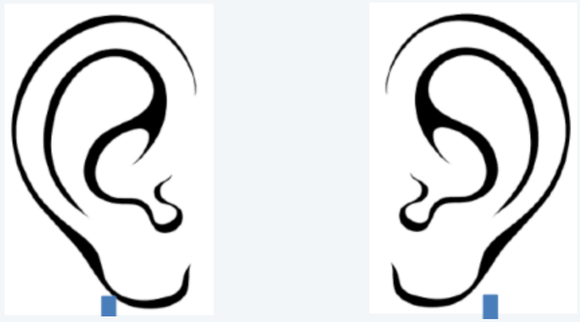
Eliminate

- Daily maintenance of a device
- Device stigma
- Costs to replace devices or accessories
- Possible surgery to replace an implant
- Use of assistive devices or technology
- Specific educational needs

Vona B 2020; Wong CL 2017; Antoni M 2016; Awad R 2019.

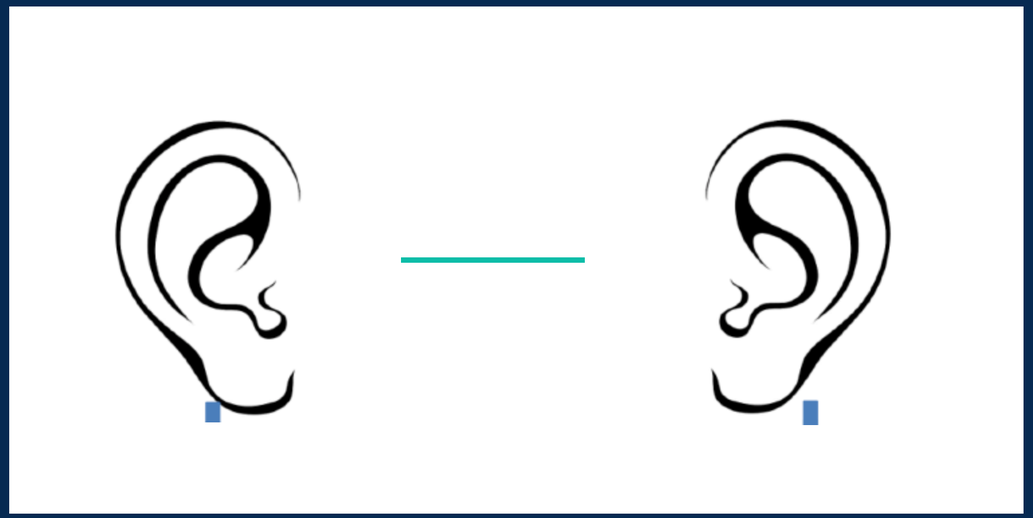
Two ears are not the same thing as hearing with two ears

Bilateral hearing



Sound is delivered to both ears. Each side is stimulated independently.

Binaural hearing



The two ears are integrated centrally. Gene therapy is what allows localization, lateralization and listening in noise.

Part 2 of HearConnex: plan for bilateral administration

1

GJB2-related hearing loss is identified at birth, and the window for intervention is measured in months

2

Cochlear implantation restores access to sound, not physiological hearing

3

Binaural integration, listening in noise and listening effort are where the significant unmet need remains

The population is identified at birth and the gap left by implants is well-defined: gene therapy addresses the cause of hearing loss instead of bypassing it

Session 2

GJB2 mouse models: decoding DFNB1A to drive therapeutic innovation

Prof. Christine Petit, Institut Pasteur, Institut de l’Audition / Institut reConnect
Professor Emeritus at the Collège de France
Laureate of the Kavli Prize in Neuroscience

“Les Causeuses”
(The Conversationalists)

Camille Claudel, 1897

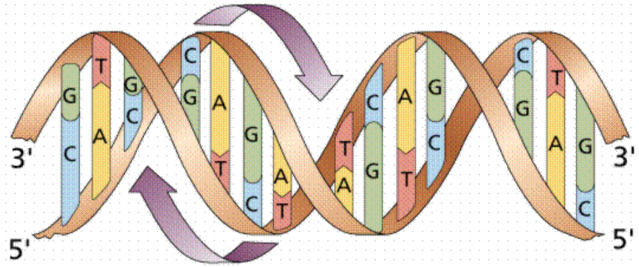
"not seeing separates us from things; not hearing, from our fellow man."

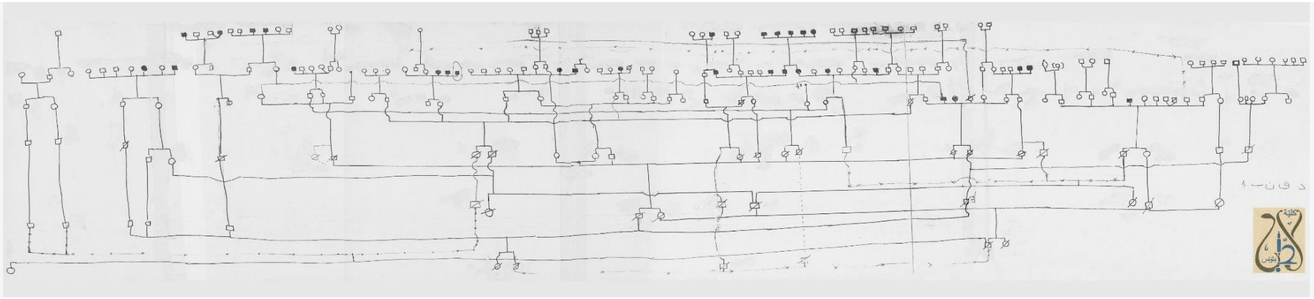
Immanuel Kant

1992 Discovery of the genes responsible for deafness

A strategy based on the study of affected blood-related families living in geographic isolates.

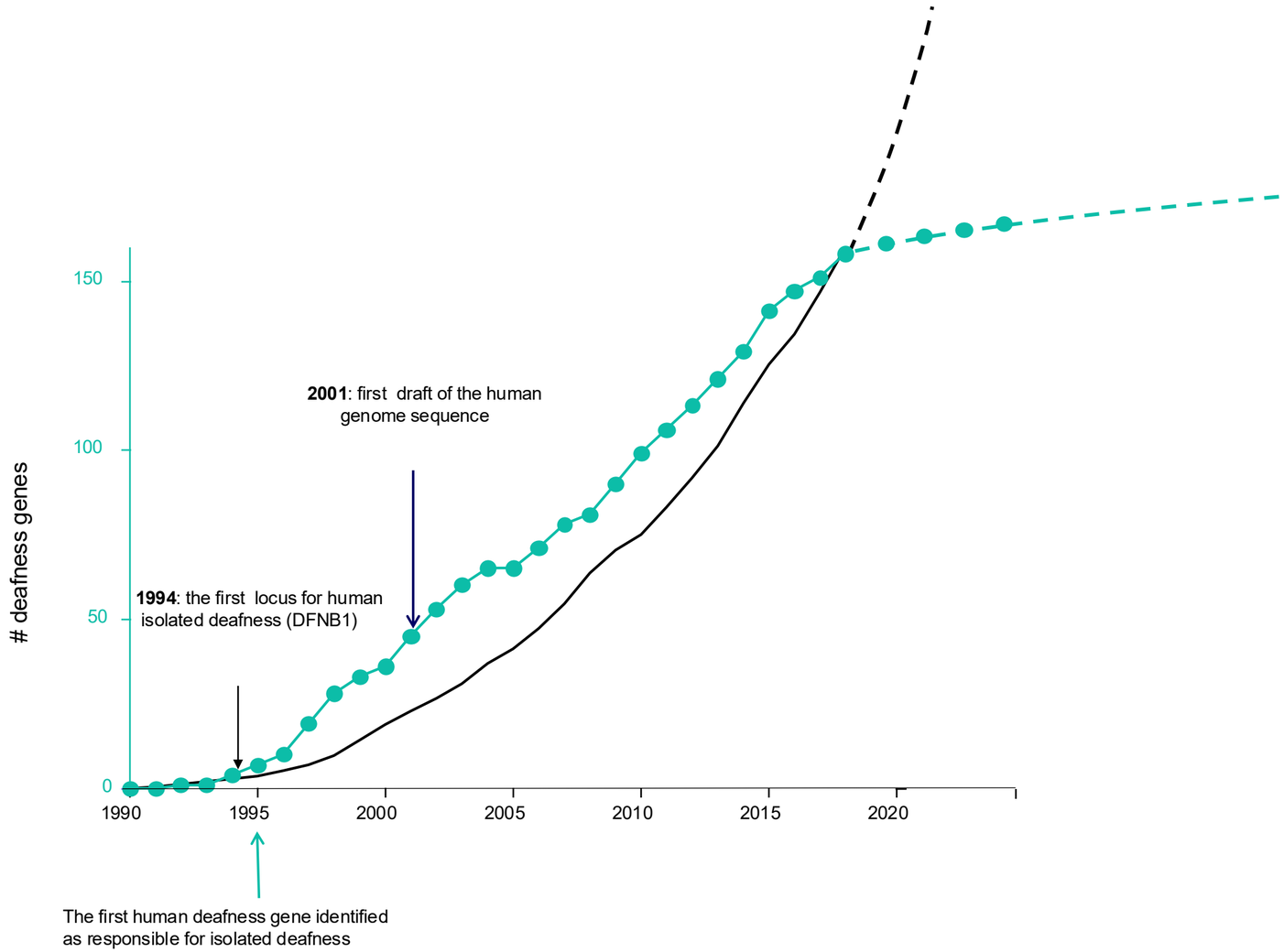
This approach, applied across isolated populations mainly around the Mediterranean sea, became the foundation for uncovering the genes responsible for deafness over the following three decades.





Causal genes for monogenic early-onset forms of non-syndromic deafness

The number of known deafness genes has climbed steadily since the first DFNB1 locus was mapped in 1994, accelerating after the 2001 first published human genome sequence and now reaching more than 150 genes.



Weil D et al. 1995 Nature, Gibson et al, 1995 Nature

Genetic architecture of hearing loss

157

Genes for monogenic congenital / prelingual / childhood / pre-adult / age-related hearing loss altogether

88

Genes for autosomal recessive (DFNB) forms — generally the most severe and with the earliest onset (congenital prelingual)

65

Genes for autosomal dominant (DFNA) forms, generally postlingual progressive and less severe

15

Genes cause for both DFNA and DFNB forms

7

Chromosome X-linked genes

1

Chromosome Y-linked genes

2

Modifier genes

9

Mitochondrial genes, incl. 2 genes predisposing to aminoglycoside ototoxicity

~300

Genes for monogenic syndromic forms of hearing loss

~100 / ~135

Genes associated with age-related hearing impairment / noise-induced hearing loss susceptibility

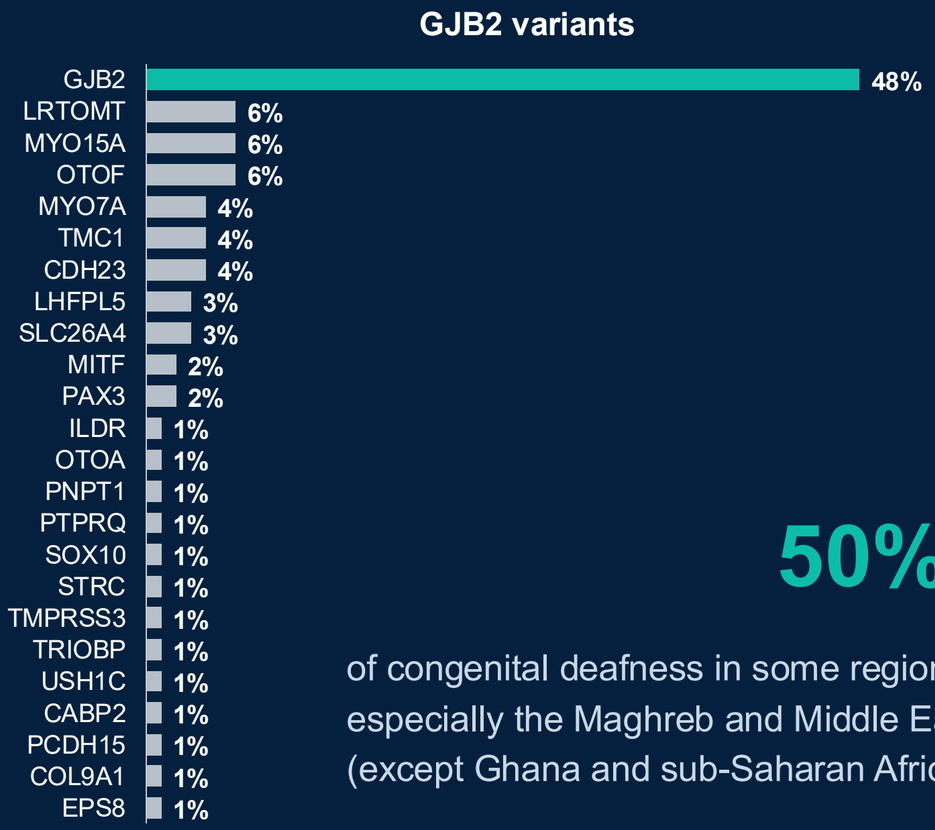
A single recessive, non syndromic form, DFNB1 caused by defect in *GJB2* accounts for up to half congenital deafness cases in some geographic areas

Clinical course

2/3

of children affected by DFNB1 born with profound deafness retain residual hearing at birth; most of them will lose it in early childhood.

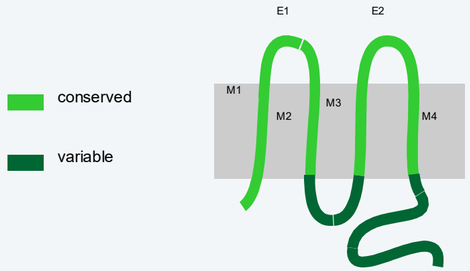
Algeria



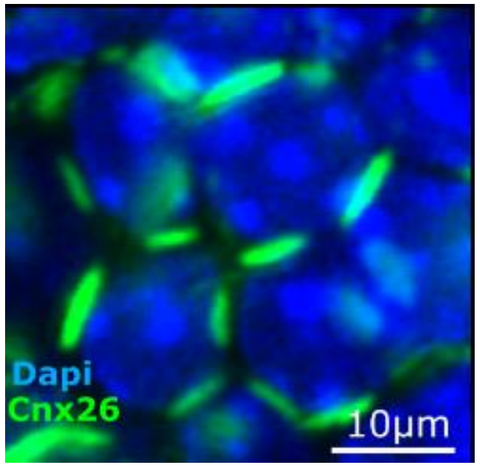
50%

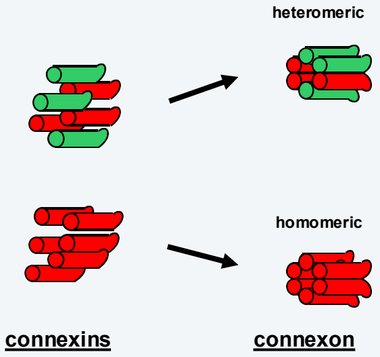
of congenital deafness in some regions, especially the Maghreb and Middle East (except Ghana and sub-Saharan Africa)

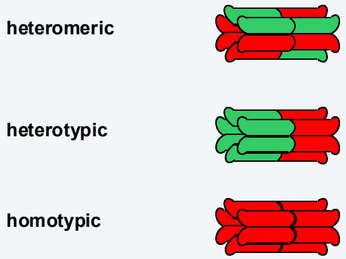
Connexin 26 (CX26) from connexons to gap junctions



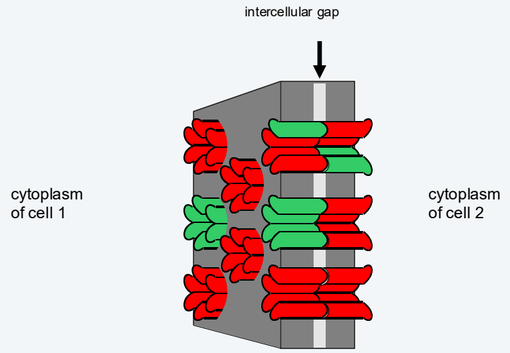
Topology of a generic connexin





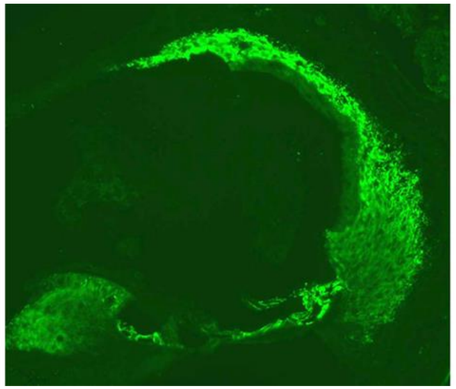


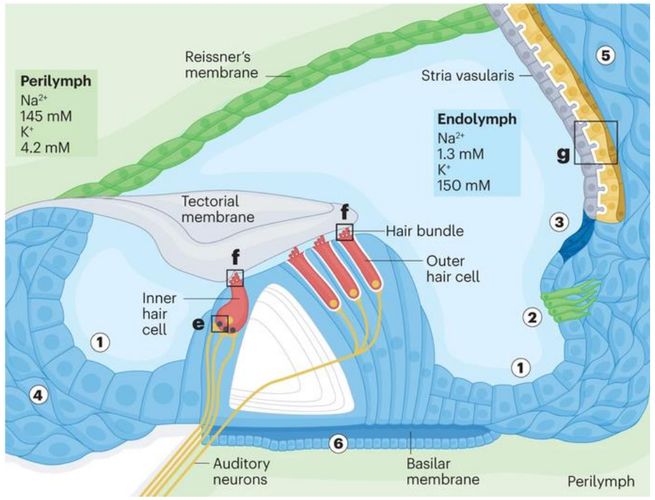
Intercellular channels

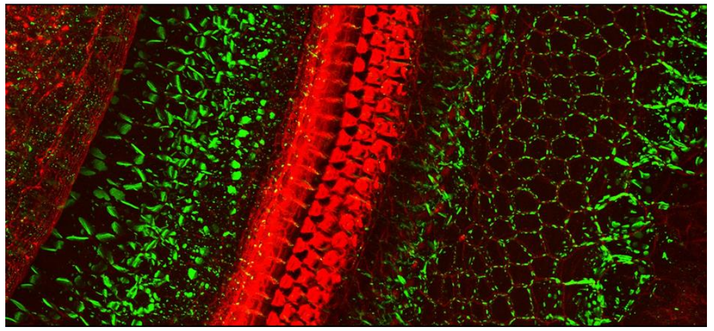


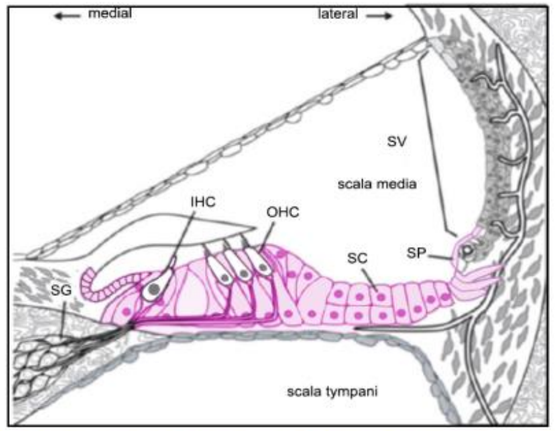
Gap junction

CX26 gap junction networks in the cochlea



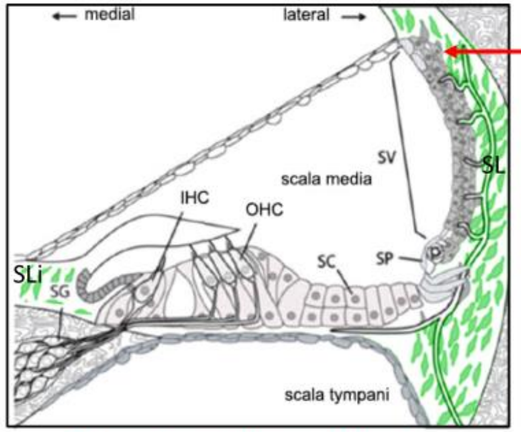






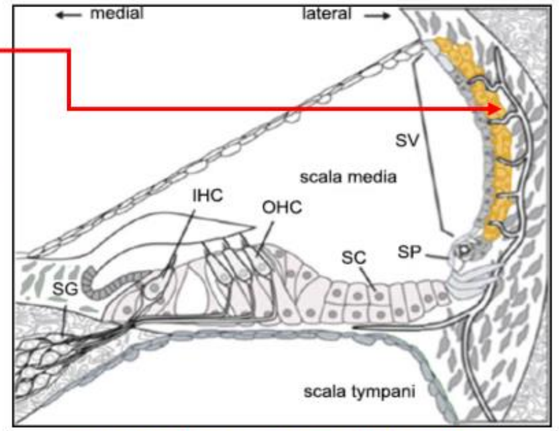
All the supporting cells of the sensory epithelium
Including interdental cells of the spiral limbus
and root cells of the spiral ligament

The epithelial gap junction network (E16)



Fibrocytes of the spiral limbus
and the spiral ligament

Gap junctions channel interconnections between the spiral ligament and the basal cells of the stria vascularis (P5-P7)



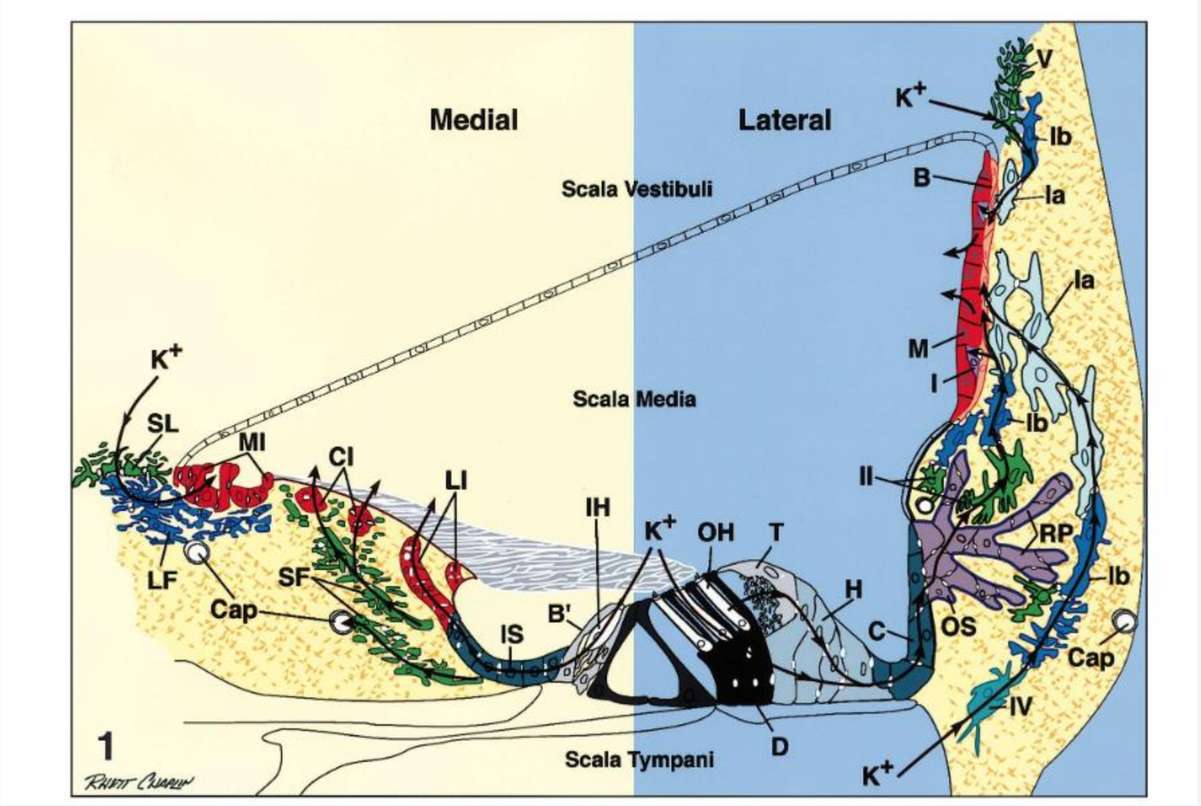
The basal and intermediate cells
of the stria vascularis

The connective tissue gap junction network (P0)

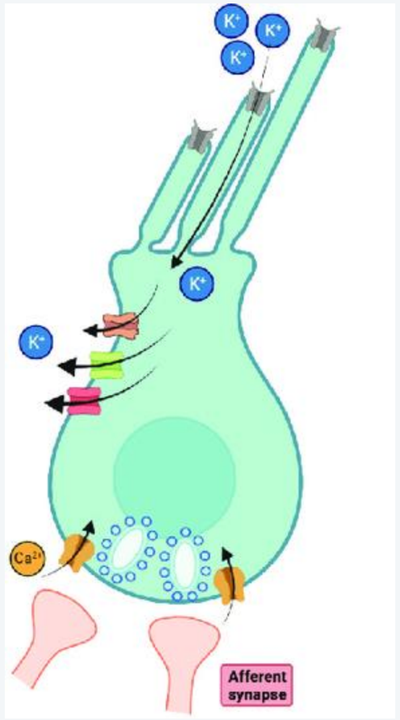
Connective tissue CX26 gap junction network in the cochlea

“Evidence for a medial K+ recycling pathway from inner hair cells.”

Spicer SS, Schulte BA. Hear Res. 1998 Apr;118(1–2):1–12.



Physiology of CX26 gap junction networks in the cochlea



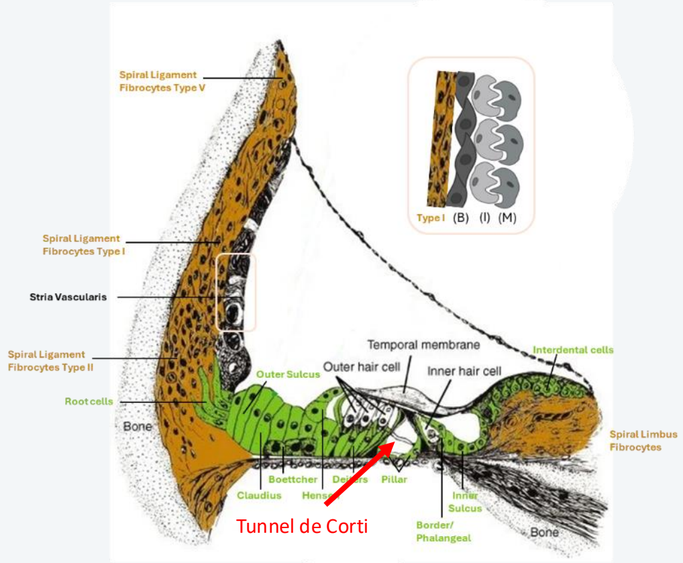
1. K+ buffering and recycling

Mechanoelectrical transduction leads to extrusion of K^+ ions from hair cells which enter supporting cells and move from cell to cell via CX26/(CX30) channels. K^+ ions also enter the connective gap junction network generating the endocochlear potential and resulting in K+ secretion into the endolymph.

2. Development

Cx26/(Cx30) channels are essential for cochlea development, notably for the tunnel of Corti formation. They are also essential for propagation of IP_3 / Ca^{2+} waves and glucose transport during embryonic development.

3. Protection of cochlear cells in adulthood



Adapted from https://www.nature.com/articles/doi:10.1038

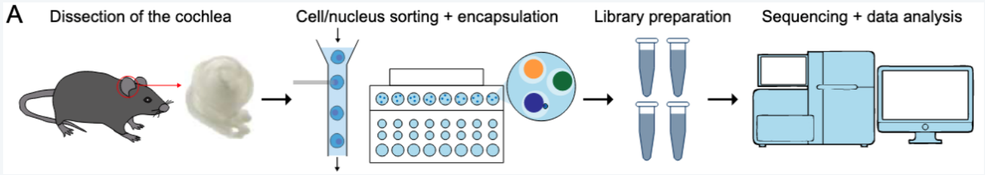
K+: potassium ion; CX26/CX30: connexins 26 and 30; IP3: inositol trisphosphate; Ca2+: calcium ion

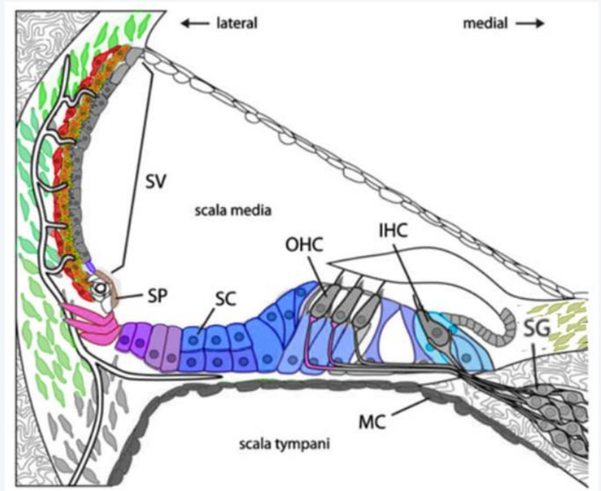
Pathophysiology of CX26 gap junction networks in the cochlea

Pathophysiological studies in animal models reveal different defects in different models.

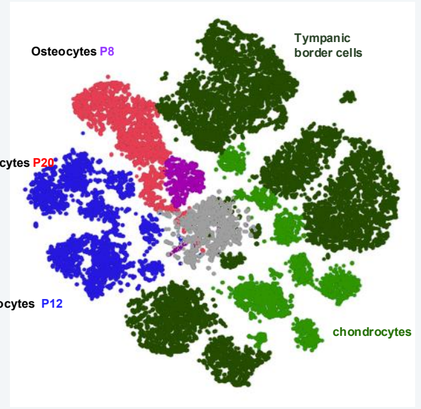
- **Absence of tunnel of Corti opening**
- **Absence of, or decrease in, endocochlear potential**
- **Functional defects of cochlear cell types, including hair cells**
- **Elevation of the hearing threshold: mild to profound deafness**

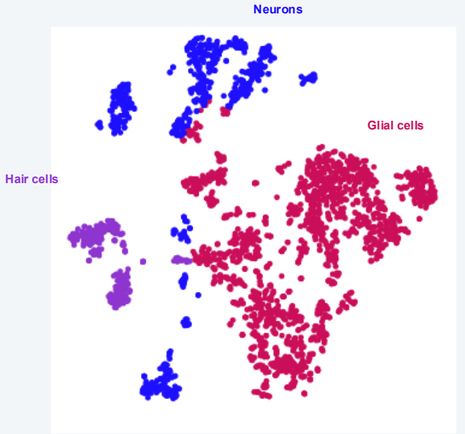
When and where is CX26 essential for cochlear cell survival and proper auditory function?

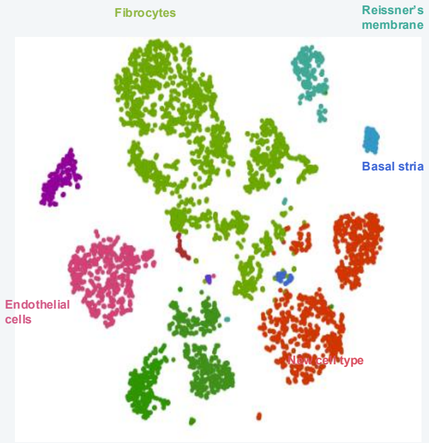


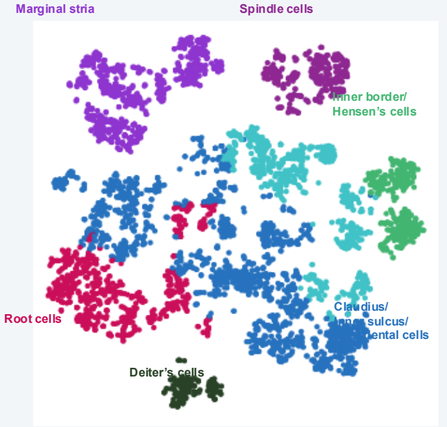


Fibrocyte type 1 Spiral proeminense Claudius cell Deiter cell Border cell Fibrocyte
Fibrocyte type 2 Spindle cell Outer sulcus Hensen cell Inner phallengeal cell
Fibrocyte type 5 Intermiedia stria Root cell Boetcher cell Pillar cell
Basal stria



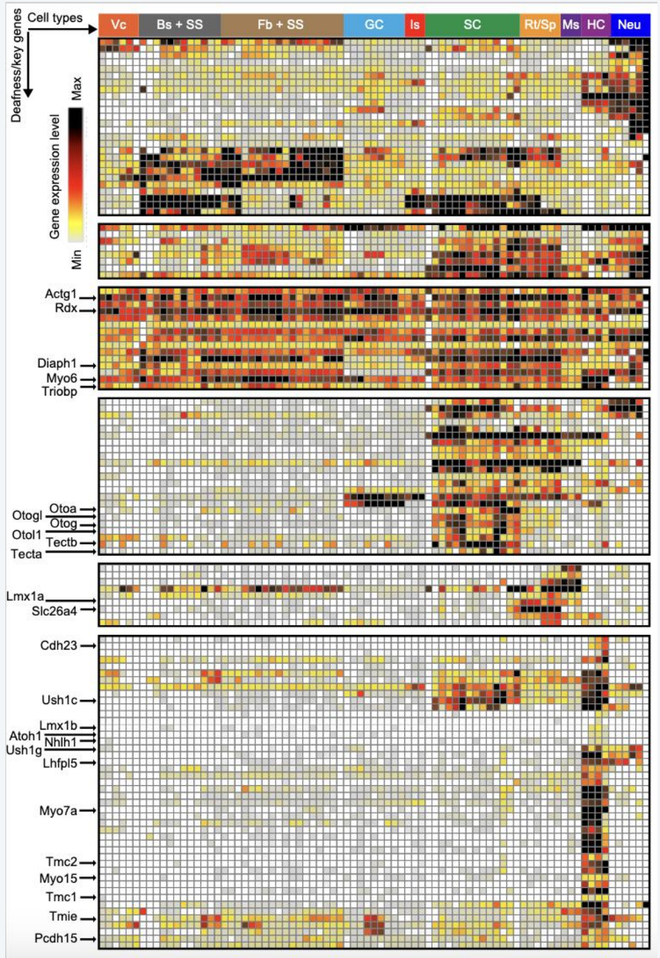






Jean P, Petit C*, Michalski N*. PNAS 2023.

When and where is CX26 essential for cochlear cell survival and proper auditory function?



The first transcriptomic atlas the mouse cochlea from postnatal to adult stages maps deafness gene expression across every cochlear cell type: hair cells, neurons, fibrocytes and stria vascularis cells…

This atlas identifies precisely which cell types express *Gjb2* and suggests the genes it may directly or indirectly interact with, providing the reference map currently used to design our targeted mouse models.

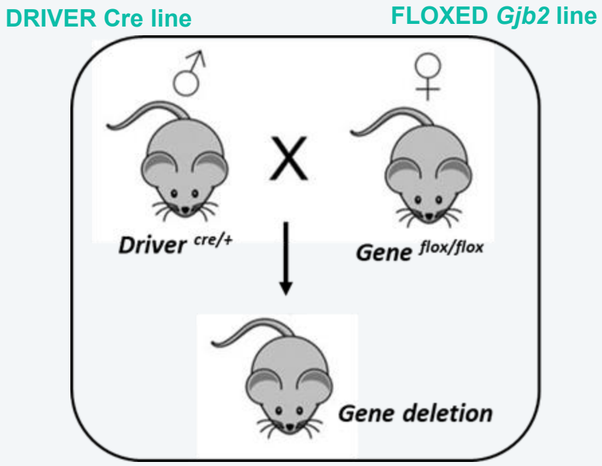
Jean P, Petit C, Michalski N*. PNAS 2023.*

K+: potassium ion; CX26/CX30: connexins 26 and 30; IP3: inositol trisphosphate, an intracellular signaling molecule. Ca2+: calcium ion

Three tailored Gjb2-deficient mouse models, built on the Cre/Lox system

Gjb2-deficient mouse models

Cre/Lox SYSTEM



Connective tissue network — a) fibrocytes

Cre driven by a promoter expressed exclusively in cochlear fibrocytes.

Connective tissue network — b) stria vascularis intermediate cells

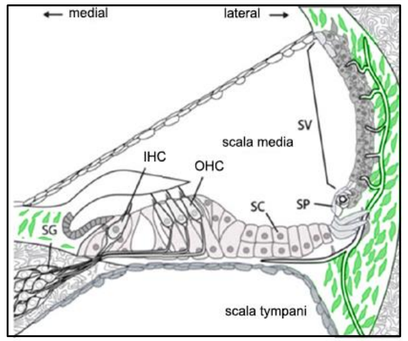
Cre driven by a promoter expressed exclusively in the intermediate cells of the stria vascularis.

Epithelial gap junction network - cKO1 model used to build the preclinical package

Cre driven by a promoter expressed exclusively in the neuroepithelium

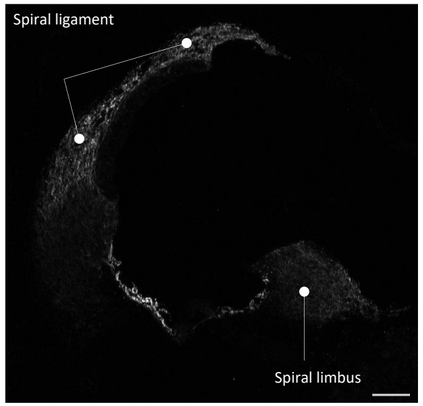
cKO1:conditional knockout model 1

Loss of CX26 in the spiral ligament and spiral limbus, with no morphological defects

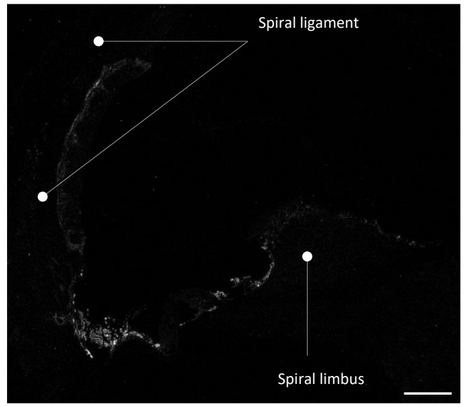


Fibrocytes of the spiral limbus (SL) and the spiral ligament (SLi)

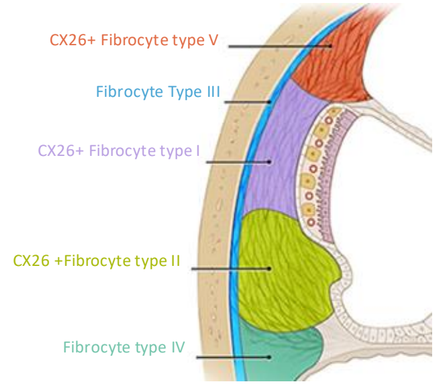
Control



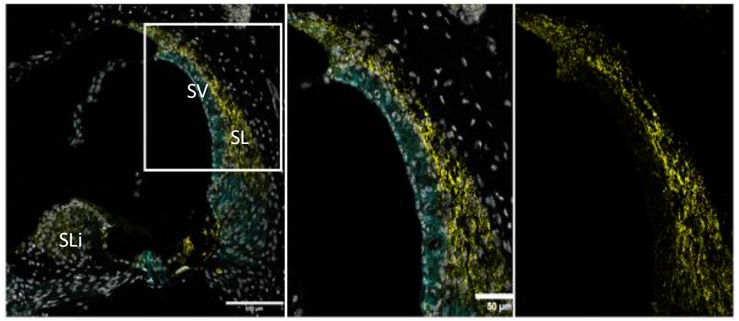
Mutant



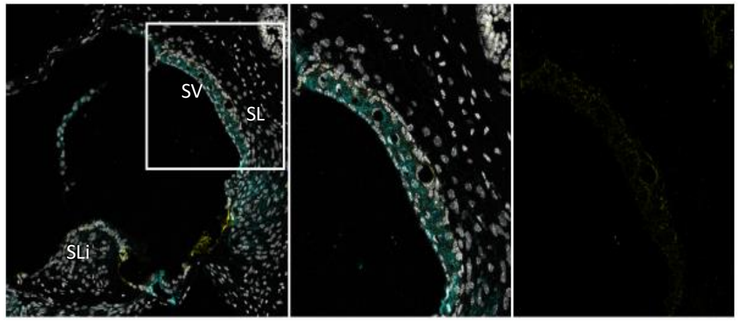
P30 cochlea cryosection; CX26 in white



Control



Mutant



CX26 Tubulin

SV: stria vascularis

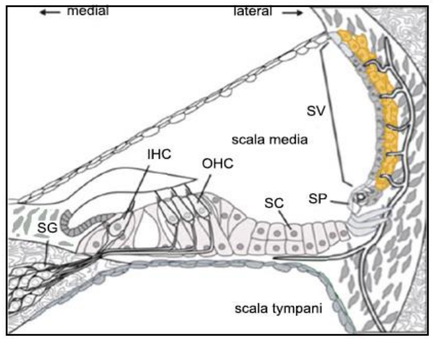
Loss of CX26 expression in the spiral ligament and spiral limbus fibrocytes.

While the expression in the epithelial gap junction network and the stria vascularis **is persisting.**

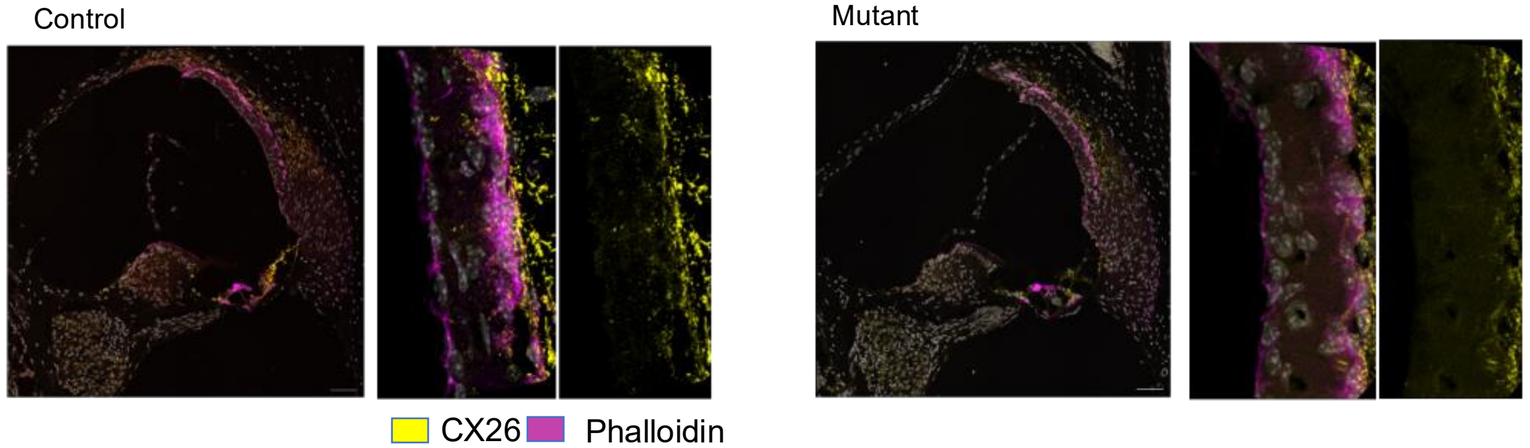
No morphological defects observed (P30 cochlea cryosection).

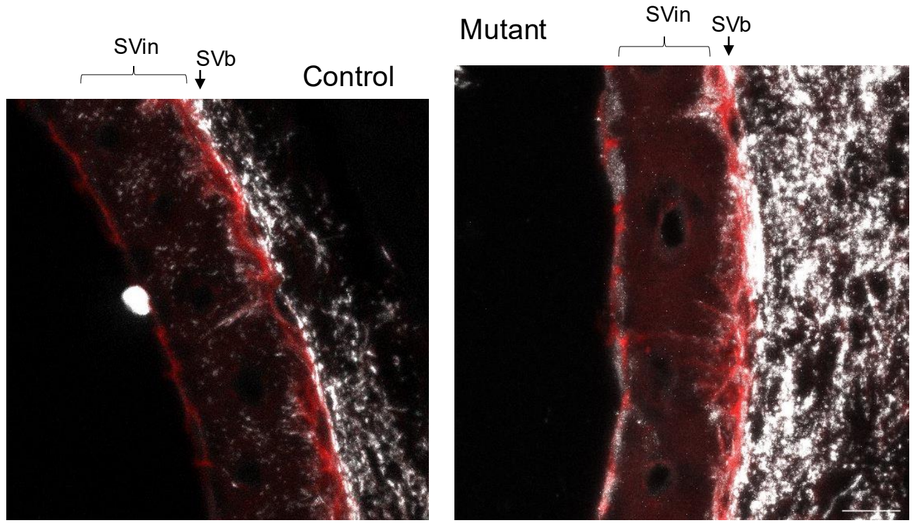
Adapted from doi.org/10.3389/fneur.2020.580639

Loss of CX26 in the intermediate cells of the stria vascularis



The basal and intermediate cells of the stria vascularis (SV)





CX26

Phalloidin

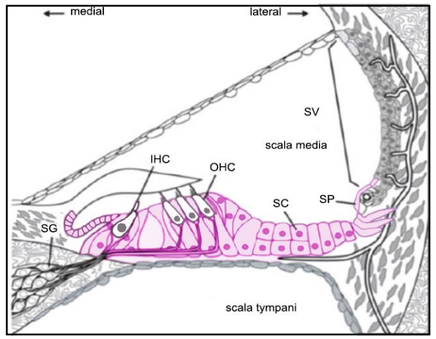
SVin: stria vascularis intermediate

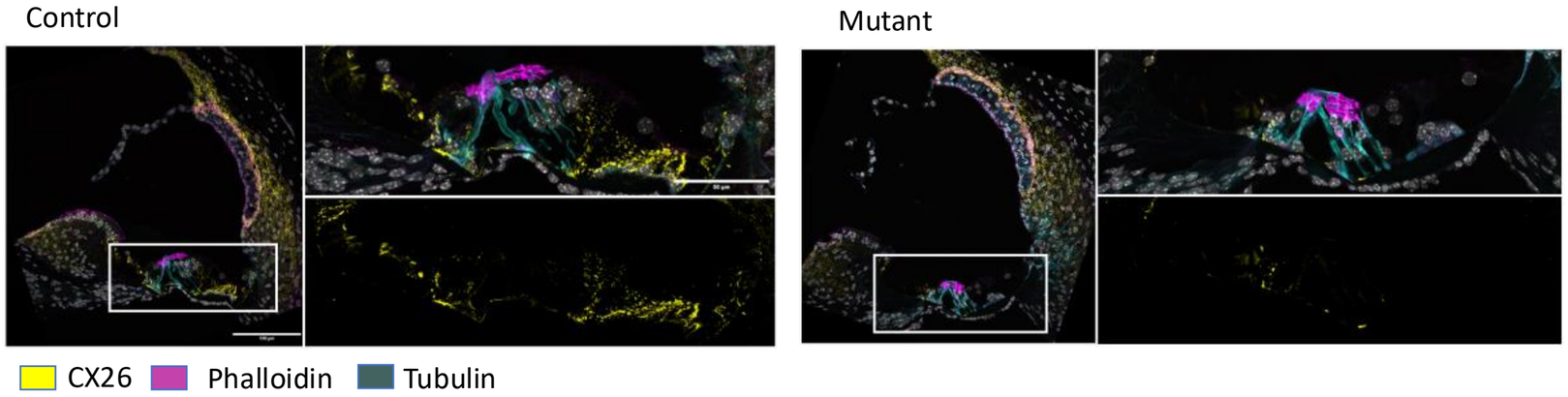
SVb: stria vascularis basal

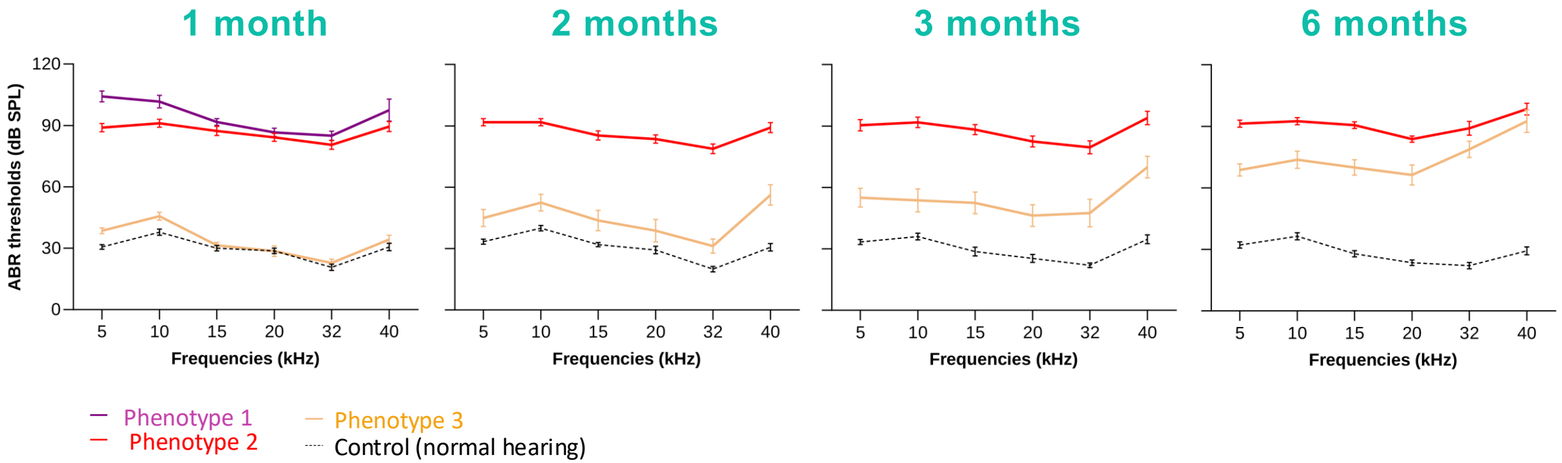
Loss of CX26 expression in the intermediate cells of the stria vascularis (SV).

Persisting expression in the epithelial gap junction network and in the fibrocytes of the spiral ligament and spiral limbus.

One model - three phenotypes based on the ABR profile







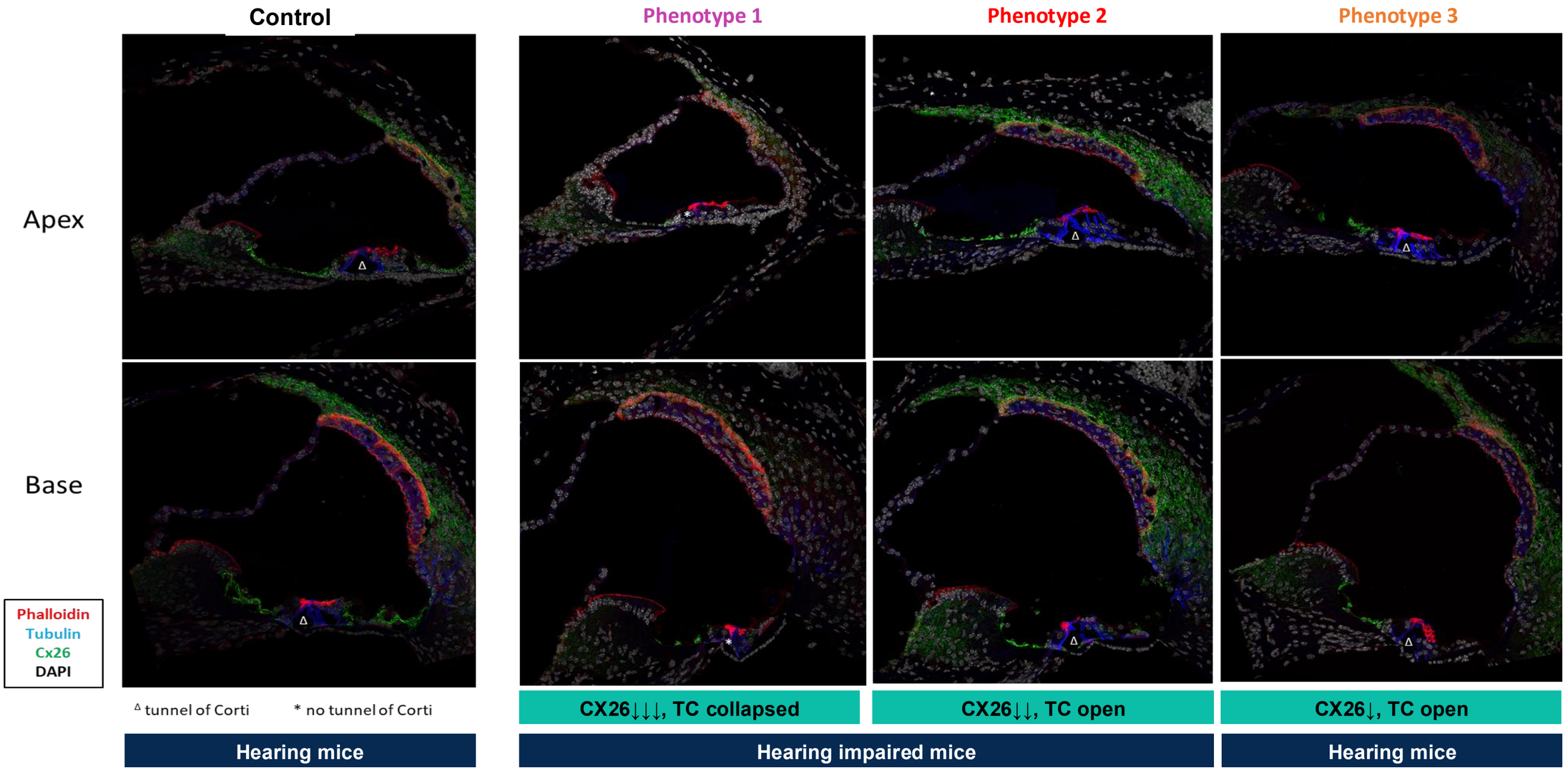
Three phenotypes (1 to 3) are classified according to their ABRs.

From P15 and at 1 month:

- phenotype 1: mice are profoundly deaf
- phenotype 2: mice are severely deaf
- phenotype 3: mice have normal hearing and become progressively deaf

ABR: Auditory Brainstem Response

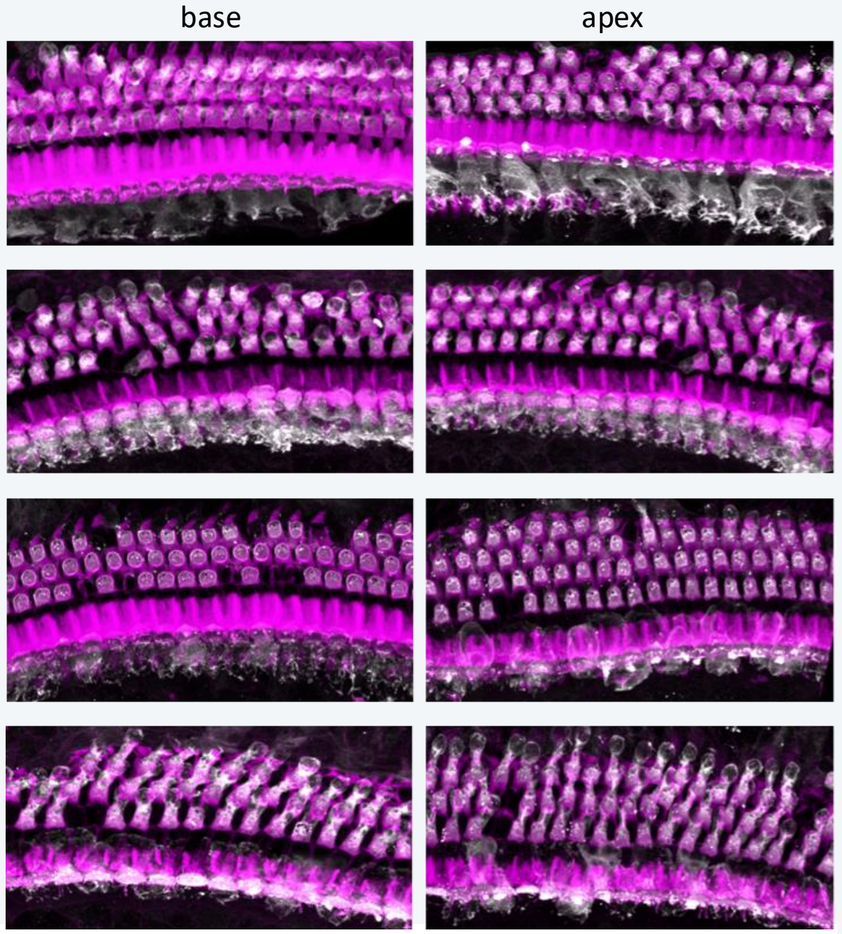
Three phenotypes - different levels of CX26 expression



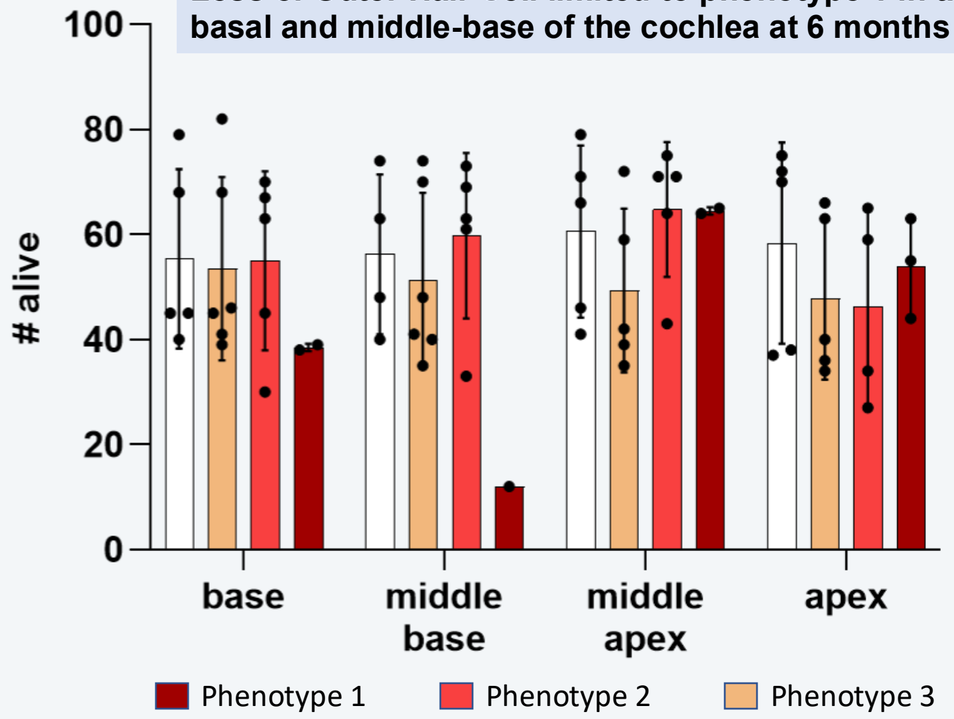
TC: Tunnel of Corti Cryosections at P16

Three phenotypes – hair cells viability

No Inner Hair Cell loss in any of the 3 phenotypes up to 6 months



Loss of Outer Hair Cell limited to phenotype 1 in the basal and middle-base of the cochlea at 6 months



Inner and outer hair cell persistence allows for therapeutic intervention

Three phenotypes – hair cells functionality

Inner hair cells (IHC)

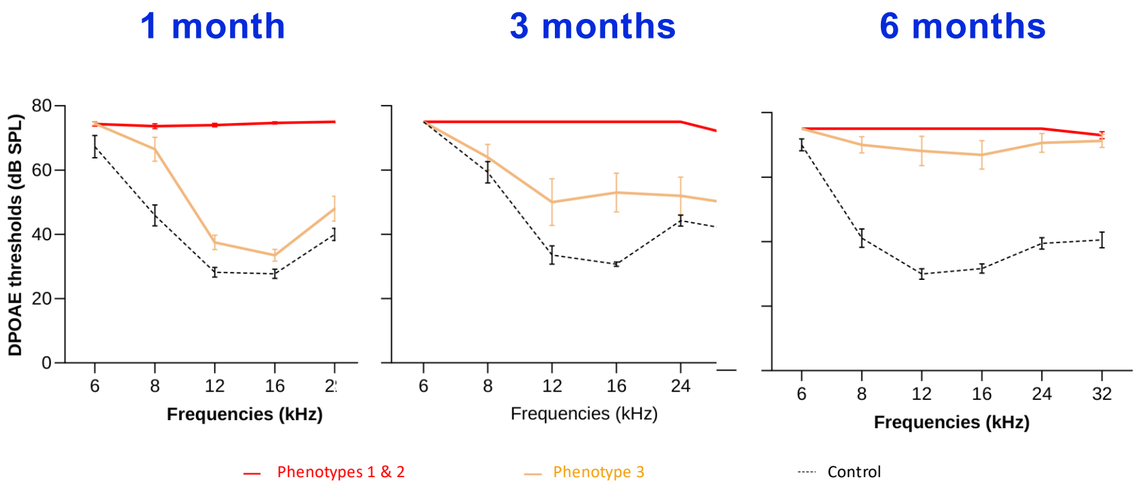
Function analyzed by suprathreshold ABR response (wave 1 amplitude), demonstrating no functional defects in any of the three models.

Outer hair cells (OHC)

Function analyzed by DPOAEs from 1 to 6 months.

In Phenotypes 1 & 2, absence of DPOAE associated with OHC persistence, indicating OHC dysfunction.

In Phenotype 3, Outer Hair Cells develop functional defects between 3 and 5 months.



Together, this GJB2 model with three phenotypes provides a unique experimental platform to assess the efficacy of gene therapy across distinct cochlear cell types within the epithelial gap junction network

DPOAEs: distortion product otoacoustic emissions

Session 2, key messages

1

A new era of innovative therapeutic solutions for patients enabled the discovery of the genes responsible for hearing loss

2

Unraveling the pathogenic mechanisms underlying DFNB1A

Identification of the cochlear cells in which CX26 expression is essential for normal hearing

3

Unique, highly relevant, exclusive mouse models developed

Our bi-allelic GJB2 deletion model with congenital severe to profound HL, mimics the most common form of DFNB1A

By decoding DFNB1A, we aim to drive therapeutic innovation and pave the way for gene therapy development for the benefit of patients

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ACKNOWLEDGMENTS

With thanks to the team behind this work

Christine Petit

Amrit Estivalet
Anne-Valérie Héritier
Lauralee Robichon
Nawel Mekdad
Jeanne Rakotopare
Nicolas Michalski
Muriel Sudres
Solène Roux
Andrea Lelli

Session 3

SENS-601: the preclinical package

Laurent Désiré, Head of Preclinical, Sensorion
Valérie Salentey, Head of Regulatory Affairs and Quality Assurance, Sensorion

The Institut Pasteur collaboration gives SENS-601 a strong scientific foundation based on unique expertise in biology and genetics of hearing

An exclusive collaboration

Exclusive license option

Co-development model on the GJB2 gene therapy program, developed with the laboratory of Prof. Christine Petit

The two teams have successfully advanced the program from target identification into a clinical candidate

What the collaboration provides

Mechanistic understanding. Cell-level mapping of CX26 expression across the cochlea, used to define which cells the vector has to reach to be able to build the most relevant construct

Disease-relevant models. Exclusive conditional knockout models, characterized for hearing phenotype and for the cochlear cell types involved

Why it matters for SENS-601

Target cell selection, the silencing strategy and the choice of efficacy readouts all come out of that work.

Partner institutions

INSTITUT Pasteur

Institut de l'Audition

Institut reConnect

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Prof. Christine Petit, Institut Pasteur, Institut de l'Audition / Institut reConnect

The construct and the design rationale

Laurent Désiré, Head of Preclinical, Sensorion

Preclinical, specific targeting

The SENS-601 vector is designed to restore Connexin 26 expression and the gap-junction network in cochlear supporting cells

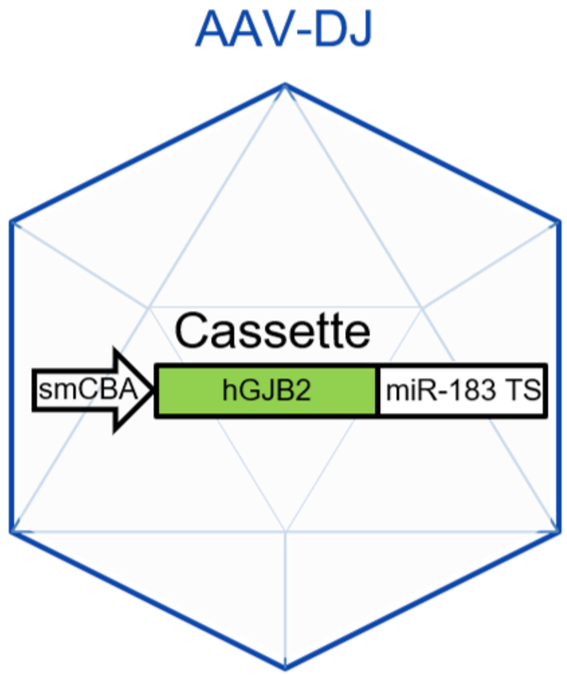
Objective. **Enabling targeted expression of the CX26 protein to restore gap junctions in supporting cells.**

AAV-DJ capsid, providing broad tropism across cochlear supporting cell types

Promoter smCBA, well characterized, ensuring strong and durable expression of the human GJB2 gene

Regulatory sequence miR-183 target sequence, silencing CX26 expression in hair cells for high selectivity and safety

Patents on SENS-601 and the miR-183 target sequence



SENS-601 vector and cassette

Preclinical, specific targeting

A miR-183 target sequence keeps expression out of the sensory hair cells

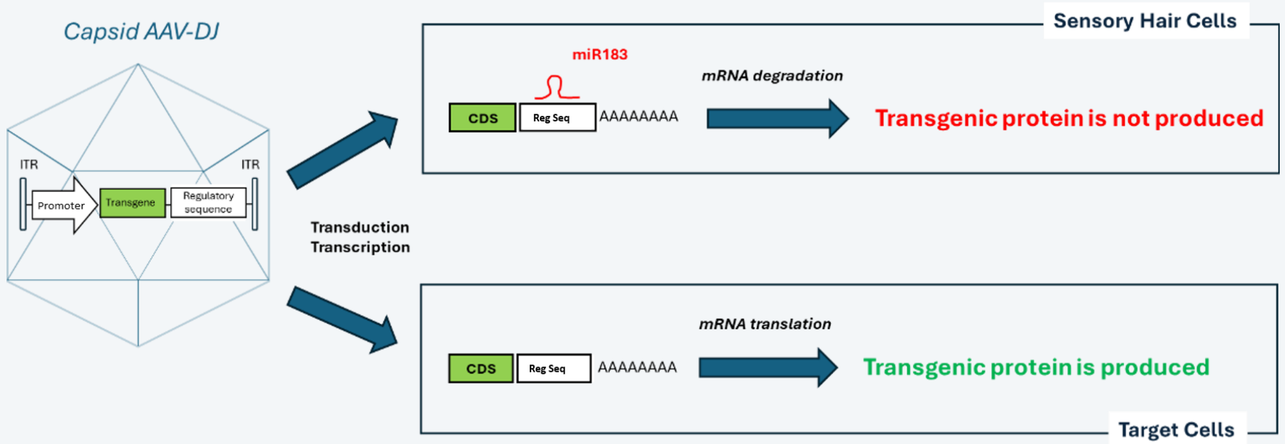
Selective. Off in sensory hair cells, on in target cells

Efficient. Transgene silencing efficacy: 85-100 %

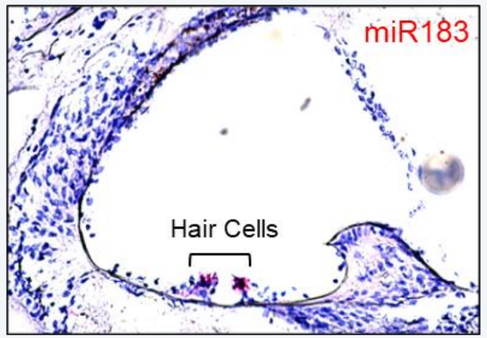
Sustained. miR-183 sequence remains expressed in aged cochlea, including human

Durable. Long-term miR-183-mediated transgene silencing in hair cells, at least 12 months after treatment

miR-183-mediated transgene expression control is well tolerated



Mouse, 1 year old

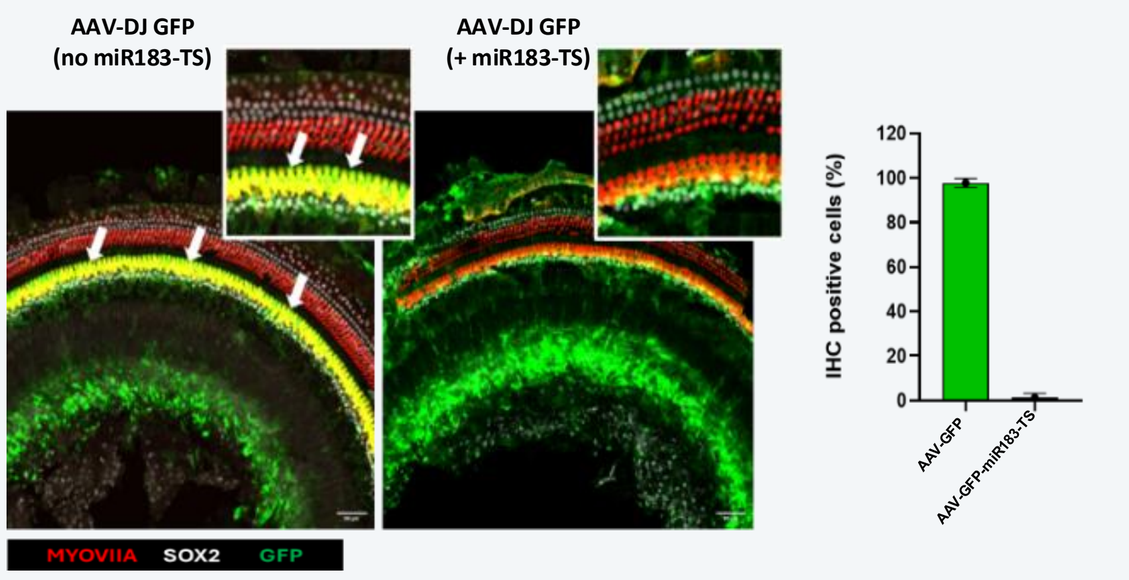


We validated by in situ hybridization that miR183 is mostly detected in cochlear sensory hair cells and maintained for at least 1 year in mice

Preclinical, specific targeting

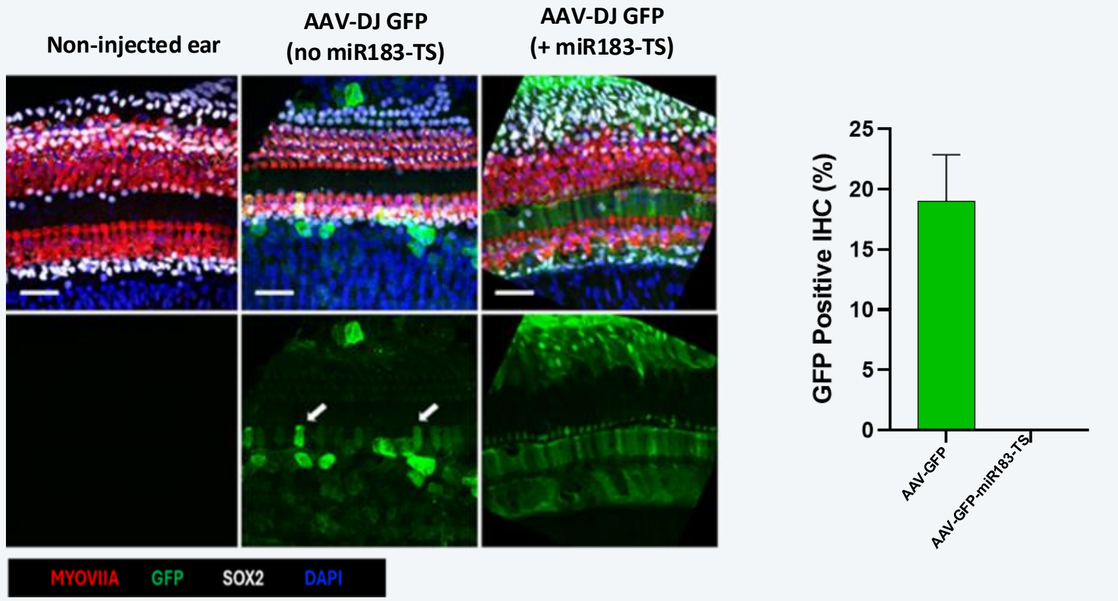
SENS-601 combines broad cochlear reach with built-in hair cell transgene silencing via miRNA

Full hair cell transgene silencing in mouse cochlea



Negative control, AAV-GFP without miR-TS, AAV-GFP with miR-183 TS. Arrows indicate transduced inner hair cells.

Full hair cell transgene silencing in NHP cochlea



Same pattern reproduced in non-human primate cochlea.

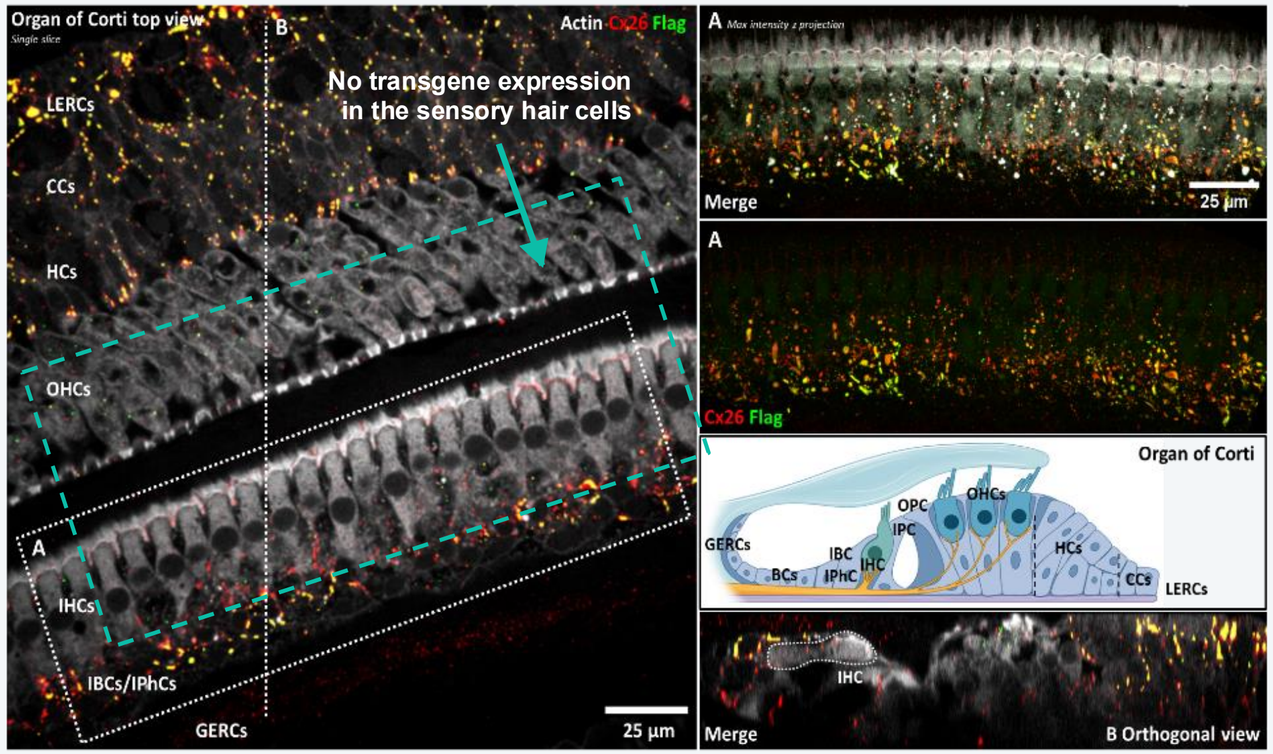
Hair cells. +++ without miR-183 TS, no expression with miR-183 TS

Supporting cells. +++ in both conditions

miR-183 allows efficient transgene expression in the target cells with virtually no expression in hair cells

NHP: non-human-primate

Connexin 26 is observed at the cell membrane of the appropriate target cells in NHP cochlea

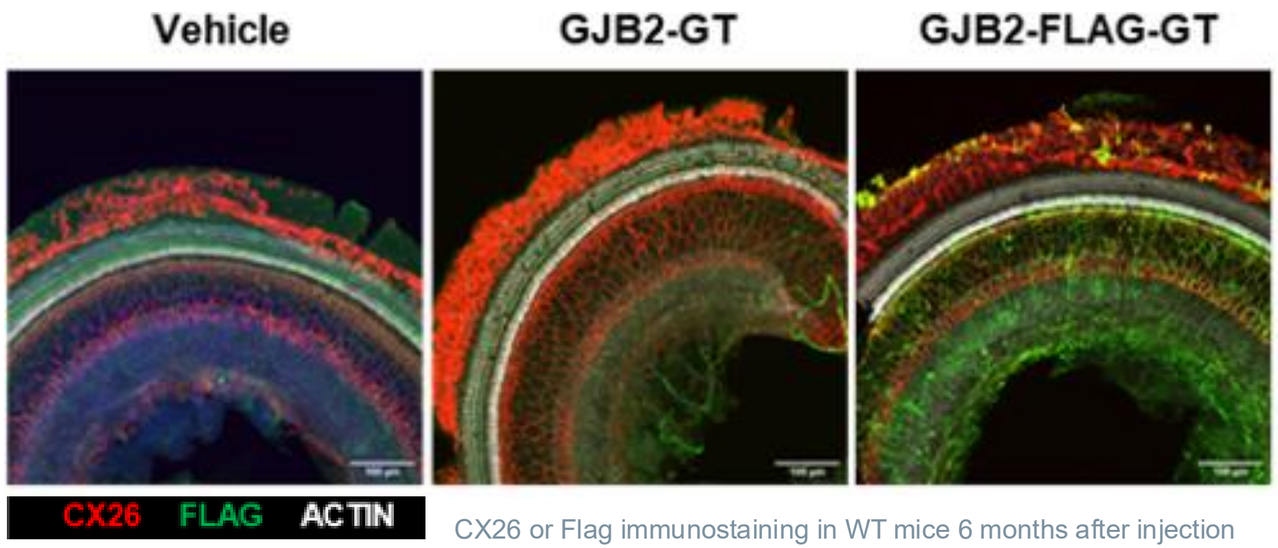


Yellow staining indicates the characteristic CX26 expression pattern at the cell membrane, overlapping with the transgene Flag

No expression of either endogenous CX26 or transgenic CX26 Flag is detected in hair cells

GJB2-Flag-GT: SENS-601 surrogate vector with CX26 fused to Flag epitope

Long-term transgene expression in mice and NHP

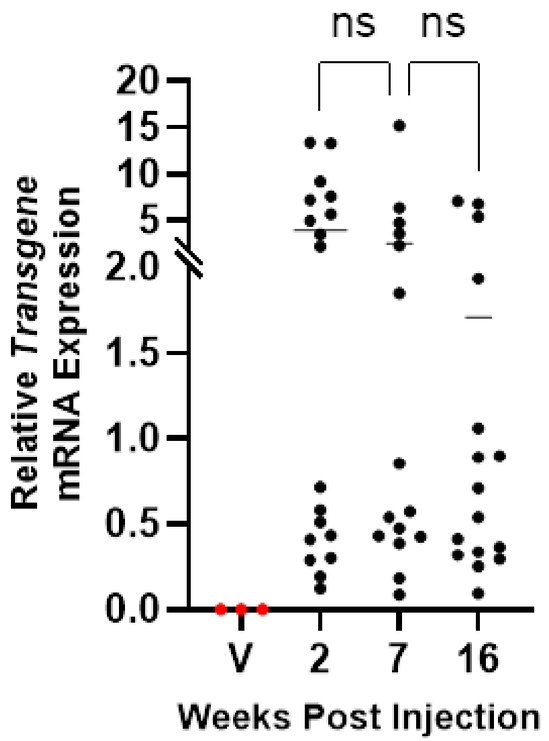


CX26 or Flag immunostaining in WT mice 6 months after injection

Protein expression remains highly expressed for at least 6 months in mice

No loss of mRNA expression detected by RT-qPCR up to 6 months in mice and NHP cochlea

Long term transgene mRNA and protein expression demonstrated in cochlea



CX26 mRNA quantification in WT mice 4 months after SENS-601 injection

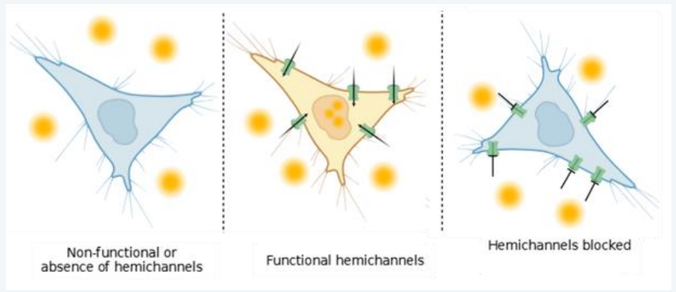
Efficacy and functional restoration

Laurent Désiré, Head of Preclinical, Sensorion

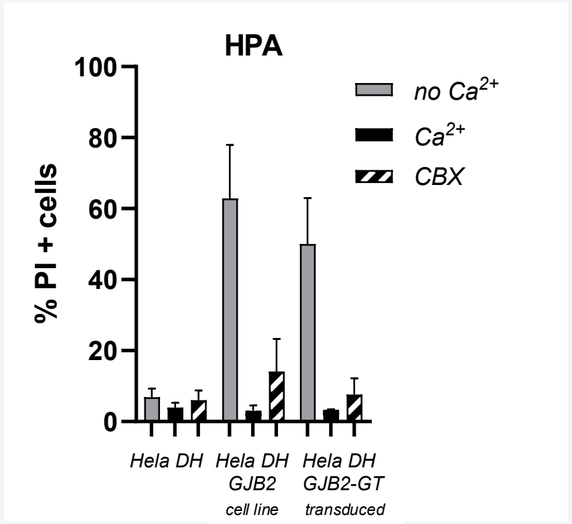
Preclinical, demonstrated restoration

SENS-601 restores functional hemichannels in Connexin-deficient HeLa cells

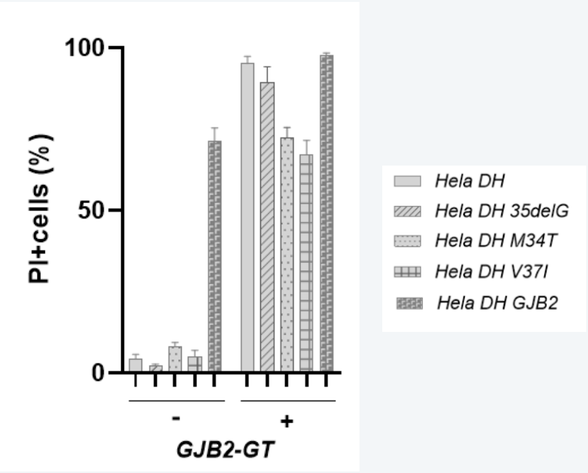
Propidium iodide assay monitors functional hemichannels



SENS-601 restores functional hemichannels

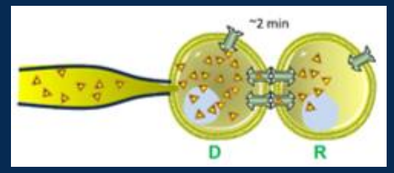


Rescue of functionality from CX26 human pathological mutants



35delG: truncating mutation, severe-to-profound HL

M34T / V37I: missense variants, mild-to-moderate HL

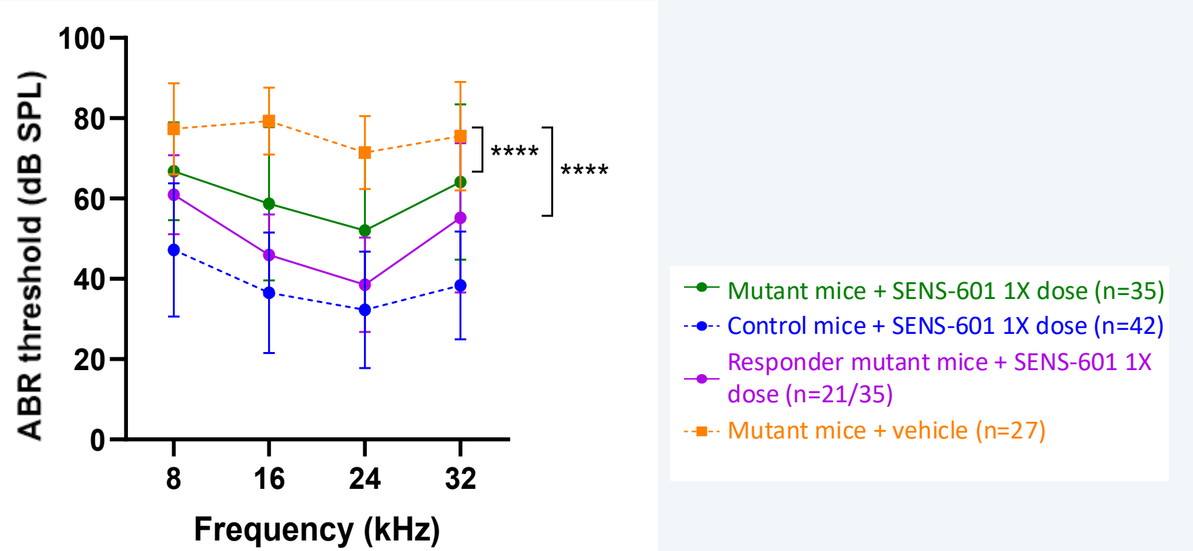


Confirmatory dye transfer assays further demonstrate that GJB2 transduction leads to functional gap junctions and associated intercellular communication.

Preclinical, demonstrated restoration

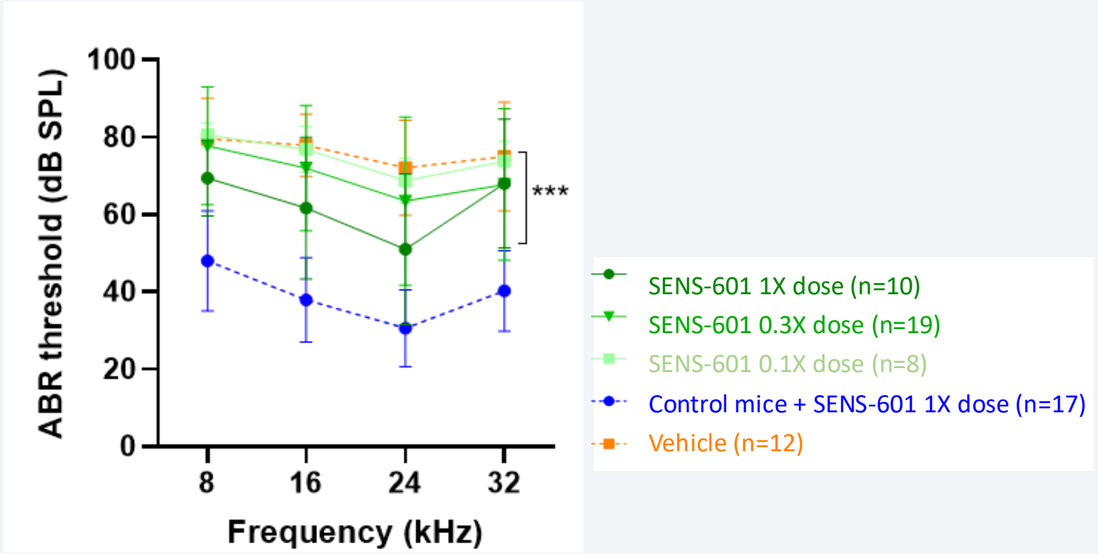
SENS-601 restores ABR thresholds across multiple frequencies in the cKO1 model with severe to profound HL

ABR threshold, 7 weeks post-injection



Pooled data from multiple studies using several batches
**** p<0.0001; two-way ANOVA multiple comparisons followed by Tukey test

ABR threshold by dose, 7 weeks post-injection



*** p<0.001; two-way ANOVA multiple comparisons followed by Tukey test

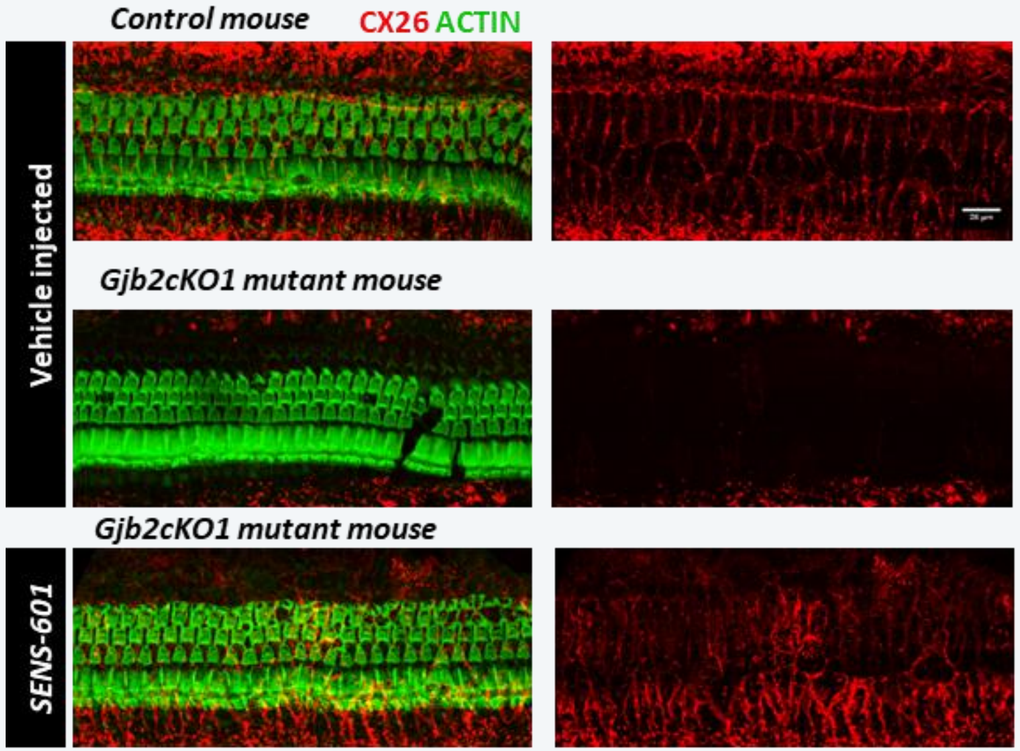
- **Efficacy within 3 weeks of injection at P0/P2, across all tested frequencies**
- **Reproduced across multiple batches, from R&D through to final manufacturing**
- **Restored ABR correlates with DPOAE and EP; effective dose confirmed in dose-ranging studies**

ABR: Auditory Brainstem Response

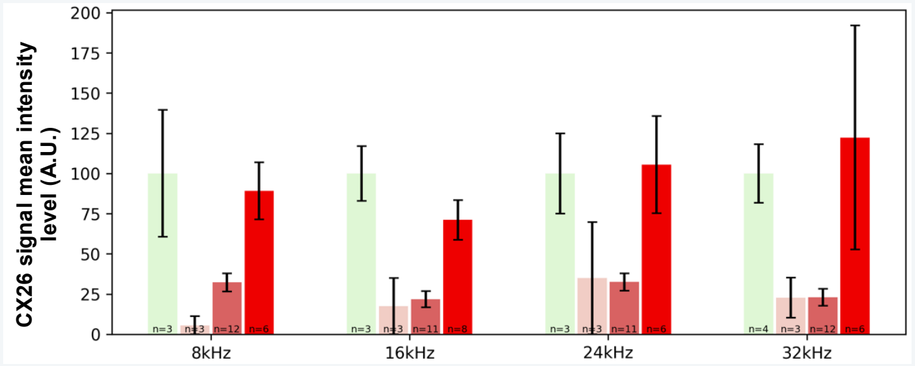
Preclinical, demonstrated restoration

Cochlear CX26 levels are normalized by SENS-601 in the cKO1 model – outer sulcus

CX26 network immunostaining



CX26 levels quantification



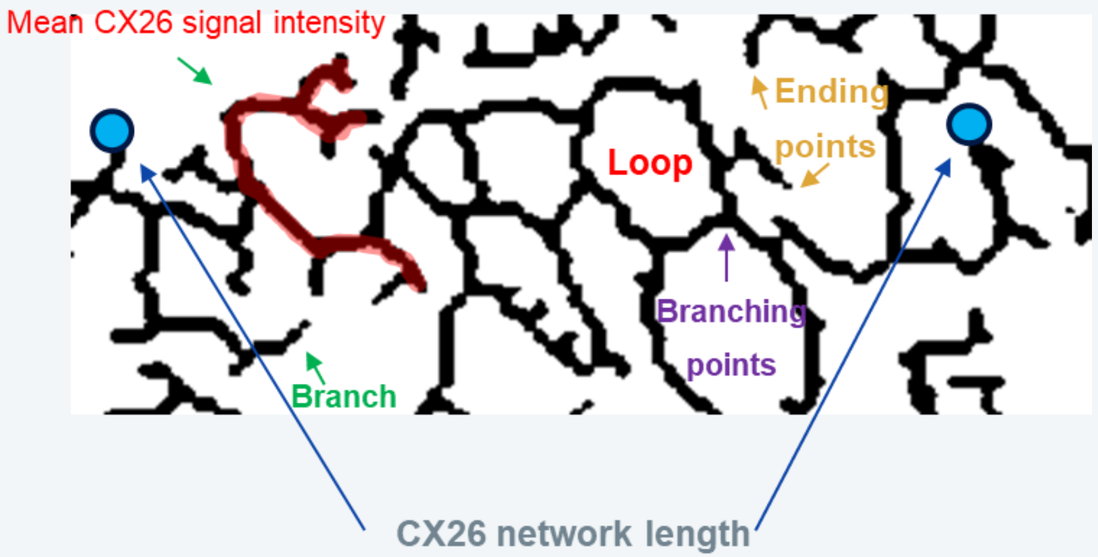
Tonotopic and dose-dependent improvement of CX26 levels, evaluated by AI-based image analysis of the cochlear CX26 network.

Analyses performed 18 weeks post-administration at P0/P2.

Preclinical, demonstrated restoration

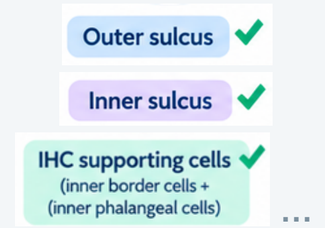
The C26 network itself is rescued, not only the level of expression

Main CX26 network metrics

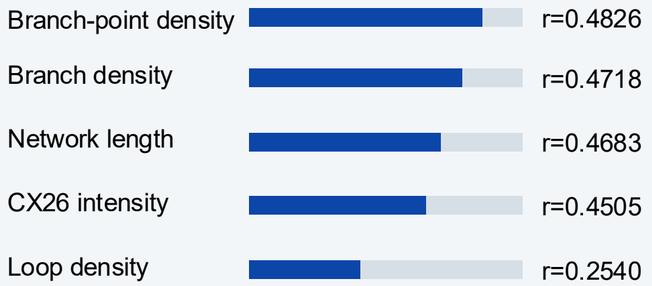


150 CX26 network features measured per sample

CX26 network metrics are improved in multiple cochlear structures and cell type



Association with ABR outcome



Associations are significant: p<0.0001; loop density p=0.0135

CX26 network tracks auditory recovery

Analyses performed 18 weeks post-administration at P0/P2.

Preclinical Observations & Tolerability

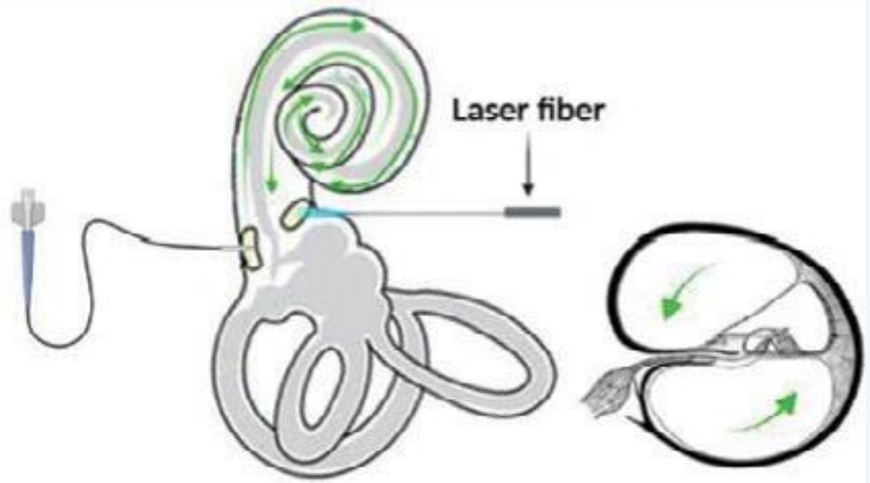
Laurent Désiré, Head of Preclinical, Sensorion

Surgical delivery approach

Dual-fenestration technique combines two familiar procedures, cochlear implantation and stapedotomy, and is designed to minimize overpressure and no backflow.

Designed to maximize vector distribution and target cell exposure along the full length of the cochlea, allowing widespread transduction.

Well tolerated in our previous gene therapy program with no serious adverse events reported; reproducibility confirmed across multiple administrations performed to date.

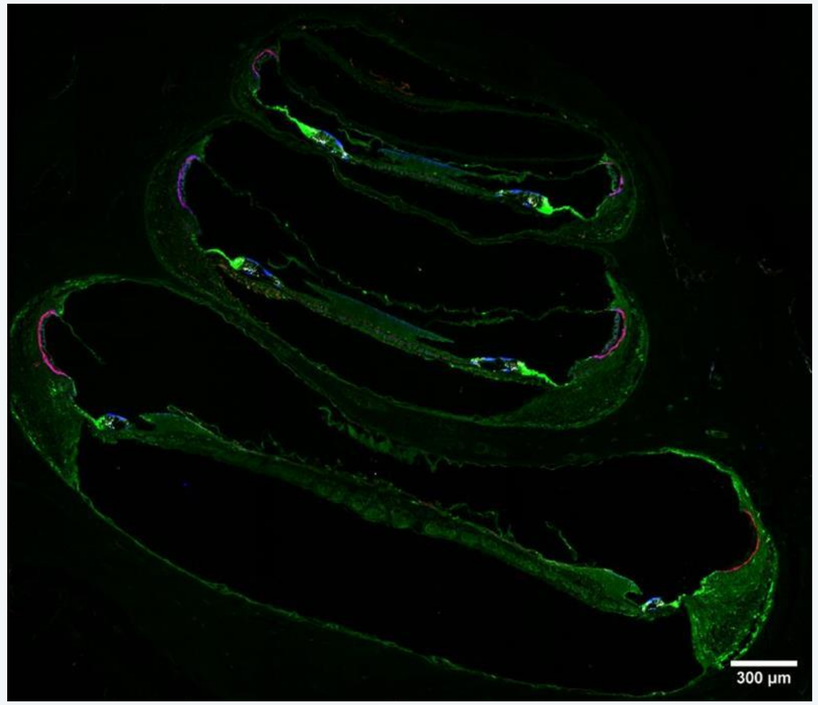


Proprietary injection system combining cochlear implantation and stapedotomy approaches

Adapted from Yoshimura et al (2018)

SENS-601 Efficiently Transduces NHP Cochlea

- Injection of SENS-601 (Flag surrogate), at the NHP equivalent effective dose, leads to strong transgene detection in the cochlea of NHP from base to apex
- Flag signal within the CX26 network was quantified across the different compartments of interest and tonotopic axis
- Transgene expression pattern in NHP is consistent to the one observed in mice



SENS-601 injected with intended surgical delivery approach efficiently transduces NHP cochlea

No safety signal was observed in SENS-601 combined 3 and 6-month GLP toxicology and biodistribution studies

Non-human primate studies

Design. Unilateral intracochlear administration. Three groups: vehicle, low dose, high dose. Two cohorts: 3 months and 6 months.

Tolerability. No mortality. Mild and transient anti-AAV-DJ response only in low-titer animals; no anti-CX26 antibodies. Weak cellular immune response against AAV-DJ, no cellular response to hGJB2.

Biodistribution and shedding. Dose-related, confined to the injected ear and draining lymph nodes. No blood or CSF detection, rapid clearance in urine and feces (undetectable by Day 15); transient positivity in nasal swabs to Day 29 and ear swabs to Week 26.

Mouse studies

Design. Intravenous administration. Three groups: vehicle, low dose, high dose. Three cohorts: 3 months, 6 months and biodistribution. c

Tolerability. No mortality. No SENS-601-related clinical or histology findings; one minor non-adverse creatinine change in females only.

Biodistribution and shedding. Dose-proportional; still detectable at 6 months, mainly at the injection site (tail) and in the liver (highest level); DNA in blood up to Week 5.

Session 3, key messages

1

The target cells are identified, and the construct is designed to reach them

Transgene is expressed in target cells but not in hair cells

2

SENS-601 studies showed functional restoration of hearing and normalization of the CX26 network

3

No safety signal was observed in GLP toxicology and biodistribution studies

Surgery was well tolerated in previous gene therapy program

With data and models translating well to humans, we are ready to take SENS-601 into the clinic

Regulatory path

Valérie Salentey, Head of Regulatory Affairs and Quality Assurance, Sensorion

First-in-human study with SENS-601 is designed as a multi-country trial across four geographies

Enrollment opens geography by geography as each regulatory clearance lands

France

CTA approved

Hôpital Necker Enfants Malades, Paris
Dr. Natalie Loundon

Canada

Application under review

Hospital for Sick Children, Toronto
Dr. Sharon Cushing

United States

IND Filing expected by YE 2026

Site to be disclosed

Australia

CTA Filing expected by YE 2026

Children’s Hospital Westmead, Sydney
Pr. Catherine Birman

Site activation follows regulatory clearance and site contracting.

Session 4

Clinical development strategy and the HearConnex design

Sharon Cushing, M.D., The Hospital for Sick Children (SickKids), Toronto
Coordinating Investigator, HearConnex

OTOCONEX: the natural history foundation

Longitudinal clinical, audiological and genetic data support endpoint selection and stratification

Otoconex

Data collected

Genetic	GJB2 genotype, molecular diagnosis
Audiological	Pure tone audiometry and objective auditory assessments over time (at least 2 years)
Functional	Functional hearing assessments and quality of life questionnaires

Population

Aged 16 years or under without a cochlear implant, and 10 years or under with an implant. Non-syndromic, bilateral, mild to profound sensorineural hearing loss, pre-lingual or post-lingual.

What it enables

Endpoint selection, patient stratification and trial design decisions, taken on data rather than on assumption.

OTOCONEX also built the patient, site and data infrastructure HearConnex runs on

Otoconex

1 Patient asset

Genotyped patients

Clinical characterization

Longitudinal follow-up

2 Site asset

Experienced investigators

Referral pathways

Standardized assessments

3 Data asset

Natural history data

Outcome measures

Disease progression insights

Patient identification, and reduced recruitment uncertainty	**A trained network of investigators and sites**	**A natural history benchmark for treatment evaluation**

HearConnex: study design

Grant from French State (Bpifrance) as part of the France 2030 investment plan

The standard of care improves access to sound; the aim here is sustained physiological hearing

The disease

GJB2-related hearing loss is the most frequent type of autosomal recessive non-syndromic hearing loss.

The limit of current care

Cochlear implantation improves access to sound and speech stimuli but does not restore physiological hearing, and some limitations still need to be overcome.

The intent of treatment

SENS-601 aims to facilitate physiological hearing while overcoming the drawbacks of implantation and is intended to last a lifetime.

Based on efficacy results from proof-of-concept studies in mouse models.

HearConnex will evaluate SENS-601 safety and efficacy, with first patient dosing anticipated in early 2027

Open-label, multicenter Phase I/II trial to assess the safety, tolerability and efficacy of intracochlear administration of SENS-601 at escalating doses and the injection system.

Trial objectives

Primary

Dose escalation. Safety and tolerability of SENS-601 and the injection system

Dose expansion. Efficacy of SENS-601 on Auditory Brainstem Response (ABR) and Pure Tone Audiometry (PTA)

Secondary

Efficacy and safety of SENS-601; injection system performance, usability and clinical benefit

Status

Regulatory

France: CTA approved 31 August 2026

Canada: CTA under review

United States: IND submission expected by YE 2026

Australia: CTA submission expected by YE 2026

Enrollment

First patient in anticipated by early 2027

Clinical data generation expected throughout 2027

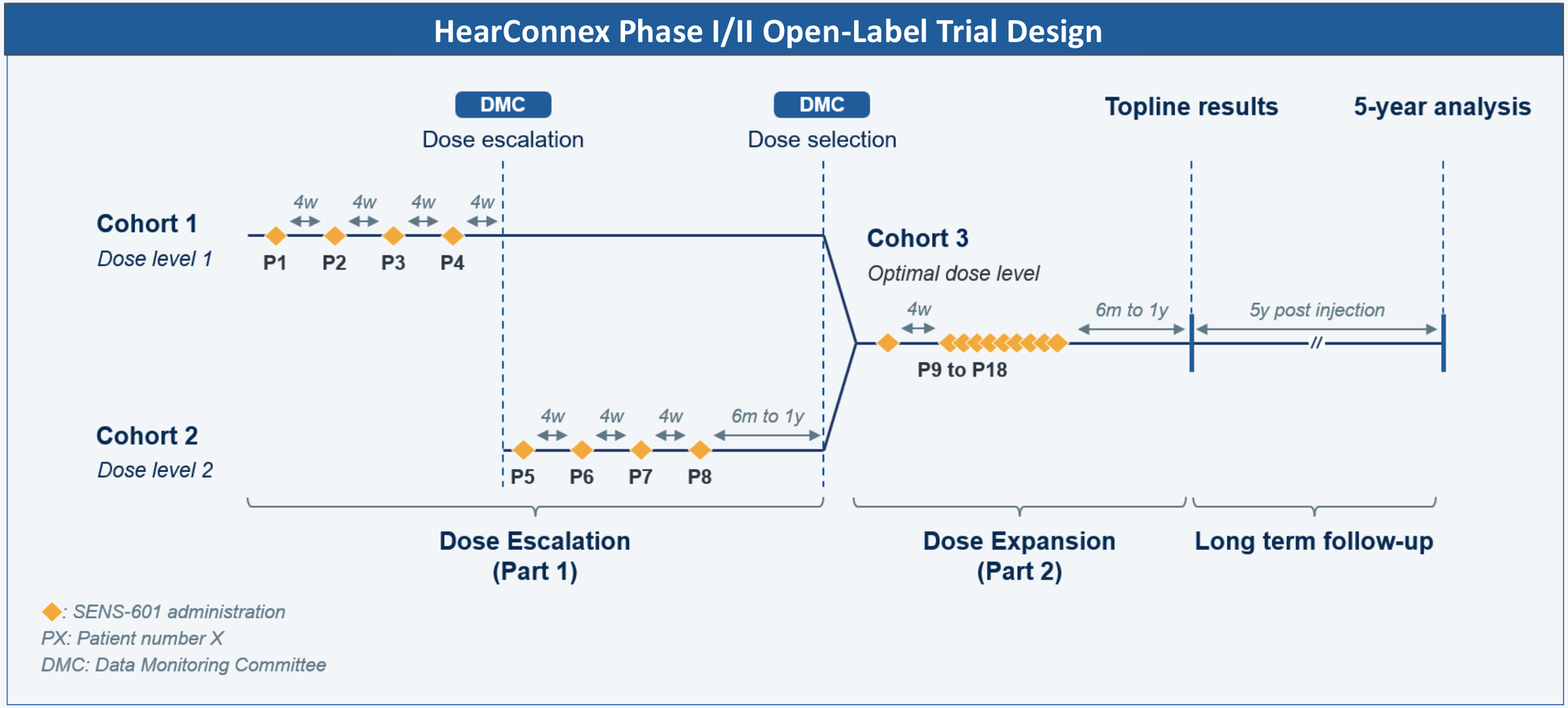
CTA: Clinical Trial Application; CTN: Clinical Trial Notification; IND: Investigational New Drug.

HearConnex Phase I/II trial design

	Part 1 – Dose Escalation	Part 2 – Dose Expansion
Study population	Children with non-syndromic, bilateral, severe sensorineural hearing loss eligible for cochlear implantation	
Patients	8 children	10 children
Age	≥ 6 months to ≤ 31 months, cochlear implant-naïve ≥ 6 months to ≤ 6 years, already unilaterally implanted within 18 months	≥ 6 months to ≤ 31 months, cochlear implant-naïve
GT procedure	Unilateral intracochlear injection	Bilateral intracochlear injection
Dosing	Dose level 1 / Dose level 2	At the optimal dose
Primary objective	Safety and tolerability	Efficacy (co-primary endpoints: ABR, PTA)

ABR: Auditory Brainstem Response, PTA: Pure-tone audiometry

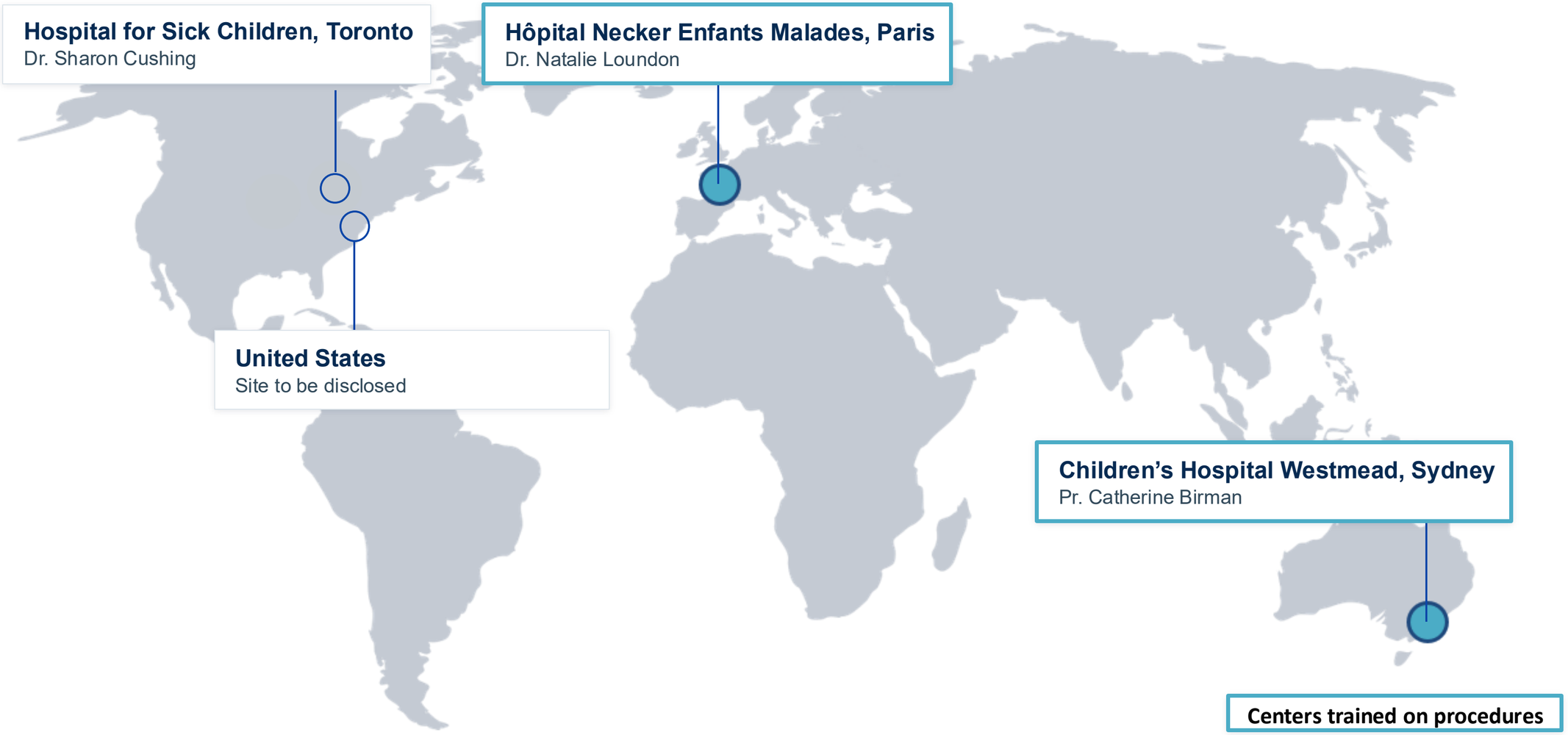
HearConnex is designed to allow fast transition to pivotal trial



PX: patient number X. DMC: Data Monitoring Committee. Part 1 uses unilateral administration, Part 2 bilateral administration.

HearConnex is planned at centers already trained on the procedure

Sensorion has long-standing relationships with an extensive global network of sites with expertise in hearing loss.



Planned investigator centres, each trained on the procedure. Site activation follows regulatory clearance and site contracting. France: CTA approved by ANSM. Canada: application under review. Australia and United States: filing targeted by YE 2026 ; outlined marker indicates site selection under way.

Positive benefit / risk assessment

The case rests on four elements, each shown earlier today.

Efficacy grounded in mechanism

- Proof-of-concept established in mouse models
- Addresses the pathophysiology of GJB2-related hearing loss directly
- Restores CX26 expression and the Cx26 network in disease-relevant models
- Recovery of ABR thresholds and improved endocochlear potential

GLP Toxicology / safety package

- Combined three- and six-month studies in mice and non-human primates
- Absence of safety signal

A surgical procedure already performed in this population

- Intracochlear administration and injection system already used in the Audiogene trial of SENS-501
- No safety signal observed for the procedure itself

The cochlear implant option is preserved

- Children who do not benefit can still receive a cochlear implant
- The schedule keeps that option open within the recommended developmental window

1

OTOCONEX defined the endpoints, the disease trajectory and the patient survey before the trial was designed

2

HearConnex leverages our experience from previous trials in the hearing field as well as our relationships with already trained centers

3

The Phase I/II trial allows flexibility to yield early data on dose selection and confirmatory data on bilateral administration to move to next steps

HearConnex was informed by learnings from previous trial and natural history study to carry a path rapidly to give access to gene therapy to the largest number of patients

Session 5

Panel discussion and Q&A

Panel discussion

Dr. Sharon Cushing

The Hospital for Sick Children (SickKids), Toronto

Pediatric otolaryngologist, Director of the Cochlear Implant Program. Coordinating Investigator, HearConnex.

Prof. Christine Petit

Institut Pasteur, Institut de l'Audition / Institut reConnect

Professor Emeritus at the Collège de France, laureate of several prizes incl. the Kavli Prize in Neuroscience.

Fred Chereau

Chief Executive Officer, Sensorion

Laurent Désiré

Head of Preclinical, Sensorion

Valérie Salentey

Head of Regulatory Affairs and Quality Assurance, Sensorion

To ask a question, dial in on the conference call number

ir.contact@sensorion-pharma.com

Session 6

Closing remarks

Fred Chereau, Chief Executive Officer, Sensorion

Three things to take from today

1

The biology is mapped and the target cells are identified.

The cochlear cell types critical to hearing in DFNB1A have been characterized in disease-relevant models, and SENS-601 is designed to reach them and to stay out of hair cells.

2

The preclinical package shows functional restoration.

CX26 expression and network restoration, ABR threshold recovery across frequencies, GLP toxicology in mice and non-human primates were well tolerated.

3

The trial is designed on Sensorion’s own experience.

Prior trials and natural history studies set the endpoints, disease trajectory and patient identification pathway.

The same investigator centers and practitioners, already trained on the delivery procedure, carry that experience into HearConnex.

With validated biology and a unique collaboration, Sensorion aims to run a best-in-class trial with HearConnex

What comes next

Under review	Health Canada CTA decision
By Q4 2026[1]	IND submission in the United States
By Q4 2026[1]	CTA submission in Australia
Early 2027	First patient dosed in HearConnex
Throughout 2027	Clinical data generation

ir.contact@sensorion-pharma.com

[1] Expected milestones.

Thank you